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Cancellation of the vestibulo-ocular reflex during horizontal combined eye-head tracking

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Case Western Reserve University, 1991
CANCELLATION OF THE VESTIBULO-OCULAR REFLEX DURING
HORIZONTAL COMBINED EYE-HEAD TRACKING

by
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Submitted in partial fulfillment of the requirements
for the Degree of Doctor of Philosophy

Department of Biomedical Engineering
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January, 1991
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GRADUATE STUDIES

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CANCELLATION OF THE VESTIBULO-OCCULAR REFLEX DURING HORIZONTAL COMBINED EYE-HEAD TRACKING

Abstract

by

WILLIAM PAUL HUEBNER

During natural activities, the vestibulo-ocular reflex (VOR) is used to provide clear vision of stationary objects by automatically causing eye rotations that are equal and opposite to head rotations. However, during combined eye-head tracking (CEHT), wherein the head rotates in synchrony with a moving object, the eyes must remain stationary in the head; this necessitates overriding the VOR. The goal of this research was to determine how the VOR is cancelled during CEHT. Specifically, I attempted to confirm or refute the hypotheses that, for human subjects in the horizontal plane, the smooth pursuit system generates the primary signal responsible for cancelling the VOR during CEHT, and that this cancellation occurs through a superposition of the smooth pursuit and VOR signals.

My strategy involved (1) developing realistic mathematical models of the VOR and smooth pursuit systems, (2) using optimal parameter estimation techniques to make the two models accurately reflect subject data, (3) joining the component models into a single model for CEHT consistent with the research hypotheses, (4) using stimulus data from CEHT experiments to drive the model and create simulated response waveforms, and (5) comparing the simulation waveforms with the
corresponding CEHT data from subjects to detect differences that might refute my research hypotheses. The eye movements from four human subjects were measured using the magnetic field/scleral search coil technique while the subjects were exposed to various combinations of transient visual and vestibular stimuli, presented under computer control. Two specific CEHT stimuli, the Chair Brake and the Delayed Target Onset, were employed to capitalize on the significantly different latencies of the VOR and smooth pursuit systems.

Based on my analyses involving residual and correlation methods, there was insufficient evidence to reject the hypothesis that a superposition of the smooth pursuit and VOR signals accounts for the observed cancellation of the VOR during transient, horizontal CEHT. However, accounting for a particular feature observed in certain responses involved slightly relaxing the premise of a strict superposition. A novel finding of this research was that VOR gain appears to change dynamically within individual responses, depending on the demands placed upon this reflex.
We want to perfect ourselves so that we can win with less struggle and increasing ease, but the strange thing is that it's not the easy wins we ostensibly seek but rather the difficult struggles to which we really look forward.

— Vincent T. Lombardi

To my family and my friends
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I. INTRODUCTION

Our brains contain a number of neural circuits which have a variety of sensory and motor functions. The *ocular motor system* comprises one collection of these circuits and is responsible for controlling the rotation of the eyes within the head. The purpose of this system is to provide clear vision of the surroundings (or, of specific objects within the surroundings) by moving the eyes in a way that maintains the stability of images on the retina, and especially the *fovea* (the photoreceptor-dense region of the retina). Stated another way, the ocular motor system tries to prevent the images of the visual surround from slipping on the retina by working to minimize *retinal slip*. Because the head (which contains the eyes) and an object of interest may both move, the ocular motor system has been equipped to deal effectively with a number of rather complicated eye movement tasks. Fortunately, to aid our understanding, the ocular motor system may be broken down into a few basic subsystems, each of which is responsible for a specific ocular motor function.

One subsystem of the ocular motor system is responsible for holding images of *stationary* objects still on the retina during rotations or perturbations of the head; this system is referred to as the *vestibulo-ocular reflex* (VOR). Additionally, the *smooth pursuit system* is available to enable the eye to hold the images of *moving* objects of interest steady on the fovea (Leigh and Zee 1991). Clearly, the purposes of these two eye movement systems are different: the VOR serves to *maintain* gaze (the direction of the eye with respect to space) on a stationary target despite perturbations of the
head, while smooth pursuit changes gaze, with the head stationary, to allow the tracking of moving targets. Another more complicated condition arises when the eyes track a moving target during simultaneous head motion. This is called combined eye-head tracking (CEHT). Although much is known about the operation of the VOR and smooth pursuit systems, there is still disagreement as to how these subsystems interact during CEHT.

My research primarily focused on investigating the mechanism used by the ocular motor system to perform CEHT. This involved characterizing the activity of the smooth pursuit and VOR ocular motor subsystems. Thus, to provide background for my research, this section will first discuss the VOR and smooth pursuit systems in more detail. Then, a discussion on the various hypotheses which have been proposed to explain CEHT will be presented. Finally, the section will conclude with a statement of my specific research hypotheses.

A. The Vestibulo-Ocular Reflex (VOR): Gaze-Holding

1. Teleology and Physiology of the VOR

Perhaps the most phylogenetically primitive component of the ocular motor system is the vestibulo-ocular reflex, or VOR (Walls 1962). By working to maintain clear vision of stationary objects despite motion of
the head, the VOR allows animals to see and move at the same time (Robinson 1978). In fact, one of the greatest potential sources of image slip is due to self-rotation. Thus, if during a rapid head rotation the eyes did not compensate by rotating in the opposite direction at the same speed, the resulting image slip would essentially prevent any sort of useful vision during the head movement (Robinson 1981). In fact, the VOR has been so useful that it has changed very little since its origin, and the same basic scheme for ocular stability can be found in widely different species of mammals, birds, and fish (Melvill Jones and Spells 1963).

This section briefly summarizes the physiological characteristics of the VOR. (For a more detailed development, see Leigh and Zee (1991), Wilson and Melvill Jones (1979), Carpenter (1988), and Robinson (1981)). We first consider the structure which transduces head motion. Within the temporal bone of the inner ear is a structure called the labyrinth. The labyrinth can be divided into three functionally distinct parts: the cochlea (with which we will not be concerned, as it is responsible for the transduction of auditory information); the semicircular canals, consisting of three (approximately) orthogonally arranged angular motion detectors; and the otolith organs, comprised of a pair of endolymphatic sacs—the utricle and saccule—responsible for detecting linear acceleration and the direction of gravity. (Because my research is not directly concerned with the eye movements generated during linear head motion nor with changes in head position with respect to gravity, only the signal originating within the semicircular canals will be discussed here.)
The *membranous labyrinth* lies within its bony counterpart and is cushioned with fluid called *perilymph*. Inside the membranous labyrinth of the canals is fluid called *endolymph* and, in the widened, bulging region each canal is found a group of epithelial receptor cells called the *cristae*. The cristae contain specialized hair cells which transduce mechanical shearing forces into neural impulses. The processes of these hair cells are embedded in the *cupula*, a gelatinous, sail-like structure. When the head rotates, the mass-dependent inertia of the endolymph tends to lag rotation of the canal, resulting in relative fluid flow within the canal. This displacement of fluid in the canals causes the cupula to bend, thereby stimulating the receptor cells which innervate the cupula. In this way, the semicircular canal-cupula system acts as a gyroscope by generating neural signals in response to angular head motion.

The VOR truly is a reflex; the signal generated in the semicircular canals traverses to the extraocular muscles via a three-neuron arc. From the receptor cells of the cupula, the neural signal encoding angular head velocity follows vestibular afferent fibers to the vestibular nucleus. From there, an interneuron transmits the vestibular signal to the ocular motor nuclei, where the final eye movement command signal is synthesized and sent to the extraocular muscles. For horizontal head rotations, the signals from the horizontal canals project to the lateral and medial recti muscles. The simplest pathway to the lateral rectus muscle is disynaptic and to the medial rectus muscle is trisynaptic. Clearly this oligosynaptic pathway can effect eye movements with a short latency (Maas *et al*. 1989). However, parallel polysynaptic projections are also of significant importance in
modifying the VOR signal. These additional pathways, some of which pass through the vestibulocerebellum, provide the capacity for long-term adaptation plasticity (based on internal comparisons with visual inputs) as well as short-term changes in VOR gain (see Berthoz and Melvill Jones (1985) for a review).

2. Models of the VOR

Although the primary spatial transformations between the semicircular canals and extraocular muscles are conceptually understood for the VOR, far less is known about the temporal signal processing in this reflex (Robinson 1981). However, this has not hindered efforts to model the system. Fundamental models of the VOR rely on known physiological characteristics. Indeed, it was the discovery of the cupula by SteinhAUSEN (1933) that prompted him to propose a *torsion-pendulum model* for the semicircular canal-cupula system that is still important today (see Wilson and Melvill Jones (1979) for a review). The essence of this model is that, although the semicircular canals sense head *acceleration*, because each canal’s lumen is small relative to its radius of curvature, and because of the hydrodynamic properties of the endolymph, a *mechanical integration* is performed in the canals; as a consequence, the cupula senses head *velocity*. Exceptions to this integration in the canals occur when the head experiences extremes in the frequency of motion. At very low frequencies (below 0.03 Hz), the elasticity of the cupula factors into the torsion-pendulum model by working against the motion of the
endolymph, effectively negating the integration. Thus, below approximately 0.03 Hz, the cupula transduces head *acceleration*. This can be modeled as a lead element with a time constant of approximately 5 sec. At very high frequencies (above about 50 Hz), the model predicts that the cupula transduces head *position*. This can be modeled with a lag element having a time constant of 0.003 sec. These results, which were originally based on psychophysical studies (van Egmond *et al.* 1949), have received confirmation by recording the activity of primary afferents of the vestibular nerve during head rotations with squirrel monkeys (Fernandez and Goldberg 1971).

Thus, at frequencies below 0.03 Hz, the cupula transduces head *acceleration*, while for frequencies between 0.03 and 50 Hz, the cupula transduces head *velocity*. Finally, above 50 Hz, the cupula transduces head *position*. Because this latter case is seldom encountered with normal head movements, its consequences are only of academic concern and most models of the VOR generally omit the corresponding lag element. It is interesting to note that the range of frequencies for which the canals transduce velocity generally corresponds to the frequencies of natural head movements (Grossman *et al.* 1988; Grossman *et al.* 1989; Melvill Jones and Spells 1963). Also, this model successfully predicts that with sustained rotation (in the dark), the signal from the VOR will dissipate with time (as would any similar system employing a lead—or, high pass filter—element), thereby causing eye velocity to fall toward zero.

Because the VOR is open loop (in that eye movements do not
influence the head movements that produced them), efforts to model its characteristics are relatively straightforward. These models generally start by providing a head velocity signal to the lead element proposed by the torsion-pendulum model of semicircular canal dynamics. From here, the signal is scaled by a gain element (usually having a value near 1.0) and inverted; the latter operation is to command the eyes to move in the opposite direction as head rotation, thereby allowing the angle of gaze to be maintained. From here, the signal continues to the common neural integrator and the plant where the command signal is manifested as an eye rotation. (For details on this latter step, see Section II.C.1.a below).

More sophisticated models involving the VOR have been developed. For example, Robinson (1977) expanded an elementary model of the VOR to include its supplementation by the optokinetic system. This model provides a possible answer to the curious observation that with sustained rotation in the dark, the eye velocity signal naturally diminishes with a time constant that is about three times as long as that predicted by the torsion-pendulum model. He explains this difference by proposing that an internal positive feedback loop helps maintain the signal from the canals, and he refers to the phenomenon as velocity storage. Then, to further supplement the VOR signal during sustained rotation in the presence of a visual surround, he suggests that a signal based on retinal slip (i.e., the velocity of the target with respect to the eye) is added to the signal in the positive feedback loop to augment the VOR signal back towards its initial level. A similar model of VOR-optokinetic interaction has been proposed by Raphan et al. (1979). Finally, advances in neural
network theory have provided new approaches to modeling which supplement more classical, block diagram modeling methods. As an example, Anastasio and Robinson (1989) have used a neural network approach to develop a model for the VOR which elucidates the distributed nature of the system's organization.

One additional characteristic of the VOR should be mentioned. The gain of the VOR is affected by the proximity of an object of regard (Viirre et al. 1986). This phenomenon is a consequence of the geometric arrangement of the eyes (eccentrically) within the head; I will address this issue in my research (Section II.B.2.b).

B. The Smooth Pursuit System: Gaze-Shifting

1. Teleology and Physiology of Smooth Pursuit

The purpose of the smooth pursuit system is to actively redirect the angle of gaze such that moving objects of interest are held steady on the fovea. This eye movement feature is only available to frontal-eyed, foveate animals, and it likely developed as a survival mechanism which improved the chances for a predator to pursue and catch its prey (Robinson 1981). In addition, because smooth pursuit allows the tracking of small objects, this system requires that retinal slip occurring from the relative "motion" of a stationary background with respect to the eye be
relatively ignored (Dodge 1903; Leigh and Zee 1991). Traditionally, smooth pursuit describes eye movements made with the head still.

Unlike the VOR which derives its afferent signals from mechanical activity in the semicircular canals, smooth pursuit eye movements are generated based on the processing of visual signals with the goal of reducing retinal slip. Then, because the visual input detector (the retina) is directly attached to the effected object of the ocular motor command signals (the eye), the smooth pursuit system inherently has a negative feedback nature. This is an important consideration when modeling smooth pursuit (see next section). By moving the eye with the appropriate speed and direction, it is possible to keep images of interest on the fovea.

The following discussion of smooth pursuit physiology is derived from Leigh (1989), Leigh and Zee (1991), and Robinson (1981). The origin of the smooth pursuit command signal lies with structures that process visual information. The visual signal is generated in the retina, where a significant amount of processing is required to convert the incident light information into neurological information. In fact, the latency between light striking the retina and the first electrical activity in visual cortex is almost 100 msec. A subdivision of the visual system has been identified that is concerned with the analysis of moving stimuli (Livingstone and Hubel 1988). This system starts with ganglion cells of the retina transmitting signals via the magnocellular layers of the lateral geniculate nuclei (LGN) to layer 4Cγ of primary visual cortex. Next, retinal information about the speed and direction of a moving target is abstracted
in the visual cortex, particularly in area MT (the *middle temporal* visual area), and is then transmitted to area MST (the *medial superior temporal* visual area), the frontal eye fields, and the *dorsolateral pontine nuclei* (DLPN). Area MST combines an internal, *afference copy* signal of eye velocity with the visual motion signal synthesized in area MT, sending the resulting information to the DLPN. The frontal eye fields may be important in programming *predictive* pursuit movements, and signals originating there may project independently to pontine nuclei, including the DLPN. Thus, the DLPN contains a mixture of processed visual information and internal eye movement signals that it subsequently passes on to the *flocculus, paraflocculus,* and *vermis* of the *cerebellum*. The cerebellum plays an important role in synthesizing the smooth pursuit command signal from visual and ocular motor inputs; in fact, bilateral lesions of the flocculus and paraflocculus greatly impair smooth pursuit (by reducing pursuit gain), while total cerebellectomy abolishes smooth pursuit altogether. The output from the cerebellum projects to the *vestibular nuclei* where, with the assistance of the *nucleus prepositus hypoglossi*, the eye *velocity* command signal is converted into an eye *position* command signal using a circuit referred to as the *neural integrator* (Cannon and Robinson 1987). Finally, the vestibular nuclei send the smooth pursuit command signal to the ocular motor nuclei, perhaps with some possible contributions from the PPRF (the *paramedian pontine reticular formation*). Movement of the eyes is effected when the ocular motor nuclei receive the eye position command signal.

Factors such as attention, motivation, and age influence how well
subjects pursue (Leigh and Zee 1991). In fact, my preliminary experiments suggest that even with target velocities as low as 30 deg/sec, some subjects have steady-state smooth pursuit gains (eye velocity divided by target velocity) that are less than one. In contrast, others have reported satisfactory tracking by humans with target velocities as high as 130 deg/sec (Lisberger et al. 1977). Although luminance and other properties of the target (e.g. color) may influence smooth pursuit eye movements (Lisberger and Westbrook 1985), these characteristics have only minor effects compared with their effects on aspects of visual perception.

Due to the various processing delays involved in creating the smooth pursuit command signal, the latency between the initiation of target motion and the corresponding change in eye motion is about 130 msec, although in extreme cases pursuit latencies as short as 75 msec have been reported (Carl et al. 1987). With non-predictable target motion, the delay resulting from this latency will cause a significant phase lag in eye movement. However, if the target motion becomes predictable, the brain can alter the smooth pursuit command signal to significantly reduce the latency and corresponding phase lag to yield eye motion that virtually coincides with target motion. The properties of this prediction mechanism are of interest (Becker and Fuchs 1985), but they confound study of the smooth pursuit pathway outlined above. Thus, to study smooth pursuit in its “pure” form, target motion should be non-predictable.

The most convenient way to study the initiation of smooth pursuit
is by measuring eye position or velocity within the first second after the presentation of a transient ramp (or a step-ramp—see Rashbass (1961)) of target motion. After the initial latency period, the first 20 msec of the pursuit response to this type of stimulus is typically unrelated to the speed of the target (i.e., initial eye acceleration is independent of target velocity), and it is less influenced by the brightness of the target or its position in the visual field than at later times in the response (Tychsen and Lisberger 1986). Thereafter, eye acceleration is a function of the velocity of the target motion across the retina. However, as target velocity is progressively increased, eye acceleration does not increase by the same amount; this has been referred to as acceleration saturation (Robinson et al. 1986). Also, the early phase of smooth pursuit frequently shows an initial velocity overshoot of the target which is usually followed by damped oscillation—or “ringing”—at frequencies of about 3 to 4 Hz. These response characteristics have been used in developing quantitative hypotheses for smooth pursuit, as described next.

2. Models of Smooth Pursuit

As explained earlier, the generation of smooth pursuit eye movements inherently involves negative feedback. Thus, efforts to model smooth pursuit must incorporate an external negative feedback pathway which supplies the retina with eye motion information. To maintain an image stationary on the fovea, there can be no difference in velocity between the target and the eye; any net difference in velocity
results in retinal slip. It is this retinal slip velocity that drives the pursuit system, and it is customarily modeled as resulting from the subtraction of the feed-back eye velocity signal from the target velocity signal. If appropriate smooth pursuit command signals are generated, eye velocity will approach target velocity and retinal slip velocity will subsequently approach zero. Therefore, smooth pursuit is usually modeled as a velocity servo system, and the differences between current models of smooth pursuit lie in the way the internal eye movement command signal is generated.

There are presently two major hypotheses about how the smooth pursuit system operates. One hypothesis uses a classic goal-oriented approach in which the system uses internal positive feedback to create an internal representation of target velocity (Young et al. 1968). This construct is then used as a goal by the system for generating the eye movement command signal. The other hypothesis assumes the use of parallel processing in a feed-forward structure (Lisberger et al. 1987). This type of system creates the command signal by pooling contributions from various internal elements and does not rely on the internal feedback of information.

a. Parallel Processing Model

Lisberger and his colleagues advocate the parallel processing approach to modeling the smooth pursuit system (Lisberger et al. 1987;
Krauzilis and Lisberger 1989). Their models concentrate on creating the eye movement command signal by subjecting the retinal slip signal (and derived forms of it, i.e., retinal slip position, velocity, and acceleration signals) to various explicit nonlinearities and transfer functions, and subsequently combining the processed results. Here, the capacity of their model to recreate and predict measured data rests in the ability to adequately define the transfer characteristics of the various system nonlinearities. They define their nonlinear elements based on behavioral measurements collected under open-loop conditions. (These “open-loop” experiments involve augmenting the target position command signal with an instantaneous measurement of eye position.) This is an interesting way to model a feedback system: design experiments that effectively remove the effects of feedback and use the measured results to define the internal transfer characteristics of the system. Then, if a condition is discovered that is not explained by the model, another parallel path can simply be added that will modify the command signal in an appropriate way.

Unlike some other models for smooth pursuit, this type of model has been shown to correctly describe unusual subject data collected using novel stimuli under open-loop conditions (D. A. Robinson, personal communication). On the other hand, because this type of model still employs an external negative feedback loop, modeled smooth pursuit gains can never be greater than 1.0; however, gains greater than 1.0 are occasionally observed in subjects (Robinson et al. 1986). Also, because this type of model uses several nonlinear elements whose definitions are
derived empirically from subject data, it would be difficult to study the influences of individual elements on the operation of the model. Such effects can be easily studied in other system models of smooth pursuit by changing the values of internal delays and time constants.

b. Goal-Oriented Model

Perhaps the most thorough goal-oriented approach to modeling the smooth pursuit system was presented by Robinson et al. (1986). Development of this model followed the lead of Young et al. (1968) who proposed using an internal positive feedback loop to effectively cancel the inherent negative feedback loop. What are the advantages of cancelling the external negative feedback loop? By eliminating the external feedback pathway, the system becomes open loop. Such a system removes inherent instabilities imposed by negative feedback and, unlike the feed-forward models discussed above, this type of system can have any value of steady-state gain (even greater than 1.0). The positive feedback of an internal motor command has a physiological basis; it has been called efference copy (von Holst and Mittelstaedt 1950) and corollary discharge (Sperry 1950).

As mentioned earlier, the degree of retinal slip is observed as the difference between target velocity and eye velocity. Then, if an internal estimate of eye velocity (manifested as the eye movement command signal) can be fed-back and added to the retinal slip signal, an internal estimate of target velocity is created. This is precisely what happens when
the internal positive feedback loop is employed to cancel the negative feedback loop. Thus, the proposed positive feedback loop also creates the goal signal used by the smooth pursuit system when it generates the eye movement command signals. In fact, Newsome et al. (1988) provide evidence that the efference copy feedback signal of eye velocity is incorporated with other signals in area MST and influences generation of the smooth pursuit command signal.

With the system effectively open loop, Robinson et al. were free to select system elements which, when combined, could generate signals representative of actual subject responses. Because ringing is often observed in smooth pursuit responses, they installed a separate internal negative feedback loop and equipped it with appropriate delay and gain characteristics to adequately represent the acceleration and ringing characteristics of responses from subjects. As part of this, they included a representation of the observed acceleration saturation phenomenon (described above). They also included plasticity parameters which could represent long-term changes in pursuit gain. Thus, this modeling approach provides realistic response simulations and allows a researcher the opportunity to "tweak" system element delays and time constants to investigate specific characteristics of the system and their overall effect on the system response. These are the main reasons I chose to use the goal-oriented model of Robinson et al. in my research.

Although the parallel processing and goal-oriented models of smooth pursuit seem to have widely varying characteristics, Deno et al.
(1989) showed that if both models are made linear, then the two models are essentially equivalent (at least in mathematical terms) and may be collapsed into a more compact structure. This has the positive effect of suggesting that neither model is fundamentally more or less correct than the other. However, even if one can accept such restrictive assumptions about linearity, performing this transformation essentially eliminates the essence of both models by removing their ability to completely describe how each system is hypothesized to operate.

Finally, all of the presented models are equipped to deal only with non-predictable smooth pursuit stimuli. As explained earlier, prediction has a significant effect on the operation of the smooth pursuit system. Thus, to my knowledge, no accepted model of smooth pursuit has incorporated elements to account for prediction.

C. Combined Eye-Head Tracking (CEHT)

Consider the circumstance in which an object of interest moves horizontally and the subject smoothly turns his or her head to track the moving object. In this case, the eyes must remain stationary with respect to the head to maintain foveation of the object. This describes combined eye-head tracking (CEHT), a phenomenon which occurs regularly in everyday life.
As described earlier, however, the VOR is a reflex which causes the angle of gaze to be maintained despite rotation of the head. When the head rotates in one direction, the vestibulo-ocular reflex invokes a compensatory eye rotation in the opposite direction. Thus, in order to allow CEHT to occur, the VOR must be somehow cancelled or suppressed to avoid the compensatory rotation of the eyes and keep them stationary in the head.

How is the VOR cancelled, suppressed, or otherwise overcome during horizontal CEHT? A number of possible explanations exist. One simple way would be to keep the vestibular command signal from reaching the ocular motor nuclei by causing an effective "open-circuit" in the VOR pathway during CEHT. However, this cannot be a valid mechanism because neurons projecting to the ocular motor nuclei continue to carry vestibular information during CEHT (Robinson 1982; Tomlinson and Robinson 1984). Thus, a more sophisticated scheme must be used to provide VOR cancellation. I will next highlight some of the proposed hypotheses for VOR cancellation during horizontal CEHT.

McKinley and Peterson (1985) suggest that the VOR is suppressed through parametric gain changes of the VOR. With this hypothesis, the suppression of the VOR signal during CEHT is achieved completely within the VOR system proper, and does not rely on interaction with other eye movement systems. No specific mechanism has yet been presented to suggest how the brain decides when to allow CEHT nor how this suppression of VOR gain actually occurs.
Another hypothesis of VOR cancellation during CEHT proposes that a copy of the internal VOR signal runs in parallel with the actual VOR signal. Then, when VOR cancellation is required, this accessory VOR signal adds negatively with the actual VOR signal, effectively causing the VOR to cancel itself (May and McCrea 1985; Tomlinson and Robinson 1981). This hypothesis also implies that the cancellation of the VOR occurs completely within the VOR system itself. Although a block diagram model of this process was presented, no specific mathematical description of the dynamics involved in VOR self-cancellation have been proposed.

The hypothesis that the VOR signal is canceled directly by a signal generated within the smooth pursuit system has received widespread support (Barnes et al. 1978; Lanman et al. 1978; Lau et al. 1978; Leigh et al. 1987; Barnes and Eason 1988; Ranalli and Sharpe 1988). In fact, it is possible that the original stimulus in the evolution of the smooth pursuit system may have been the need to cancel the effect of the VOR rather than to track targets when the head was still (Robinson 1978; Robinson 1981). The proposed means of combining the VOR and smooth pursuit signals with this hypothesis is through a superposition (or, a simple linear addition) of the component signals. This is supported by the observation that when the head rotation of a monkey is suddenly braked during CEHT, the eye starts to track the moving target with a latency of less than 15 msec (Lanman et al. 1978). The prompt initiation of eye movements in response to the moving target suggests that a smooth pursuit signal is already
actively being relayed to ocular motor neurons during CEHT. Thus, the smooth pursuit signal may be used to cancel the VOR. Because mathematical models exist for both the VOR and smooth pursuit systems, it is possible to test this hypothesis quantitatively by comparing subject response data with model simulation data comprised of the superposition between the output signals from the VOR and smooth pursuit models.

Robinson (1982) extended the superposition model just described to include CEHT in which the head motion is initiated by the subject (head-free tracking). He realized that the internal, self-mediated generation of head movements can cause alteration in the way the VOR and smooth pursuit are manifested, and he subsequently modified the superposition model to account for these changes. However, in cases of passive head rotation (in which the subject's head is rotated by an external means), Robinson's model of VOR cancellation simplifies to be structurally identical to the superposition hypothesis presented above.

Finally, Cullen et al. (1989) propose that the VOR is partially cancelled using an accessory VOR suppression pathway under certain CEHT conditions. This accessory pathway would work in consort with a smooth pursuit signal when successful CEHT is interrupted by an additional head perturbation. Again, although a block diagram model of this process was proposed, no specific mathematical description of the dynamics involved in the accessory VOR suppression pathway has been presented. Thus, it is not possible to quantitatively test this hypothesis.
D. Statement of Research Hypotheses

The goal of my research was to investigate how the VOR is cancelled during horizontal combined eye-head tracking. My working hypotheses assumed that (1) the smooth pursuit system provides the primary signal responsible for cancelling the VOR during CEHT, and (2) this cancellation occurs as a simple summation (superposition) of the two component ocular motor signals. I then searched for evidence to refute these hypotheses. Although these hypotheses have been proposed by other researchers (see above), this was the first attempt we know of to confirm them rigorously in humans.

My strategy was to obtain realistic mathematical models for the component VOR and smooth pursuit systems, and, using optimal parameter estimation techniques, to individualize these models for each subject. By joining the outputs of the two component models, I created a combined model for each subject capable of generating simulated CEHT eye movement signals consistent with my working research hypotheses.

To test the combined model, I exposed each subject to various CEHT paradigms and compared the subject response data with the corresponding model simulation data. By carefully designing the experimental paradigms to employ, I took advantage of the significant difference in the response latencies of smooth pursuit and the VOR to elucidate when each system was active. Also, all head rotations were passive and all stimuli were randomized to minimize the possible effects of prediction.
II. METHODS

A. Experimental Methods

This section introduces the design of experiments used to test the hypotheses formulated in Section I.D, above. It discusses the experimental stimuli, the specific test paradigms, as well as the hardware and software used to perform experiments and collect data.

1. Experimental Apparatus

a. Hardware

i. The Visual Stimulus

The visual target consisted of a spot of white light projected onto a semi-translucent display screen in a darkened room. A slide projector was used as the light source, and its beam was projected through a small pin hole in the center of an opaque plastic slide. The resulting display spot subtended 0.3 degrees and had a luminance of 0.77 ft-lamberts.

This light beam emerged from the projector adjacent to the bottom of the display screen and parallel to it. To allow modification of the light spot's horizontal position on the display screen, the beam was next reflected from a mirror galvanometer (General Scanning, model CCX-660,
Watertown, MA; bandwidth: 0-150 Hz) under computer control. A uniform change in the signal to the galvanometer caused a uniform rotation of the mirror and thus, a uniform rotation of the light beam. The galvanometer’s controller permitted adjustment of the gain and offset of the computer’s command signal for calibration purposes. From the galvanometer-mounted mirror the beam was projected backward (and slightly upward), away from the display screen toward another large mirror mounted to a wall, parallel to the display screen (see Fig. II-1). Use of this back-projection mirror effectively increased the distance from the light source to the display screen and allowed the galvanometer-mounted mirror to project the light beam the entire width of the display screen. The beam was projected slightly upward to avoid contacting the projector apparatus when reflected back from the wall-mounted mirror to the display screen. Although the distance from the light source to the screen varied, because the light spot was small, any distortions of the spot due to projecting it on the screen at an oblique angle were negligible.

The display screen consisted of a semi-opaque sheet of plastic (Filmscreen 200, Stewart Filmscreen Corp., Torrance, CA) that formed a flat wall in front of the subject. The screen was placed 1.3 m away from the subject; at this distance, the screen subtended approximately ±40 deg with respect to the subject.

The projector was fitted with a computer-controlled mechanical shutter (A. W. Vincent Associates, Inc., Rochester, N.Y.; model 214L, transfer opening time: 1.5 msec) to allow the light spot to be displayed or
Fig. II-1. Diagrammatic representation of the visual display apparatus showing the optical path of the target light spot from the projector to the subject. After the light beam reflects from the galvanometer-mounted mirror, it emanates backwards toward another large, wall-mounted mirror. From there, the beam is deflected forward to create the target image on the semi-opaque display screen.
hidden, as necessary. Because all of the experiments employed with this research used the light spot as a continual visual stimulus, the computer commanded the shutter to remain open throughout each experiment.

ii. The Vestibular Stimulus

Vestibular stimulation was provided to the subject using a vestibular chair (Templin Engineering, Laytonville, CA). This belt-driven chair is connected to a motor capable of providing 30 ft-lbs of torque and chair rotations of up to ±150 deg/s. The chair controller converts input voltages from the computer to angular velocity command signals to the chair. Then, to provide chair position information, I equipped the chair with a 12-bit optical encoder (Model M25G-F4-LSS-4096N-G-D1-CR-EC14-X-5, BEI Motion Systems Co., Cincinnati, OH) capable of resolving chair rotations of 0.088 deg.

Because body weight can affect angular acceleration rate, the chair controller is equipped with a dial to allow for uniform acceleration, independent of the subject’s weight. I installed a switch to override any imposed controller limits on acceleration to provide as large a vestibular stimulus as possible. However, we found that using the chair in this way produced high frequency vibrations of the head-support system that led to artifactual coil signals. Therefore, I reverted to performing the experiments under limited-acceleration conditions. For uniformity, all experiments were performed at the 85 Kg setting.
iii. The Eye-Coil Measurement System

The gaze (G) and head position (H) data were collected using the magnetic field/scleral search coil technique (CNC Engineering, Seattle, WA). This method (Collewijn 1977) essentially involves placing a small coil of copper wire, embedded within a silastic annulus, around the cornea of the subject's viewing eye and fastening another similar coil to his or her forehead. Then, two orthogonal alternating magnetic fields are established about the subject. These fields are generated horizontally, one parallel and the other perpendicular to the display screen, and they are 90 degrees apart in phase. This creates a rotating magnetic vector around the subject in the horizontal plane. When either (or both) of the eye and head search coils rotates in the magnetic field, a small current is induced in the wire. A phase detector compares the phase of the sinusoidal signal induced in the search coils with the phase of the signal induced in a stationary reference coil. The difference between the phases of the search and reference coils is linearly related to the angle of the search coil in the horizontal plane, and the output of the phase detectors is a voltage proportional to this angle. Thus, a uniform angular rotation of a search coil in the horizontal plane will generate a uniformly changing voltage signal, which can be sent to the computer for digitization and storage. This method is insensitive to variations in the magnitudes of the induced currents and thus, does not depend directly on knowing the coil size or the number of turns in the coil. The generated signals are also insensitive to
linear translations of the coils (less than 0.03 deg/cm) within the normal operating region of the coils. (This normal operating region corresponds to subjects' eye locations in space as they are seated in the vestibular chair. It is situated within a 30 cm cube at the center of the applied magnetic field.)

(If I had been interested in measuring vertical eye and head movements, we have available another system (Robinson 1963) which uses the same rotating magnetic vector, but it instead considers the amplitude of a search coil signal and relates it to the projection of the search coil on the vertical plane. In this case, after phase detection is performed to remove the common field signal from a search coil's induced signal relative to the appropriate vertical reference coil, the output voltage is proportional to the sine of the angle the coil makes with the vertical. This method is sensitive to the size of the search coils and to the number of turns of wire in the coils, and it requires an arcsine correction to yield the angle of interest.)

iv. The Computer System

Once the search coil displacements have been converted into voltages, they are available for measurement and analysis by a number of devices. I chose to digitize the voltages and store them for subsequent analysis using a computer.
We employed an IBM-PC/AT operating under DOS 3.3 as our primary computer resource. To facilitate digitization of the coil position signals, we equipped the computer with two Data Translation DT-2801 input-output boards. One was used primarily for data acquisition: it was a DT-2801/5716A board configured to sample my three input signals (gaze, head, and target position) at 1002.5063 Hz with 16-bit resolution. The other board was a standard DT-2801 used primarily to control the experimental stimuli: specifically, it decoded the target position command signal, the chair velocity command signal, and the commands to the projector and laser spot shutters (the latter of which was not used in this set of experiments). This board also monitored the signal from the optical encoder to provide information about the vestibular chair's angular position. To aid in making connections to the boards, we equipped each with a Data Translation DT-707 screw terminal panel. Table II-1 lists all external connections to the input-output boards.

In addition to the input-output hardware, the PC-AT was equipped with BocaRAM expanded memory boards to provide maximum computing power to ASYST™, my primary data analysis software environment (Hary et al. 1987).

v. The Analog Filters

To prevent aliasing, I filtered the input signals (target, gaze, and head) with a set of matched Krohn-Hite analog low-pass filters (Model
Table II-1. External connections to the computer’s input-output boards

**Board 1 (DT 2801)**

*Digital-to-Analog Converters (12-bits):*

<table>
<thead>
<tr>
<th>DAC 0</th>
<th>Output Position Signal to Target Controller</th>
</tr>
</thead>
<tbody>
<tr>
<td>DAC 1</td>
<td>Output Velocity Signal to Chair Controller</td>
</tr>
</tbody>
</table>

*Digital I/O:*

<table>
<thead>
<tr>
<th>Port 0, Bit 0</th>
<th>Shutter for Gaze-Initiating Laser</th>
</tr>
</thead>
<tbody>
<tr>
<td>Port 0, Bit 1</td>
<td>Shutter for Target Projector</td>
</tr>
<tr>
<td>Port 1, Bit 0</td>
<td>Optical Encoder Latch</td>
</tr>
<tr>
<td>Port 1, Bit 1</td>
<td>Optical Encoder Rotation Control</td>
</tr>
</tbody>
</table>

**Board 2 (DT 2801/5716A)**

*Analog-to-Digital Converters (16-bits):*

<table>
<thead>
<tr>
<th>ADC Channel 0</th>
<th>Input Target Position Signal</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADC Channel 1</td>
<td>Input Gaze Position Signal</td>
</tr>
<tr>
<td>ADC Channel 2</td>
<td>Input Head Position Signal</td>
</tr>
</tbody>
</table>

*Digital-to-Analog Converters (12-bits):*

| DAC 0          | Output Position Signal to Laser Controller (not used) |

*Digital I/O (Both ports):*

| Bits 0-11      | Optical Encoder Chair Position Data (straight binary) |
3321) before digitization. Although most of the signal energy lies below 20 Hz, I decided to maintain as wide a bandwidth as possible to be certain that observed response features (such as delays or changes in acceleration) were real and not an artifact from using too narrow a bandwidth. Thus, each signal was low-pass filtered with a cutoff frequency of 100 Hz (well below the folding frequency of approximately 500 Hz) using the filter’s “Low-Pass, Max Flat” setting. This was equivalent to using a 4-pole Butterworth filter.

These filters can only deal with a maximum input voltage of 7 volts. Thus, although the search coils were calibrated to represent the full-scale operating range of ±50 degrees with full-scale output voltages of ±10 volts, the use of these filters limited the effective operating range to ±35 degrees.

b. Software

To facilitate collection of the eye, head, and target motion data, I created a group of three routines, written in Microsoft FORTRAN (version 4.01), which interfaced with the input-output boards using the Data Translation subroutine package PC-LAB. These routines (1) aided calibration of the target and chair stimuli, (2) provided an interface for designing flexible experimental paradigms, and (3) interpreted a specified set of paradigm instructions, carried out the experiment, and stored the collected data in a file for subsequent analysis. Each of these routines are
identified by the characters SPVOR, indicating that they are part of the package for testing Smooth Pursuit, the VOR, and the interaction between them.

Finally, this section will conclude by describing a mathematical modification to the target command signals, implemented within the above routines, which simulates use of a curved display screen. This modification is to provide non-uniform linear target motion on the flat display screen that effectively resembles uniform angular target rotation on a curved screen about the subject.

i. SPVORCAL—Calibration of Target and Chair Stimuli

The routine SPVORCAL aided calibration of the target and chair stimuli by outputting static command signals to the controllers of these devices, as specified by the user. First, the user chooses a position to place the target, and the computer generates an appropriate command signal to the General Scanning controller to move the target to that position. This is repeated for different positions to allow setting the controller's gain and offset settings. To aid in the calibration procedure, I suspended plumb lines from the display screen at precalculated positions corresponding to known angular displacements (e.g., ±15, ±20, and ±25 deg). The settings of the controller were altered until the target repeatedly aligned with the plumb lines. After target calibration the plumb lines were removed.
Also, the user is given the option of modifying the command signal to the target controller in a way that simulates the use of a curved display screen. If this (default) option is chosen, the geometric correction described below in Section iv would be employed, thereby enabling targets displayed on the flat display screen to be presented at calibration locations that would appear the same to the subject as targets displayed on a curved screen at a uniform distance from the subject. If this option is not used, calibration of the target would still be possible, but target positions other than the calibration positions would not be at the expected positions with respect to the subject. (The later condition was retained to maintain compatibility with paradigms used before the geometric correction was available.)

After calibration of the target, the command signal to the chair controller is calibrated. The chair controller was factory calibrated such that an input signal of +1 volt yields a clockwise chair rotation of 30 deg/sec; we confirmed this by timing several rotations when the chair controller was provided with a known voltage. I discovered that when the computer was used to control the chair directly, a power buffer was necessary to boost the command signal. For this reason, I passed the chair velocity command signal through an amplifier (Tektronix, Model AM 502) to provide the necessary signal buffering. Thus, calibration of the command signal to the chair consisted of sending known voltages from the computer to the amplifier and adjusting the amplifier settings using a voltmeter. SPVORCAL provides a simple interface for this procedure by having the user specify a desired chair velocity and then displaying the
value the voltmeter should read. This is repeated until the user is satisfied with the amplifier settings.

ii. SPVORSTM—Creation of Experimental Stimulus Paradigms

To make the data acquisition program (SPVOR) as flexible as possible, I designed it to allow users to “plug-in” any variety of available experimental paradigms. This is accomplished by creating special stimulus paradigm files containing specifications for each stimulus used by SPVOR in a given experiment as well as the order in which these stimuli are to be presented. The routine SPVORSTM is used to create these STiMulus paradigm files for SPVOR.

The user creates one or more stimulus paradigm files before the actual experiment is performed. Then, when the experiment is to be performed, the user simply specifies the name of the stimulus paradigm file to use. Thus, the nature of a given experiment is governed by the contents of the specified stimulus paradigm file.

Each stimulus used in an experiment is defined in terms of a configuration. A configuration is a set of specifications which completely define a given stimulus. These include:

- The stimulus type (e.g., Smooth Pursuit Onset, Chair Brake)
The sampling frequency to be used for collecting data

The sampling time prior to the trial onset

The target status between trials (on or off)

The target status during trials (on or off; when applicable, e.g., for VOR stimulus types)

The initial target and chair positions

The final target and chair positions

The target and chair velocities when each is moving

Whether or not the actual final target position should be randomized beyond the specified final target position (a stimulus feature that has been shown to be necessary to maintain highest smooth pursuit gain (Robinson et al. 1986))

Laser fixation spot status before and during trials (on or off; used primarily for step-ramp (Rashbass type) stimuli (Rashbass 1961))

A list of all the available stimulus types is given in Table II-2.

To create a stimulus paradigm file, the user defines several different
<table>
<thead>
<tr>
<th>Stimulus Type Number</th>
<th>Stimulus Name</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Smooth Pursuit Onset</td>
<td>Target moves, Chair stationary</td>
</tr>
<tr>
<td>2</td>
<td>Smooth Pursuit Offset</td>
<td>Target moves, Chair stationary; Recording continues after target stops</td>
</tr>
<tr>
<td>3</td>
<td>VOR Onset</td>
<td>Target stationary, Chair moves</td>
</tr>
<tr>
<td>4</td>
<td>VOR Offset</td>
<td>Target stationary, Chair moves; Recording continues after chair stops</td>
</tr>
<tr>
<td>5</td>
<td>CEHT Enhance VOR</td>
<td>Target moves; Chair moves some specified time later</td>
</tr>
<tr>
<td>6</td>
<td>CEHT Onset</td>
<td>Target and Chair begin moving at the same time</td>
</tr>
<tr>
<td>7</td>
<td>Chair Brake</td>
<td>Target and Chair begin moving at the same time; Chair stops midway through</td>
</tr>
<tr>
<td>8</td>
<td>Target Brake</td>
<td>Target and Chair begin moving at the same time; Target stops midway through</td>
</tr>
<tr>
<td>9</td>
<td>Delayed Chair Onset</td>
<td>Target starts to move; Chair also begins to move when Target and Chair align</td>
</tr>
<tr>
<td>10</td>
<td>Delayed Target Onset</td>
<td>Chair starts to move; Target also begins to move when Chair and Target align</td>
</tr>
<tr>
<td>11</td>
<td>CEHT Generic</td>
<td>Target and/or Chair may remain stationary, move, or stop at arbitrary positions; Multiple velocities available</td>
</tr>
</tbody>
</table>
configurations, each with its own specific stimulus characteristics. Each configuration is identified by the order in which it was created (i.e., the first configuration defined becomes Configuration 1, the second becomes 2, and so on). Then, after all the desired configurations have been defined, the user lists the order in which the configurations are to be presented during an experiment. Each configuration may be used as many times as desired by the user, and the order of the configurations may be made unpredictable, if so desired. Also, the user has the option of specifying a pause in the experiment any time between trials to allow periodic monitoring of the subject's condition, particularly to check the comfort of the eye coil. After the order of the configurations (and pauses) is specified, the user may specify the minimum time to wait between trials. This provides the user the opportunity to allow extra time between trials, for example, to allow for the dissipation of after-images or to allow the cupulae to return to rest following a head rotation. Finally, the user specifies the name of the stimulus paradigm file.

This program affords considerable flexibility for creating the stimuli for an experiment. For example, different sampling rates may be used for different trials depending on the expected frequency content of the stimuli or response data, VOR studies may be conducted both with and without a target during the same experiment, and the same stimulus type may be presented with several different velocities in either direction. Thus, SPVORSTM can be used to design many types of experiments calling for transient, horizontal target and/or head rotations in which the stimuli move as a ramp of position (step of velocity).
iii. **SPVOR—Performing Combined Eye-Head Tracking Experiments**

Assuming that a stimulus paradigm file has been created using SPVORSTM, that the target and chair command signals have been calibrated using SPVORCAL, and that the head and eye coils have been calibrated (using other means), the user may conduct an experiment using SPVOR.

When invoked, SPVOR asks the user for the name of a stimulus paradigm file. Next, if any of the trials requires chair motion, the routine asks if the chair acceleration will be limited or maximal. If limited chair acceleration is used, the chair controller causes a small time delay between the time that the chair is commanded to move (or stop) and the time that the chair actually begins to move (or stop). This may be an undesirable effect, particularly if the target and chair are to move in synchrony. For this reason, the routine allows the user to enter a compensation time between target and chair motion which causes commands to the chair controller to be executed earlier. If an appropriate compensation time is entered, the final result of this modification causes the target and chair to begin moving together. Based on preliminary measurements, I determined that an appropriate value for this compensation time was 25 msec for my experiments.

The response data file is created next. The user specifies the name of
the file; by convention, the file extension is usually specified as "SPV" (for Smooth Pursuit, VOR). The current time and date are then automatically stored to the file, along with relevant information about whether the geometric correction to simulate a curved display screen is used and whether the time compensation is used. The following information is then entered: subject name, the eye containing the search coil (left or right), characteristics of the target slide (e.g., light spot, random dots), analog filter settings (e.g., 100 Hz, low-pass max-flat), and any other miscellaneous comments which may be of value in interpreting the data at a later time. If an artifact correction look-up table was created, its file name is stored next to provide association between the response data and the look-up table when analysis is performed (see Section II.B.1 for a discussion on artifact correction). Finally, relevant systems data are stored for later reference. This includes the number of analog-to-digital (A/D) converter bits used, a gain value representing the full-scale number of volts the A/D converters were set to accept, the maximum full-scale range of coil and target position values, the number of different stimulus configurations, and the total number of trials in the experiment. Because the measured position values will be stored in the file in their raw, binary format (to minimize the file size), many of these system values are stored to allow conversion of the data into useful units (degrees or seconds) within the analysis program.

When ready to begin the experiment the user presses (Enter). The first trial is executed, after which the measured target, eye, and head position data are plotted on the computer monitor to verify the integrity
of all input channels. After verification, \( \langle \text{Enter} \rangle \) is again pressed, and the
experiment proceeds. If a pause was called for after a given trial, the
program will suspend operation when the pause is reached. At this point,
the user may continue the routine by pressing \( \langle \text{Enter} \rangle \) or abort the routine
by pressing \( \text{A-} \langle \text{Enter} \rangle \). The pause may be useful for attending to the needs
of the subject or for resolving problems with the experimental apparatus
(\textit{e.g.}, replacing a burned-out projector lamp). When the experiment
terminates, either after all the designated trials have been completed or
after being aborted by the user, all the collected data are stored in the data
file for subsequent analysis.

For this research, each experiment was comprised of between 48 and
60 trials and lasted between 20 and 30 minutes. One guide for determining
the number of trials to use in an experiment is to limit the experiment
time to last only as long as the anticipated subject population can
comfortably wear the search coil (about 30 minutes). Also, each floppy-
diskette is capable of storing a maximum of 1.2 MB of data. Because it is
advisable to back-up the data from each experiment on a floppy-diskette,
the data file from an experiment should not be larger than 1.2 MB. The
file size is determined not only by the number of trials but the amount of
data collected in each trial. The latter is governed by several variables,
including the trial’s sampling rate, the distance traveled by the trial’s
stimuli and the velocity at which they traveled, and the amount of data
collected before and after stimulus motion. Because there is no easy way
to determine the size of a data file when the stimulus paradigm file is
created, the user may wish to first run a blank experiment, without a
subject, and check the size of the resulting data file. (Such an experiment may also be useful for verifying that the stimulus paradigms were defined correctly.) If the resulting data file is too large, the stimulus paradigm file may be edited to remove some trials.

iv. Target Signal Modification to Provide Uniform Angular Target Motion

Our visuo-vestibular stimulus system includes an apparatus that back-projects a spot of light onto a flat, translucent display screen for use as a visual target. The display screen is 1.3 m in front of the subject, and the light spot (which subtends 0.3 deg visual angle) is deflected horizontally using a mirror galvanometer that is rotated under computer control (Fig. II-1). Because a flat display screen is used, our range of target motion is limited to approximately ±40 deg with respect to the subject.

In order for a visual target to appear to move at a constant angular velocity with respect to the subject (as required by my experiments), the target must move at a non-constant velocity across the flat display screen, slower in the middle and faster near the ends of the screen. This is achieved by rotating the mirror galvanometer in a nonlinear way.

For a given instant in time, consider what galvanometer deflection \( T' \) is required to present an observed target deflection \( T \). An equivalent configuration of our experimental apparatus is shown in Fig. II-2. From this, we see that knowing \( T, D \) (the minimum distance from the subject to
Fig. II-2. Geometry used to calculate the appropriate angle of galvanometer deflection $T'$ required to create an observed angle of target deflection $T$. $D$, minimum distance from the subject to the tangent screen; $\delta_1$ and $\delta_2$, minimum distances from the galvanometer mounted mirror to the back-projecting mirror and from the back-projecting mirror to the display screen, respectively (see Fig. II-1). $\delta_1$ and $\delta_2$ are used to calculate $\Delta$, the effective minimum distance from the mirror galvanometer to the display screen.
the tangent screen), and \( \Delta \) (the effective minimum distance from the mirror galvanometer to the display screen), we can calculate \( T' \) using simple trigonometry:

\[
D \tan T = \Delta \tan T' \quad \text{II.1a}
\]

\[
T' = \tan^{-1} \left( \frac{D}{\Delta} \tan T \right) \quad \text{II.1b}
\]

Thus, to obtain the desired target deflection characteristics for a given trial, I first determine how quickly the computer will change the voltage values sent to the galvanometer to (discretely) change the position of the mirror. I then construct an array containing a sequence of desired target deflection angles \( T_i \) for the trial, which, when output from the computer to the galvanometer in a time sequence using the computer's D/A converter, will create motion of the target. Next, I modify each array value by the relation given in Eqn. II.1b to obtain the necessary sequence of galvanometer position values \( T'_i \) that will make the target appear to move at the desired velocity.

Calibration of the mirror galvanometer controller is performed using a similar procedure. Marked calibration positions are chosen on the display screen (distance \( d \) in Fig. II-2, usually corresponding to \( T=0 \) deg and \( T=\pm25 \) deg) and the modification of galvanometer position given in Eqn. II.1b is made. Then, the gain and offset controls of the galvanometer are adjusted to move the light spot to the desired calibration location on the
display screen.

Note that unless \( \Delta \) equals \( D \), the operating ranges of \( T \) and \( T' \) are different. For example, if \( \Delta \) is twice the length of \( D \), then an observed full-scale target position of 40 deg would only require a galvanometer deflection of about 22.8 deg. Thus, if \( T' \) is multiplied by an appropriate scale factor before calibration, it will span the same range as \( T \) (and thus, span the entire D/A range, which has advantages when considering quantization noise). Any corresponding increase in gain will be compensated for during the subsequent calibration procedure. If the maximum deflection of \( T \) is \( T^* \) (40 deg for my experiments), then the (constant) scale factor

\[
\frac{T^*}{T'(T^*)} = \tan^{-1}\left(\frac{D}{\Delta \tan T^*}\right)
\]

II.2

will adjust \( T' \) to use the entire available operating range. Again, each element of the \( T'_i \) array will be multiplied by this scale factor before calibration is performed.
2. Experimental Protocol

Four normal male subjects between the ages of 23 and 42 were used for this experiment. All gave informed consent. Subject 1 was a myope who habitually wore contact lenses (correction: OD -4.50, OS -5.00), subjects 2 and 3 were emmetropes, and subject 4 was a myope who wore spectacle corrections (OD -2.00, OS -1.25 -0.50x006). No subject wore his correction during experiments and none was taking any medication.

At the time of each experiment, the subject was seated in the vestibular chair and restrained with a Velcro™ lap belt. Two to three drops of proparacaine HCl 0.5% were placed in the subject's dominant eye as an anesthetic. The search coil for measuring gaze was then lubricated with a few drops of artificial tears and carefully placed on the subject's dominant eye. To prevent possible confounding effects of binocular viewing, I placed an eye patch over the subject's non-dominant eye. The search coil used to record head position was secured to the center of the subject's forehead with cloth adhesive tape. The vestibular chair's head restraint was then fastened to immobilize the head. Fastening the head restraint also made the head coil fit more tightly to the subject's forehead, so that I could be sure that head coil rotations accurately reflected true head rotations.

The subjects were told that a spot of white light would occasionally move across the display screen and that the chair in which they were seated would occasionally rotate; they were instructed to follow the spot of
white light with their eyes—to not let the light spot out of their sight. They were also asked to count or otherwise utter aloud to maintain alertness (Weissman et al. 1989). Finally, the room lights were turned off and the subject was allowed a brief time to acclimate to the darkness before the experiment began.

3. Experimental Stimuli

Again, for this research, I was only concerned with the ocular motor system’s response to transient, horizontal stimuli. Thus, each of the paradigms used in the experiment involved having the target and/or head begin from rest and move horizontally for a maximum of 4 sec. The experiments consisted of various trials using the stimuli described in Table II-2. Because I was interested in the VOR, the smooth pursuit system, and the possible interaction between them, I mainly used stimulus speeds of 15 deg/sec to insure that we were working within the normal operating ranges of these systems. To eliminate the possible effects of prediction, I randomized the order of the stimulus types, their direction (leftward or rightward), the time that stimulus motion began, and I even included trials with stimulus speeds of 30 deg/sec. Also, the experimental apparatus did not provide any extraneous auditory cues which might help the subject predict when a trial was about to commence. When asked after an experiment, none of the subjects expressed the ability to predict the nature of an upcoming stimulus.
Although most of the different stimulus types described in Table II-2 were used in the experiments, only the Smooth Pursuit Offset (Type 2, measuring both smooth pursuit onset and offset activity), the VOR Offset (Type 4, measuring both VOR onset and offset activity), the Chair Brake (Type 7), and the Delayed Target Onset (Type 10) were of principal concern for the subsequent analysis. The remaining trials were either used to provide data for additional analysis at a later time or to act as stimulus masks to reduce the possibility of prediction by increasing the variety of stimuli experienced by the user.

a. The VOR Stimulus

To collect data representing both the onset and offset phases of the VOR, I used the VOR Offset stimulus (stimulus Type 4; see Fig. II-3). For this, the target was held stationary at 0 deg and the vestibular chair was rotated to either −30 deg (for a rightward response) or +30 deg (for a leftward response). After the subject was given a few seconds to fixate the centrally-located target and dissipate any residual VOR signals resulting from motion to the starting position, the vestibular chair rotated toward the center of the operating range at 15 deg/sec for 2 sec until it stopped at 0 deg. Target, gaze, and head position data were collected from 100 msec before chair motion until 1 sec after the chair stopped rotating. This captured the VOR responses to both the onset and offset of head motion.

Notice that this was not technically a “pure” VOR stimulus as it was
Fig. II-3. Diagrammatic representation of the VOR Offset paradigm used for collecting VOR responses to both the onset and offset of head motion. \( H \), head position; \( \dot{H} \), head velocity.
not performed in the dark. Rather, it was visually-enhanced VOR, involving the feedback of visual information to maintain fixation of a stationary, earth-fixed target. I employed this type of stimulus because the CEHT paradigms I used to test my hypothesis were actually composed of combinations of the more basic VOR and smooth pursuit paradigms. Thus, it was important for me to use visually-enhanced VOR as a control condition for the corresponding CEHT paradigms involving both head and target motion. However, despite the use of visual feedback, remember that the VOR system still operates effectively open-loop for at least the first 75 msec of a response (and, more reasonably, the first 130 msec) because it takes that long for a visual input to affect the response due to processing delays (Carl et al. 1987). Also, under natural circumstances, the visual and vestibular systems work together to hold gaze steady.

Qualitative comparison of responses from this type of VOR stimulus (Type 4) with responses collected using the VOR onset paradigm (stimulus Type 3), in which the chair began in the center of the operating range and moved eccentrically, showed no difference in terms of either the stimulus or the response characteristics. Thus, I chose to analyze VOR data using Type 4 stimuli because I could obtain both onset and offset response data from the same trial. Although not anticipated at the beginning of the experiments, I later found that it was essential to have responses to both VOR onset and offset from the same trial when I tried to model the VOR (see Section II.C.1.c.iii).
b. The Smooth Pursuit Stimulus

To collect data representing both the onset and offset phases of smooth pursuit, I used the smooth pursuit offset stimulus (stimulus Type 2; see Fig. II-4). For this, the vestibular chair was held stationary at 0 deg (the center of the experimental range) and the target was positioned at either −30 deg (for a rightward response) or +30 deg (for a leftward response). After the subject was given a few seconds to fixate the eccentric target, the target moved toward the center of the screen at 15 deg/sec for 2 sec; the target then stopped at 0 deg, directly in front of the subject. Target, gaze, and head position data were collected from 150 msec before target motion began until 1 sec after the target stopped. This captured both the pursuit response to the onset of target motion as well as the response to having the target stop (offset of target motion).

Qualitative comparison of responses from this type of stimulus (Type 2) with responses collected using the smooth pursuit onset paradigm (stimulus Type 1), in which the target began in the center of the screen (directly in front of the subject) and moved eccentrically, showed no difference, except for occasional low-level gaze evoked drift. Thus, I chose to analyze pursuit data using Type 2 stimuli because I could obtain both onset and offset response data from the same trial.
Fig. II-4. Diagrammatic representation of the Smooth Pursuit Offset paradigm used for collecting smooth pursuit responses to both the onset and offset of target motion. $T$, target position; $\dot{T}$, target velocity.
c. The Chair Brake Stimulus

To investigate the possible interaction of the VOR and smooth pursuit systems during CEHT, I selected stimuli that would take advantage of the dramatic differences in the latencies of smooth pursuit and the VOR. One paradigm that proved to be useful was the Chair Brake paradigm (Table II-2, stimulus Type 7). Lanman et al. (1978) originally employed this type of stimulus to study the coordination of eye and head movements in monkeys while they tracked moving targets. Briefly, my implementation of the Chair Brake paradigm involves rotating the chair and revolving the target in synchrony until, at some point, the chair is stopped, or braked, and the target continues moving alone. For this, at the point when the chair (head) is stopped and the target continues to move alone, the sole input to the ocular motor system is the moving target. If the smooth pursuit system is not active during CEHT, then one would expect to see an eye movement response with a morphology similar to that seen at the onset of smooth pursuit (i.e., a latency of about 130 msec, with possible ringing in the response). On the other hand, if the smooth pursuit system is active during CEHT, then it does not need to get "charged-up" in response to the moving target, and the eye will continue to track the target with a transient morphology resembling that of VOR offset.

To perform the Chair Brake paradigm (Fig. II-5), the chair and target were both positioned at either \(-30\) deg (for a rightward response) or \(+30\) deg (for a leftward response). After the subject was given a few seconds to
Chair Brake Paradigm

Fig. II-5. Diagrammatic representation of the Chair Brake paradigm used for determining the relative activity of the smooth pursuit system during CEHT. After CEHT is initiated, the chair is abruptly stopped or braked while the target continues moving. Symbols as in Figs. II-3 and II-4.
d. The Delayed Target Onset Stimulus

Another paradigm I found useful for investigating the possible interaction of the VOR and smooth pursuit systems during CEHT was the Delayed Target Onset paradigm (Table II-2, stimulus Type 10). This involved first rotating the chair alone, as with a visually-enhanced VOR stimulus. When the chair eventually aligned with the stationary target, the target began to move in synchrony with the chair to evoke CEHT. For this, the onset of target motion occurs 2 sec after the onset of chair motion, and the behavioral paradigm switches from being visually-enhanced VOR to CEHT. Thus, if smooth pursuit is involved in cancelling the VOR during CEHT, the morphology of the eye signal during cancellation will resemble the onset of smooth pursuit (i.e., a latency of about 130 msec, with possible ringing in the response). Otherwise, if smooth pursuit is not involved in cancelling the VOR during CEHT, the morphology of the
eye signal during cancellation will not likely resemble that of smooth pursuit onset.

To perform the Delayed Target Onset paradigm (Fig. II-6), the chair was positioned at either -30 deg (for a rightward response) or +30 deg (for a leftward response) and the target was located in the center of the screen (at 0 deg). After the subject was given a few seconds to dissipate any residual VOR signals resulting from motion to the starting location and to fixate the centrally located target, the vestibular chair was rotated toward the target at 15 deg/sec. When the chair aligned with the target (after 2 sec), the target began to move in synchrony with the chair for an additional 2 sec. Target, gaze, and head position data were collected from the beginning of stimuli motion for 4 sec (until the chair had moved through 60 deg). This captured the eye movement response as it changed from visually-enhanced VOR to CEHT.

B. Data Processing Methods

After an experiment, the target, head, and gaze position data for each trial must be processed prior to subsequent analysis (parameter estimation and hypothesis testing). Before the processing, each trial was screened for suitability. Any response containing excessive noise or eye movement artifacts (e.g., blinks), so that the waveform was clearly not similar to others of the same kind, was excluded from further
Fig. II-6. Diagrammatic representation of the Delayed Target Onset paradigm used to investigate how the VOR is cancelled upon the initiation of CEHT. After a period of VOR stimulation, CEHT is initiated when target motion commences. Symbols as in Fig. II-5.
consideration. Also, I scrutinized responses for the possible effects of anticipation (Kowler et al. 1984); trials exhibiting unrealistically short pursuit latencies were discarded.

The processing performed on the measured data involved the following seven procedures: (1) artifact correction, to adjust for nonlinearities in the magnetic field; (2) geometric correction, to adjust for the eccentric position of the eye relative to the center of the head, and to allow presentation of the visual target on a flat display screen; (3) removal of saccades from gaze position waveforms; (4) digital filtering, to remove unwanted noise; (5) digital differentiation, to obtain velocity waveforms; (6) removal of saccades from gaze velocity waveforms, if necessary; and (7) cleaning of the target velocity waveforms. To expedite analysis, I integrated all seven procedures into a unified data processing program using the ASYST™ software environment (Hary et al. 1987) on an AST Premium 286 computer. These processing steps are described next.

1. **Coil Artifact Identification and Correction**

Due to inhomogeneities in the magnetic field, the measured search coil voltages do not exactly correspond to the coil’s angular displacement within the field. Instead, a constant coil rotation will yield a linearly changing voltage with small variations due to the field nonlinearities. This variation in measured position is small (usually less than 1 degree peak-to-peak through ±35 degrees, and cycling about four times over a 360
degree rotation), and its characteristics change insignificantly over several hours.

Although this artifact due to field nonlinearities is small, we decided that it was significant enough to deal with because small position differences may manifest themselves as large velocity differences upon differentiation. So, to reduce systematic errors as much as possible, I developed a procedure to effectively remove the field inhomogeneity artifact by creating a look-up table for each measured coil signal that associates a set of measured, artifact-influenced coil positions with a corresponding set of true coil positions.

The precise angular position information needed to create the artifact correction look-up tables was provided by the vestibular chair, whose built-in optical angular-position encoder was used as a position standard. The chair position signal was quantized using 12 bits—4096 levels—over 360 degrees, giving a chair position resolution of 0.088 deg. After the usual calibration of the head and eye coils (at ±25 degrees), the search coils were fixed at zero-degrees with respect to the chair. Next, the chair was programmed to cycle slowly back and forth over the experimental operating range (±35 degrees; see Section II.A.1.a.iv) at approximately 1.5 deg/sec. Meanwhile, for each known chair position (defined using each quantization level of the chair’s optical encoder), the measured position values from the head and eye coils were saved to arrays. After seven to ten sweeps, the measured position values were averaged for each coil, and the resulting arrays provided a mapping from
true angular position to measured, artifact-altered position.

As the desired look-up tables require a mapping from measured, artifact-altered position to true angular position, I needed to invert the above mapping. To do this, I treated the measured position arrays as abscissae. This was complicated somewhat due to the higher resolution of the search coils: the coil position signals were quantized using 16 bits—65536 levels—over ±50 degrees, yielding a coil position resolution of 0.0015 deg. Because this resolution is higher than that of the known chair position values (whose resolution is 0.088 deg), there were many coil positions that could be associated with each chair position. To rectify this disparity, linear interpolation and scaling techniques were employed to effectively increase the number of chair positions and provide a one-to-one mapping from measured coil position to true (chair) position. After inversion, the resulting look-up table arrays for the head and eye coils map each possible measured coil position to the one expected coil position that would be obtained directly if the magnetic field were homogeneous and linear.

To test the procedure, I created artifact-correction look-up tables as described above. I then collected separate arrays of artifact-altered eye and head coil data by slowly sweeping the chair-fixed coils through the same ±35 deg range used to make the correction look-up tables. By subtracting the expected coil positions from the measured coil positions, I obtained profiles of the inhomogeneity artifact; these often showed an almost sinusoidal variation of about 1 deg peak-to-peak over the measured ±35
deg range. After remapping the measured values using the look-up tables, this sinusoidal variation virtually disappeared (within quantization noise levels), indicating that the procedure was effective in removing the artifact due to the inhomogeneities in our search coil system.

This procedure to remove artifacts due to the field inhomogeneities was routinely performed on all gaze and head data analyzed for this research.

2. Geometric Correction

a. Target Signal Modification

Due to the nature of the target display apparatus, it was necessary to modify the command signal governing target position (as described in Section II.A.1.b.iv). This involved causing the target to move across the flat display screen in a way that made it appear to revolve about the subject at a constant angular rate. During an experiment, the only sources of a measurable target signal are either the target command signal direct from the computer or the signal fed back from the target’s mirror galvanometer; both of these contained the position modification. Because I wished to actually sample the target signal simultaneously with the head and eye signals to preserve their time relationship, I needed to re-create the true target positions (as observed by the subject) from the measured target position data. This simply involved applying an inverted form of the
original modification equation to each data element of the measured target array:

\[ T = \tan^{-1} \left( \frac{A \tan T'}{D} \right) \]  \( \text{II.3} \)

where \( T \) is the array of desired target positions as seen by the observer, \( T' \) is the array of measured target positions, \( D \) is the minimum distance from the subject to the display screen, and \( A \) is the effective minimum distance from the mirror galvanometer to the display screen. This inverse modification caused some slight ringing at various places in otherwise flat target velocity waveforms due to quantization and round-off effects. (These artifactual fluctuations are later edited interactively from the target signals, after filtering and differentiation.)

b. Eye Eccentricity Correction

When the search coil is positioned on a subject’s eye, it is naturally displaced somewhat from the center of rotation of the head due to the eye’s eccentricity. Although these distances may seem negligible when compared with the other metrics of the experimental apparatus, it may be desirable to modify the measured gaze position data to adjust for the different axes of rotation of the eyes and of the head. Using a simple geometric correction based on some easily-obtained measurements, it is
possible to *effectively* move the eye coil to the center of rotation of the head. By doing this, it is possible to directly compare data collected from different subjects or collected from the same subject measuring from different eyes. Also, gaze or eye position data collected during trials in which the head is stationary (*e.g.* smooth pursuit eye movements) may be compared directly with trials in which the head is moving (*e.g.* tracking movements or the vestibulo-ocular reflex) by simply adjusting all gaze or eye data as if they were collected from a common point, at the center of the head. Such comparisons are essential in interpreting the data for this research.

Although a number of authors have offered geometric equations to deal with certain aspects of this problem, no comprehensive solution has been reported. Thus, I derived a general geometric equation that can be incorporated into the data analysis program, and I developed a simplified but realistic method to measure values that will serve as parameters for this geometric correction for any subject.

The general approach is to transform signals recorded from one or both eye coils so that the standardized data are appropriate for the rotation of an eye lying at the center of rotation of the head. A number of previous attempts have been made to deal with this issue (*e.g.*, Blakemore and Donaghy 1980). Wist *et al.* (1983) suggest the adjustments that are necessary while maintaining fixation on a stationary target during head movements. This method assumes a cyclopean eye and derives its head geometry metrics from a simple calculation of head circumference. Another method demonstrates how the VOR gain is different for both
eyes when viewing a common target nearer than optical infinity (Viirre et al. 1986). Although this approach departs from the assumption of a cyclopean eye, it requires a circumferential display screen situated at a uniform distance from the subject, and it relates eye position with head position using a specific target position on the display screen. Because their equations assume that the line-of-sight is always directed towards a visible target, their method is correct only at times when the target lies along the line-of-sight. At other times, such as during the first 130 msec of a smooth pursuit response when the eye typically has not yet begun to follow the moving target, the method is not valid. Therefore, this approach is most appropriate for modifying eye signals measured under stationary target conditions, such as during testing of the VOR; it is not well-suited for modifying eye movement data collected using paradigms involving target motion (e.g., during smooth pursuit or combined eye-head tracking).

Collewijn et al. (1982) develop a linear approximation to the transformation assuming a cyclopean eye and the use of a circumferential display screen. This method does not assume foveation of a specific target; it depends solely on the distance from the center of head rotation to the eye, the distance from the center of rotation to the display screen (both constants), and the measured eye position signal. Unfortunately, this method is not useful for measurements made using a flat display screen, and it does not take into account the separation of the eyes (the interocular distance). Thus, I developed a mathematical expression for modifying gaze (or eye) position measurements that (1) may be used with
either a flat target display screen or a circumferential display screen situated at a uniform distance from the subject, (2) accounts for the different axes of rotation of the head and eyes as well as the separation of the eyes, (3) assumes nothing about the expected position of a target on the display screen nor about the capacity of a subject to foveate such a target, (4) may be used with any combination of horizontal eye and head movements, and (5) is not merely a simplified approximation. The only major assumptions made by this procedure are that the axis of head rotation passes through the center of the head and that the line-of-sight after modification should intersect the display screen in the same place as the original, unmodified line-of-sight. This does not assume a target is positioned at the point where the two lines-of-sight intersect the screen; however, if such a target on the screen were being foveated, the modification to the gaze or eye signals should maintain the foveation. This provides my approach with the capacity to deal correctly with circumstances in which the eye does not rotate in synchrony with a moving target (as occurs, for example, at the onset of smooth pursuit).

Figure II-7 diagrammatically illustrates how the measured angle of gaze \(G\) depends on the eccentric location of the eye coil with respect to the center of rotation of the head. Notice, for a given screen intersection point (the place where the line-of-sight intersects the display screen), how angular head position \(H\) and head geometry affect the location of the eye in space and thus, the measured angle of gaze. Also note that by effectively moving the eye to the head's center of rotation it is possible to eliminate these effects by creating a standardized angle of gaze \(G'\), thereby
Fig. II-7. Geometric relationships used to derive an expression for the eye eccentricity compensation term $\beta$. See text for details.
allowing responses obtained under different conditions to be directly compared. (Dimensions in Figs. II-7 through II-9 have been distorted for visual clarity.) Because the gaze and eye signals are directly related by

\[ E = G - H \]  

the eye signal \( E \) can similarly have the eye eccentricity effects removed \( E' \).

To calculate the standardized angle of gaze \( G' \), we see from Fig. II-7 that

\[ G = E + H = 90^\circ - \alpha \]  

\[ \text{II.5a} \]

and

\[ G' = E' + H = 90^\circ - (\alpha + \beta) \]
\[ = (90^\circ - \alpha) - \beta \]  

\[ \text{II.5b} \]

(Note that these relationships are generally true as long as the line-of-sight passes through the screen intersection point. If a circumferential display screen is used, the line labeled "Display Screen" in Fig. II-7 may be interpreted as a construction line.) Substituting Eqn. (II.5a) into Eqn. (II.5b), we see that the standardized angle of gaze \( G' \) may be obtained from the measured angle of gaze \( G \) by subtracting from \( G \) the angle \( \beta \). That is,
\[ G' = G - \beta \quad \text{II.6a} \]

If eye position \( E \) is measured instead, the transformation may be expressed as

\[ E' = E - \beta \quad \text{II.6b} \]

where \( E' \) is the standardized angle of eye position. As we shall see, \( \beta \) can be derived from the constant values \( D_1, m, \) and \( \gamma \), and from the measured quantities \( H \) and \( E \). The value \( D_1 \) is the minimum distance from the display screen to the center of head rotation. The values \( m \) and \( \gamma \) are specific head geometry parameters that are measured separately for each subject: \( m \) is measured directly as one-half the inter-ocular distance, and \( \gamma \) is the angle between the naso-occipital axis and the line extending from the center of the head to the eye. (Note that, by convention, the values for \( m \) and \( \gamma \) are positive for measurements from the right eye and negative from the left eye.)

One way to accurately measure \( \gamma \) is to place the subject in a vestibular chair capable of providing accurate angular position measurements and restrain the head in the center of rotation of the chair, thereby allowing head position to be expressed by chair position. (For example, if the chair is equipped with an optical angular-position encoder having 12-bit resolution, it is possible to resolve head rotations of 0.088
deg.) Next, suspend two plumb lines along the line $D_1$, one adjacent to the subject's head and the other adjacent to the display screen (Fig. II-8). Allow the subject to view from one eye and self-rotate in the chair until the two plumb lines superimpose; when this happens, the line-of-sight of the viewing eye corresponds to the line extending from the center of head rotation and passing through the eye. Then measure the angle of chair rotation; this angle is an approximation to $\gamma$, and its accuracy depends on the ability to position the chair at zero-degrees (along the line $D_1$) before the self-rotation. A value for $\gamma$ can be determined that eliminates such potential offset errors by first obtaining $\gamma$ for the right eye, then for the left eye, and finally averaging the two values. Notice that this will also correct for any offset between the head and chair positions.

After determination of $m$ and $\gamma$, a value for $n$, the distance from the center of rotation to the eye, can be obtained from

$$n = \frac{m}{\sin \gamma} \quad \text{(II.7)}$$

(Because it is difficult to directly measure a value for $n$, other authors have performed their calculations using a value of $n$ that is simply derived from a measurement of head circumference. This is less reliable than calculating $n$ using the directly-measurable parameters $m$ and $\gamma$ with Eqn. II.7.)

To determine an expression for $\beta$, we use the law of sines
Fig. II-8. Procedure to determine a value for $\gamma$, the angle between the naso-occipital axis and the line extending from the center of head rotation to the eye. See text for details.
\[
\frac{\sin \beta}{n} = \frac{\sin \sigma}{D_2}
\]

and solve for \( \beta \)

\[
\beta = \sin^{-1}\left( \frac{n}{D_2} \sin \sigma \right)
\]

Although \( n \) has already been calculated, we still need to determine values for \( \sigma \) (or, \( \sin \sigma \)) and \( D_2 \). From Fig. II-7 we see that

\[
\sigma = 180^\circ - (E - \gamma)
\]

but using the identity

\[
\sin (180^\circ - \theta) = \sin \theta
\]

we obtain an expression for \( \sin \sigma \)

\[
\sin \sigma = \sin (E - \gamma)
\]

The value for \( \gamma \) was previously determined and \( E \) is the measured eye signal. (If the gaze signal \( G \) is measured instead, \( E \) can be obtained using \( G \) and the head signal \( H \) with Eqn. II.4). Thus, substituting Eqn. II.12 into Eqn. II.9, the expression for \( \beta \) becomes
\[ \beta = \sin^{-1} \left( \frac{n}{D_2} \sin (E - \gamma) \right) \]  

II.13

If a circumferential display screen is used, the length \( D_2 \) equals the known length \( D_1 \), and all the values needed to calculate \( \beta \) are determined. In fact, if Collewijn et al. (1982) had not assumed a cyclopean eye and had continued their calculations without making the linear approximation, they could have reached an equivalent result.

If the display screen is flat, the expression for \( \beta \) becomes somewhat more complicated. Using the geometric relationships depicted in Fig. II-9, we will determine an expression for \( D_2 \) using the Pythagorean theorem. One leg of the right triangle is simply \( D_1 \). One component of the other leg can be obtained knowing

\[ \tan (E + H) = \frac{k}{D_1 - n \cos (H + \gamma)} \]  

II.14

where the denominator is derived using trigonometric relationships of the head geometry. From this, an expression for \( k \) is

\[ k = \tan (E + H) [D_1 - n \cos (H + \gamma)] \]  

II.15

Using similar trigonometric relationships to those given in the
Fig. II-9. Angles and distances used to calculate $D_2$, the distance from the center of head rotation to the display screen along the modified line-of-sight.
denominator of Eqn. II.14, the second leg can be expressed as

\[ n \sin(H + \gamma) + k \]  

Thus, \( D_2 \) is given by

\[
D_2 = \sqrt{D_1^2 + \left( n \sin(H + \gamma) + \tan(E + H)[D_1 - n \cos(H + \gamma)] \right)^2}
\]

and the resulting expression for \( \beta \) in which a flat display screen is used is

\[
\beta = \sin^{-1}\left[ \frac{n \sin(E - \gamma)}{\sqrt{D_1^2 + \left( n \sin(H + \gamma) + \tan(E + H)[D_1 - n \cos(H + \gamma)] \right)^2}} \right]
\]

Notice that \( \beta \) is not constant but rather varies with changes in the values of \( E \) and \( H \) during a response. Thus, use of this modification method is somewhat calculation intensive, although not prohibitive (particularly if array processing capabilities, such as those provided by the ASYST™ software package (Hary et al. 1987), are available).

In the data processing procedure for this research, the modifications described in this section were routinely performed before any subsequent analysis.

This research is mainly concerned with investigating the VOR, smooth pursuit, and the possible interaction between them. Although the semi-circular canals in the inner ear sense rotational head accelerations, we shall later see how, for the frequencies of rotations corresponding to natural head movements, the vestibular nerve encodes head velocity (Fernandez and Goldberg 1971; Melvill Jones and Milsum 1971). Similarly, a popular model of the smooth pursuit system suggests that it too responds mainly to velocity signals (Robinson et al. 1986).

The saccadic system is yet another eye-movement system; it is responsible for correcting for position errors by generating high velocity eye movements to allow rapid foveation of targets. Although the saccadic system does contribute to the general tracking mechanisms (see Bloomberg 1989), because I am mainly interested in analyzing the eye velocity signals generated by the ocular motor system in response to smoothly-moving stimuli, I have chosen to simplify my analysis by disregarding the effects the saccadic system has on smooth tracking. Because saccades are generated from sources in the brain independently of smooth pursuit and the VOR, it is not uncommon for ocular motor researchers to treat saccades as "noise" and remove them from eye and gaze waveforms (e.g., Newsome et al. 1985). Thus, one stage of the data processing was the removal of saccades from the measured gaze signals.

When during the data processing procedure is the best time to
remove saccades and how is the best way to do it? Many researchers obtain eye velocity waveforms by differentiating the raw eye position signal on-line using analog methods and then directly sample the eye data in velocity. With this approach, typically the only option for saccade removal available is to specify the endpoints of each saccade spike and either linearly interpolate values between the endpoints to maintain a continuous waveform or remove these saccade points altogether leaving a gap in the response data.

Another approach is to digitize eye position signals and then digitally differentiate to obtain velocity waveforms; this method provides the option of removing saccades in either position or velocity. The easiest and most direct method is to remove the saccades in velocity, as described above. The advantage of performing saccade removal with velocity waveforms is that the magnitudes of the eye velocities generated during a saccade are generally much greater than the simultaneous velocity contributions from either the VOR or smooth pursuit systems. Thus, the ability to detect saccade occurrences and to specify accurately the times at which they begin and end is greatly enhanced if the eye or gaze data are represented in terms of velocity. However, to prepare an eye movement waveform for saccade removal in velocity, preliminary digital filtering must be performed to remove powerline noise and other contaminants that are accentuated upon subsequent differentiation, which is used to convert the position waveforms to velocity waveforms. (Details of these procedures will be discussed later.) Due to theoretical limitations in the capacity of digital processing techniques to provide ideal filtering or differentiation
characteristics, the tradeoff for having good saccade detection capability is the alteration of the saccade morphology within the velocity data due to the digital processing. This distortion is manifest as a broadening of the saccade peak, with significant rippling of the waveform either after the saccade, or both before and after the saccade, depending on the nature of the digital filter. These effects can stretch a processed saccade as much as two to three times its unprocessed width. Thus, the subsequent saccade removal process will leave far less of the original waveform intact than if the processing distortions had not occurred. Despite this major disadvantage, many researchers still choose to digitally remove saccades from their eye velocity data.

In an attempt to avoid the problems filtering and differentiation can pose to saccade removal, I initially tried to remove saccades from the original position waveforms, before filtering and differentiation were performed. How can saccades be removed from position data? Remember that a saccade provides a rapid redirection of eye position. This is manifest as a (band-limited) discontinuity in an otherwise smooth eye position waveform. Thus, removing a saccade from an eye position record involves removing the position discontinuity. Assume for a moment that the saccadic system (including the ocular motor plant) is not band-limited but rather, is capable of generating eye movements which instantly foveate an eccentric target. Removal of such a saccade would simply require that the entire response after the saccade be offset by an amount equal to the magnitude of the saccade. This would bring the data points on either side of the saccade next to each other, thereby eliminating the
position discontinuity.

This was the general approach I used to remove position saccades. However, because saccades are band-limited and require time for the fovea to capture an eccentric target (on the order of 30 msec), the method just described becomes somewhat more complicated. In addition to providing an appropriate offset to the post-saccade waveform, the \textit{saccade interval} (i.e., the part of a response waveform during which a saccade occurs) must be filled with suitable values to properly join the pre- and post-saccade response segments. I chose to fill the saccade interval with a series of small sub-segments having slopes intermediate between those calculated for the response just before and just after the saccade interval. The goal was to provide an approximate continuity in the velocity signal by using piecewise linear segments within the position waveform's saccade interval. Then, the post-saccade response was offset, as described above, to maintain continuity of the position waveform.

Although this method did succeed in reducing much of the distortion and ringing described above, it essentially replaced one large discontinuity with a series of smaller discontinuities which, after differentiation, still contributed a smaller but perceptible distortion to the resulting waveform. Also, the characteristics of this distortion were less regular than those occurring with velocity saccade removal and thus, it was more difficult to determine which waveform variations resulted from actual eye movements and which arose due to desaccading artifacts. In addition, although it is fairly easy to choose the beginning and end points
of a saccade when it is displayed in velocity, accurately determining these limits when the saccade is displayed in position is rather difficult. Similarly, signal noise made it difficult to determine accurate values for the slopes on either side of the saccade, thereby allowing large potential variation in the way the values within the saccade interval are calculated. Thus, saccade removal using position information alone was only slightly more satisfactory than removing saccades from velocity waveforms.

To take advantage of the favorable characteristics of saccade removal in position, I developed an integrated technique for removing saccades from gaze position waveforms using gaze velocity data. Utilizing the flexibility of the ASYST™ programming environment (Hary et al. 1987), I designed a routine that displayed a gaze velocity waveform (unfiltered) while simultaneously allowing me to operate back on the corresponding gaze position data. (Although the unfiltered gaze velocity data were noisy, saccades were still readily apparent.) In this way, I interactively chose the beginning and ending times of a saccade while viewing the gaze velocity data ($t_1$ and $t_2$), and obtained the corresponding gaze velocity values at these times ($\dot{E}(t_1)$ and $\dot{E}(t_2)$). Assuming that the gaze velocity data underlying the saccade could be adequately reconstructed using a linear interpolation between the endpoints, the velocity saccade segment for these data could be defined using

$$\dot{E}(t) = mt + b$$  \hspace{1cm} II.19
where \( m \) is the slope of the interpolation segment, calculated from

\[
m = \frac{\dot{E}(t_2) - \dot{E}(t_1)}{t_2 - t_1}
\]

and \( b \) is the \( y \)-intercept, given by

\[
b = \dot{E}(t_1) - mt_1
\]

The goal is to remove the saccade from the gaze position plot. Thus, given the characteristics of the required gaze velocity interpolation segment, we must obtain an expression from which the position values within the saccade interval may be calculated. This is done simply by integration:

\[
E(t) = \int \dot{E}(t) \, dt + c = \int (mt + b) \, dt + c
\]

where \( c \) is a constant whose value is yet to be determined. Performing the integration yields

\[
E(t) = \frac{mt^2}{2} + bt + c
\]
Because both the position and velocity waveforms are available simultaneously, we can determine a value for $c$ using the first end point of the position waveform's saccade interval, $E(t_1)$, from

$$c = E(t_1) - \frac{m}{2}t_1^2 - bt_1$$

Thus, the values of the position waveform within the saccade interval $t_1$ to $t_2$ may be calculated using Eqn. II.23 equipped with the calculated values for $m$, $b$, and $c$ (Eqns. II.20, II.21, and II.24). This way, the saccade within the position waveform is replaced by a parabola whose characteristics are based on the linear saccade segment specified within the corresponding velocity waveform. Notice that when differentiation is later performed, this same linear segment will be created as a replacement for the saccade in the velocity waveform.

After the equation for the parabola (Eqn. II.23) is used to calculate values within the saccade interval of the position waveform, there remains a discontinuity between the last element of the saccade interval (at $t_2$) and the first position array element after the saccade interval (at time $t_2 + \tau$, where $\tau$ is the time interval between adjacent samples, equal to the reciprocal of the sampling rate). To reconcile this difference, the position array values corresponding to times after $t_2$ must be offset by a constant value, as described above. To do this, Eqn. II.23 is evaluated at $t_2 + \tau$; from this the actual position array value $E(t_2 + \tau)$ is subtracted to yield a correction factor $\Delta E$. Then, the final adjustment requires that $\Delta E$ be added
to all array values corresponding to times after $t_2$. This results in a continuous position waveform with the saccade removed. For additional saccades, the overall procedure is simply repeated.

This method of saccade removal is particularly well suited for removing saccades in a relatively noise-free environment. However, for signals with considerable noise components (e.g., 60 Hz), the task of choosing appropriate end points for the saccade interval becomes more complicated. In fact, even carefully selected saccade intervals may result in unusual signal perturbations after filtering and differentiation. (Nonetheless, the procedure is still far superior to simple desaccading in velocity.)

Because I used an analog low-pass filter with a cutoff frequency of 100 Hz prior to sampling, the signals are susceptible to considerable 60 Hz powerline noise contamination. As the described method of saccade removal must be performed before any digital filtering, I devised a modification to the saccade removal procedure which improves its performance in the presence of noise dominated by one specific frequency. Replacing a saccade with a straight line segment within a sinusoidally varying signal may cause undesirable effects upon filtering, particularly at the transitions points. Also, if the line segment is displaced from the centerline around which the sinusoid oscillates, discontinuities are effectively introduced at the transition points as well. Thus, I reasoned that rather than filling the velocity saccade region with a simple line segment, if I instead use a line segment superimposed with a cosine of the
predominant noise frequency, I can make the saccade interval meld with the surrounding signal and thereby eliminate potential end-effect problems.

This modification is based on a few assumptions. First, the method assumes that if the predominant noise frequency were removed, a simple line segment would be an adequate replacement for the removed saccade. (This was the same assumption made above for the noise-free condition.) Next, by adding "noise" in the form of a cosine wave, we assume that subsequent filtering procedures can be used to remove any noise energy we are introducing to the signal at this stage. (The validity of this assumption will be discussed and verified in the following section on filtering.) Thus, this modification effectively adds noise (that will later be removed) to make the correction blend with the signal's inherent noise.

Why is a cosine used instead of a sine? The easiest way to replace an interval within a waveform containing a dominant noise component is to substitute an integer number of cycles of a (co-)sinusoid with the appropriate amplitude and frequency. Although any two points separated by an integer number of cycles could be used as the endpoints for the interval, I reasoned that peaks of the noise could be chosen with less ambiguity than any other pair of points. Thus, a cosine, rather than a sine, is the appropriate function to use because its cycles begin at peaks without the need to consider the complicating effects of phase.

To remove the saccade, we choose two noise peaks on either side of
the saccade and connect the peaks with a straight line. This line is then modulated by a cosine wave to make the replaced saccade interval blend with the surrounding signal. When the signal is later filtered, the noise components will be removed, giving the same results as for the noise-free saccade removal procedure above. Depending upon the duration of the saccade, several cycles of the cosine wave may be used.

To obtain a revised equation for calculating the position values within the saccade interval, we will combine the equation for a line with the equation for a cosine wave and integrate the result. As before, the equation for the line is

\[ \dot{E}_s(t) = mt + b \]  \hspace{1cm} \text{II.25} \]

where the subscript \( s \) denotes the contribution from a line segment and the constants \( m \) and \( b \) are calculated as above, using Eqns. II.20 and II.21. Superimposed with this is a cosine wave whose equation is

\[ \dot{E}_c(t) = A \cos(2\pi f t) - A \]  \hspace{1cm} \text{II.26} \]

where the subscript \( c \) denotes the cosine component and \( A \) and \( f \) are the amplitude and frequency of the cosine, respectively. The value for \( f \) should match the observed frequency of the predominant noise component; for my work \( f \) equaled 60 Hz. Also, \( A \) is a parameter that must be specified to match the observed amplitude of the noise in the
neighborhood of the saccade. I found that the value for $A$ sometimes varied as much as 5 deg/sec within different regions of the same waveform. Note in Eqn. II.26 that the amplitude $A$ is subtracted. This is to make the cosine range between 0 and $-2A$; when this cosine is added to the line segment connecting noise peaks (Eqn. II.25), the resulting waveform appropriately oscillates between and below these peaks and matches with the oscillating signal on either side.

Using Eqns. II.25 and II.26, the overall expression for defining the saccade interval in velocity is

$$\dot{E}(t) = mt + (b - A) + A \cos(2\pi ft)$$  \hspace{1cm} \text{(II.27)}$$

and integrating, the corresponding correction in position is given by

$$E(t) = \frac{mt^2}{2} + (b - A)t + \frac{A}{2\pi f} \sin(2\pi ft) + c$$  \hspace{1cm} \text{(II.28)}$$

A value for the constant $c$ is determined using the left endpoint of the saccade region, $E(t_1)$, as before:

$$c = E(t_1) - \frac{mt_1^2}{2} - (b - A)t_1 + \frac{A}{2\pi f} \sin(2\pi ft_1)$$  \hspace{1cm} \text{(II.29)}$$

Again, with values for $m$, $b$, and $c$ (calculated from Eqns. II.20, II.21, and
II.29), the values within the saccade interval may be calculated using Eqn. II.28. Finally, the remaining elements of $E(i)$ (i.e., those corresponding to times after $t_2$) are adjusted for the position discontinuity resulting from the recalculation of the saccade interval values. This uses the same procedure of determining a correction factor $AE$ and adding this correction to the remaining array values as described above.

Note that, unlike the earlier method, one cannot simply specify the onset and offset points of the saccade to define the saccade interval. For this procedure, the saccade interval must be widened to include the nearest neighboring noise peaks. Although this will make the saccade interval slightly wider than the saccade itself, the benefits of using this method will likely far outweigh the cost of widening the interval, particularly if the noise frequency $f$ is high enough to provide peaks close to the true saccade endpoints. Also, care must be taken to include close to an integer number of cycles of the cosine wave within the saccade interval. If not, unusual discontinuities may be introduced resulting in undesirable distortions. To add flexibility and perhaps help reduce the width of the saccade interval, I programmed this procedure to allow the saccade interval endpoints to be chosen as either cosine maxima or cosine minima.

To test the procedure, I developed a routine to generate a stylized smooth pursuit response complete with saccades. The design characteristics of the pursuit response were based on actual pursuit data (Robinson et al. 1986), and the location and width of the saccades can be
specified by the user (see Fig. II-10a). Desaccading the stylized response in velocity, after filtering and differentiation, required removing a segment almost three times the width of the original saccade due to ringing introduced when the saccadic discontinuity was filtered (Figs. II-10b and II-10c). On the other hand, simple desaccading in position caused a slight but quite noticeable distortion after filtering and differentiation, which lasted about twice the saccade width (Fig. II-10d). Using the special desaccading procedure for the noise-free condition (Eqn. II.23) recovered the pursuit response with no noticeable distortion due to the saccade, other than the saccade interval being replaced with a line segment (Fig. II-10e).

When a 60 Hz noise component with realistic amplitude was added to the stylized signal (Fig. II-11a), the basic desaccading procedure (Eqn. II.23) was tested first. It resulted in significant distortions of the pursuit response lasting about twice the width of the saccade interval (Fig. II-11c). These distortions arose from the inability to choose appropriate endpoints for the saccade line segment (Eqn. II.19) within the noise (Fig. II-11b), and the characteristics of the distortions changed significantly depending upon the chosen saccade endpoints (Figs. II-11c and II-11d). Next, the enhanced desaccading procedure designed to work with noise (Eqn. II.28) was used. Through careful choice of the noise peaks on either side of the saccade (Fig. II-12b), the procedure was able to completely remove the saccade (Fig. II-12c) with no noticeable distortion of the pursuit waveform upon subsequent filtering and differentiation (Fig. II-12d). This was tested several times by changing the position and width of the saccade, and the results were the same. Thus, as long as a velocity signal can have the
Fig II-10. Saccade removal in a noise-free condition. 

(a) Stylized smooth pursuit velocity waveform containing a saccade. 
(b) Filtered version of (a). Notice that the saccade has become about three times as wide as in the unfiltered waveform. 

Next page: (c) Saccade removed from the velocity waveform in (b). A substantial amount of data has been replaced by this procedure. 
(d) Saccade removed in position, before filtering and differentiation, using the simple desaccading procedure. A slight artifact is apparent within the saccade interval. 
(e) Saccade removed in position, before filtering and differentiation, using the special desaccading procedure for the noise-free condition. This procedure has recovered the pursuit response with no noticeable distortion due to the saccade.
Fig. II-10, continued.
Fig. II-11. Saccade removal with noisy data using the basic procedure. a) Stylized smooth pursuit velocity waveform containing a saccade and sinusoidal noise with a realistic amplitude. b) Selection of endpoints for the saccade interval. It is difficult to match the slope of the underlying data by arbitrarily choosing such pairs of points. (Although the limits of the saccade interval are chosen while observing velocity waveforms, saccade removal is actually performed in position using this procedure.) Next page: c) and d) Results of applying the basic procedure for removing saccades from position waveforms when the data are contaminated with noise. The differences in the nature of the artifacts in the two plots stem from the different choices of saccade interval endpoints.
Fig. II-11, continued.
Fig. II-12. Saccade removal with noisy data using the enhanced procedure.  

a) Stylized smooth pursuit velocity waveform containing a saccade and sinusoidal noise with a realistic amplitude.  
b) Selection of saccade interval endpoints. Noise peaks on either side of the saccade are chosen. (Note that the peak of the saccade has been removed.)  

Next page:

c) Section of the velocity waveform, corresponding to the position waveform whose saccade was removed using the enhanced desaccading procedure, prior to filtering.  
d) Velocity waveform following enhanced desaccading, filtering, and differentiation. Notice that the procedure was able to completely remove the saccade with no noticeable distortion of the pursuit waveform.
Fig. II-12, continued.
saccade interval replaced with a line segment, and if the signal is
contaminated by noise with a predominant component at one specific
frequency (e.g., 60 Hz), then this procedure may be quite useful for
removing saccades. Although the procedure is a bit more involved than
simple desaccading in velocity, the processed data have greater integrity,
particularly due to the more limited range of the distortions originating
from the desaccading procedure.

4. Digital Filtering

In my research, I am interested in understanding the possible
interaction between the VOR and the smooth pursuit eye movement
systems. To test these systems I subjected them to steps of either head or
target velocity. Such velocity steps contain relatively high frequency
components, so an accurate assessment of the responses to these stimuli
requires that the responses maintain all of their frequency components.
Thus, the analog filters that I used to eliminate aliasing upon digitization
were set to have a wide bandwidth (0-100 Hz) relative to the expected
frequency content of the measured eye movements (the major energy of
which lies below 20 Hz). In this way, I could insure that any observed
effects such as reduced gain or reduced response acceleration were real and
not due to the confounding effects of having too narrow a filtering
bandwidth.

The major disadvantage of maintaining a high bandwidth for the
signal is the possible contamination of the signal by additional, higher frequency noise components. Because I used an analog upper cutoff frequency of 100 Hz, the 60 Hz component of powerline noise was passed essentially unattenuated, and the 120 Hz component was only slightly attenuated. In addition, a significant (perhaps aliased) harmonic of the field coil generator was present at approximately 45.85 Hz. If allowed to remain in the signal, these specific noise components would be so greatly magnified upon differentiation that the resulting velocity waveforms would be rendered virtually useless. Thus, I was faced with the dilemma of requiring a wide bandwidth to maintain the signal while needing high attenuation at 45.85, 60, and 120 Hz to remove the major noise components.

At first, I investigated using simple finite impulse response (FIR) filters (either Hamming or Blackman-Harris) to help remove noise (Oppenheim and Schafer 1975). However, despite the excellent phase characteristics of these filters, the fact that they cause oscillations in the filtered waveform both after and before a major transition of a response caused me to reject their use. It was important for me to be able to detect accurately the onset of a response (e.g., in determining the latency of smooth pursuit), and this would be obscured if the data showed ringing before the response began. For this reason, I opted to consider instead using infinite impulse response (IIR) filters.

Due to the causality of IIR filters, no filter-generated activity is seen before a transition. However, with the more peculiar phase characteristics
of these filters, it is important that all signals be treated with the same filter (or set of filters), and that all response data have generally the same frequency spectra. This way, any phase shifts will be seen by all the recorded waveforms in the same way, and events within the waveforms will remain in synchrony with each other.

By using a simple low-pass IIR filter (such as a Butterworth), I could attenuate the unwanted noise components at the expense of possibly losing some high frequency signal components. If I instead implemented a notch (band-stop) IIR filter, I could eliminate the noise at a specific frequency but at the same time induce undesirable ringing in my data, as often occurs with notch filters. I finally decided to use a Chebychev type II low-pass filter. As we have shown elsewhere (Huebner et al. 1988; Thomas et al. 1988), these low-pass filters can be designed so that one of the filter’s natural frequency nulls occurs at a coherent noise frequency thereby providing theoretically infinite attenuation at that frequency. If a low-order filter is used, the overall frequency attenuation in the filter’s stopband is still quite small despite the large attenuation at the frequency null. Figure II-13 shows an example of the frequency characteristics for such a filter. Thus, using special filter design techniques, I was able to use low-pass filters to provide notch filter rejection of unwanted noise components while maintaining a relatively wide bandwidth. An example of the noise rejection capabilities of this type of filter was shown above, where a Chebychev type II low-pass filter (with a null at 60 Hz) was used to filter a stylized, desaccaded waveform (see Figs. II-12c and II-12d for pre- and post-filtering waveforms, respectively).
Fig. II-13. Bode plots of two implemented Chebychev Type II low-pass filters. 

a) second-order filter with a cutoff frequency of 40 Hz and a null at 60 Hz. 

b) a fourth-order filter with a cutoff frequency of 80 Hz and a null at 120 Hz.
For this research, I used Chebychev type II filters with the following characteristics: the 45.85 Hz noise component was removed with a second-order filter whose cutoff frequency (~3 dB point) was at 30 Hz; a second-order filter with a 40 Hz cutoff frequency was used to remove the 60 Hz noise component (as well as any "noise" added by the desaccading procedure); and the 120 Hz noise component was removed using a fourth-order filter with a cutoff frequency of 80 Hz. The first two filters are second-order to provide minimal attenuation beyond the specified noise rejection while the last filter was fourth-order to provide some degree of high frequency noise attenuation above 80 Hz. Thus, by successively applying these three IIR filters, I was able to remove most of the unwanted noise without sacrificing a wide signal bandwidth.

5. Digital Differentiation

Although there are several elaborate and sophisticated algorithms available for digital differentiation, I chose to use probably the most rudimentary of these, the two-point central difference algorithm (Bahill and McDonald 1983). For all pairs of data array elements separated by \( n \) values (where \( n \) is a user-defined parameter), this algorithm simply calculates the slope of the line between the elements, relative to the scale of their independent variable.

In essence, the two-point central difference algorithm is an ideal
differentiator in series with a low-pass filter. The low-pass filter characteristic is governed by the value of \( n \). If \( n \) is large, the slopes are calculated using widely separated data points, and local, high-frequency differences will tend to be lost. On the other hand, if \( n \) is small, local high-frequency perturbations will be maintained and manifested in the slope calculations. Thus, to decide if this algorithm can be used, one must understand the ramifications of its inherent low-pass filter characteristics.

To do this, I extended the work of Bahill and McDonald (1983). They showed that the low-pass filter characteristic of this differentiator behaves like a sinc function (i.e., \( \sin(x)/x \)). The first null of this function occurs at \( f_s/(2n) \) Hz, where \( f_s \) represents the frequency at which the data were sampled. To define the low-pass characteristic, we need to know the filter's -3 dB cutoff frequency; that is, we must determine the frequency at which the filter's gain falls to \( 1/\sqrt{2} \), or approximately 0.707. Thus, we must determine the value of \( f_c \) such that

\[
\frac{1}{\sqrt{2}} = \frac{\sin\left(2\pi n \frac{f_c}{f_s}\right)}{2\pi n \frac{f_c}{f_s}}
\]

II.30

in terms of \( n \) and for a given sampling frequency, \( f_s \). To aid in this process, we create an auxiliary variable \( f_c \) defined as
\[ f^* = 2n \left( \frac{f_c}{f_s} \right) \]  

which allows Eqn. II.30 to be rewritten as

\[ \frac{1}{\sqrt{2}} = \frac{\sin(\pi f^*)}{\pi f^*} \]

I next determined the value of \( f^* \) to be approximately 0.44295 using iterative techniques. From this, we can use Eqn. II.31 to obtain an expression for the cutoff frequency, \( f_c \), of the two point central difference algorithm's inherent low-pass filter based on the sampling frequency, \( f_s \), and the parameter \( n \):

\[ f_c = \frac{0.22147 f_s}{n} \]

Notice that, as expected, there is an inverse relationship between \( n \) and the filter cutoff frequency.

Some users may wish to utilize the inherent low-pass filtering characteristics of the two point central difference algorithm in tandem with its differentiation function. To do this, the values for the sampling frequency and the (approximate) desired cutoff frequency are used to solve for the parameter \( n \) using Eqn. II.33. Note that \( n \) will not likely be an
integer; thus, the user must choose the most appropriate integer value of \( n \) that will yield filter frequency characteristics nearest those desired. The user should understand, though, that although this type of filter can provide attenuation of noise, its mediocre frequency characteristics are far from the quality of those commonly used in standard digital signal processing. For this reason, I chose to let \( n \) equal 1, thereby giving the inherent filter as wide a bandwidth as possible, and to use separate digital filters whose frequency characteristics I carefully designed to suit my specific needs (as described above).

Is the two point central difference algorithm appropriate for my differentiation needs? Because this method essentially provides an ideal differentiator in series with a low-pass filter, the degree to which we can assume it acts solely as an ideal differentiator is governed by the low-pass filter characteristic: if the filter’s cutoff frequency is significantly greater than the frequency content of the signal, we can assume that the filter has minimal effect and that the differentiator is, for all practical purposes, ideal. Using Eqn. II.33 with my sampling rate of 1002.5063 Hz and the value of \( n \) equal to 1, the inherent filter cutoff frequency for the data would be approximately 222 Hz. Because this is significantly greater than even my analog cutoff frequency of 100 Hz, I feel justified in using the two point central difference algorithm to perform my differentiation.

After the target, gaze, and head position waveforms were filtered (as described above), they were differentiated to obtain their corresponding velocity equivalents. As a specific note about implementation, I
discovered that the ASYST™ differentiator, invoked by the command
DIFFERENTIATE.DATA (with SET.DEGREE equal to 2 and with the
default setting of SET.ORDER), is equivalent to the two point central
difference differentiator (with the value of n set to 1). Although the
elements of the resulting array must subsequently be multiplied by the
sampling frequency to maintain proper scaling, this procedure for
differentiation is significantly faster than using a method which
implements the algorithm explicitly.

6. Saccade Removal Techniques: Velocity Waveforms

After filtering and differentiation have been performed on the gaze
data, an occasional low-amplitude saccade appears. I provided the option
for removing these saccades and other anomalies from the velocity
waveforms using the simple linear interpolation method described above
in the section on removing position saccades (Section II.B.3).

7. Cleaning Target Velocity Waveforms

As described in the section on target signal correction (Section
II.A.1.b.iv), the target position command signal is intentionally made to
vary at a non-uniform rate so the target will appear to move at a constant
velocity along the display screen. Because this non-uniform signal is the
only signal available for recording target positions, a mathematical re-
transformation of the target data is necessary to recover target motion as seen by the subject (see Section II.B.2.a). Although the re-transformation does grossly recreate the expected target signal, quantization and round-off effects add jitter to the signal. (This was verified by noting that the jitter virtually vanished when a constant-velocity command signal was applied, measured, and processed.) Thus, after filtering and differentiation to obtain the (artificially noisy) target velocity signal, the target data are cleaned of the jitter by replacing flat segments of the data with the expected constant value. For example, all data points after the target had attained its expected, steady-state velocity were replaced with that velocity value.

The cleaning of the target velocity signals was more than cosmetic: when the measured target signals were later used as input signals to the model simulation routines, unremoved high-frequency jitter occasionally caused problems for the simulation program's integrator. This usually resulted in the simulation run terminating prematurely.

C. Analysis Methods

As stated earlier, the main hypotheses of this research are that the smooth pursuit system provides the primary signal responsible for cancelling the vestibulo-ocular reflex (VOR) during combined eye-head tracking (CEHT) and that this cancellation occurs as a simple superposition of the internal VOR and smooth pursuit signals. To test these hypotheses,
I decided to use a modeling approach: mathematical models of the VOR and smooth pursuit systems were separately adapted to accurately reflect subjects' responses to these fundamental types of stimuli by systematically calculating optimal values for various model parameters. Next, these two basic system models were fit together to form a model hypothesis for CEHT by assuming that the command signals from the VOR and smooth pursuit sub-systems simply add at a final summing junction (proposed to be at the ocular motor nuclei). Finally, actual CEHT eye movement data were compared with simulation data generated from the CEHT model using actual target and head movement signals as model inputs. Using these methods, I searched for evidence to refute the smooth pursuit cancellation of the VOR by superposition hypotheses.

The analysis procedure may be broken down into three basic steps: (1) develop realistic models of the VOR and smooth pursuit systems, (2) obtain optimal values for specific parameters of these models to make the model outputs accurately reflect subjects' responses, (3) combine these two component models into a comprehensive model for combined eye-head tracking by assuming a simple superposition of the VOR and smooth pursuit signals, and (4) verify whether or not simulations from the comprehensive model (generated using actual target and head velocity data as the model stimuli) compared well with the corresponding eye movement responses measured during CEHT paradigms. This overall procedure is summarized in Fig. II-14.
Overall Research Scheme

Collect S.P. Data  
|↓| |↓| |↓| |↓|  
Obtain S.P. Model  
|↓| |↓| |↓|  
Determine Optimal S.P. Model Parameters Using Collected Data  
|↓| |↓|  
Adapted S.P. Model

Collect VOR Data  
|↓| |↓|  
Create VOR Model  
|↓|  
Determine Optimal VOR Model Parameters Using Collected Data  
|↓|  
Adapted VOR Model

Fit Models Together (ACSL)

CEHT Model

\[ \dot{T}, \dot{H} \] (System Inputs)

COMPARE

\[ \hat{\dot{\varepsilon}}_{\text{model}}, \hat{\dot{\varepsilon}}_{\text{measured}} \]

Does the CEHT Model Describe the Data?

Fig. II-14. Overall scheme used to test the research hypotheses.
1. Preparing the Fundamental Models

The first phase of the hypothesis testing process involved obtaining realistic models for the VOR and smooth pursuit systems. Although I could have done this in a number of ways, I felt that it was important to use models whose structures each have a strong physiological basis (i.e., are homeomorphic). For this reason, the model implementations of both the horizontal smooth pursuit and VOR systems are based on well-accepted system models known to accurately reflect the transient response characteristics of these systems. Then, I used optimal parameter estimation methods to adjust each model's parameters so that actual subject responses could be precisely characterized by simulations from the appropriate model.

a. Considerations for Modeling the Plant

Before describing how the models of the various ocular motor sub-systems are defined, I first describe how I dealt with modeling the ocular motor plant, an element common to all sub-systems. The purpose of the ocular motor system is to send appropriate signals to the extraocular muscles to cause desired eye rotations. In terms of classical systems and control, the ocular motor system may be viewed as a controller, and that which is effected by a controller is called the plant. Because the eye in its orbit and its associated musculature are the target of the ocular motor system's command signals, these structures are collectively known as the
ocular motor plant (or simply the plant). No matter what controller the
ocular system uses in a particular circumstance (e.g., the VOR or the
saccadic system), the common structure affected by all ocular motor
command signals is the plant. Thus, obtaining a reasonable model for the
plant is of prime importance in modeling any of the ocular motor system’s
component eye movement types.

Various methods exist for modeling the plant. Goldstein (1987)
outlined the background of three different plant models. The most
elementary of these is a simple single-pole passive plant model proposed
by Bahill et al. (1975). Modeling the plant this way is supported by single-
unit recordings of motoneurons during slow eye movement (Fuchs and
Luschei 1970; Robinson 1970), and it takes the form

\[
\frac{1}{sT + 1}
\]

where the value of \( T \) is approximately 240 msec. A two-pole model for the
plant was proposed by Keller and Robinson (1972) based on vergence eye
movements. This model may be represented as

\[
\frac{1}{(sT_1 + 1)(sT_2 + 1)}
\]

where \( T_1 \) has a value of approximately 240 msec and \( T_2 \) is about 15 msec.
Finally, another model originally proposed by Robinson (1964) has more recently received support from two studies investigating saccades (Goldstein 1983; Optican and Miles 1985). This is another passive plant model, which is comprised of two visco-elastic elements in series and characterized mathematically by

\[
\frac{(sT_2 + 1)}{(sT_1 + 1)(sT_2 + 1)}
\]

where \(T_1\), \(T_2\), and \(T_3\) have values of 260 msec, 12 msec, and 72 msec respectively.

Now, before we choose one of these models to represent the plant, it would be beneficial to review a bit more about the internal operation of the ocular motor system. Most of the models currently presented to describe various characteristics of the ocular motor system represent their input and output signals in terms of velocity. Although this may be quite useful, it ignores the reality that the ocular motor system actually acts to change the position of the eye with respect to the head. Thus, each of these models assumes the presence of an internal neural integrator which converts the output velocity command signal into a position signal to be used by the extraocular muscles. Theoretical and experimental evidence suggest that a common neural network actually exists in the brain which is responsible for decoding the internal velocity signals to create position command signals (Cannon and Robinson 1987; Robinson 1975).
Because the neural integrator and the plant are intimately related in terms of function, it is useful to consider modeling them together, as demonstrated by Robinson et al. with their model of the smooth pursuit system (1986). How should the neural integrator be modeled? The commonly accepted approach is to treat the neural integrator as a pure integrator in parallel with a feedforward gain element (Robinson 1981). When this type of neural integrator is coupled with the single pole plant model, the overall transfer function reduces to $1/s$, or, perfect integration (see Fig. II-15a). Unfortunately, Goldstein explains that this plant model results in unphysiological torque profiles and thus, should not be used (1987).

If the same pulse-step neural integrator is coupled with the two-pole plant model (Fig. II-15b), the overall transfer function reduces to

$$\frac{1}{s} \left( \frac{1}{sT_2 + 1} \right)$$

\[
\text{II.37}
\]

which demonstrates band-limited integration. (This assumes, not unreasonably, that the value of the time constant in the neural integrator's feedforward pathway equals $T_1$.) This is the scheme used by Robinson et al. for defining the plant of their smooth pursuit model (1986). However, because they represent their output in terms of velocity, they simply drop the integration term $1/s$ and leave the remaining lag element to represent the plant. Goldstein points out that this two-pole plant model
Fig. II-15. Various ways to model the ocular motor plant. Shown are schemes which combine the operations of the neural integrator and the plant. 

a) Pulse-step neural integrator coupled with the single pole plant model. 

b) Pulse-step neural integrator coupled with the two-pole plant model. 

c) Pulse-slide-step integrator coupled with the two visco-elastic element plant model.
Goldstein supports the two visco-elastic element plant model, and he proposes an alternate model of the neural integrator to be coupled with it (1983). Figure II-15c depicts Goldstein's pulse-slide-step integrator coupled with the two visco-elastic element plant model. It can be shown that if appropriate values are chosen for the neural integrator parameters $T$ and $\alpha$, it is possible to obtain pure integration ($1/s$) and effectively cancel all the plant dynamics. Following this development, I chose to incorporate a similar integrator/plant combination in my modeling to effectively eliminate any dynamic effects caused by the plant, and, because I represent the eye movement output signals in terms of velocity, I simply dropped the $1/s$ integration element (as is standard practice in the ocular motor literature). Also, to help simplify the subsequent calculations involving the models, I grouped the delay attributed to the plant with other model delay elements knowing that this delay element could be recreated whenever necessary. Thus, for my modeling purposes, the plant element dynamics may be ignored altogether.

b. Overview of the Parameter Estimation Procedure

Because I chose to test my hypotheses about the interaction between the VOR and smooth pursuit systems using modeling techniques, I felt that it was important to make the models for these systems reflect actual subject responses as accurately as possible. Thus, I employed optimal
parameter estimation techniques to help tune the models to each individual subject. A detailed description of the parameter estimation procedure I used for the smooth pursuit model has been published elsewhere (Huebner et al. 1990). In this, we outline why this method is superior to other less-rigorous techniques, and we provide details about how to implement the procedure.

Basically, we must first choose which model parameters we wish to optimize. For the models I am concerned with, it was not possible to represent the model outputs as linear combinations of the parameters to optimize. Thus, it was necessary for me to use nonlinear parameter estimation techniques. The heart of the nonlinear parameter estimation process is a software routine called NL2SOL (Dennis et al. 1981a; Dennis et al. 1981b). When NL2SOL is provided with an initial set of parameter values, a residual vector (an array containing the value-by-value differences between a set of response data and the corresponding simulation data generated by the model), and a complete set of sensitivity functions (arrays describing how changes in the model output are effected by changes in each parameter as a function of time during a simulation), it computes a new, updated set of parameter values. After a new set of parameter values is made available by NL2SOL, a model simulation is performed in ACSL (the Advanced Continuous Simulation Language, Mitchell and Gauthier Associates, Concord, MA) using the new values. The simulation is used to calculate a new residual vector and an updated set of sensitivity functions; these new arrays can then be used by NL2SOL to obtain yet another updated set of parameter values. This cyclic process of performing
simulations and recalculting values for the parameters continues until
NL2SOL's convergence criteria are satisfied. When this occurs, the
parameter values are considered optimal in a least-squares sense. (For
details about this procedure, see Hubeber et al. (1990).)

To facilitate the parameter estimation process, I created a general
FORTRAN program which integrated the simulation capabilities of ACSL
with the parameter estimation function of NL2SOL. This general program
mediated the interaction between the two routines and performed all the
necessary file management. The latter involved reading a file containing
a set of response data (created during the data processing procedure
described above and to which the model will be adapted) and another file
containing an initial set of parameter values. It also involved creating
separate output files consisting of (1) the optimal parameter values for a
given set of response data along with the corresponding convergence
information, (2) arrays containing the response data and the
responding model generated using the optimal parameter values,
and (3) the final set of sensitivity functions for each parameter. Then, for
each of the fundamental models described below, the general program was
made specific by including the necessary ACSL code for describing the
model as well as the code to calculate the required sensitivity functions.

A set of optimal parameter values was obtained for every suitable
smooth pursuit onset, smooth pursuit offset, and VOR response. Then,
for each subject and for each direction (leftward or rightward), the
parameter values were averaged. All appropriate trials were used in
calculating the mean parameter values unless (1) a response contained excessive noise or eye movement artifacts (e.g., blinks) so that the waveform was clearly not similar to others of the same kind, or (2) the result of the parameter estimation process indicated anything other than absolute convergence to the optimal parameter vector. This yielded one representative set of parameter values for each subject, for each direction. (I chose to maintain a distinction between leftward and rightward responses to guard against the possibility that subjects may have a directional response asymmetry.)

c. The Fundamental Models

By using modeling techniques to test the superposition hypothesis of the smooth pursuit signal cancelling the VOR during CEHT, I needed to define specific models for both the onset and offset phases of smooth pursuit and for the VOR. These models were incorporated in the parameter estimation process to make their simulation outputs resemble subject responses as closely as possible. Then, after parameter estimation, they were combined to form the comprehensive CEHT model used for testing the research hypotheses.

i. Smooth Pursuit Onset Model

Probably the most realistic model of transient smooth pursuit dynamics available is one proposed by D. A. Robinson and colleagues
(1986). This model, depicted in Fig. II-16, is a goal-oriented model which uses an internal positive feedback loop to provide for the internal recreation of a target velocity signal. This internal recreation of target velocity serves as the "goal" for the smooth pursuit system. Also, the internal positive feedback loop is postulated to directly cancel the inherent outer negative feedback loop (resulting from the retina's direct connection to the eye) thereby making the overall model effectively open-loop (and thus stable). The elements in the forward path of the model provide for the observed dynamic features of smooth pursuit responses. These features include the overall latency of smooth pursuit (represented by several distributed time delay elements), the ringing often observed in pursuit responses (provided by the internal negative feedback loop), and the acceleration characteristics of smooth pursuit (guided mainly by the lag element and an acceleration-saturation element).

One case against the smooth pursuit model of Robinson et al. is that a relatively large time delay (on the order of 80 msec) is required in the positive feedback path used to cancel the outer negative feedback loop. Although it may be unreasonable to expect the brain to cause a signal to be delayed this long, if one instead thinks of the delay as providing a phase shift of the feedback signal to cause time alignment of two signals, the approach is not all that unpalatable. Because a pure time delay may be represented in the frequency domain as an element having unity gain and a linear phase shift (Lathi (1974), p. 138), if the time delay element can be replaced with a set of simple elements which collectively provide the same frequency characteristics as the delay (i.e., a linear phase shift) over
Fig. II-16. The smooth pursuit model of Robinson et al. (1986) and the incorporated parameter values. CP, central processing felt to reflect the activity of the central nervous system; CNS; PMC, premotor circuitry; AS, acceleration saturation; VS, velocity saturation; Ṫ, target velocity (input); Ŕ, eye velocity (output); ̇e, retinal error velocity; Ṫ', centrally reconstructed copy of target velocity; ̇Em, desired eye velocity; ̇em, motor error; ̇Em', desired eye acceleration; ̇E', eye velocity command signal; numbers in boxes refer to the number of msec of pure delay; τ1, τ2, τ3, internal delays; Tc, time constant of CP; ̇e0, break in nonlinearity AS; A, PMC loop gain; K, gain of the oculomotor plant; Tc2, time constant of oculomotor plant; P₁ and P₂, plasticity gain parameters. The input-output relation of AS is

\[ \dot{\hat{E}}' = 40 + 5 \dot{\hat{e}}_m, \quad \dot{\hat{e}}_m > \dot{e}_0; \quad \dot{\hat{E}}' = (5 + 40/\dot{\hat{e}}_m) \cdot \dot{\hat{e}}_m, \quad \dot{\hat{e}}_m \leq \dot{e}_0 \]

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the bandwidth of interest, then the incorporation of such an internal positive feedback loop is still warranted. Using techniques outlined by Deno et al. (1989), the delay element may be re-expressed by a Padé approximation in the form of a rational polynomial transfer function. Although such an approximation is only valid over a certain bandwidth, it may be formulated to encompass the entire bandwidth of the smooth pursuit system. No matter how complicated this transfer function is, it may be broken down into the sum of simple linear lag and lead elements which provide the necessary phase shifting of the feedback signal to be equivalent to the pure time delay. The use of such a sum of simple linear elements to represent activity in the nervous system has gained popularity with the advent of neural network theory. Thus, although Robinson et al. propose a large, pure time delay, it can be replaced theoretically with several simple linear elements which collectively serve the same purpose and which have a plausible theoretical basis for existing in the brain. Therefore, for simplicity, I retain the (unrealistically) large time delay element in the model’s positive feedback loop knowing that it could be replaced with an equivalent (realistic) network of simple, linear lag and lead elements.

To simplify my parameter estimation calculations, I removed the internal positive and external negative feedback loops from the model (Fig. II-17). Note that this modification affects neither the overall system response nor the individual system elements containing the parameters I chose to optimize (Huebner et al. 1990). Then, in addition to the four parameters we optimized in our paper (the internal loop gain A, the lag
Fig. II-17. Equivalent representation of the Robinson et al. model of smooth pursuit (Fig. II-16) used in the parameter estimation procedure. \( \dot{T} \), input target velocity; \( \dot{E} \), output eye velocity; \( \tau_R \), retinal time delay. Other symbols as in Fig. II-16. Note that a plant element is included for completeness, but its dynamics can be ignored (see text).
element time constant $T_c$, the piecewise-linear breakpoint in the acceleration-saturation element $\dot{\theta}_0$, and the plasticity parameter governing the overall response gain $P_1$), I also added calculations to optimize values for the central delay element $\tau_1$ and the internal feedback delay element $\tau_2$. The velocity saturation element ("VS") was included for completeness but was of no consequence because the value of the breakpoint velocity $\dot{E}_0$ (approximately 90 deg/sec) is much higher than any of the pursuit velocities achieved in my experiments. Thus, by optimizing these six parameters I felt that I could obtain as good a match between actual smooth pursuit data and the generated model output data as possible.

Finally, Fig. II-17 contains a block representing the plant. Actually, this is only provided for completeness: as outlined in Section 1a, above, the actual plant dynamics can be ignored.

ii. Smooth Pursuit Offset Model

The smooth pursuit onset model introduced above is well-suited for describing subject responses to the onset of target motion when the head is stationary. However, Robinson et al. point out that responses to a cessation of target motion do not resemble responses to the initiation of target motion (1986). They suggest that different neural circuitry is probably responsible for mediating the offset of smooth pursuit, and, although they did not propose a specific model, they indicate that the dynamics resemble those due to a first-order lag element. Because some of my stimulus paradigms involved having the target stop after traversing a
significant portion of the subject's ocular motor range, it was necessary for me to define a model for the offset of smooth pursuit. Also, because I was invoking visually-enhanced VOR, I needed to introduce a visual mechanism into the model by which fixation of the stationary target could be maintained. A smooth pursuit offset model could additionally offer a way to stabilize gaze on a stationary target despite head perturbations. For this reason, I loosely refer to this pursuit offset model as a "fixation" model. Support for a separate fixation pathway was presented by Luebke and Robinson with their finding that fixation in not simply pursuit at zero velocity (1988).

Figure II-18a depicts my effort to model smooth pursuit offset. Note that this model also assumes the existence of an internal positive feedback loop to cancel the inherent outer negative feedback loop, as is the case with the smooth pursuit onset model. Again, to simplify my calculations, I have removed these two feedback loops from the model (Fig. II-18b). Also, as with the smooth pursuit onset model, plant dynamics can be ignored.

As proposed by Robinson et al. (1986), this model contains a single pole lag element governed by the time constant parameter $T_f$. Also included is a unique delay element $\tau_f$ for this pathway (in addition to the retinal delay $\tau_r$), and, to be consistent with the smooth pursuit onset model, the velocity-saturation element "VS" is incorporated. (Note that the gain of the linear segment of the velocity-saturation element is also $P_1$; the value of $P_1$ is determined during the smooth pursuit onset model's
Fig. II-18. Proposed model for smooth pursuit offset. a) The entire model. b) Equivalent model with the outer feedback loops removed. Symbols as in Fig. II-16.
parameter estimation procedure and is subsequently included here as a constant.) Thus, parameter estimation for the smooth pursuit offset model simply involved finding optimal values for the parameters $\tau_f$ and $T_f$.

For simplicity, I assumed that this pursuit offset model serves as an adequate approximation for the fixation system as well. Although I could have implemented a more detailed hypotheses for fixation (e.g., de Bie and van den Brink (1986)), I believed that doing so was beyond the scope of this research.

iii. VOR Model

Although several sophisticated models have been developed for the VOR (e.g., Robinson 1977), most of these contain elements and pathways to explain phenomena (such as velocity storage) that are not relevant to the present project. Again, for computational efficiency I chose to model the VOR in as simple a way as possible.

Figure II-19 depicts my initial attempt at modeling the VOR. The VOR is truly a reflex, and the most direct pathway between its sensors (the semicircular canals of the inner ear) and its innervation of the extra-ocular muscles consists of a simple three neuron arc. This supports keeping the model simple. Also, the VOR is open-loop in that its normal operation does not rely on visual feedback. (Technically speaking, the VOR does use
Fig. II-19. Initial attempt to model the VOR. $\dot{H}$, head velocity (input); $\dot{E}$, eye velocity (output); $\tau_{VOR}$, delay element related to the latency of the VOR; $T_1$ and $T_2$, time constants governing the lag and lead elements that represent the dynamics of the semicircular canals; $A$, VOR gain.
visual feedback, but only for the purposes of long-term recalibration.)

The lag and lead elements, governed by the parameters $T_1$ and $T_2$, represent known dynamics of the semicircular canals (Wilson and Melvill Jones 1979). The lag element limits the bandwidth of these VOR transducers, and the value of its time constant $T_1$ is approximately 3 msec. The lead element is incorporated to account for the fact that after the cupula is displaced due to a head rotation, it will be passively restored to its resting zero position with a time constant $T_2$ of approximately 5 sec. Actually, when subjects experience sustained rotation in the dark, their corresponding eye movements show a similar decay, but with a time constant about three times as long—on the order of 15 sec. Because this time constant can be easily measured experimentally, and because I am interested in describing the global characteristics of the VOR in this research, we measured $T_2$ directly for each subject and let the corresponding lead element represent that for the entire VOR, not just for the semicircular canals. Making this simplification eliminated the need to provide complex additions to the VOR model while still maintaining realistic VOR characteristics. Thus, $T_1$ was assigned a constant value of 3 msec while $T_2$ was measured directly for each subject and held constant during the subsequent parameter estimation procedure.

A delay element was included to account for the latency of the VOR; the value of its time delay, $\tau_{VOR}$, was determined for each subject by parameter estimation and was expected to be less than 15 msec (Maas et al. 1989). Also, a gain element $-A$ was incorporated in the model to allow
subjects' VOR gains to vary somewhat from the ideal value of \(-1\). (Note that a minus sign is included so that eye rotations will be in the opposite direction from head rotations, as expected for a normal VOR response.) For completeness, Fig. II-19 contains a block representing the plant. Practically, however, the plant dynamics can be ignored (following the development in Section 1a, above).

For this research I must characterize the VOR while it works in consort with vision, as head perturbations occur while the subject fixates upon a stationary spot of light. This is *visually-enhanced* VOR. Thus, to obtain VOR model parameters that characterize the activity of the VOR while enhanced by vision, I coupled the Smooth Pursuit Offset model (*i.e.*, "fixation" model) with the VOR model. In this way, deficiencies in the ability of the VOR to maintain fixation on the stationary light spot could be corrected with a contribution from the Smooth Pursuit Offset system, driven by vision. In this case, the Smooth Pursuit Offset model was equipped with *fixed* model parameter values determined from its own parameter estimation procedure. The only modifications required to provide this coupling were (1) to change the input of Smooth Pursuit Offset from absolute target velocity \(\dot{T}\) to target velocity with respect to the head \(\ddot{T}-\dot{H}\), and (2) to sum the final Smooth Pursuit Offset signal with the signal from the VOR.

After performing some preliminary experiments, I realized that it was necessary to modify the model of the VOR. If the VOR acts perfectly
with a gain near -1 (or 1, assuming an inherent inversion of the signal), one would expect that gaze would not be significantly perturbed, despite perturbations of the head. However, as shown by the data in Fig. II-20, gaze is clearly perturbed when the head begins to move from rest. However, when the head stops again after being in motion, gaze is only slightly perturbed. I investigated some possible causes which may explain this nonlinear behavior. One of these including looking for differences in the acceleration and deceleration characteristics of the vestibular chair; I found that the differences were negligible and thus, could not account for the observed variations in gaze perturbation. Also, the improved performance during VOR offset could not arise due to augmentation of the VOR with signals derived from visual inputs. This is because the latency of visual processing (taking at least 50 msec for retinal processing alone (Robinson et al. 1986)) would delay such visual contributions until long after the observed eye motion has already occurred.

Barr et al. (1976) have demonstrated that subjects can modulate their VOR gains during rotations in the dark based on various instructions. When instructed to perform mental arithmetic the subjects’ VOR gains were approximately 0.65. However, the VOR gains rose to near 0.95 when the same subjects were asked to fixate imaginary targets that were stationary in space. This suggests that VOR gain is not constant but rather can vary depending upon its required use. From this, I theorized that when the VOR is not being used (i.e., when the head is stationary), its gain approaches some quiescent resting level, perhaps near 0.65. However, when the head begins to move and gaze stabilization is required, the brain
Fig. II-20. Sample data from a trial of VOR Offset demonstrating the need to include a variable gain element in the VOR model. When the head begins to move (at 0.0 sec), gaze is perturbed, indicating VOR gain is less than 1.0. However, when the head later stops (at 2.0 sec), gaze is not perturbed, indicating VOR gain has risen to near 1.0. Note that, for display convenience, the eye velocity waveform has been inverted (~ Eye).
realizes the need to get the maximum utility from the VOR and thus, increases the VOR gain to near 1.0. Similar results have been recently reported, but not specifically commented on, by Gauthier and Vercher (1990).

Although it is not unreasonable to presume the brain works in such a manner, can it explain why we observe gaze perturbations at the onset of head motion but not at the offset of head motion? If the VOR gain is much less than 1.0 (e.g., near 0.65), when a head rotation is initiated the magnitude of the induced eye rotations will be less the magnitude of the corresponding head rotation. In this case, gaze will be perturbed in the direction of head motion because the compensatory eye rotation cannot keep up with the head movement. This is consistent with the VOR data shown in Fig. II-20. Then, when the brain senses head motion, the VOR gain is increased to near 1.0 and the resulting eye rotations become equal and opposite to the head rotation, yielding a near-zero change in gaze. This also can be seen in Fig. II-20. Note that the actual dynamics of this increase in gain are obscured somewhat by the simultaneous contribution to the eye movement signal from the "fixation" pathway, which starts to contribute about 75-120 msec into the response (see Section ii above). Next, because the VOR gain has increased to near 1.0, when the head is stopped the VOR causes the eyes to stop at virtually the same rate. This will result in a negligible perturbation of gaze, as can be observed in Fig. II-20 at the 2 sec point of the response. Finally, because the head is at rest, the brain can then reduce the VOR gain back to its resting level. Thus, the hypothesis of dynamically changing VOR gain is consistent with my
observation of varying degrees of gaze perturbation relative to the onset and offset of head motion.

To model the proposed phenomenon of dynamically changing VOR gain, I added a variable gain element (see Fig. II-21). To change the VOR gain, I assume the brain monitors the internal head velocity signal, $\dot{H}'$. If the magnitude of the internal head velocity signal is less than some threshold value $\dot{H}'_0$, then the VOR gain is set to the resting value $A_{off}$. This allows for maintaining a lower VOR gain despite small head movements (which do not cause significant changes in the visual field). For my analyses, $\dot{H}'_0$ was set to 5 deg/sec. On the other hand, if the magnitude of the internal head velocity signal is greater than $\dot{H}'_0$, then the brain causes an increase in VOR gain up to the active value of $A_{on}$. A delay element $\tau_{A_{vor}}$ was added to allow the brain time to make decisions about which level to set the VOR gain. Also, it is unreasonable to expect the VOR gain to change instantaneously; thus, I included a lag element, governed by the time constant $T_{A_{vor}}$ to make the VOR gain change gradual. Therefore, adapting the VOR model to subject responses involved optimizing five parameters: $\tau_{VOR}, A_{off}, A_{on}, \tau_{A_{vor}}$ and $T_{A_{vor}}$.

Although the observed phenomenon of gaze perturbation at the onset but not the offset of head motion may actually be manifested by some other mechanism, my approach provided a reasonable working hypothesis from which my VOR modeling and parameter estimation efforts could proceed. Whether or not this hypothesis is correct actually
Fig. II-21. Revised model of the VOR incorporating a variable gain element. \( \dot{H} \), internal estimate of head velocity; \( A_{off} \), resting VOR gain value; \( A_{on} \), active VOR gain value; \( \dot{H}_0 \), threshold head velocity for eliciting a change in VOR gain; \( \tau_{Avor} \), delay element to allow the brain time to decide at which level to set VOR gain; \( T_{Avor} \), lag element time constant to make the gain changes gradual. Other symbols as in Fig. II-19.
has little bearing on the validity of my overall research hypotheses. In fact, the variable VOR gain element was mainly included to suggest one possible explanation for the observed phenomenon and to provide a means for allowing the VOR model simulations to more closely resemble actual subject responses.

2. Creating the Combined Eye-Head Tracking Model

After the fundamental models for smooth pursuit (both onset and offset) and the VOR were specified, the next task was to fit them together in a way which might describe combined eye-head tracking. The manner in which this was done was prescribed by one of my experimental hypotheses: specifically, that the VOR is cancelled through superposition with an internal smooth pursuit command signal. Thus, this hypothesis of combined eye-head tracking requires that the internal signal from the smooth pursuit system simply adds to the internal VOR command signal to yield the overall eye movement command signal. This is depicted in Fig. II-22, which shows a block diagram for the proposed model for CEHT. The model for each sub-system is simply inserted into the appropriate block of this overall model. The superposition between the smooth pursuit command signal $\dot{E}'_{SP}$ and the VOR command signal $\dot{E}'_{VOR}$ is clearly shown at the summing junction just before the plant. For horizontal eye movements, this is theorized to occur in the abducens nucleus of the ocular motor system. (Although blocks representing the plant have been included in the figure for completeness, remember from
Fig. II-22. Overall model hypothesis for combined eye-head tracking. The VOR signal ($\dot{E}_{\text{VOR}}'$) is simply cancelled by adding with a signal generated by the smooth pursuit system ($\dot{E}_{\text{sp}}'$). Other symbols as in Figs. II-16 and II-21. See text for details.
Section 1a, above, that the actual plant dynamics may be ignored.) The delay element \( r_e \) corresponds to retinal processing delay. The retinal error velocity signal \( \dot{e} \) still drives the pursuit system as before, but now the target velocity is judged with respect to the head (\( \dot{T} - \dot{H} \)), not with respect to an earth-fixed reference (\( \dot{T} \), as was the case with the original, head-stationary smooth pursuit model).

One additional issue to address in defining this overall model was to specify how the smooth pursuit onset and offset sub-systems interact. Clearly, based on the way in which they were defined, each of these two sub-systems is meant to perform exclusive of the other. Thus, I reasoned that the brain monitors its internal representation of target velocity, \( \dot{T} \). If the brain determines that the target is moving faster than some threshold amount \( \dot{T}_{0} \), it uses its system designed to quickly acquire and track moving targets: the onset branch of smooth pursuit. On the other hand, if the brain determines that the target is no longer moving (or moving at a very slow speed, below \( \dot{T}_{0} \)), it instead utilizes a different pathway responsible for providing smooth pursuit offset signals. In a loose sense, the latter may also be considered a “fixation” system (see Section ii, above). For this model, I chose to set the threshold value \( \dot{T}_{0} \) to 5 deg/sec based roughly on the Luebke and Robinson study of fixation (1988). Then, for modeling simplicity, I chose to represent the smooth pursuit command signal \( \dot{E}_{sp} \) as coming from a switch set to one or the other pursuit sub-systems, where the direction of the switch is based on the current value of
\( \hat{T} \) (Fig. II-22). Again, this interaction could have been modeled in a more complicated way. However, I decided to develop a scheme that was as conceptually simple as possible without being unreasonable.

3. **Comparing Subject Data with Simulated Model Data**

The final phase of my analysis involved comparing simulated data from the CEHT model with actual subject data (Fig. II-14). To do this, I took the head and target velocity waveforms for each trial (that were processed concurrently with the Eye velocity data using the methods of Section B) and used them as input signals to the CEHT model described in the previous section. The resulting model simulation from a given trial was then compared *directly* with (a processed form of) the actual data measured from the subject in response to the *same* experimental stimuli. Thus, there was a direct pairing between subject data and model simulations for a specific trial of target and head motion. It was important to maintain this association because trial-to-trial variations in the stimuli (particularly in the head motion stimulus) should show corresponding variations in both the subject and model responses, and other means of comparison would obscure this fundamental relationship. Again, with my comparison I am looking for evidence to *refute* the hypothesis that the smooth pursuit system provides the primary signal responsible for cancelling the VOR during combined eye-head tracking. I used two methods of comparison, one descriptive and one quantitative. These methods are presented next.
a. Residual Analysis

One way of probing the similarity between the model and subject data for a given set of stimulus waveforms was to calculate residuals. For each trial I created a residual waveform which was comprised of the point-by-point differences between the model simulation data and the actual subject data. If the model exactly predicted the subject's response, each point-by-point difference would be zero, and the residual waveform would be a constant with a value of zero. Variations between the simulation and the subject data would add features to the residual waveform. If these features were due to random effects, one would expect that averaging a collection of residuals (derived from similar experimental stimuli) would tend to cancel the features. On the other hand, if a feature in a residual waveform is maintained after averaging, a systematic error in the model's capacity to describe the subject data is indicated. Thus, I averaged the residuals from similar trials and looked for consistent differences between the model simulation and subject data.

Although this means of comparison is rather descriptive and not well-suited for quantitative hypothesis testing, it is perhaps the purest way to analyze the data. Instead of providing a simple yes-no answer to a rather complicated question, presentation of the residuals allows one to assess the relative strengths and weaknesses in the model's capacity to describe the data over the span of the responses. For my purposes, I could
consider rejecting the superposition hypothesis if the average residual waveforms showed gross, consistent features (having values similar to the magnitude of the stimulus perturbation—15 deg/sec for these experiments) for all subjects. However, smaller features in the residuals may only indicate that refinement of elements in the component models may be necessary and that the overall hypothesis of superposition may still be valid. To a degree, conclusions are drawn based on the collaborative judgments of various informed observers.

b. Correlation Methods

Although residuals can tell a great deal about a model's capacity to describe a specific phenomenon, the aforementioned method of drawing conclusions from residual plots lacks the ability to make concrete statements about the similarity between the simulated and measured data based on quantitave measures. In an attempt to bridge this gap, I searched for an accepted means of comparing the similarity of two waveforms (e.g., model and data). Unfortunately, I was unable to find such a method that is used with any regularity in the scientific community. Thus, after careful consideration, I opted to develop my own procedure for comparison based on correlation methods.

Correlation involves measuring the strength of the linear relationship between two variables. Although a subject's response waveform and the corresponding model simulation waveform are both
functions with respect to time, I am interested in determining the degree
of linear association between these two deterministic signals (Lathi 1965).
One way to do this is to treat the two waveforms as a pairwise collection of
variable values, where each pair of values corresponds to a unique time in
a given response. Then, the correlation between the two waveforms is
calculated using the collection of these pairings. Although using pairs of
deterministic variables with an underlying time dependence is not how
correlation statistics are commonly used, I felt that this procedure provides
me with a useful indicator of the similarity between measured and
simulated waveforms which is not unreasonable.

The degree of correlation between the two waveforms was
estimated by calculating the Pearson correlation coefficient $r$. From this, I
also calculated the statistic $r^2$, sometimes referred to as the coefficient of
determination. The coefficient of determination expresses (as a percentage)
the variation in one variable that can be attributed to a linear relationship
between the two variables (Milton and Tsokos (1983), p.362). If $r^2$ has a
value near 1, one can conclude that a strong relationship exists between
the variables (or, the two waveforms).

One caveat about using this procedure involves choosing an
appropriate response interval from which the statistics are calculated. For
example, assume that a given model does a poor job of describing the
transition region of a subject's response (i.e., the time interval of the subject's
transient response to the trial's intended perturbation). However, assume
the model does eventually provide simulation values that reach the
correct steady-state value. If calculations of $r$ and $r^2$ are performed using several data points both before the perturbation and after the model attains steady-state (relative to the number of points in the transition region), then the response data indicating poor model performance will be overwhelmed by the values at the ends of the response, and the calculated correlation values will be erroneously high. For this reason, I limited the range of the response data over which I calculated the statistics to entail only the anticipated transition region, because this is where the model is most likely to fail. To be certain that I was working with as much of the transition region as possible while eliminating significant portions of the steady-state responses, the actual choices of the transition region size for Smooth Pursuit Onset and Offset, VOR Onset and Offset, as well as for both the Chair Brake and Delayed Target Onset paradigms were made based on observations of various measured responses. For the VOR Onset, VOR Offset, and Chair Brake paradigms, I used only the model and response data from the first 100 msec after the chair began to brake. Because subjects took varying amounts of time to reach peak eye velocity in response to the onset of target motion, I used from 350 to 400 msec after the target began to move, depending on the subject, for the Smooth Pursuit Onset and Delayed Target Onset paradigms. Finally, I used the first 600 msec after the onset of target motion to evaluate the performance of the model for Smooth Pursuit Offset in describing subject response data.

This research is investigating the plausibility of a superposition model involving smooth pursuit to explain VOR cancellation during CEHT. Because an alternative mathematical hypothesis is not available at
this time, I am unable to quantitatively compare the superposition model with another specific model. However, I performed an indirect quality comparison based on the anticipated eye movement characteristics if the smooth pursuit superposition hypothesis is not correct.

Recall that if smooth pursuit is not responsible for cancelling the VOR during combined eye-head tracking, then when the head stops during the Chair Brake paradigm one would expect that at least 75 msec (and more like 130 msec) would pass before the eye again begins to track the still-moving target. As a crude alternative test to the superposition hypothesis, for the Chair Brake paradigm I separately time-shifted (delayed) the model simulation waveforms 75 msec relative to the eye signal and recalculated the correlation statistics. If the calculated correlations increase significantly, it suggests that an additional response latency of about 75 msec should be accounted for by the model. In this case, we may wish to conclude that the superposition hypothesis should be rejected in favor of an alternative hypothesis which cancels the VOR using some other accessory pathway. However, if the correlations decrease significantly, the additional latency required to "charge-up" the smooth pursuit system is not necessary, perhaps because pursuit is already active. Under this circumstance, we may wish to conclude that the superposition hypothesis should be accepted (at least until another quantitative hypothesis is proposed to challenge it).

A similar alternative test of the superposition hypothesis was employed for the Delayed Target Onset paradigm. Remember that if the
smooth pursuit system is responsible for cancelling the VOR during CEHT, then, after the initial phase of visually-enhanced VOR in the Delayed Target Onset paradigm, the cancellation of the VOR signal in response to the onset of target motion should have a morphology resembling that of smooth pursuit onset, complete with a latency of at least 75 msec. On the other hand, if smooth pursuit is not responsible for VOR cancellation, then cancellation of the VOR signal would occur in some other manner, most likely with a latency less than 75 msec. Based on an alternative scheme for VOR cancellation proposed by Cullen et al., a non-visual mechanism acts to cancel the VOR at an early latency of less than 30 msec (Cullen et al., in press). Thus, I recalculated the correlations for the Delayed Target Onset trials by shifting the model simulation waveforms in time so that the onset of the model's "response" to the target motion occurred 30 msec after the target began to move. Again, if the calculated correlations increase significantly, it suggests that a response latency greater than 30 msec is not required before VOR cancellation occurs, and thus, that smooth pursuit does not provide the cancellation signal. In this case, we may wish to conclude that the superposition hypothesis should be rejected in favor of an alternative hypothesis which cancels the VOR using some other accessory pathway. However, if the correlations decrease significantly, it suggests that a latency greater than 30 msec is required to "charge-up" the smooth pursuit system in this paradigm. Under this circumstance, we may wish to conclude that the superposition hypothesis should be accepted.
III. RESULTS

A. Parameter Estimation Results

Using the methods described in Section II.C, optimal values for the Smooth Pursuit Onset, Smooth Pursuit Offset, and VOR model parameters were obtained from appropriate response trials for each subject.

A typical Smooth Pursuit Onset response from Subject 1 is shown in Fig. III-1 along with the corresponding Smooth Pursuit Onset model simulation, which used parameter values calculated to be optimal for that response. The model successfully describes the corresponding data, to a first approximation, in terms of matching the response latency and initial acceleration characteristics.

To quantify the similarity between response data and the corresponding model simulation data, I employed a correlation technique (developed in Section II.C.3.b). For the onset and offset phases of both smooth pursuit and VOR responses, the average correlation coefficient and coefficient of determination values calculated using this procedure indicate how capable the parameter estimation procedure is of finding an optimal set of parameter values to allow each model to adequately describe the related types of response waveforms. If the simulated model velocity waveform exactly predicted the measured eye velocity values (over the transition region of each response), then the calculated correlation
Fig. III-1. Typical response demonstrating the onset of smooth pursuit. Also shown is the corresponding model simulation derived using optimal parameter values calculated for this response. Notice that after the step in target velocity, about 130 msec elapses before either waveform responds to track the target. This latency is characteristic of smooth pursuit responses to changes in target velocity.
coefficient and coefficient of determination would be 1.0 and 100%, respectively. The degrees to which these coefficients deviate from such ideal values gives us an indication of the relative similarity (or difference) between the measured eye velocity values and the corresponding simulated model velocity values.

The average correlation coefficient and coefficient of determination values comparing the similarity between subjects' responses during the onset of smooth pursuit with the corresponding model simulations are given in Table III-1, for all four subjects and for both rightward and leftward motion. Note from this table that all conditions show that the degree of linear association between the model simulation and the subject data is at least 97.5%. This indicates that simulations of smooth pursuit onset adequately describe actual pursuit onset behavior (at least during the critical transition region of the responses). This strong relationship also gave me confidence that I could use modeling techniques to adequately describe subject responses, and that I could later base credible conclusions on simulations derived from such models.

Table III-2 lists the mean parameter values of the smooth pursuit onset model for each subject, for both leftward and rightward responses. (These correspond to parameters depicted in Fig. II-17.) Also shown in the table for completeness are the lumped results of both rightward and leftward responses, (although these values were never actually used in the subsequent analysis). Notice that the optimal parameter values qualitatively do not deviate to any great degree from the parameter values
Table III-1. Mean correlation statistics describing the similarity between subject response data and model simulation data for the transition region of Smooth Pursuit Onset

<table>
<thead>
<tr>
<th></th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rightward Target Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.996 ±0.001</td>
<td>0.992 ±0.006</td>
<td>0.988 ±0.009</td>
<td>0.989 ±0.005</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>99.15 ±0.20</td>
<td>98.47 ±1.12</td>
<td>97.52 ±1.67</td>
<td>97.82 ±1.06</td>
</tr>
<tr>
<td>n</td>
<td>8</td>
<td>5</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

| **Leftward Target Motion** |           |           |           |           |
| Correlation Coefficient | 0.993 ±0.006 | 0.991 ±0.007 | 0.991 ±0.007 | 0.993 ±0.003 |
| Coefficient of Determination | 98.67 ±1.10   | 98.24 ±1.46  | 98.21 ±1.36  | 98.61 ±0.60  |
| n                    | 5         | 6          | 5          | 4          |
Table III-2. Mean optimal values for the parameters of the Smooth Pursuit Onset model (standard deviations listed in parentheses; time constant and delays are given in seconds)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>A</th>
<th>Tc</th>
<th>Td</th>
<th>P1</th>
<th>D1</th>
<th>D2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subject 1</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Rightward n=8</td>
<td>1.219 (0.126)</td>
<td>0.043 (0.041)</td>
<td>9.664 (3.837)</td>
<td>1.004 (0.020)</td>
<td>0.047 (0.007)</td>
<td>0.036 (0.028)</td>
</tr>
<tr>
<td>Leftward n=5</td>
<td>1.201 (0.162)</td>
<td>0.109 (0.024)</td>
<td>7.873 (2.547)</td>
<td>0.933 (0.023)</td>
<td>0.051 (0.008)</td>
<td>0.030 (0.021)</td>
</tr>
<tr>
<td>Lumped n=13</td>
<td>1.212 (0.141)</td>
<td>0.069 (0.048)</td>
<td>8.975 (3.509)</td>
<td>0.976 (0.040)</td>
<td>0.049 (0.008)</td>
<td>0.034 (0.026)</td>
</tr>
<tr>
<td><strong>Subject 2</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Rightward n=5</td>
<td>1.136 (0.202)</td>
<td>0.031 (0.022)</td>
<td>14.225 (5.069)</td>
<td>1.014 (0.017)</td>
<td>0.052 (0.021)</td>
<td>0.049 (0.015)</td>
</tr>
<tr>
<td>Leftward n=6</td>
<td>0.995 (0.223)</td>
<td>0.025 (0.018)</td>
<td>12.257 (3.816)</td>
<td>1.003 (0.028)</td>
<td>0.062 (0.013)</td>
<td>0.051 (0.023)</td>
</tr>
<tr>
<td>Lumped n=11</td>
<td>1.059 (0.225)</td>
<td>0.028 (0.020)</td>
<td>13.151 (4.537)</td>
<td>1.008 (0.024)</td>
<td>0.057 (0.018)</td>
<td>0.051 (0.019)</td>
</tr>
<tr>
<td><strong>Subject 3</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward n=4</td>
<td>1.006 (0.175)</td>
<td>0.082 (0.045)</td>
<td>11.565 (2.919)</td>
<td>0.980 (0.027)</td>
<td>0.064 (0.012)</td>
<td>0.019 (0.019)</td>
</tr>
<tr>
<td>Leftward n=6</td>
<td>0.923 (0.131)</td>
<td>0.038 (0.037)</td>
<td>7.337 (5.106)</td>
<td>1.004 (0.031)</td>
<td>0.041 (0.020)</td>
<td>0.044 (0.028)</td>
</tr>
<tr>
<td>Lumped n=10</td>
<td>0.956 (0.155)</td>
<td>0.055 (0.045)</td>
<td>9.028 (4.831)</td>
<td>0.995 (0.032)</td>
<td>0.050 (0.021)</td>
<td>0.034 (0.028)</td>
</tr>
<tr>
<td><strong>Subject 4</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward n=5</td>
<td>0.912 (0.097)</td>
<td>0.001 (0.000)</td>
<td>17.058 (3.212)</td>
<td>1.068 (0.048)</td>
<td>0.096 (0.019)</td>
<td>0.073 (0.019)</td>
</tr>
<tr>
<td>Leftward n=4</td>
<td>0.876 (0.167)</td>
<td>0.007 (0.007)</td>
<td>9.710 (7.722)</td>
<td>0.980 (0.019)</td>
<td>0.072 (0.018)</td>
<td>0.065 (0.023)</td>
</tr>
<tr>
<td>Lumped n=9</td>
<td>0.896 (0.134)</td>
<td>0.004 (0.005)</td>
<td>13.792 (6.750)</td>
<td>1.029 (0.058)</td>
<td>0.085 (0.022)</td>
<td>0.059 (0.021)</td>
</tr>
</tbody>
</table>
published by Robinson et al. (1986) for their smooth pursuit model (Fig. II-16). The value for \( \dot{e}_0 \) was generally greater than the published value of 4.0, while \( T_c \) was typically much less than the published value of 0.07; this is consistent with observations I made in our paper describing the overall parameter estimation procedure (Huebner et al. 1990).

Figure III-2 shows a typical Smooth Pursuit Offset response from Subject 1 along with the corresponding smooth pursuit model simulation (again using the optimal parameter values calculated for that response). Although this was the first attempt we know of to model the phenomenon, it appears as though the suggestion of Robinson et al. (1986) to use a simple first-order lag element is generally satisfactory. Table III-3 gives the average correlation coefficient and coefficient of determination values which compare the similarity between subjects' responses during the offset of smooth pursuit with the corresponding model simulations, for all four subjects and for both rightward and leftward motion. Note from this table that all conditions show that the degree of linear association between the model simulation and the subject data is at least 97.7%. This indicates that simulations of smooth pursuit offset adequately describe actual pursuit offset behavior, at least during the critical transition region of the responses.

The mean parameter values of the smooth pursuit offset model for each subject, for both leftward and rightward responses, are given in Table III-4. (These correspond to parameters depicted in Fig. II-18.) Again, also shown in the table for completeness are the lumped results of both
Fig. III-2. Typical response demonstrating the offset of smooth pursuit. Also shown is the corresponding model simulation derived using optimal parameter values calculated for this response. Notice that a simple exponential model for smooth pursuit offset does a reasonable job of representing the data.
Table III-3. Mean correlation statistics describing the similarity between subject response data and model simulation data for the transition region of Smooth Pursuit Offset

<table>
<thead>
<tr>
<th></th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rightward</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Target Motion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.991 ±0.004</td>
<td>0.993 ±0.002</td>
<td>0.985 ±0.003</td>
<td>0.989 ±0.004</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>98.29 ±0.76</td>
<td>98.67 ±0.34</td>
<td>98.90 ±1.67</td>
<td>97.87 ±0.71</td>
</tr>
<tr>
<td><strong>Leftward</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Target Motion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.992 ±0.003</td>
<td>0.993 ±0.003</td>
<td>0.988 ±0.003</td>
<td>0.989 ±0.008</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>98.43 ±0.53</td>
<td>98.64 ±0.55</td>
<td>97.69 ±0.54</td>
<td>97.76 ±1.56</td>
</tr>
<tr>
<td>n</td>
<td>8</td>
<td>9</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>
Table III-4. Mean optimal values for the parameters of the Smooth Pursuit Offset model (standard deviations listed in parentheses; values are given in seconds)

<table>
<thead>
<tr>
<th>Parameter:</th>
<th>tps</th>
<th>Tps</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subject 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward</td>
<td>0.055</td>
<td>0.081</td>
</tr>
<tr>
<td>n=8</td>
<td>(0.015)</td>
<td>(0.018)</td>
</tr>
<tr>
<td>Leftward</td>
<td>0.060</td>
<td>0.050</td>
</tr>
<tr>
<td>n=8</td>
<td>(0.012)</td>
<td>(0.022)</td>
</tr>
<tr>
<td>Lumped</td>
<td>0.058</td>
<td>0.065</td>
</tr>
<tr>
<td>n=16</td>
<td>(0.014)</td>
<td>(0.025)</td>
</tr>
<tr>
<td><strong>Subject 2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward</td>
<td>0.083</td>
<td>0.088</td>
</tr>
<tr>
<td>n=9</td>
<td>(0.018)</td>
<td>(0.018)</td>
</tr>
<tr>
<td>Leftward</td>
<td>0.061</td>
<td>0.067</td>
</tr>
<tr>
<td>n=9</td>
<td>(0.011)</td>
<td>(0.015)</td>
</tr>
<tr>
<td>Lumped</td>
<td>0.072</td>
<td>0.078</td>
</tr>
<tr>
<td>n=18</td>
<td>(0.018)</td>
<td>(0.020)</td>
</tr>
<tr>
<td><strong>Subject 3</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward</td>
<td>0.048</td>
<td>0.104</td>
</tr>
<tr>
<td>n=7</td>
<td>(0.012)</td>
<td>(0.015)</td>
</tr>
<tr>
<td>Leftward</td>
<td>0.042</td>
<td>0.062</td>
</tr>
<tr>
<td>n=6</td>
<td>(0.012)</td>
<td>(0.012)</td>
</tr>
<tr>
<td>Lumped</td>
<td>0.045</td>
<td>0.085</td>
</tr>
<tr>
<td>n=13</td>
<td>(0.012)</td>
<td>(0.025)</td>
</tr>
<tr>
<td><strong>Subject 4</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward</td>
<td>0.064</td>
<td>0.058</td>
</tr>
<tr>
<td>n=7</td>
<td>(0.017)</td>
<td>(0.014)</td>
</tr>
<tr>
<td>Leftward</td>
<td>0.059</td>
<td>0.089</td>
</tr>
<tr>
<td>n=5</td>
<td>(0.009)</td>
<td>(0.010)</td>
</tr>
<tr>
<td>Lumped</td>
<td>0.062</td>
<td>0.071</td>
</tr>
<tr>
<td>n=12</td>
<td>(0.015)</td>
<td>(0.020)</td>
</tr>
</tbody>
</table>
rightward and leftward responses.

Finally, Fig. III-3 presents a typical response to VOR Onset and Offset from Subject 2 (the same response as that shown in Fig. II-20) as well as the corresponding model simulation (again using the optimal parameter values calculated for that response). Notice that the revised VOR model, incorporating a variable gain element, can adequately describe the actual VOR data during both the onset and offset of head motion. This can be verified quantitatively by observing the average correlation coefficient and coefficient of determination values in Table III-5. These values compare the similarity between subjects' responses during VOR Onset and VOR Offset with the corresponding model simulations, for all four subjects and for both rightward and leftward motion. Note from this table that all conditions show that the degree of linear association between the model simulation and the subject data for VOR Onset is at least 95.0% (and, if Subject 4-Leftward values are excluded, at least 97.4%). For VOR Offset, the degree of linear association is at least 97.7% (and again, if Subject 4-Leftward values are excluded, at least 99.0%). This indicates that simulations of both VOR Onset and Offset adequately describe actual VOR response behavior, at least during the critical transition region of the responses.

The average optimal VOR parameter values for each subject, for both leftward and rightward responses, are listed in Table III-6 along with the lumped results of both rightward and leftward responses together. Notice first that the VOR latencies are generally very short, much less than
Fig. III-3. Typical response of the VOR to the onset and offset of head motion. Shown with the eye and gaze velocity waveforms are the corresponding model simulations derived using optimal parameter values calculated for this response. (The eye and gaze velocity responses were shown with their head movement stimulus in Fig. II-20, and, to be consistent with this figure, the eye and corresponding model velocity waveforms have again been inverted.) Notice that the model reasonably reflects the actual eye movement activity for both the onset and offset of head motion.
Table III-5. Mean correlation statistics describing the similarity between subject response data and model simulation data for the transition regions of VOR Onset and VOR Offset

<table>
<thead>
<tr>
<th>Head Motion: Rightward</th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VOR ONSET</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.994 ± 0.003</td>
<td>0.993 ± 0.005</td>
<td>0.990 ± 0.009</td>
<td>0.991 ± 0.005</td>
</tr>
<tr>
<td>Coeff. of Determination</td>
<td>98.75 ± 0.70</td>
<td>98.51 ± 0.94</td>
<td>98.00 ± 1.71</td>
<td>98.28 ± 1.04</td>
</tr>
<tr>
<td>n</td>
<td>12</td>
<td>7</td>
<td>12</td>
<td>8</td>
</tr>
<tr>
<td><strong>Leftward</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.993 ± 0.005</td>
<td>0.987 ± 0.009</td>
<td>0.992 ± 0.007</td>
<td>0.975 ± 0.014</td>
</tr>
<tr>
<td>Coeff. of Determination</td>
<td>98.64 ± 0.96</td>
<td>97.43 ± 1.84</td>
<td>98.49 ± 1.41</td>
<td>94.98 ± 2.72</td>
</tr>
<tr>
<td>n</td>
<td>10</td>
<td>11</td>
<td>5</td>
<td>7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Head Motion: Rightward</th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VOR OFFSET</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.997 ± 0.004</td>
<td>0.996 ± 0.003</td>
<td>0.995 ± 0.005</td>
<td>0.996 ± 0.003</td>
</tr>
<tr>
<td>Coeff. of Determination</td>
<td>99.35 ± 0.72</td>
<td>99.17 ± 0.55</td>
<td>98.96 ± 1.06</td>
<td>99.17 ± 0.61</td>
</tr>
<tr>
<td>n</td>
<td>12</td>
<td>7</td>
<td>12</td>
<td>8</td>
</tr>
<tr>
<td><strong>Leftward</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.996 ± 0.003</td>
<td>0.996 ± 0.003</td>
<td>0.997 ± 0.001</td>
<td>0.989 ± 0.006</td>
</tr>
<tr>
<td>Coeff. of Determination</td>
<td>99.13 ± 0.68</td>
<td>99.16 ± 0.63</td>
<td>99.46 ± 0.20</td>
<td>97.73 ± 1.27</td>
</tr>
<tr>
<td>n</td>
<td>10</td>
<td>11</td>
<td>5</td>
<td>7</td>
</tr>
</tbody>
</table>
Table III-6. Mean optimal values for the parameters of the variable-gain VOR model (standard deviations listed in parentheses; time constant and delays are given in seconds)

<table>
<thead>
<tr>
<th>Parameter:</th>
<th>( \text{Time} )</th>
<th>( \Delta v )</th>
<th>( \Delta \alpha )</th>
<th>( T_{\text{time}} )</th>
<th>( T_{\text{delay}} )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subject 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward</td>
<td>0.0001</td>
<td>0.814</td>
<td>0.985</td>
<td>0.277</td>
<td>0.082</td>
</tr>
<tr>
<td>n=12</td>
<td>(0.0002)</td>
<td>(0.064)</td>
<td>(0.024)</td>
<td>(0.420)</td>
<td>(0.065)</td>
</tr>
<tr>
<td>Leftward</td>
<td>0.0008</td>
<td>0.769</td>
<td>0.960</td>
<td>0.232</td>
<td>0.068</td>
</tr>
<tr>
<td>n=10</td>
<td>(0.0010)</td>
<td>(0.128)</td>
<td>(0.024)</td>
<td>(0.303)</td>
<td>(0.071)</td>
</tr>
<tr>
<td>Lumped</td>
<td>0.0004</td>
<td>0.793</td>
<td>0.973</td>
<td>0.256</td>
<td>0.076</td>
</tr>
<tr>
<td>n=22</td>
<td>(0.0008)</td>
<td>(0.101)</td>
<td>(0.027)</td>
<td>(0.372)</td>
<td>(0.068)</td>
</tr>
<tr>
<td><strong>Subject 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward</td>
<td>0.0000</td>
<td>0.779</td>
<td>0.960</td>
<td>0.468</td>
<td>0.072</td>
</tr>
<tr>
<td>n=7</td>
<td>(0.0000)</td>
<td>(0.031)</td>
<td>(0.035)</td>
<td>(0.235)</td>
<td>(0.040)</td>
</tr>
<tr>
<td>Leftward</td>
<td>0.0005</td>
<td>0.685</td>
<td>0.914</td>
<td>0.081</td>
<td>0.094</td>
</tr>
<tr>
<td>n=11</td>
<td>(0.0008)</td>
<td>(0.098)</td>
<td>(0.026)</td>
<td>(0.112)</td>
<td>(0.036)</td>
</tr>
<tr>
<td>Lumped</td>
<td>0.0003</td>
<td>0.721</td>
<td>0.932</td>
<td>0.231</td>
<td>0.086</td>
</tr>
<tr>
<td>n=18</td>
<td>(0.0006)</td>
<td>(0.092)</td>
<td>(0.038)</td>
<td>(0.254)</td>
<td>(0.039)</td>
</tr>
<tr>
<td><strong>Subject 3</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward</td>
<td>0.0000</td>
<td>0.771</td>
<td>0.990</td>
<td>0.358</td>
<td>0.079</td>
</tr>
<tr>
<td>n=12</td>
<td>(0.0000)</td>
<td>(0.057)</td>
<td>(0.019)</td>
<td>(0.217)</td>
<td>(0.070)</td>
</tr>
<tr>
<td>Leftward</td>
<td>0.0014</td>
<td>0.863</td>
<td>0.977</td>
<td>0.175</td>
<td>0.033</td>
</tr>
<tr>
<td>n=5</td>
<td>(0.0010)</td>
<td>(0.069)</td>
<td>(0.022)</td>
<td>(0.166)</td>
<td>(0.034)</td>
</tr>
<tr>
<td>Lumped</td>
<td>0.0004</td>
<td>0.798</td>
<td>0.986</td>
<td>0.304</td>
<td>0.066</td>
</tr>
<tr>
<td>n=17</td>
<td>(0.0008)</td>
<td>(0.074)</td>
<td>(0.021)</td>
<td>(0.220)</td>
<td>(0.065)</td>
</tr>
<tr>
<td><strong>Subject 4</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rightward</td>
<td>0.0001</td>
<td>0.754</td>
<td>0.916</td>
<td>0.283</td>
<td>0.093</td>
</tr>
<tr>
<td>n=8</td>
<td>(0.0003)</td>
<td>(0.062)</td>
<td>(0.041)</td>
<td>(0.176)</td>
<td>(0.050)</td>
</tr>
<tr>
<td>Leftward</td>
<td>0.0002</td>
<td>0.735</td>
<td>0.963</td>
<td>0.100</td>
<td>0.094</td>
</tr>
<tr>
<td>n=7</td>
<td>(0.0003)</td>
<td>(0.052)</td>
<td>(0.055)</td>
<td>(0.047)</td>
<td>(0.051)</td>
</tr>
<tr>
<td>Lumped</td>
<td>0.0001</td>
<td>0.746</td>
<td>0.938</td>
<td>0.198</td>
<td>0.093</td>
</tr>
<tr>
<td>n=15</td>
<td>(0.0003)</td>
<td>(0.058)</td>
<td>(0.054)</td>
<td>(0.161)</td>
<td>(0.050)</td>
</tr>
</tbody>
</table>
the observed maximum value of 15 msec. One possible explanation for this is that lag elements in the model may tend to stretch the onset of a response so that an observed latency of some time may actually be implemented in a model by a much shorter pure delay in series with a lag element. Also of interest are the resting and active VOR gain values. The resting gain values are generally 0.2 less than the active values. These values, ranging from 0.68 to 0.86 for the resting gains and from 0.91 to 0.99 for the active gains, are not inconsistent with the values reported by Barr et al. (1976). Finally, the values of the time constant that govern the transition between resting and active VOR gain values, $T_{AVoR}$ (see Fig. II-21), show about a five-fold variation in value (from 0.081 to 0.468). At first this variability surprised me. However, consider that the VOR signal is tempered by the signal from "fixation" (Smooth Pursuit Offset). This "fixation" signal tends to bring the eye velocity towards the apparent target velocity at the same time VOR gain is changing from its resting level to its active level. By performing some auxiliary model simulations using the model of visually-enhanced VOR, I found that no matter what the value of $T_{AVoR}$ over the range 0.010 to 1.000, its overall effect seems to only last, at most, 300 msec because of the contribution from the "fixation" system. However, I also found that this large range of values is necessary to accurately characterize the variability found in subject responses. Short time constants (small values of $T_{AVoR}$) reflect responses that rise quickly to the full-scale level and perhaps even overshoot. On the other hand, long time constants (large values of $T_{AVoR}$) reflect responses that rise monotonically towards the full-scale level and, with the help of the "fixation" system, reach it after about 300 msec.
B. Comparison of Model Simulations with
Subject Combined Eye-Head Tracking Data

The means of testing the hypotheses posed by this research involved comparing actual subject response data with simulations generated using a model structured as the superposition of two component models. This section details the results of these comparisons for two revealing stimulus types: the Chair Brake stimulus and the Delayed Target Onset stimulus.

1. The Chair Brake Stimulus

The purpose of the Chair Brake stimulus was to isolate how the ocular motor system changes from performing combined eye-head tracking to performing smooth pursuit alone. The stimulus achieved this by initiating CEHT and then abruptly terminating head rotation, leaving the moving target as the sole input to the ocular motor system. Since the latency of smooth pursuit onset is long (greater than 75 msec) relative to the cessation of head motion (evident in less than 10 msec), then the brake in head motion will unmask whatever contribution smooth pursuit provides to CEHT. Thus, I am mainly interested in comparing the eye movement response when the head is stopped during CEHT with what the superposition model predicts the eye will do during this transition.
Figure III-4 presents a typical set of plots of target, head and eye waveforms for an entire rightward Chair Brake stimulus. To compare how the eye and model each respond to this type of stimulus, Fig. III-5 shows some representative plots of responses to the Chair Brake stimulus along with the corresponding model simulations for Subject 1. For convenience in interpretation, gaze velocity is plotted in addition to eye velocity. Also, to focus on the sole effects of the chair brake, the plots range from 1.00 sec before until 1.25 sec after the chair began to stop.

Several characteristics about the plots in Fig. III-5 are worth mentioning. First, it is evident in all plots that when the head is stopped (at the 2.0 sec point in the responses), both the eye and the model begin to move almost immediately (after a short delay comparable to the latency of the VOR). Next, note that unlike the offset of visually-guided VOR, gaze is perturbed from its established steady-state value when the head is stopped. Also, many responses showed virtually no ringing in the response after the brake (Fig. III-5a), while some other responses displayed slight degrees of ringing (Fig. III-5b), and still others showed appreciable amounts of sustained ringing (Fig III-5c). Generally, however, the simulated model response values tend to change in synchrony with the corresponding eye movement response values.

These results were obtained during passive head rotations (in which the head was constrained to rotate along with the vestibular chair). The observed effect of an immediate change in eye velocity when the chair was braked is similar to that described by Lanman et al. (1978) for active (head
Fig. III-4. Typical eye velocity response and the corresponding target and head stimulus waveforms for a rightward Chair Brake stimulus. Notice that when the head is stopped (at the 2.0 sec point in the response), the eye responds almost immediately to begin tracking the still-moving target.
Fig. III-5. Some representative plots of responses to the Chair Brake stimulus along with their corresponding model simulations. For convenience in interpretation, gaze velocity is plotted in addition to eye velocity, and the plots focus on the responses in the vicinity of the chair brake. Various degrees of ringing can be seen in the post-brake response waveforms: 
a) virtually no ringing; next page: b) slight ringing; and c) appreciable ringing.
Fig. III-5, continued.
free) tracking in monkeys (wherein the head was braked directly using electromechanical means). However, unlike my results, they reported no detectable change in gaze velocity subsequent to the brake. (Actually, Lanman et al. based their interpretation on eye position data; perhaps gaze perturbations would have been more apparent in the corresponding velocity waveforms. This minor difference between our results may also be due to species variations. In addition, their monkeys were performing active head rotations, while our subjects were passively rotated.)

a. Residuals

By plotting average residuals (see Section II.C.3.a), we can quickly get a descriptive idea of where and to what degree the model simulations fail to recreate the measured subject responses to the Chair Brake. Figure III-6 plots the average residuals from the Chair Brake trials for each of the four subjects, over the same time range as the plots in Fig. III-5. It is evident from these residual plots (and the residual feature which consistently appears to begin at 2.0 sec in the plots) that the gaze perturbations shown in Fig. III-5 keep the model from accurately describing the actual eye movement profiles over approximately the first 200 msec after the chair began to stop. Note however that the relative magnitude of even this largest feature of the residuals is generally much smaller than the overall magnitude of the response (which for these experiments is about 15 deg/sec). Also note that some of the residuals contain a low-level sinusoidal component, indicating that the model does not account for
Fig. III-6. Average residuals from the Chair Brake trials for each of the four subjects, over the same time range as the plots in Fig. III-5. See text for details.
some occasionally observed ringing in the responses. These observations indicate that the model generally does reflect the corresponding eye movement activity, although some refinements in the model may improve its performance and reduce the magnitude of some of the features in its residuals.

One interesting observation is that Subjects 1, 2, and 4 all tend to exhibit post-brake ringing in the residuals corresponding to leftward motion but not in the residuals of rightward motion. The opposite seems to be true for Subject 3. Although it may be coincidental, Subjects 1, 2, and 4 are all left-handed (sinistral) while Subject 3 is right-handed (dextral).

b. Correlations

In an attempt to quantify just how well the model simulations predict actual eye movement responses, I employed a correlation technique (see Section II.C.3.b). Figure III-7 shows the transition region of a sample Chair Brake response (the first 100 msec after the chair began to stop) over which the correlation coefficient values were calculated. While Fig. III-7 demonstrates how eye and model velocity change with respect to time inside this region, Fig. III-8 uses the same data to depict how model velocity changes with respect to eye velocity. If the simulated model velocity waveform exactly predicted the measured eye velocity values, then we would see a straight line with a slope of 1.0 in Fig. III-8, and the calculated correlation coefficient and coefficient of determination would
Fig. III-7. Example of how eye and model velocity change with respect to time inside the *transition region* of a Chair Brake response (the first 100 msec after the chair began to stop). The correlation coefficients were calculated using the values in this region.
Fig. III-8. The same data from Fig. III-7 depicting how model velocity changes with respect to eye velocity. If model velocity *exactly* predicted eye velocity, this plot would show a line with a slope of 1.0 passing through the origin.
be 1.0 and 100%, respectively. The degrees to which these coefficients deviate from such ideal values gives us an indication of the relative similarity (or difference) between the measured eye velocity values and the corresponding simulated model velocity values. For the data depicted in Figs. III-7 and III-8, the correlation coefficient was 0.975 while the coefficient of determination was 95.1%, indicating a rather strong similarity.

Table III-7 lists, for all four subjects, the average correlation coefficients and coefficients of determination which compare the similarity of the immediate subject response to the chair brake with the corresponding model simulations for both rightward and leftward motion. Note from this table that all conditions show that the degree of linear relationship between the model simulation and the subject data is at least 90%, and most show about 98% similarity.

To contrast these results with those which may be obtained from a model derived using an alternative hypothesis of how the VOR might be cancelled during CEHT (see Section II.C.3.b), I recalculated the correlation coefficients and coefficients of determination after shifting the model waveform 75 msec relative to the actual eye data. (This 75 msec shift corresponds to the minimum latency expected before a normal smooth pursuit onset response can be elicited; it is included to try to align the model simulation with data that would result if the post-brake response required smooth pursuit to begin from a quiescent state.) Figure III-9 shows how the eye and (shifted) model velocity waveforms change with
Table III-7. Mean correlation statistics describing the similarity between subject response data and model simulation data for the transition region of the Chair Brake paradigm

<table>
<thead>
<tr>
<th></th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rightward Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.988 ±0.007</td>
<td>0.980 ±0.017</td>
<td>0.995 ±0.006</td>
<td>0.984 ±0.009</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>97.61 ±1.35</td>
<td>96.06 ±3.29</td>
<td>98.96 ±1.11</td>
<td>96.83 ±1.82</td>
</tr>
<tr>
<td>n</td>
<td>10</td>
<td>10</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td><strong>Leftward Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.992 ±0.006</td>
<td>0.993 ±0.004</td>
<td>0.987 ±0.006</td>
<td>0.952 ±0.048</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>98.49 ±1.14</td>
<td>98.65 ±0.86</td>
<td>97.39 ±1.19</td>
<td>90.92 ±8.99</td>
</tr>
<tr>
<td>n</td>
<td>10</td>
<td>9</td>
<td>12</td>
<td>7</td>
</tr>
</tbody>
</table>
Fig. III-9. Example of how the eye and (shifted) model velocity waveforms may change with respect to time in the revised transition region of a Chair Brake response (the first 175 msec after the chair began to stop) using an alternative hypothesis of how the VOR might be cancelled during CEHT. See text for details.
respect to time in the new transition region (the first 175 msec after the
chair began to stop), while Fig. III-10 presents how (shifted) model velocity
changes with respect to eye velocity in this region. From the data in Fig.
III-9 it seems clear that a model which waits (at least) 75 msec for smooth
pursuit to become active after the chair is braked does an unsatisfactory job
of predicting the course of the actual eye movement data. This is also
shown by Fig. III-10 in which the association between model and eye
velocity seems far from linear. To quantify this association, the correlation
coefficient for the data in Fig. III-10 is 0.672, while the corresponding
coefficient of determination is 45.2%. (This example actually shows one of
the stronger associations.)

The average correlation coefficient and coefficient of determination
values, recalculated after shifting the model waveforms, are shown in
Table III-8. These values are generally much lower than those calculated
for the unshifted model waveforms: the degrees of linear relationship
between model and data range from about 45% to as low as about 20%.
This demonstrates that a much weaker relationship exists between the
subject and model data when the model data are shifted (to try to emulate
a hypothetical model which might be constructed assuming an alternative
hypothesis). In fact, the shifted correlation coefficient and coefficient of
determination values are significantly less than the corresponding
unshifted values, with a maximum significance of $p \leq 0.0004$. This
supports the overall hypothesis that cancellation of the VOR during CEHT
is performed by simply summing the VOR signal with a signal from the
smooth pursuit system.
Fig. III-10. The same data from Fig. III-9 depicting how (shifted) model velocity changes with respect to eye velocity. Again, if model velocity \textit{exactly} predicted eye velocity, this plot would show a line with a slope of 1.0 passing through the origin.
Table III-8. Mean correlation statistics describing the similarity between subject response data and shifted model simulation data for the transition region of the Chair Brake paradigm

<table>
<thead>
<tr>
<th></th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rightward Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.668 ±0.057</td>
<td>0.642 ±0.085</td>
<td>0.606 ±0.064</td>
<td>0.524 ±0.097</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>44.99 ±7.26</td>
<td>41.93 ±10.69</td>
<td>37.16 ±7.50</td>
<td>28.43 ±9.94</td>
</tr>
<tr>
<td>n</td>
<td>10</td>
<td>10</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td><strong>Leftward Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.439 ±0.107</td>
<td>0.513 ±0.128</td>
<td>0.461 ±0.081</td>
<td>0.432 ±0.142</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>20.39 ±9.43</td>
<td>28.01 ±11.31</td>
<td>21.93 ±7.81</td>
<td>20.70 ±12.11</td>
</tr>
<tr>
<td>n</td>
<td>10</td>
<td>9</td>
<td>12</td>
<td>7</td>
</tr>
</tbody>
</table>
2. The Delayed Target Onset Stimulus

The Delayed Target Onset stimulus was used to isolate how the ocular motor system changes from performing visually-enhanced VOR to performing combined eye-head tracking. The stimulus achieved this by initiating synchronous target motion with an already-moving head. Does the morphology of the eye movement response when initiating CEHT resemble the onset of smooth pursuit, or does the response appear appreciably different than smooth pursuit, suggesting that the VOR is cancelled by a different mechanism? Thus, I am mainly interested in seeing how the eye movement response changes when the target begins to move along with the head as well as what the superposition model predicts the eye will do during this transition.

Figure III-11 presents a typical set of plots of Target, Head and Eye waveforms for an entire rightward Delayed Target Onset stimulus. (Note: this response contains artifacts in the first few hundred milliseconds because it was collected when chair acceleration was still maximal—see Section II.A.1.a.ii). To compare how the eye and model each respond to this type of stimulus, Fig. III-12 shows some representative plots of responses to the leftward Delayed Target Onset stimulus along with the corresponding model simulations for Subject 2. These plots range from 1.00 sec before until 1.5 sec after the onset of target motion to focus on the sole effects of the transition form VOR to CEHT.

A few characteristics about the plots in Fig. III-12 are worth
Fig. III-11. Typical eye velocity response and the corresponding target and head stimulus waveforms for a rightward Delayed Target Onset stimulus. Notice that after the target begins to move (at the 2.0 sec point in the response), the eye requires about 160 msec before it responds to the onset of target motion. Also, notice that the ringing in the response after the onset of target motion resembles that seen in the onset of smooth pursuit.
Fig. III-12. Some representative plots of responses to the Delayed Target Onset stimulus along with their corresponding model simulations. For convenience in interpretation, the plots focus on the responses in the vicinity of the onset in target motion. Various degrees of ringing can be seen in the post-brake response waveforms: a) some ringing, and b) little ringing.
mentioning. First, it is evident in both plots that when the target begins to
move (at the 2.0 sec point in the responses), both the eye and the model
begin to move only after a considerable amount of time (approximately
160 msec). Also, many responses showed some degree of ringing in the
response after the onset of target motion (Fig. III-12a), while some other
responses displayed very little ringing (Fig. III-12b). Again, like the Chair
Brake responses, the model waveforms generally tend to move in
synchrony with the corresponding eye movement responses.

a. Residuals

Like the analysis for the Chair Brake, I also calculated the residuals
from trials using the Delayed Target Onset stimulus. Figure III-13 plots the
average residuals from the Delayed Target Onset trials, for each of the four
subjects, over the same time range as the plots in Fig. III-12. Of great
importance in this research is the magnitude of the residuals immediately
after the onset of target motion (which occurs at the 2.0 sec point in Figs.
III-12 and III-13). Notice that for all subjects the residuals remain near zero
(i.e., they remain at the same level as that seen before target motion) for at
least 100 msec after the target begins to move. This suggests that the model
does a satisfactory job of predicting the actual eye movement response
during this critical time.

After this initial interval (about 2.1 sec into the response), most
residual plots show various peaks. The presence of these peaks can be
Fig. III-13. Average residuals from the Delayed Target Onset trials for each of the four subjects, over the same time range as the plots in Fig. III-12. See text for details.
attributed to the variability of subject response latencies relative to the average model latency implemented for the simulations. That is, if a model simulation exactly resembled the corresponding subject data except that a slight difference in latency shifted one waveform with respect to the other, then this difference in latency would be manifested as a peak in the residual plot. Greater differences in response latency would result in wider and higher amplitude peaks in the residual waveform. Realize however that the relative magnitude of even the largest of these peaks in the residuals is generally much smaller than the overall magnitude of the response (which again for these experiments is about 15 deg/sec). Also, in addition to the peaks, note that some of the residuals contain a low-level oscillatory component, indicating that the model may provide ringing which is not always seen as prevalently in the responses.

Despite these observed features in the residuals, overall analysis of the residuals indicates that the model generally does reflect the corresponding eye movement activity. This becomes clearer when one notes the low-level of the residuals during the first 100 msec after the onset of target motion, and when one understands that the major features in the residual plots stem from the natural variability of smooth pursuit latency.

b. Correlations

To quantitatively test the similarity between model and measured
data waveforms for Delayed Target Onset, I used the same correlation technique as that employed above for the Chair Brake stimulus (see Sections II.C.3.b and III.B.1.b). Figure III-14 shows the transition region of a sample Delayed Target Onset response (the first 400 msec after the target began to move) over which the correlation coefficient values were calculated. While Fig. III-14 demonstrates how eye and model velocity change with respect to time inside this region, Fig. III-15 uses the same data to depict how model velocity changes with respect to eye velocity. If the simulated model velocity waveform exactly predicted the measured eye velocity values, then, as before, we would see a straight line with a slope of 1.0 in Fig. III-15. For the data depicted in Figs. III-14 and III-15, the correlation coefficient was 0.994 while the coefficient of determination was 98.8%, indicating a rather strong similarity.

Table III-9 lists, for all four subjects, the average correlation coefficients and coefficients of determination which compare the similarity of the subject response to the onset of target motion with the corresponding model simulations for both rightward and leftward motion. Note from this table that all conditions show that the degree of linear relationship between the model simulation and the subject data is at least 90%, and most show about 95% similarity.

To contrast these results with those which may be obtained from a model derived using an alternative hypothesis of how the VOR is cancelled during CEHT (see Section II.C.3.b), I again recalculated the correlation coefficients and coefficients of determination upon fixing the
Fig. III-14. Example of how eye and model velocity change with respect to time inside the transition region of a Delayed Target Onset response (the first 350-400 msec after the target began to move). The correlation coefficients were calculated using the values in this region.
Fig. III-15. The same data from Fig. III-14 depicting how model velocity changes with respect to eye velocity. If model velocity exactly predicted eye velocity, this plot would show a line with a slope of 1.0 passing through the origin.
Table III-9. Mean correlation statistics describing the similarity between subject response data and model simulation data for the transition region of the Delayed Target Onset paradigm

<table>
<thead>
<tr>
<th></th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rightward Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.950 ±0.020</td>
<td>0.970 ±0.013</td>
<td>0.961 ±0.023</td>
<td>0.988 ±0.005</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>90.37 ±3.74</td>
<td>94.08 ±2.55</td>
<td>92.34 ±4.32</td>
<td>97.69 ±0.92</td>
</tr>
<tr>
<td>( n )</td>
<td>11</td>
<td>9</td>
<td>11</td>
<td>10</td>
</tr>
</tbody>
</table>

| **Leftward Motion** |           |           |           |           |
| Correlation Coefficient | 0.965 ±0.027 | 0.985 ±0.005 | 0.987 ±0.004 | 0.978 ±0.025 |
| Coefficient of Determination | 93.10 ±5.19 | 96.97 ±0.92 | 97.34 ±0.73 | 95.79 ±4.75 |
| \( n \) | 12 | 10 | 9 | 7 |
onset of the model response to be 30 msec after the onset of target motion. (This 30 msec corresponds to the maximum latency expected by Cullen et al. (in press) before VOR suppression is observed using their proposed accessory pathway.) Figure III-16 shows how the eye and (shifted) model velocity waveforms change with respect to time in the transition region, while Fig. III-17 presents how (shifted) model velocity changes with respect to eye velocity in this region. From the data in Fig. III-16 it seems clear that a model which does not allow sufficient time for smooth pursuit to become active after the onset of target motion does an unsatisfactory job of predicting the course of the actual eye movement data. This is also shown by Fig. III-17 in which the association between model and eye velocity again seems far from linear. To quantify this association, the correlation coefficient for the data in Fig. III-17 is 0.770, while the corresponding coefficient of determination is 59.3%.

The average correlation coefficient and coefficient of determination values, recalculated after shifting the model waveforms, are shown in Table III-10. These values are generally much lower than those calculated for the unshifted model waveforms: the degrees of linear relationship between model and data range from about 83% to as low as about 54%. This demonstrates that a much weaker relationship exists between the subject and model data when the model data are shifted (to try to emulate a hypothetical model which might be constructed assuming an alternative hypothesis). In fact, the shifted correlation coefficient and coefficient of determination values are significantly less than the corresponding unshifted values for Delayed Target Onset: the maximum significance
Fig. III-16. Example of how the eye and (shifted) model velocity waveforms may change with respect to time in the transition region of a Delayed Target Onset response using an alternative hypothesis of how the VOR might be cancelled during CEHT. Here, the model response was shifted so that its onset would occur 30 msec after the onset of target motion. See text for details.
Fig. III-17. The same data from Fig. III-16 depicting how (shifted) model velocity changes with respect to eye velocity. Again, if model velocity exactly predicted eye velocity, this plot would show a line with a slope of 1.0 passing through the origin.
Table III-10. Mean correlation statistics describing the similarity between subject response data and shifted model simulation data for the transition region of the Delayed Target Onset paradigm

<table>
<thead>
<tr>
<th></th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rightward Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.813 ±0.052</td>
<td>0.781 ±0.060</td>
<td>0.909 ±0.027</td>
<td>0.795 ±0.038</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>56.45 ±8.39</td>
<td>61.38 ±8.87</td>
<td>82.77 ±4.97</td>
<td>63.37 ±6.24</td>
</tr>
<tr>
<td>n</td>
<td>11</td>
<td>9</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td><strong>Leftward Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.844 ±0.053</td>
<td>0.737 ±0.064</td>
<td>0.788 ±0.041</td>
<td>0.759 ±0.063</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>71.51 ±8.99</td>
<td>54.70 ±9.23</td>
<td>62.24 ±6.32</td>
<td>58.05 ±9.27</td>
</tr>
<tr>
<td>n</td>
<td>12</td>
<td>10</td>
<td>9</td>
<td>7</td>
</tr>
</tbody>
</table>
level for these data is $p \leq 0.007$. (In fact, if the rightward trials for Subject 3 are excluded, the maximum significance level drops to $p \leq 0.0006$.) Again, this supports the overall hypothesis that cancellation of the VOR during CEHT is performed by simply summing the VOR signal with a signal from the smooth pursuit system.

c. Latencies and Average Accelerations

The evidence presented from the residuals and correlation statistics suggests that I do not have sufficient cause to reject the hypothesis that the smooth pursuit system is responsible for cancelling the VOR during combined eye-head tracking, and that this cancellation occurs as a simple summation of these two signals. If the smooth pursuit system is in fact responsible for cancelling the VOR during CEHT, how similar is the onset of CEHT during the Delayed Target Onset paradigm to the onset of smooth pursuit, performed with the head stationary? To examine this question, I determined the onset times of the eye and model responses and compared them with the onset time of target motion, from which I calculated the eye and model response latencies as well as the relative latency difference. Because the model simulations were based on smooth pursuit characteristics, any observed differences could be attributed to influences of head motion. Table III-11 lists the average eye latencies, model latencies, and the average differences between them for both rightward and leftward motion in all subjects. Notice that in all cases, the latency during CEHT is less than during smooth pursuit. Although many of these differences are
Table III-1. Average eye and model latency data (in seconds) from responses to the Delayed Target Onset paradigm

<table>
<thead>
<tr>
<th></th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rightward Motion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eye Latency</td>
<td>0.137 ±0.008</td>
<td>0.134 ±0.017</td>
<td>0.119 ±0.008</td>
<td>0.159 ±0.011</td>
</tr>
<tr>
<td>Model Latency</td>
<td>0.152 ±0.007</td>
<td>0.160 ±0.011</td>
<td>0.145 ±0.005</td>
<td>0.165 ±0.009</td>
</tr>
<tr>
<td>Difference</td>
<td>-0.015 ±0.008</td>
<td>-0.026 ±0.016</td>
<td>-0.026 ±0.011</td>
<td>-0.005 ±0.004</td>
</tr>
<tr>
<td>p Value</td>
<td>0.0001</td>
<td>0.0013</td>
<td>0.0000</td>
<td>0.0025</td>
</tr>
</tbody>
</table>

|                |               |               |               |               |
| **Leftward Motion** |             |               |               |               |
| Eye Latency    | 0.130 ±0.006  | 0.150 ±0.013  | 0.146 ±0.015  | 0.155 ±0.014  |
| Model Latency  | 0.150 ±0.010  | 0.161 ±0.011  | 0.151 ±0.015  | 0.160 ±0.009  |
| Difference     | -0.020 ±0.011 | -0.011 ±0.013 | -0.006 ±0.004 | -0.005 ±0.008 |
| p Value        | 0.0000        | 0.0318        | 0.0012        | 0.2297        |
quite small, only the leftward response for Subject 4 is not significantly different \( (p < 0.032 \text{ for all except Subject 4-leftward, where } p = 0.230) \).

I also compared the average accelerations of the eye and model responses for the main transition phase of the Delayed Target Onset paradigm. Again, any observed differences could be attributed to the influences of head motion. The average eye accelerations, model accelerations, and the average differences between them for both rightward and leftward motion in all subjects are listed in Table III-12. For Subjects 3 and 4 there were no statistically significant differences in the average accelerations between the model (based on smooth pursuit) and the measured eye data \( (p = 0.050 \text{ for Subject 4-rightward, and } p \geq 0.447 \text{ for the remaining three conditions}) \). However, the average eye accelerations of Subject 1 were significantly \textit{greater} than the corresponding model accelerations \( (p \leq 0.038) \), while the average eye accelerations of Subject 2 were significantly \textit{less} than the corresponding model accelerations \( (p \leq 0.003) \). From this, as well as from the previous comparison of response latencies, it seems that if the smooth pursuit signal is indeed responsible for cancelling the VOR during CEHT, then head motion may affect the way smooth pursuit is manifested by the brain, particularly in terms of reducing the response latency.
Table III-12. Average eye and model acceleration data (in deg/sec/sec) from responses to the Delayed Target Onset paradigm

<table>
<thead>
<tr>
<th>Motion</th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rightward</td>
<td>148.0 ±22.3</td>
<td>85.0 ±20.8</td>
<td>93.7 ±20.4</td>
<td>91.7 ±14.9</td>
</tr>
<tr>
<td>Motion</td>
<td>123.5 ± 3.5</td>
<td>118.1 ± 7.2</td>
<td>89.3 ± 6.3</td>
<td>103.7 ± 6.5</td>
</tr>
<tr>
<td>Difference</td>
<td>24.5 ±23.9</td>
<td>-33.1 ±22.8</td>
<td>4.4 ±17.7</td>
<td>-12.0 ±16.7</td>
</tr>
<tr>
<td>p Value</td>
<td>0.0067</td>
<td>0.0024</td>
<td>0.4465</td>
<td>0.0496</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Motion</th>
<th>Subject 1</th>
<th>Subject 2</th>
<th>Subject 3</th>
<th>Subject 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leftward</td>
<td>105.7 ±22.5</td>
<td>86.2 ±17.0</td>
<td>93.6 ±16.4</td>
<td>101.0 ±18.2</td>
</tr>
<tr>
<td>Motion*</td>
<td>90.3 ± 5.4</td>
<td>109.0 ± 6.3</td>
<td>95.6 ±12.7</td>
<td>99.0 ± 9.3</td>
</tr>
<tr>
<td>Difference</td>
<td>15.3 ±22.5</td>
<td>-22.8 ±16.9</td>
<td>-2.0 ±11.2</td>
<td>2.0 ±12.5</td>
</tr>
<tr>
<td>p Value</td>
<td>0.0381</td>
<td>0.0021</td>
<td>0.6113</td>
<td>0.7159</td>
</tr>
</tbody>
</table>

*Leftward acceleration values shown as positive
IV. DISCUSSION

A. Capacity of the Superposition Model to Predict Subject Responses During Combined Eye-Head Tracking

Based on the results of the residual and correlation analyses, presented for both the Chair Brake and Delayed Target Onset paradigms (Section III.B), I have not found sufficient evidence to reject the hypotheses that (1) the smooth pursuit system provides the primary signal responsible for cancelling the VOR during CEHT, and (2) this cancellation occurs as a simple summation (superposition) of the two component ocular motor signals. Specifically, none of the residual plots corresponding to either test paradigm showed gross, consistent features (with magnitudes comparable to that of the 15 deg/sec stimuli) for any of the four subjects. Also, the correlation values, used to quantify the similarity between the model simulations and the corresponding subject data within the transition regions of Chair Brake and Delayed Target Onset responses, showed the degree of linear association between model and data was at least 90% for all subjects, for motion in either direction. These correlations were significantly greater than those derived using model values which may result from an alternative hypothesis of VOR cancellation (p ≤ 0.007 in all cases). Again, although other researchers have offered the hypotheses that the VOR is canceled through superposition with a signal from the smooth pursuit system (e.g., Barnes et al. 1978; Lanman et al. 1978; Lau et al. 1978; Leigh et al. 1987; Ranalli and Sharpe 1987; Barnes and Eason 1988), this was the first attempt we know of to confirm them rigorously in humans.
Since the completion of my modeling and subsequent analysis, Gauthier and Vercher (1990) published a paper that supports my hypotheses of VOR cancellation and refutes the contention that the VOR is suppressed by parametric or other non-visual means before it is cancelled by smooth pursuit. They also demonstrate that VOR gain cannot be set to a value less than 0.6, even when the paradigm is fully predictable. However, their paper does not provide any sort of specific quantitative hypothesis (model) about how VOR cancellation occurs. Also, although they suggest that VOR gain can vary between 0.9 for unpredictable, transient stimulations and 0.6 with fully predictable rotations when the subject fixates a head-fixed target, they do not specifically comment on the dynamics of VOR gain change. Thus, my research provides substantial results that extend beyond the work of Gauthier and Vercher.

1. Comparison with Other Hypotheses

Although the results from this research support my hypotheses about VOR cancellation during CEHT, how compatible are these results with other proposed mechanisms of VOR cancellation or suppression?

I have intentionally limited my research to involve only transient, non-predictable stimuli. Also, all head rotations were passive and were not internally generated by the subjects themselves. This was to insure that I observed the component eye movement signals in their most
fundamental forms, not "contaminated" with the consequences of prediction or other non-quantifiable effects. Thus, this research does not allow me to confirm nor refute Robinson's proposed model for VOR cancellation (1982), because his model deviates from a simple superposition hypothesis only in the way it deals with planned head movements (which I did not allow).

a. VOR Suppression Through Parametric Gain Change

McKinley and Peterson (1985) suggest that during CEHT, the VOR is suppressed through parametric gain changes of the VOR, and that smooth pursuit does not appear to contribute significantly to VOR cancellation. With my research, I have demonstrated that VOR gain may be modulated between various "active" and "resting" levels (approximately 0.7 and 1.0, respectively), and that this modulation may be governed by changes in the demands placed on the VOR by the ocular motor system. However, total reliance on reducing VOR gain to near 0.0 to suppress the VOR during CEHT is not indicated by my data. In fact, my results from the Chair Brake paradigm and comparable results reported by Lanman et al. (1978) both conflict directly with this notion. For these paradigms, CEHT is initiated first, followed by an abrupt stopping of the chair. This leaves the moving target as the only input to the ocular motor system. If McKinley and Peterson are correct, VOR gain is reduced to near zero during the CEHT phase of the Chair Brake paradigm, thereby allowing the eye to passively rotate along with the head to maintain fixation on the synchronously
moving target. When the chair is subsequently stopped, the eye must begin moving to track the still-moving target. To do this, their proposed scheme requires that the smooth pursuit system be initiated from rest (because the VOR cannot aid in pursuing moving targets with the head stationary, no matter what the VOR gain is). We know that it takes at least 75 msec to commence smooth pursuit eye movements (Carl et al. 1987). However, the data from my research indicate that when the head is stopped, the eye accelerates almost immediately (after a delay comparable to the latency of the VOR) to match target velocity (e.g., see Figs. III-4 and III-5). To do this, smooth pursuit must already be active when the head is braked, thereby contradicting the assertion of McKinley and Peterson that smooth pursuit is not involved in VOR cancellation.

For the Delayed Target Onset paradigms, I showed that the onset of CEHT has a morphology generally similar to the onset of smooth pursuit (e.g., see Figs III-11 and III-12). However, because McKinley and Peterson suggest that smooth pursuit is not involved in VOR cancellation during CEHT, the only way their scheme could explain my data would be if the dynamics with which the VOR parametrically changes its gain are coincidentally similar to the onset of smooth pursuit (a very unlikely possibility). Thus, our data contradict the proposal of McKinley and Peterson that the VOR is mainly suppressed during CEHT by parametrically reducing VOR gain.
b. VOR Self-Cancellation

Another hypothesis of VOR cancellation during CEHT proposes that a copy of the internal VOR signal runs in parallel with the actual VOR signal. Then, when VOR cancellation is required, this accessory VOR signal adds negatively with the actual VOR signal, effectively causing the VOR to cancel itself (May and McCrea 1985; Tomlinson and Robinson 1981). Because this scheme does not involve smooth pursuit either, our data contradict it for the same reasons mentioned above. Additionally, such a system would only work correctly if target motion were exactly synchronized with head motion. If this is not the case, the parallel VOR signal must either be switched-off and another scheme employed for VOR cancellation, or a mismatch in gains between the two VOR pathways must be created. In either case, some sort of visual signal must be processed to enable the system to judge the velocity of the target relative to the velocity of the head when deciding how to utilize the accessory VOR pathway. If a smooth pursuit signal were being used in this scheme, its magnitude would be small relative to target velocity and thus, could not account for the observed eye movements generated when the head is braked. Therefore, the VOR self-cancellation hypothesis is not a reasonable proposal for suggesting how the VOR is cancelled during CEHT.

c. Accessory Pathway for VOR Suppression

The hypothesis that an accessory suppression pathway is used in
conjunction with smooth pursuit to cancel the VOR during transient CEHT is supported by Cullen et al. (1989). They assert that if a visual target is being stabilized on the retina while the head is moving (i.e., while the VOR is being cancelled), then when the head experiences an additional perturbation, the gain of the compensatory cancellation signal is reduced. Based on this, they propose that a separate non-visual mechanism, in the form of an accessory suppression pathway employing vestibular afferent signals, operates at a very short latency to help smooth pursuit cancel the VOR.

Cullen et al. interpret their data with the expectation that, after a head perturbation, eye velocity should be equal and opposite to head velocity in the interval before the smooth pursuit signal contributes to cancel any remaining VOR signal (which occurs after about 130 msec). In the case where head movement starts from rest (i.e., at the onset of a standard VOR stimulus), their expectation is valid, since measured eye velocity is essentially equal to the opposite of head velocity (see Fig. III-3). However, in the case where the head is perturbed while smooth pursuit is cancelling the VOR, they still expect eye velocity to be equal and opposite to head velocity immediately after the perturbation. Because their data show that the magnitude of eye velocity is considerably less than the magnitude of head velocity, they attribute this difference to suppression from their proposed accessory VOR suppression pathway.

Is it reasonable to expect eye velocity to be equal and opposite to head velocity immediately after a head perturbation that occurs during
CEHT? If the VOR is being completely cancelled by a signal from smooth pursuit, then the two component signals (VOR and smooth pursuit) are equal in magnitude and they exactly cancel each other, allowing the eye to remain stationary with respect to the head. So, if the head is perturbed during CEHT, the command signal to the eye immediately after the perturbation (i.e., before the smooth pursuit component can change) will show an imbalance between the VOR and smooth pursuit signals by an amount equal to the change in the VOR signal elicited by the head perturbation. This net difference in VOR and smooth pursuit command signals provides the observed change in eye velocity. Thus, one should not expect eye velocity to always be equal and opposite to head velocity, particularly in response to head perturbations occurring during CEHT. In fact, the suppression signal that Cullen et al. attribute to an accessory vestibular pathway is most likely the smooth pursuit cancellation signal that was already active before the head perturbation. Thus, I believe that a proposed accessory pathway for VOR suppression is not necessary to explain their data, and that the hypothesis asserting VOR cancellation through superposition of the VOR and smooth pursuit signals is generally correct.

2. Comparison with Vertical and Torsional VOR Cancellation

I have demonstrated that the VOR is cancelled by a smooth pursuit signal during horizontal CEHT. How does this compare with our (limited) understanding of cancellation of the vertical and torsional VOR signals?
Cancellation of the vertical VOR was investigated by Ranalli and Sharpe (1988) in subjects as well as in patients having lesions that hinder propagation of vertical VOR and smooth pursuit signals. They used an algebraic model of VOR cancellation (which also employs an internal smooth pursuit command signal), and they found that the model satisfactorily predicted gaze gain \([\hat{E} + \hat{H}/\dot{T}]\) for vertical head-free tracking by normal subjects but not by patients. They then describe how certain lesions (e.g., internuclear ophthalmoplegia) disrupt a major component of the signal mediating cancellation of the vertical VOR during head-free tracking, but these lesions spare most of the signal that generates vertical smooth pursuit, with the head immobile. These results cast doubt on the notion that the vertical VOR is cancelled by vertical smooth pursuit because if cancellation is deficient, one would expect that the signal responsible for the cancellation (smooth pursuit) might also be deficient. However, they find that smooth pursuit remains largely intact.

One key point about these results is that they were derived using head-free tracking. Such a condition does not look solely at the interaction between smooth pursuit and the VOR during CEHT because, by the nature of head-free tracking, the subject (or patient) has internal control over the scheme employed to perform cancellation. Thus, these experiments did not control for effects due to subject prediction, which could have significant effects on system gains and other parameter values as well as on the initiation or other accessory pathways. Therefore, the scheme by which the vertical VOR is cancelled during vertical CEHT is still unclear.
The torsional system presents an interesting challenge for explaining cancellation of the torsional VOR. Because there is no torsional smooth pursuit system (and only a very weak torsional optokinetic system), torsional VOR must clearly be cancelled or suppressed by a different mechanism. Leigh et al. (1989) demonstrated that humans can increase and decrease their torsional VOR gains in situations in which the smooth pursuit system can be ruled out as contributing the primary modification component. However, the modification of the torsional VOR signal is not nearly as complete as during horizontal VOR cancellation: under various conditions, mean VOR gains ranged from 0.72 to 0.46. They also showed that mental effort has little influence on the torsional VOR, but that vision does play a role, although not by pursuit. Thus, the way in which torsional VOR is enhanced or cancelled likely uses a mechanism quite different than that employed by the horizontal VOR.

B. Possible Modifications to the Component Models

In the course of this research, I was unable to find sufficient evidence to reject the hypotheses that the smooth pursuit system provides the primary signal responsible for cancelling the VOR during CEHT and that this cancellation occurs as a simple summation (superposition) of the two component ocular motor signals. Generally, the simulations obtained
using the combined model compare quite well with actual subject data (as confirmed using the residual and correlation analyses). However, I noted a few circumstances in which performance of the combined model might be further improved. This section proposes some possible modifications to the component models which could provide such improvements, and it discusses the implications of these modifications on the overall research conclusions.

1. VOR Model

Examination of the Chair Brake response data (Fig. III-5) and the Chair Brake residuals (Fig. III-6) showed that, unlike visually-enhanced VOR, gaze is perturbed when the head is stopped during combined eye-head tracking. Also from Fig. III-5 we saw that the current configuration of the combined eye-head tracking model does not account for this perturbation of gaze when the head is braked. How might this discrepancy be explained, and how might the combined model be modified to deal correctly with both the VOR Offset and the Chair Brake conditions?

While constructing the model for visually-enhanced VOR, I reasoned that the brain determines when to boost VOR gain based on need. If the head is stationary, then the VOR is not needed to provide visual stability to any great degree, and VOR gain remains at some resting level. On the other hand, I postulate that when the head is moving and the brain determines that gaze stability is required, the VOR gain is
increased to an active level of near 1.0. Thus, I introduced elements into the model of the VOR that modulate VOR gain based on the brain’s internal estimate of head velocity. This modification to the model was successful in describing both visually-enhanced VOR onset and offset data. (Since obtaining these results which support my notion that VOR gain does change dynamically during a response, two other papers have been published which concur with this view (Gauthier and Vercher 1990; Lisberger 1990).)

Because I modeled the modification of VOR gain to describe data collected under ideal conditions for visually-enhanced VOR (i.e., with a stationary target), no provisions were included for circumstances in which the target also moves. However, Fig. III-5 clearly demonstrates my typical observation that gaze is perturbed when the head is stopped during CEHT (wherein the target moves in synchrony with the head), while the combined model does not reflect this gaze perturbation. Thus, we may wish to reconsider the conditions under which the brain may cause VOR gain to change.

Again, the purpose of the ocular motor system is to hold images of interest steady upon the retina. For visually-enhanced VOR, I postulated that the brain increases VOR gain to a high level (near 1.0) to get the maximum utility from this system (whose purpose is to maintain clear vision of stationary targets by providing eye movements equal and opposite to head movements). However, if the target of interest moves in synchrony with the head (as during my CEHT paradigms), then the brain
may realize that a high VOR gain is not necessary to maintain clear vision of the target moving along with the head, and thus, may allow VOR gain to remain at or near its resting level (near 0.7). As I noted when developing the model I used to represent the VOR (Section II.C.1.c.iii), a VOR gain less than 1.0 causes gaze perturbation when the head is suddenly stopped. Allowing VOR gain to increase to its active level when the target is stationary (i.e., during visually-enhanced VOR) has already been demonstrated to keep gaze from being perturbed when the chair is braked (Fig. III-3). On the other hand, keeping VOR gain at or near its resting level during CEHT could explain why gaze is perturbed when the chair is braked.

How might this be implemented in the combined model? For visually-enhanced VOR, I proposed that the brain makes its decision to increase or maintain VOR gain based on its internal estimate of head velocity ($\dot{H}'$ in Fig. II-21). Actually, because the target is stationary for visually-enhanced VOR, $\dot{H}'$ can be considered to represent the motion of head relative to the target. Then, extending this to CEHT in which the target also moves, the motion of the head relative to the target is given by ($\dot{H}' - \dot{T}'$) where $\dot{T}'$ is the brain's internal estimate of target velocity (see Figs. II-16, II-17, and II-22). Note that if the target is stationary, $\dot{T}'$ equals zero and ($\dot{H}' - \dot{T}'$) reduces to $\dot{H}'$, which is the designated input to the VOR gain modification system for visually-enhanced VOR (see Fig. II-21). On the other hand, during my CEHT paradigms in which the target moves in
synchrony with the head, \( \dot{T'} \) equals \( \dot{H'} \) so that \( (\dot{H'} - \dot{T'}) \) reduces to zero. If the VOR gain modification system is presented with zero input, the resulting VOR gain will tend to remain at, or move towards, its resting level. As we have seen in the CEHT Chair Brake data (Fig. III-5), when \( \dot{T} \) equals \( \dot{H} \) and the head is suddenly stopped, we observe a perturbation of gaze indicating that VOR gain may have returned to its resting level. Thus, by changing the input to the VOR gain modification system from *absolute* head velocity \( \dot{H'} \) to *relative* head velocity \( (\dot{H'} - \dot{T'}) \), we may be able to account for the differences in gaze perturbation seen between the VOR Offset and Chair Brake paradigms.

A revised model of CEHT using the proposed modification is shown in Fig. IV-1. To test the effectiveness of this revised model for predicting CEHT responses, I provided the model with the same sets of stimulus data I earlier supplied to the original model of CEHT. Then, I employed the same residual analysis and correlation methods used to test the original model, the specific results of which are given in the appendix.

Figure IV-2 presents example sets of subject data and model simulations using the Chair Brake paradigm for both the original and revised CEHT models. One can clearly see from both the eye and gaze velocity plots that the model simulations using the revised model better predict the data than the simulations obtained from the original model. This improvement is most striking (as expected) when predicting the gaze perturbation in response to the head brake. The transition region of a
Fig. IV-1. Revised version of the CEHT model shown in Fig. II-22. This model incorporates the estimated target velocity signal $\dot{T}'$ as a component of the input to the VOR gain modification circuit.
Fig. IV-2. Example sets of subject eye and gaze data with the corresponding model simulations using the Chair Brake paradigm over the same time frame used in Fig. III-5. a) original CEHT model, b) revised CEHT model.
Chair Brake response, presented in Fig. IV-3, shows in detail how the revision improves the model's capacity to represent eye velocity during this critical part of the response.

To objectively describe this improvement in performance, I briefly summarize the results given in the appendix. The correlation values for the Chair Brake paradigm using the revised model are virtually identical to those derived using the original model. (The largest difference in the correlation coefficients between the original and revised models for both the normal and shifted-model cases is less than 0.015 while the largest difference in the coefficients of determination is less than 0.2%.) However, the most striking improvement is seen in the Chair Brake residuals, where the peak magnitudes of the residuals decrease roughly by a factor of two.

How well does this revised model explain other CEHT phenomena, such as data from the Delayed Target Onset paradigm? Again summarizing from the appendix, there is virtually no change in the characteristics of the residuals between the original and revised models for the Delayed Target Onset paradigm. Interestingly enough, the correlation values for the revised model are slightly higher than those for the original model in all cases but one, indicating that the proposed modification may even slightly improve the model's capacity to predict subject responses to the Delayed Target Onset paradigm.

I have demonstrated how to improve the capacity of the proposed
Fig. IV-3. Example of the improvement in the capacity of the revised model to describe changes in eye velocity during the transition region of a Chair Brake response. Shown with the eye velocity waveform are plots of model velocity for both the original and revised models. Again, correlation coefficients for the revised model were calculated using the values in this region.
CEHT model to predict subtle characteristics of subject's CEHT responses. How does this modification of the model impact the overall results of the research? Although the proposed modification can be implemented simply and only acts to fine-tune one small feature of the model's response, it does affect one conclusion drawn from my results. Specifically, I stated that I could not reject the hypothesis that VOR cancellation occurs due to the superposition of two ocular motor signals because of the apparent success of the original, combined model in predicting subject CEHT data. I now present a different model (a modified version of the original model) which does a slightly better job of predicting subject CEHT data. However, strictly speaking, the revised model does not employ superposition because a signal from one pathway (\( \dot{T} \) from smooth pursuit) is used to modify the characteristics of the other pathway (the gain element of VOR). Thus, a superposition of the smooth pursuit and VOR signals does reasonably provide for the cancellation of the VOR during CEHT, but only in a general sense. On the other hand, if one accepts the revised CEHT model as an explanation for how the VOR is cancelled during CEHT, then the notion of a strict superposition of two signals must be relaxed somewhat.

2. Smooth Pursuit

As Robinson et al. discuss in their smooth pursuit model paper (1986), an expected characteristic of smooth pursuit (onset) responses is ringing. In fact, modeling the ringing in smooth pursuit was a major focus
of their paper. However, in the course of my data analysis, I noticed some peculiar ringing characteristics. Specifically, I noticed several smooth pursuit onset responses which showed little or no ringing, while I occasionally observed response ringing become quite pronounced after the head has stopped in a Chair Brake paradigm (for example, see Fig. III-5c). One important fact to note was that these responses with peculiar ringing characteristics did not occur in any regular pattern but were more or less randomly distributed throughout the set of responses for a given experiment. Thus, I was unable to relate variations in the observed degree of ringing with such variables as increasing subject fatigue during an experiment or increased adaptation to the experimental conditions. From this, I suspect that variations in the degree of response ringing occur due to idiosyncratic changes in the strategy of the subject's ocular motor system to acquire and maintain clear vision of the target.

Figure III-5 showed three different responses from the same subject to the Chair Brake stimulus. Although the nature of the head and target stimuli were quite similar for all three trials, the data displayed various degrees of post-brake ringing. What might cause this variation, and how might the CEHT model be adapted to yield the various degrees of ringing in the responses?

Unfortunately, we have no clear understanding of why smooth pursuit responses generally exhibit ringing. One possible explanation may involve the fact that smooth pursuit is inherently a negative feedback system (because the retina is attached to the eye) which contains various
delay and phase-shift (low-pass filter) elements. Depending on the overall loop phase lag and loop gain, such a negative feedback system can oscillate, perhaps causing the observed ringing characteristics. However, Robinson et al. point out that the observed ringing characteristics *cannot* be explained by relying on the inherent negative feedback nature of smooth pursuit, mainly because such a structure cannot simultaneously match both the response acceleration characteristics *and* the observed frequency of ringing (Robinson et al. 1986). They also point out that such a configuration *cannot* account for smooth pursuit gains greater than 1.0, a phenomenon which can be observed occasionally in subject data. For these reasons, they postulated the existence of an internal *positive* feedback loop to cancel the effects of the outer negative feedback loop (to make the system stable), and they provided a separate *internal* negative feedback loop whose main purpose is to create the observed ringing characteristics (see Section II.C.1.c.i). This is a large conceptual step. Rather than proposing that ringing is a natural side-effect due to the inherent negative feedback structure of the system, they instead suggest that the system *intentionally* generates the observed ringing.

What teleological reasons might explain the existence of a specific circuit in the brain that causes ringing? Although it is beyond the scope of this research to test any possible hypotheses analytically, one might argue that because smooth pursuit has a rather long latency (due to the time involved in processing the visual information), when the system *can* respond the brain makes it respond as quickly as possible. Although this scheme would cause the eye to reach the desired velocity rather rapidly,
the eye velocity may overshoot and subsequently ring until it settles to the required steady-state level. (Conceptually, this is similar to the step-response of a second-order linear system.) On the other hand, if the brain lacks this sort of urgency (perhaps due to varying degrees of subject alertness or to the slow speed of a visual target), it may allow eye velocity to match target velocity more slowly and thus, reduce the chance for overshoot and ringing. Thus, the brain may engage one or the other of two parallel smooth pursuit pathways: one which employs a special circuit that allows reaching target velocity quickly with the observed effects of overshoot and ringing, and the other which lacks (or bypasses) such a "ringing" circuit thereby allowing target velocity to be attained somewhat slower without causing the response to ring.

This possible explanation is consistent with the observed variation in the levels of ringing in both the smooth pursuit and chair brake responses. For smooth pursuit, the 15 deg/sec target velocities were not excessively fast and thus, the brain may have idiosyncratically chosen to use one pathway or the other, resulting in ringing or no ringing. Similarly, to explain the ringing seen in the chair brake data of Fig. III-5c, when the brain was involved in the initial CEHT phase of the Chair Brake paradigm, the brain may have used the non-ringing pathway when smooth pursuit worked in consort with the VOR. Then, when the chair stopped and smooth pursuit was the only active system, the brain may have switched to the other pursuit pathway, resulting in the observed ringing.
How might this parallel approach to smooth pursuit be modeled? If one closely compares the model for smooth pursuit onset (Fig. II-16) with the proposed model for smooth pursuit offset (Fig. II-18a), it is evident that the only structural difference is the additional feedback circuit (labeled "PMC" in Fig. II-16) in the pursuit onset model. This circuit is exactly the one intentionally introduced by Robinson et al. to cause the observed ringing characteristics (1986). Thus, if the smooth pursuit offset parameters are appropriately adjusted, this pathway could be used to provide smooth pursuit onset responses, but without the characteristic ringing. By placing these two pathways is parallel and switching between them as necessary, we could then model the observed variation in smooth pursuit ringing. However, we see from Fig. II-22 that this parallel structure is already in place! Therefore, we would only need to make a minor (structural) change in the combined model to explain the variation in smooth pursuit ringing. This would involve replacing the switch that presently chooses to use either the onset or offset pathway of smooth pursuit based strictly on the current value of the internal estimate of target velocity ($\hat{T}$) with a "smart" switch that can somehow predict whether or not the brain chooses to employ the ringing pathway. The activation of this new switch may rely on the current mental state of the subject (involving relative levels of alertness or fatigue) as well as on perceived characteristics of the visual scene (Pola and Wyatt 1985; Keller and Khan 1986; Miles et al. 1986; Wyatt and Pola 1987). If we could then move beyond classical systems modeling techniques, we might propose that the brain actually has available a continuum of possible responses (using a hybrid between the two pursuit pathways), ranging from a high degree of ringing to no
C. Future Work

Future work stemming from this research may be broken down into three major areas: (1) additional efforts to substantiate the superposition hypothesis using different experimental paradigms, (2) attempts to confirm or improve upon the characteristics of the component smooth pursuit and VOR models, and (3) possible applications in testing abnormal patients. Although only the first of these directly relates to the original question proposed by this research, the second builds upon insights developed while this research was being performed, while the third explores the practical significance of the research conclusions.

1. Testing the Hypothesis with Alternate Stimulus Paradigms

I chose to test the smooth pursuit/superposition hypotheses of VOR cancellation during CEHT by comparing the prediction of a combined smooth pursuit-VOR model with actual subject data in response to two specific experimental paradigms, the Chair Brake and the Delayed Target Onset. These paradigms were selected because they could help determine whether or not smooth pursuit was active during CEHT by isolating...
critical CEHT offset and onset response characteristics, either by stopping 
head motion during CEHT (Chair Brake), or by starting target motion, 
with the head already moving, to initiate CEHT (Delayed Target Onset). 
Although these two paradigms provided substantial insight into the 
interaction between the VOR and smooth pursuit during CEHT, the use of 
other experimental paradigms may lead to additional understanding.

Table II-2 listed all of the various paradigms available for 
experimentation with the SPVOR family of programs (see Section II.A.1.b). 
One of these, Target Brake, can investigate the transition from CEHT to 
visually-enhanced VOR. If smooth pursuit does cancel the VOR during 
CEHT (as this research indicates), then one would expect that when the 
target stops, the eye will remain stationary with respect to the head for a 
time and then assume a velocity equal and opposite to head velocity with 
a latency and rate similar to that of smooth pursuit offset. (Preliminary 
results suggest that this is, in fact, how the eye behaves.)

Another paradigm, Delayed Chair Onset, can investigate how the 
VOR is cancelled during CEHT by first invoking smooth pursuit and then 
initiating head motion to create CEHT. If smooth pursuit does cancel the 
VOR during CEHT, then, because smooth pursuit is already active when 
the head starts to move, the onset of CEHT should resemble VOR onset. 
(Again, preliminary results endorse this prediction.)

Throughout this research, I have initiated CEHT in which the target 
and head move in the same direction and at the same speed. Although
this is very similar to how humans actually track moving targets, additional insight into the cancellation of the VOR during CEHT might be obtained by moving the target and head at different speeds (Cullen et al. 1988), or even in different directions (Lisberger 1990). Also, I performed all the experiments using target and head velocities that allowed each of the component systems (smooth pursuit and the VOR) to operate well within their linear ranges (±15 deg/sec). We might be able to gain more insight into the interaction between smooth pursuit and the VOR during CEHT by pushing either of these systems to the edge of its effective operating range. For example, at high target velocities, the steady-state smooth pursuit gain normally falls to a value clearly less than 1.0. If we can demonstrate that, during CEHT at similar velocities, the VOR is not completely cancelled by an amount related to this reduction in smooth pursuit gain, then we have found additional evidence that smooth pursuit is indeed responsible for VOR cancellation during CEHT.

2. Modifications to the Component Models Suggested by this Research

This research uncovered two unexpected phenomena. The first of these was discovered when we compared the responses to the onset and offset of the VOR and discovered substantial gaze perturbation with the former but not the latter (see Section II.C.1.c.iii). I presumed that this is a consequence of dynamically changing VOR gain and modified the VOR model accordingly. The second phenomenon was the observation that the presence of observable ringing in smooth pursuit responses and the
smooth pursuit component of CEHT responses was irregular and unpredictable (see Section III.B.1). Although neither of these phenomena are critical to the substantiation of my overall hypotheses, they are interesting enough to warrant additional attention.

a. Further Investigation of the Dynamic Change in VOR Gain

As explained in Section II.C.1.c.iii, gaze was observed to be substantially perturbed when VOR was initiated from rest, but later in the same trial when the chair was stopped, gaze was only slightly perturbed (if it was perturbed at all). I therein provided my rationale for proposing that this observed difference in gaze perturbation to stimuli having virtually identical dynamic characteristics was due to dynamically changing VOR gain. Although the main focus of this research depends very little on the validity of this proposition, it may be of general interest to determine (1) if the VOR does change its gain dynamically, and (2) how this gain change is modulated by the brain.

Since my initial observations of this phenomenon and my subsequent attempt to model it, two independent papers have been published which report evidence that VOR gain does change dynamically: during the onset of the VOR (Gauthier and Vercher 1990) and if CEHT is being performed (Lisberger 1990). These findings, coupled with other reported observations by Barr et al. (1976) of dynamically changing VOR gain in the dark (based simply on the instructions given to the subject),
suggest that if this phenomenon is due to changes within the VOR, it is
due to changes in the VOR gain. However, as I discussed earlier, this
difference in gaze perturbation can not be due to a contribution from a
separate system relying on vision; due to the long latency of visual
processing, such a system could not manifest its contribution rapidly
enough to prevent gaze perturbation upon VOR offset. Therefore, I
believe that the observed phenomenon is due to changes within the VOR,
and thus, it is due to changes in the VOR gain. Future research efforts may
focus on this issue in an attempt to refute the hypothesis of variable VOR
gain.

How might this change in VOR gain be modulated by the brain?
Due to the nature of our VOR stimulus, head velocity did not reach 15
deg/sec instantaneously but rather accelerated more slowly (so as to
guarantee that we remained within the linear range of the VOR). Because
it took a finite amount of time for the head velocity to reach the intended
value, then the reconciliation of gaze (after its initial perturbation)
occurred from both the proposed increase in VOR gain as well as from the
contribution to the eye signal generated within the visually-driven
“fixation” system. Thus, the way in which VOR gain changed was masked
by an accessory eye movement signal (which began contributing at least 75
msec after the onset of VOR). If experiments could be re-performed with
higher head accelerations (to cause the gaze perturbation to occur earlier),
perhaps one could trace the reduction in the degree of gaze perturbation
before contributions from a system driven by vision can interfere. If the
decrease in gaze occurred inverse-exponentially, then one could conclude
that a simple lag element is a satisfactory way to model the change in VOR gain. One caution about performing such experiments is to be sure that the head movements stay within the linear range of the VOR; if head acceleration (or frequency) is too high, then gaze perturbation would occur from the inability of the VOR to deal with the rapidly changing stimulus, and conclusions drawn about contributions to gaze perturbation due to changes in VOR gain would not necessarily be valid.

What internal signal does the brain monitor when deciding to change VOR gain? As I mentioned above in Section IV.B.1, I found that more accurate simulation results could be obtained if the circuit responsible for modulating VOR gain based its decision to change VOR gain on the velocity of the head with respect to the target (relative head velocity, $\dot{H}' - \dot{\hat{H}}$) rather than on head velocity alone (absolute head velocity, $\dot{H}'$). One way to test the validity of this change in the model is to perform experiments employing the Delayed Chair Onset stimulus (see Sections II.A.1.b.ii and IV.C.1). This paradigm provides a VOR stimulus after target motion has already begun. Because the first half of the paradigm involves target motion but no head motion, ($\dot{H}' - \dot{\hat{H}}$) would be non-zero causing VOR gain to rise to its active level. Then, if the proposed modification to the model is correct, the subsequent head perturbation should result in little if any perturbation of gaze. Also, by applying a chair brake after initiation of CEHT in which the target and head move at reasonable different velocities (~10 deg/sec difference), it would be interesting to observe whether VOR gain still falls back to its resting level, or whether
VOR gain remains near its active level. If the VOR gain modification circuit does monitor \((\dot{H} - \dot{T})\), then we would expect the latter to occur, manifested by only minimal gaze perturbation. Clearly, the issue of determining what actually drives the VOR gain modification circuit is one requiring additional attention.

Another way to probe this issue about what drives the VOR gain modification circuit is to perform additional experiments which place the VOR in various "initial" states. One technique, used by Cullen et al. (1988), involves initiating VOR (or CEHT) at one velocity and then abruptly stepping the chair to a new velocity while maintaining the same target velocity. This causes the VOR to become active (and presumably, causes VOR gain to rise to its active level). To what degree will gaze be perturbed, if at all, when the chair (and head) are again accelerated? Will the results be different if the head is accelerated in the same direction than if the head is accelerated in the opposite direction? Does motion of the target cause a difference? Answers to these questions will likely confirm or refute the hypothesis that the VOR gain modification system monitors head velocity with respect to the target. Additionally, it may be possible to use optimal experiment design techniques (e.g., Endrenyi 1981) to define an optimal set of target and head motion stimuli for probing this question.

b. Effort to Explain Variations in Smooth Pursuit Ringing

Although the occurrence of smooth pursuit ringing (or the lack
thereof) was not generally predictable in my experiments (see Section IV.B.2), perhaps more can be learned about this variability, as well as about the nature of smooth pursuit ringing in general, by performing experiments which change the smooth pursuit paradigm. It is still essential to randomize the direction and duration of smooth pursuit trials to avoid the effects of prediction (Robinson et al. 1986). In addition, randomly changing the target speed to various levels may show more characteristic ringing patterns associated with each speed. This would indicate that the underlying strategy used by the ocular motor system may change relative to the demands placed upon it.

3. **Applications in Testing Abnormal Patients**

The model for combined eye-head tracking developed in this research may be useful for testing patients with visual and/or vestibular lesions. For example, patients with labyrinthine deficiencies (*i.e.*, neurological deficits due to problems with the semi-circular canals or the primary vestibular afferent nerve, resulting in the absence of a VOR signal) but having intact smooth pursuit systems would respond differently to the Chair Brake paradigm than would normal subjects. The model would predict, because there is no VOR signal to cancel, that smooth pursuit would remain relatively inactive during the initial phase of CEHT. Then, because smooth pursuit is inactive, eye velocity would *not* immediately increase towards target velocity when the chair is braked (as it does with normal subjects); in fact, the model would predict that the
eye would not move for a time equivalent to the latency of smooth pursuit. This could be simulated with the model by manually setting both the active and resting VOR gains, $A_{off}$ and $A_{on'}$ to zero (see Fig. II-21). Results of preliminary experiments on subjects known to have labyrinthine deficiencies agree with simulations made after altering the model in this way.

Similarly, subjects with hemispheric lesions which cause smooth pursuit deficits would have difficulty cancelling their VORs during CEHT. The model could simulate this condition by having the value of the overall gain parameter $P_1$ manually set to 0.5 (see Fig. II-16 through II-18). Again, the results of preliminary experiments on subjects known to have smooth pursuit deficits agree with simulations made after altering the model's gain element as described. Thus, by carefully changing model parameters to generate simulations that match patient data, clinicians may be able to discern the nature and severity of a patient's neurological illness involving the VOR, the smooth pursuit system, or both.
V. SUMMARY AND CONCLUSIONS

This research comprised the first rigorous attempt to confirm or refute the hypotheses that, for human subjects in the horizontal plane, the smooth pursuit system generates the primary signal responsible for cancelling the vestibulo-ocular reflex (VOR) during combined eye-head tracking (CEHT), and that this cancellation occurs through a superposition of the component smooth pursuit and VOR signals.

To test these hypotheses, I first developed realistic mathematical models to describe the characteristic behaviour of the component VOR and smooth pursuit systems. Second, by employing optimal techniques for parameter estimation, I determined values for various model parameters that caused the simulation results of each model to accurately reflect each subject's responses. Third, after each component model was equipped with a set of average optimal parameter values for a given subject, I joined the two component models by summing their output signals together to create a combined model. This model constituted a quantitative representation of the hypotheses of this research that could be tested by comparison with subject data. Fourth, I used actual stimulus data (head and target velocity), collected along with the subject response data during experimental trials employing various CEHT paradigms, to drive the combined model and create simulated response waveforms. Fifth, I compared the generated simulation waveforms with the corresponding subject data in an effort to detect similarities or differences which would confirm or refute my research hypotheses. The comparisons first
involved creating residual waveforms to give a graphical representation of the relative similarity between the subject data and the corresponding model prediction. In addition, I also used a technique based on correlation to quantitatively compare the actual and simulated eye movement waveforms. Based on these analyses, there was insufficient evidence for me to reject the hypothesis that a superposition of the smooth pursuit and VOR signals accounts for the observed cancellation of the VOR during transient, horizontal CEHT. However, one way to account for a particular feature observed in certain responses involved slightly relaxing the premise of a strict superposition.

The comprehensive CEHT model I developed to test my research hypotheses successfully describes responses to horizontal smooth pursuit, visually-enhanced VOR, and at least two different CEHT paradigms. When supplied with appropriate model parameters, this model could be used to make predictions about CEHT activity not yet investigated. Also, by systematically varying the model parameters (or by using more rigorous parameter estimation methods) to match model simulations with response data from patients, this model could help identify the specific nature of various patient abnormalities.

In addition to fulfilling my research objectives, I gained additional insights about certain characteristics of the component smooth pursuit and VOR systems. First, VOR gain seems to change dynamically within individual responses, depending on the demands placed upon the VOR. For example, if a subject's head is at rest, then his or her VOR is not being
used to maintain gaze stability (to any great degree), and the VOR gain can assume a resting level (about 0.75). On the other hand, if the subject uses the VOR to help maintain gaze stability of a stationary target during sustained head motion, my data indicate the possibility that the VOR gain can rise to a level which makes the VOR most useful (approximately 0.95).

An additional finding concerns CEHT when the head and target move in synchrony. Although the head is moving, because the target is also moving along with the head, my results indicate that the VOR gain remains at, or falls back to, its resting level; in this circumstance, a high VOR gain is not needed to maintain gaze stability. (However, my results do not indicate that VOR gain ever falls below its resting level of approximately 0.75). Adapting the combined model to deal with this latter case involved supplying a signal from the smooth pursuit system (i.e., the brain’s internal estimate of target velocity) to the circuit in the VOR pathway responsible for gain modification. Thus, because a signal from smooth pursuit is being used to influence the generation of the VOR signal, the premise assuming the existence of a strict superposition between the two component systems must be relaxed somewhat.

In this research, I developed a model to characterize the offset of smooth pursuit. Although the structure of such a model had been suggested elsewhere (Robinson et al. 1986), my efforts resulted in the first successful implementation we know of for modeling this phenomenon. Also, I was able to use this pursuit offset pathway as an approximation of a “fixation” system; this allowed me to model visually-enhanced VOR by
augmenting the signal from the VOR pathway with the signal from the "fixation" pathway. Simulations of visually-enhanced VOR compared well with subject responses to this activity.
APPENDIX

Residual and Correlation Results Using the Revised CEHT Model

This appendix contains the results of the residual analysis and correlation methods I used to test the capacity of the revised CEHT model (which employs the internal estimate of target velocity as an input to the VOR gain modification circuit) to describe subject responses measured during CEHT paradigms (see Section IV.B.1). Although this modification to the model was implemented to improve the model’s performance in characterizing responses to the Chair Brake paradigm, for completeness analysis results are also provided here for the Delayed Target Onset paradigm.

By plotting average residuals (see Section II.C.3.a), we can quickly get both an appreciation for the improvement provided by the revised model as well as a descriptive idea of where and to what degree the revised model simulations fail to recreate the measured subject responses. Then, in an attempt to quantify just how well the revised model simulations predict actual eye movement responses, I employed the same correlation technique used to test the simulations from the original model (see Section II.C.3.b). As before, if the simulated model velocity waveform exactly predicted the measured eye velocity values, then the calculated correlation coefficient and coefficient of determination would be 1.0 and 100%, respectively. The degrees to which these coefficients deviate from such ideal values gives us an indication of the relative similarity (or
difference) between the measured eye velocity values and the corresponding simulated model velocity values.

Figure A-1 plots the average residuals from the Chair Brake trials for each of the four subjects, over the same time range as the residual plots for the original model (Fig. III-6). By focusing on the section of the plots immediately after initiation of the chair brake (at 2.0 sec), it is evident that the modification has improved the capacity of the model for describing subject responses to the Chair Brake paradigm. Comparing with the corresponding residual plots shown in Fig. III-6, the magnitudes of the peak residual features have decreased considerably, often by at least a factor of two. Also note that some of the residuals still contain a low-level sinusoidal component; this is expected, as the revised model has not been modified in any way to account for the occasionally observed ringing in the responses.

Table A-1 lists, for all four subjects, the average correlation coefficients and coefficients of determination which compare the similarity of the immediate subject response to the chair brake with the corresponding revised model simulations for both rightward and leftward motion. Note from this table that all conditions show that the degree of linear relationship between the model simulation and the subject data is again at least 90%, and most show about 98% similarity. Comparing with the data in Table III-4, we see that the values are almost identical to those for the original model.
Fig. A-1. Average residuals from the Chair Brake trials, calculated using the revised CEHT model (over the same time range as the plots in Fig. III-5), for each of the four subjects. See text for details.
Table A-1. Mean correlation statistics describing the similarity between subject response data and model simulation data for the transition region of the Chair Brake paradigm using the revised model

<table>
<thead>
<tr>
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<th>Subject 1</th>
<th>Subject 2</th>
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<tbody>
<tr>
<td><strong>Rightward Motion</strong></td>
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<tr>
<td>Correlation Coefficient</td>
<td>0.988 ±0.007</td>
<td>0.980 ±0.017</td>
<td>0.995 ±0.006</td>
<td>0.984 ±0.009</td>
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<td>Coefficient of Determination</td>
<td>97.60 ±1.35</td>
<td>96.04 ±3.29</td>
<td>98.96 ±1.12</td>
<td>96.82 ±1.82</td>
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<tbody>
<tr>
<td><strong>Leftward Motion</strong></td>
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</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.992 ±0.007</td>
<td>0.993 ±0.004</td>
<td>0.987 ±0.006</td>
<td>0.964 ±0.043</td>
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<tr>
<td>Coefficient of Determination</td>
<td>98.48 ±1.14</td>
<td>98.65 ±0.86</td>
<td>97.33 ±1.22</td>
<td>90.80 ±9.11</td>
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The average residuals from the Delayed Target Onset trials are plotted in Figure A-2, for each of the four subjects, over the same time range as the residual plots for the original model (Fig. III-13). Again, of great importance in this research is the magnitude of the residuals immediately after the onset of target motion (which occurs at the 2.0 sec point in the responses). Notice that the residuals of the revised model are almost identical to those calculated for the original model. For all subjects the residuals still remain near zero (i.e., they remain at the same level as that seen before target motion) for at least 100 msec after the target begins to move. This suggests that the revised model also does a satisfactory job of predicting the actual eye movement response during this critical time.

Table A-2 lists, for all four subjects, the average correlation coefficients and coefficients of determination which compare the similarity of the subject response to the onset of target motion with the corresponding model simulations for both rightward and leftward motion. Note from this table that all conditions show that the degree of linear relationship between the model simulation and the subject data is still at least 90%, and most show about 95% similarity. Interestingly enough, when compared with the data in Table III-6, the correlation values for the revised model are slightly higher than those for the original model in all cases but one, indicating that the proposed modification may even slightly improve the model’s capacity to predict subject responses to the Delayed Target Onset paradigm.
Fig. A-2. Average residuals from the Delayed Target Onset trials, calculated using the revised CEHT model (over the same time range as the plots in Fig. III-5), for each of the four subjects. See text for details.
Table A-2. Mean correlation statistics describing the similarity between subject response data and model simulation data for the transition region of the Delayed Target Onset paradigm using the revised model.

<table>
<thead>
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<tbody>
<tr>
<td><strong>Rightward Motion</strong></td>
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<tr>
<td>Correlation Coefficient</td>
<td>0.952 ±0.019</td>
<td>0.971 ±0.013</td>
<td>0.962 ±0.022</td>
<td>0.989 ±0.005</td>
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<tr>
<td>Coefficient of Determination</td>
<td>90.60 ±3.66</td>
<td>94.32 ±2.44</td>
<td>92.57 ±4.28</td>
<td>97.71 ±0.91</td>
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<td>11</td>
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<tr>
<td><strong>Leftward Motion</strong></td>
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</tr>
<tr>
<td>Correlation Coefficient</td>
<td>0.969 ±0.025</td>
<td>0.984 ±0.005</td>
<td>0.989 ±0.003</td>
<td>0.981 ±0.021</td>
</tr>
<tr>
<td>Coefficient of Determination</td>
<td>93.95 ±4.83</td>
<td>96.91 ±1.09</td>
<td>97.74 ±0.60</td>
<td>96.36 ±4.04</td>
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REFERENCES


