

Chapter 11

Models of Saccade-Vergence Interactions

George K. Hung¹ and Kenneth J. Ciuffreda²

¹ Dept. of Biomedical Engineering, Rutgers University, 617 Bowser Rd., Piscataway, NJ 08854-8014, PH: (732) 445-4137, FX: (732) 445-3753, EM: shoane@rci.rutgers.edu

² Dept. of Vision Sciences, State University of New York, State College of Optometry, 33 West 42nd St. New York, NY 10036; PH: (212) 780-5132, FX: (212) 780-5124; EM: kciuffreda@sunyopt.edu

11.1 INTRODUCTION

Eye fixations in daily life are controlled by two types of eye movements. Saccades rotate the two eyes in the same direction (i.e., conjugately; Fig. 11.1A), such as during reading and scanning of a scene, whereas vergence movements rotate the two eyes in opposite-directions (i.e., disjunctively; Fig. 11.1B), such as during tracking of objects moving in depth. Together, they provide control of binocular fixation in three-dimensional space

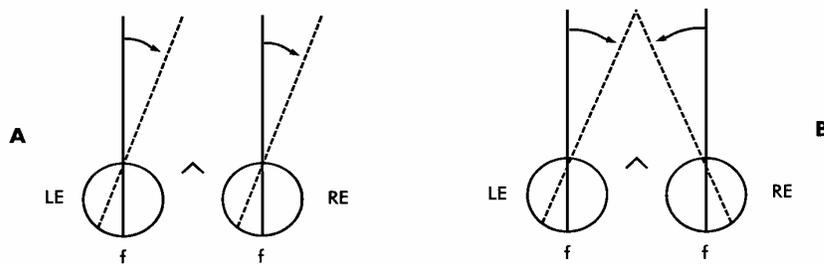


Figure 11.1. Schematic drawing of pure versional saccadic (A) and pure symmetric vergence (B) eye movements. LE, left eye; RE, right eye, f, fovea. Reprinted from Ciuffreda and Tannen (1995), pg. 2, Fig. 1-1, with permission of Harcourt Health Sciences.

(Ciuffreda and Tannen, 1995). Since nearly all of our naturally-occurring target shifts are asymmetrical in nature, responses generally involve both saccade and vergence and their interactions (see Fig. 11.2), rather than either purely saccadic or vergence eye movements (Fig. 11.1).

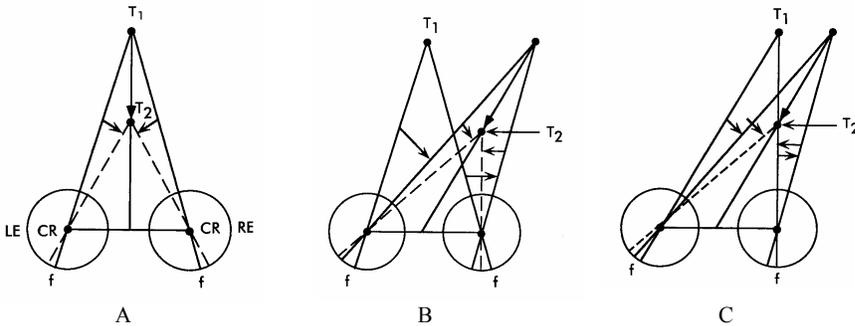


Figure 11.2. Symmetrical versus asymmetrical disparity stimuli and overall response patterns. A. Symmetric vergence. B. Asymmetric vergence. C. Line-of-sight asymmetric vergence. T_1 , Initially fixated target; T_2 , subsequently fixated target; f , fovea; CR , center of rotation of the eye; LE , left eye; RE , right eye. Reprinted from Ciuffreda and Tannen (1995), pg. 132, Fig. 6-6, with permission of Harcourt Health Sciences.

Eye movements have been monitored non-invasively using a variety of techniques, including infrared reflection, video, and magnetic search coil (Ciuffreda and Tannen, 1995). The measured responses accurately reflect the brain's control strategy for directing the eye movements, thereby providing insight into the underlying neuronal control of the oculomotor responses. It would appear that the saccadic and vergence neuronal control programs can be inferred directly from the oculomotor responses. Indeed, when the saccadic and vergence movements occur at different times, it is easy to distinguish between the rapid (typically 20 to 60 msec) conjugate eye rotations of the saccade (Yarbus, 1967; Bahill 1981; Fig. 11.3A) and the relatively slow (typically 800-1200 msec) disjunctive eye rotations of vergence (Hung et al, 1997; Fig. 11.3B). However, since the saccadic and vergence systems share a common plant (i.e., the extraocular muscles and eyeball), when these movements occur simultaneously, their individual control programs may not be so readily discerned. Moreover, some simultaneously-occurring eye movements contain a component that cannot be accounted for by linear superposition of saccade and vergence contributions (Kenyon et al, 1980; Miller et al, 1980).

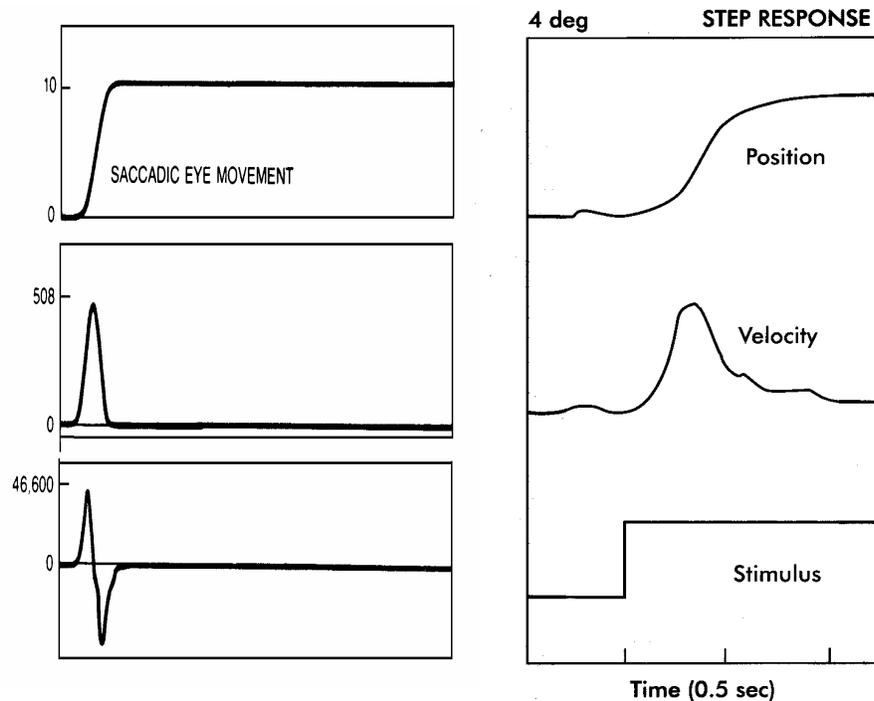


Figure 11.3. (Left) Model simulation showing dynamics of saccadic eye movement. Eye position (top, deg), velocity (middle, deg/sec), and acceleration (bottom, deg/sec²) traces are shown over a time range of 500 msec. Reprinted from Bahill and Stark (1979), pg. 112, with permission of Sci. Am. (Right) A 4-degree convergence step stimulus (bottom) resulted in disparity vergence response (top) with latency of 180 msec and peak velocity of 12 deg/sec (middle) occurring 150 msec after the vergence response begins. The total time range for the plot is 2 sec. Reprinted from Schor et al (1986), pg. 615, Fig. 4, with permission of Optom. Vis. Sci.

Some investigators (Enright, 1984, 1986, 1992; Erkelens, 1989; Zee et al, 1992) believed that this was due to vergence being facilitated by saccades, as evidenced by the transient, higher-velocity, disjunctive time course seen during combined saccade-vergence responses to asymmetrical targets (Kenyon and Ciuffreda, 1978). Yet, it is difficult to reconcile this facilitation notion with the approximately 200 msec latency of saccades, since it would take another 200 msec to respond to the transient dynamics when saccade and vergence movements coincided. If facilitation based on feedback were to occur, the ongoing vergence movement would be augmented by a signal meant to assist its position 200 msec earlier. Thus,

instead of facilitating the vergence movement, it could actually lead to system instability. Others have argued that facilitation is pre-programmed based on the target positions. However, this cannot explain why in a number of situations in which there is an asymmetrical target change, a transient divergence occurs when convergence would be required to facilitate the vergence movement. Moreover, saccadic (Zuber and Stark, 1966) and vergence (Hung et al, 1989, 1990) suppression during the actual combined movements preclude accurate guidance of vergence movements by saccades. Hence, there is considerable evidence against the facilitation notion.

To clarify these apparently contradictory results, a more systematic approach is needed. Models of saccade and vergence movements and their interactions can provide the necessary quantitative analysis for the following reasons: First, a saccade-vergence model requires a formal structuring of the important components in the system. This demands a deep conceptual understanding of visual optics, anatomy, muscle biomechanics, and neurophysiology of the oculomotor components. Also, the interconnections within a model require understanding and insight into the unique anatomical and neurological connectivity between the saccadic and vergence systems. Second, a model is needed because of the complexity of the system. Since eye movement responses involve temporal dynamic interactions between the two systems, only a model can keep track of the multitude of rapid changes in parameter values within the model. And, third, model simulations may provide new insight and understanding of these systems that would not have been possible using traditional descriptive or graphical techniques.

Some of the earlier saccade-vergence models are discussed below. In addition, a recent robust quantitative model of saccade-vergence interactions, which provides significant insight into the underlying mechanisms in helping to resolve the previous controversies, is discussed in detail (Hung, 1998a,b). The control processes developed in this model were based on known neurophysiological signal pathways and extraocular muscle innervations. In essence, it was found that the small difference in latency between ipsilateral and contralateral signal pathways was responsible for the transient divergence mentioned earlier. Model responses under different stimulus conditions were found to be consistent with known experimental results. Perhaps most significantly, the model did not require complicated higher-center processes to modify the eye movement dynamic timecourse, as others (Enright, 1984, 1986, 1992; Erkelens, 1989; Zee et al, 1992) have hypothesized. Thus, this saccade-vergence model, which is discussed later in the chapter, provides a realistic, relatively simple, clearly-defined, and robust framework for both the understanding and quantitative assessment of saccade-vergence interactions.

11.2 BACKGROUND

11.2.1 Stimulus Configuration

Target displacements equivalent to those occurring in free-space can be re-created optically in the laboratory (Fig. 11.4). Linear displacements of the targets on the oscilloscopes presented to each eye result in the percept of a target shift in 3-D space. In this way, various types of target movements can be presented to the subject.

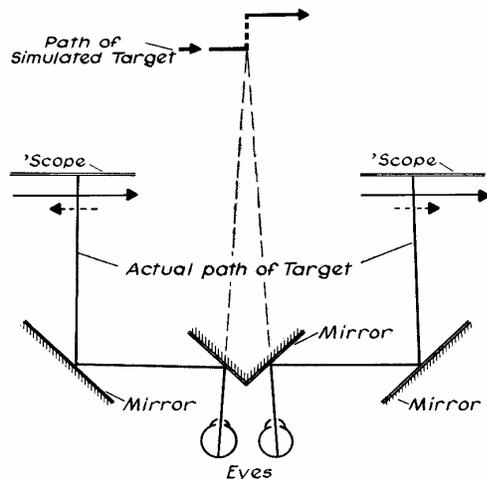


Figure 11.4. Mirror stereoscope through which stimuli are viewed. Stimuli moving at the same velocity but in different directions on the scope (dotted arrows) appear as a single stimulus moving toward or away from the viewer. Stimuli moving at the same velocity in the same direction (solid arrows) appear as a single stimulus moving to the left or right in front of the viewer. Reprinted from Ono (1983), pg. 382, Fig. 11.3, with permission of K. J. Ciuffreda, the copyright holder.

11.2.2 Saccadic Eye Movements

Saccadic eye movements refer to the rapid conjugate rotations of the eyes in response to target changes in the same depth, or isovergence plane (see Figs. 11.1A and 11.4). They have been found to exhibit a latency of about 180 to 220 msec (Stark, 1968), and durations ranging from 25 msec for 0.1 deg amplitude to 125 msec for 50 deg amplitude (Bahill & Stark, 1979). Furthermore, saccade dynamics exhibit general characteristics described by its peak velocity-amplitude relationship, or main sequence (Bahill & Stark, 1979), having a slope of about 50 (deg/sec)/deg.

Two types of saccadic models have been proposed. In the sampled-data model, saccades consist of open-loop movements, i.e., once they are initiated, their dynamics cannot be modified over the intervening 200 msec

interval (Young and Stark, 1962; Semmlow & Venkiteswaren, 1976). This is applicable under normal viewing conditions. On the other hand, in the continuous feedback model, target changes can alter the saccadic responses during the 200 msec interval. This occurs under special conditions in which target pulses are introduced (Aslin & Shea, 1987; Becker & Jurgens, 1979; Hou & Fender, 1979; Wheelles et al, 1966).

To test whether the open- (i.e., sampled-data model) or the closed-loop (continuous feedback) model of the controller is more applicable, a technique in which the variability of amplitude and duration of saccadic step responses has been used (Jurgens et al, 1981). The source of the variation has been attributed to changes in firing frequency or recruitment of the neurons that constitute the pulse generator (Keller, 1977; Keller & Robinson, 1972; King & Fuchs, 1977; van Gisbergen et al, 1981). A preprogrammed, open-loop model would be expected to show variations in both amplitude and duration, whereas a local feedback closed-loop model would be expected to vary in duration only. The results for both normal and Diazepam (a tranquilizer known to reduce saccadic velocity) experiments favored the local feedback model.

In reality, experimental results suggest that probably both open- and closed-loop models are correct. This is because the refractory period of the saccadic system appears to have an early relative portion, in which rapid stimulus changes can re-trigger and modify the saccade, and a later absolute portion (during the last 75 msec or so) in which the saccade is not re-triggerable (Ciuffreda and Tannen, 1995).

11.2.3 Vergence Eye Movements

Vergence eye movements are relatively slow, oppositely-directed, or disjunctive, rotations of the eyes that bring about a single, binocular 3-dimensional percept of objects moving in depth (Riggs and Niehls, 1960; Rashbass & Westheimer, 1961b; Westheimer & Mitchell, 1956) (see Figs. 11.1B & 11.3B). These disjunctive movements have been thought previously as being controlled by a simple continuous feedback system (Krishnan & Stark, 1977; Rashbass & Westheimer, 1961b; Schor, 1979; Zuber & Stark, 1968). However, such a continuous model could not account for a number of experimental results. For example, the model response to sinusoidal input exhibited a phase lag that was much larger than that found in the experimental data (Rashbass & Westheimer, 1961b). Also, the response to a step followed by a ramp (i.e., smooth, continuous target change of disparity) in the opposite direction exhibited a reversal in direction well before the target moved past the zero disparity level, which indicated that

velocity information was used to anticipate target position (Rashbass & Westheimer, 1961b). Furthermore, the response to a ramp of disparity exhibited multiple movements that could not be simulated simply as visual feedback oscillations (Hung et al, 1986). The primary problem with a continuous feedback model was that the time constant of a step response (about 200 msec) was equal or nearly equal to the response latency (160 to 200 msec) (Hung et al, 1983). Thus, the response to a rapidly changing periodic stimulus, such as a sinusoid, would not be fast enough to account for the measured short experimental latencies if it simply consisted of a passive continuous-feedback slow movement following a 200 msec time delay. Furthermore, due to its relatively long latency and time constant, a conflict could arise between instantaneous error and actual target position, so that the model responses to either steps or ramps would show marked instability oscillations.

Over the past decade, we have accumulated a body of evidence clearly demonstrating that the vergence system is composed of two components or subsystems: a fast, open-loop movement which brings the eyes near the target position (Hung et al, 1986; Semmlow et al, 1986, 1993, 1994), followed by a much slower movement under visual feedback control that reduces the residual error (or fixation disparity) to a few minutes of arc, i.e., within foveal Panum's fusional area (Panum, 1858). This was clearly shown using ramp disparity stimuli of various velocities (Hung et al, 1986; Semmlow et al, 1986) (see Fig. 11.5a). For velocities less than about 1.4 degrees of disparity per second (deg/sec), the response consisted mainly of smooth ramp tracking of the target, whereas for a velocity greater than 2.7 deg/sec, the response consisted of multiple-steps in which the termination of each step approximately matched the ramp target position. The latter indicated that the vergence system used an estimate of target velocity to generate the appropriate vergence step movement (Hung et al, 1986). Intermediate velocities resulted in responses containing a mixture of step and ramp movements. Also, the steps in the multiple-step responses were found to lie on the normal main sequence for vergence eye movements (Bahill & Stark, 1979), having a slope of about 4 (deg/sec)/deg. Thus, the open-loop component estimated the future position of the ramp stimulus, and then generated a normal step movement to match the position of the stimulus. As the ramp stimulus progressed, successive steps were generated to follow the ramp accurately. Moreover, when instrument feedback was used to open the vergence feedback loop (Semmlow et al, 1993, 1994), the response to a step disparity also consisted of a series of steps that were on the main sequence, which lent further support to the concept of an open-loop fast component.

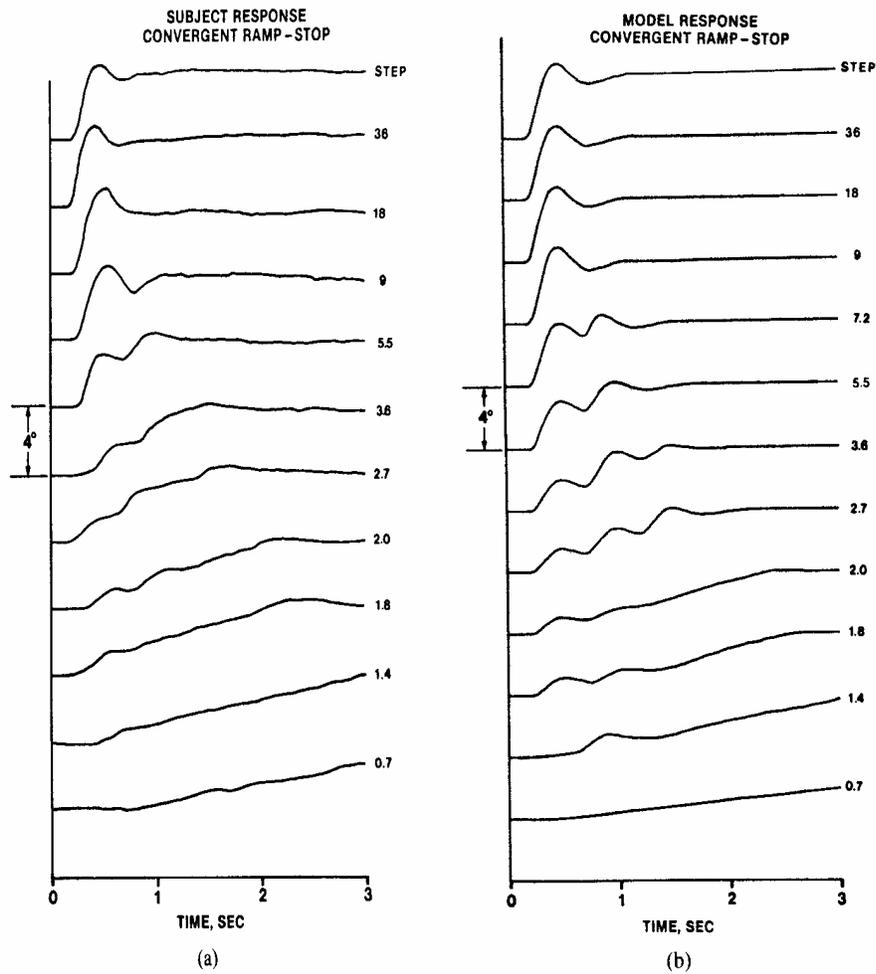


Figure 11.5. Convergent ramp responses up to 4 deg amplitude are shown for (a) experimental and (b) model simulation conditions. Experimental curves are individual responses of a subject. Ramp velocity, in deg/sec, is shown next to each curve. Top curve for convergent step responses. Reprinted from Hung et al (1986), pg. 1025, Fig. 3, with permission of © IEEE.

To simulate the experimental responses to various stimuli accurately, a dual-mode vergence model was developed by Hung et al (1984) (see Fig. 11.6a-c). In this model, both the fast and slow components have disparity (or vergence error) and velocity thresholds to provide operation within their respective ranges. The fast open-loop component used target position and velocity information to produce the appropriate step movement, and the slow closed-loop component used visual feedback to reduce the disparity and fuse the binocular retinal images. Simulation results showed accurate fit to the experimental responses for pulse, step, and ramp stimuli, and in addition showed reduced phase lag to the sinusoidal stimulation mentioned earlier (Hung et al, 1984) (see Fig. 11.5b). Thus, this dual-mode model provided an accurate representation of the vergence system that simulated vergence responses to a variety of stimuli. Moreover, recent findings in our laboratory showed that vergence responses to symmetric step disparities under a variety of naturalistic conditions (e.g., in the light and dark, under voluntary control, etc.) also fell on the main sequence for simple step disparities, thus indicating a common motoneuronal controller signal for the generation of symmetric vergence step responses regardless of the stimulus spatial pattern or mode of initiation (Hung et al, 1994).

Figure 11.6. (See next page). (a) The overall model of the vergence system showing slow and fast components in the forward loop. The slow and fast component responses are summed to give VR1. Internal feedback from VR1 is summed with vergence error VE to give an estimate of target position. The plant represents the mechanical properties of the eyeball and musculature and is assumed to have unity gain for the vergence stimulation. The vergence response VR is subtracted from the vergence stimulus VS to give the vergence error VE. (b) Slow component in the forward loop: delayed vergence error (VE1) is VE delayed by 200 msec (DELAY1). Error magnitude limiter (up to 1 deg) and error velocity limiter gain element (up to 2 deg/sec) simulate range of slow component dynamics. The time constant $1/A$ is 10 sec. Gain G1 was determined via simulation to be 30. (c) Fast component in the forward loop: the vergence error VE is summed with VR1 to give an estimate of the target position. The delay element (DELAY2) represents the effective delay throughout the fast component. The estimated target velocity above a threshold of 1.7 deg/sec is used to trigger the sampler. The sampler enables the predictor to use estimated target position and velocity to predict the future position of, for example, the ramp stimulus. After triggering, the threshold increases slightly to 2.1 deg/sec. This accounts for the initial step but subsequent smooth following seen in response to 1.8 deg/sec ramp stimulus. If estimated stimulus velocity remains constant, the sampler repeats every 0.5 sec. This accounts for the staircase step-like responses to ramp stimuli. Sudden large changes in velocity will reset the sampler. This accounts for the ramp-pulse data. The predictor also reduces its calculation time, thus reducing DELAY2, for repetitive stimuli such as sinusoids. This accounts for the small phase lag found in sinusoidal responses. Reprinted from Hung et al (1986), pg. 1023, Fig. 1, with permission of © IEEE.

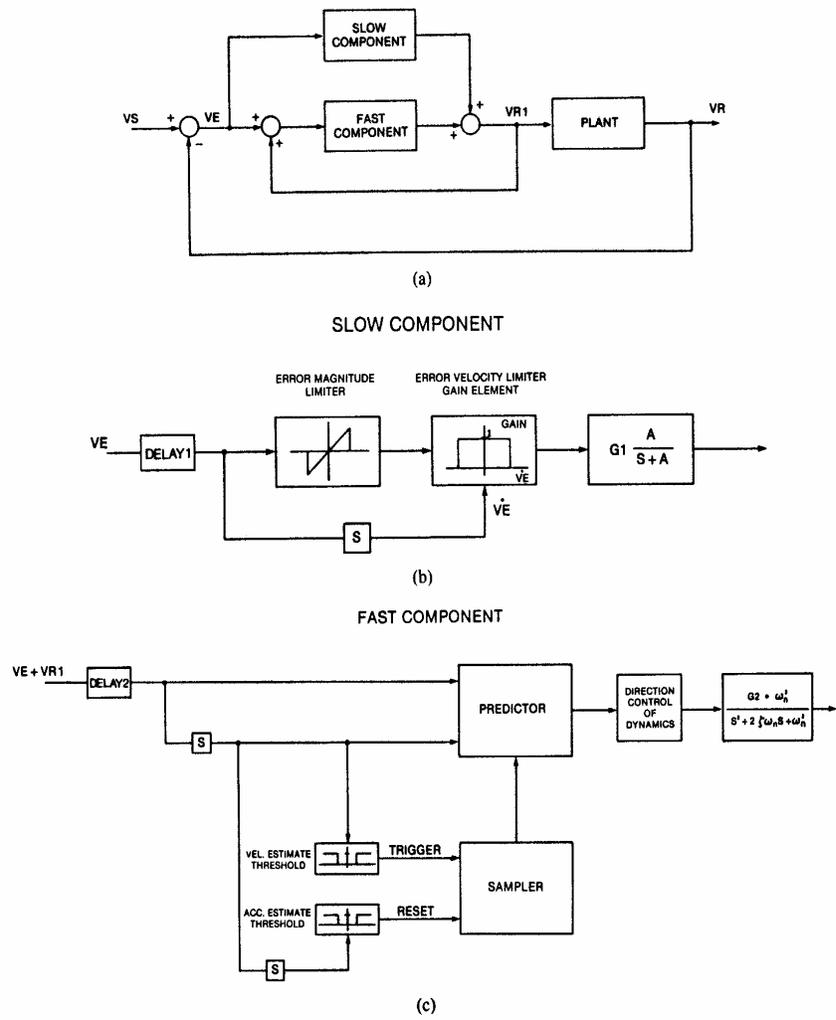


Figure 11.6 - See figure legend on previous page.

11.2.4 Saccade-Vergence Interactions

If we had only one eye, the innervational pattern for directing one's gaze, for example to the right, would be relatively simple. It can be describe by the Descartes-Sherrington principle of reciprocal innervation (Ciuffreda and Stark, 1975) (see Fig. 11.7A), whereby the right extraocular muscle (EOM) receives a positive increment, and the left EOM simultaneously receives a negative increment, of innervation. However, since we have two eyes, both eyes need to be innervated to direct one's gaze in space appropriately (see Fig. 11.7B), and this could result in relatively complicated innervational patterns, especially for asymmetrical target changes. Two primary innervational scenarios are possible. In the first, the two eyes are innervated separately as two independent units. The brain pre-calculates the exact amount of innervation needed in the extraocular muscles of each eye, so that the two eyes are driven independently and directly to their final bifixation position. In the second, the innervations retain their separate drives to the saccadic and vergence systems. The brain in essence calculates the "cyclopean" (i.e., single common eye) amount of version (i.e., lateral shift) and vergence (i.e., disparity-based depth change) needed, and distributes half of the these version and vergence amounts to each eye, and then drives the two eyes using the separate saccade and vergence signals that are combined at each eye to arrive at the final bifixation position (see Figs. 11.1A,B. Therefore, although two sets of signals (saccade and vergence) are used, they innervate the two eyes together as if they were one unit.

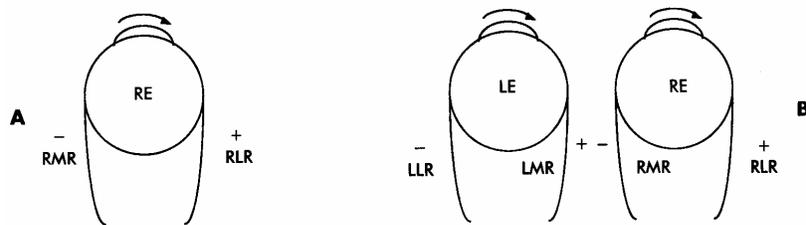


Figure 11.7. A. Descartes-Sherrington law of reciprocal innervation. B. Hering's Law of Equal Innervation. *RE*, right eye; *LE*, left eye; +, increased innervation or excitation; -, decreased innervation or inhibition; *RLR*, right lateral rectus; *RMR*, right medial rectus; *LLR*, left lateral rectus; and *LMR*, left medial rectus. Reprinted from Ciuffreda and Tannen (1995), pg. 6, Fig. 1-3, with permission of Harcourt Health Sciences.

Hering's theory of binocular vision is based on the second scenario (Hering, 1977), whereby the two eyes are innervated as though they were one. This concept of equal innervations for saccade and vergence movements in the two eyes was codified as Hering's Law of Equal Innervation (Hering, 1977; Ono, 1983; Findlay and Harris, 1993). It should be noted that Hering did not consider unequal movement of the two eyes to be inconsistent with his hypothesis (Ono, 1983). This is because summation of saccade and vergence signals could result in unequal interocular movement amplitudes, even though the underlying control signals were simply the required cyclopean-determined amounts of version and vergence innervations.

Hering's law, however, requires an intact vergence system. For example, Alpern and Ellen (1956) found that under normal binocular viewing conditions, the eye movements were consistent with Hering's law. However, for accommodative vergence movements (with the non-fixating eye occluded, and therefore with disparity vergence feedback rendered ineffective), the viewing eye remained relatively fixed during such a movement, thus suggesting a gross violation of Hering's law. The main difference is that the disparity vergence feedback loop is open during accommodative vergence, but is closed under binocular viewing.

Some investigators (Clark and Crane, 1978; Steinman and Collewijn, 1980; Ono, 1983; and Erkelens et al, 1989) have considered unequal movements of the two eyes under binocular viewing to be a violation of Hering's law. For example, Ono (1983) set out to investigate the implications of Hering's law on a combination of version and vergence movements. An example of one of his recorded asymmetrical eye movement traces along with a geometrical view of the movement is shown in Fig. 11.8. Ono (1983) concluded from his analyses that "... when the disjunctive movement combines with the slow conjunctive movement, the difference in velocities for the two eyes is slightly less than that predicted from the additivity proposition. When the disjunctive movement combines with the saccadic movement, the difference in magnitude and velocity are larger than those predicted." Thus, he observed several different patterns that did not fit the additivity hypothesis, which suggested a violation of Hering's law. Erkelens et al (1989) measured oculomotor responses to large target shifts of up to 40 and 50 deg and found that unequal saccades could account for 95% of the vergence required for divergence and 75% for convergence. Again, this suggested that the movements violated Hering's law. However, it can be shown that these apparent violations of Hering's law can be explained by the difference in latencies between the two eyes due to differences in their individual neuronal pathways (see Differential Latency

Theory, detailed in Section 11.3; Hung, 1998a,b). In fact, these movements can actually be shown to conform with Hering's law.

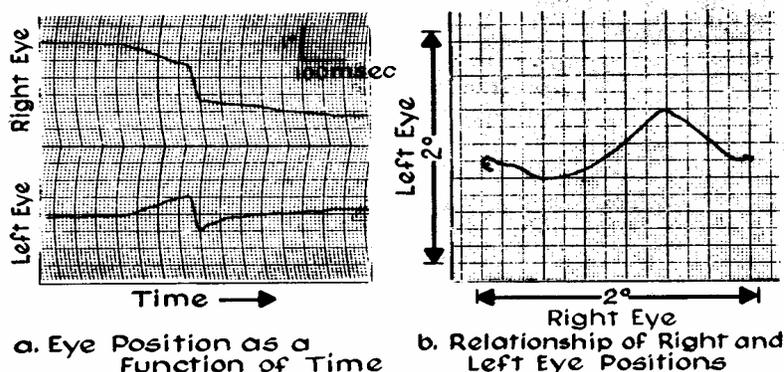


Figure 11.8. a) Sample record of eye movement when the target is abruptly moved to a different distance and different direction, equivalent to 0 deg in the LE and 2 deg leftward in the RE b) Representation of the same movement in Cartesian (LE - rightward is up, vs. RE - rightwards is to the right) coordinates. Movement begins at right with an initial convergence (RE leftward and LE rightward), followed by saccade (both RE and LE leftward), and then followed by additional convergence to the final position. Reprinted from Ono (1983), pg. 383, Fig. 11.4, with permission of K. J. Ciuffreda, the copyright holder.

Other investigators (Enright, 1984, 1986, 1992; Collewijn et al, 1988; Maxwell and King, 1992; Zee et al, 1992; Erkelens et al, 1989) proposed that not only some movements violated Hering's law, but that the vergence movement was in fact facilitated by the ongoing saccade. For example, Maxwell and King (1992) found in macaque monkeys that for symmetric stimuli, vergence peak velocities fell on the normal main sequence (Hung et al, 1997). However, for asymmetrical stimuli, the vergence peak velocities, which were taken simply as the difference between the right and left eyes, were much higher than main sequence values. They believed that the oculomotor system was able to use these disjunctive transients to shorten the length of time required to complete the vergence movements. Zee et al (1992) investigated oculomotor responses to step stimuli in space. They used a graphical method, which projected a combined vergence trace onto a pure vergence trace of the expected amplitude, and measured the difference. Any difference was presumed to reflect a facilitation process. They concluded that horizontal vergence was facilitated by both horizontal and vertical saccades, although more so with horizontal saccades. However, the transient vergence movements found in these studies often occurred in the

opposite direction to that required for the intended vergence movement. Thus, they did not assist, and in many cases actually opposed, the ongoing vergence movement. As discussed in detail in Section 11.3 (Hung, 1998a,b), transient divergence during combined saccade-vergence movements is an inherent consequence of the latency difference between the neural pathways in the two eyes, and not due to facilitation of the vergence movement.

Enright (1984; 1986; 1992) examined changes in vergence movements during responses to asymmetrical targets, with normal disparity, blur, and proximal cues present. He used a video recording technique at a relatively slow frame interval of 33 msec to record eye movements. He found that a large portion of the total change in vergence occurred during saccades, with values ranging from 40% for certain tasks to 100% when large version was combined with a small vergence stimulus. Enright also noted that additivity of vergence and saccadic movements was violated, since the rate of vergence change during saccades was much greater than that found either before or after the saccade. He therefore concluded that vergence was facilitated by the saccade during the combined movement. However, since dynamic changes, such as small to moderate saccadic movements, could occur within the 33 msec. sampling interval he used, the presumed facilitation may have occurred, but it could also have been an artefact of under-sampling.

11.2.5 Saccadic Suppression/Omission and Vergence Suppression

The numerous oculomotor movements one makes in daily life produce rapid changes in the retinal image of the visual scene. For example, a saccade is accompanied by a complete lateral shift in the visual frame of reference, a blink briefly darkens the field of view, and a vergence movement changes the binocular reference to depth. Nevertheless, an observer perceives the world as being both continuous and stable. Much of this sensation of perceptual continuity can be attributed to the correlated transient reduction in visual sensitivity both immediately before, during, and after the motor response. This has been observed for saccades (Alpern, 1953; Stark, 1968, Campbell and Wurtz, 1978), blinks (Volkman et al, 1980), and vergence (Manning and Riggs, 1986; Hung et al, 1989, 1990). Thus, despite the apparent continuity of perception, during a combined saccade-vergence movement, there is a true reduction in visual sensitivity for up to 100 msec or so. Such a disruption would preclude accurate visual feedback guidance and control of vergence by saccades. Thus, this too does not support the notion of dynamic facilitation.

11.2.6 The Fast Learning Mechanism for Asymmetrical Targets

van der Steen and Bruno (1995) noted that “When the objects of interest are at optical infinity, saccades are essentially conjugate, with the exception of a small transient divergence during the saccade (Collewyn et al, 1988; Zee et al, 1992). However, when we change fixation between objects at different directions and distances, as we do under natural circumstances, saccades become disconjugate which allows to accomplish most of the required vergence during the saccade (Erkelens et al, 1989)”. Recently, investigators proposed a fast learning mechanism to provide appropriate disconjugacy in response to asymmetrical targets following brief periods of training (Eggert and Kapoula 1995; Kapoula et al, 1995; van der Steen and Bruno, 1995; Averbauch-Heller et al, 1999; Bucci et al, 1999). The training consisted of, for example, viewing through different parts of prisms in the two eyes which displaced the images, or viewing different-size images in the two eyes. These results indicate that some type of motor learning may have occurred. Yet, these peripheral modifications, which occur perhaps at or beyond the final common pathway for each eye, are still consistent with Hering’s law of equal central innervation to the two eyes.

11.2.7 The Neurophysiology of Saccade-Vergence Interactions

Tamler et al (1958) used electromyography to examine whether the lack of movement in the viewing eye during asymmetrical vergence violated Hering’s law. This involved inserting a small microelectrode into selected extraocular muscles and observing changes in firing frequency of muscle motor units. They found that there was co-contraction of the opposing horizontal recti muscles of the viewing eye during asymmetrical vergence. Hence, they concluded that this confirmed Hering’s view that there was a peripheral adjustment (i.e., cancellation) of opposing version and vergence central innervations, and thus Hering’s law was not violated during asymmetrical vergence.

Keller and Robinson (1972) found that in the monkey abducens units there was “no type of eye movements, version or vergence”, that was “the exclusive product of a particular subset of oculomotor neurons”. In an additional experiment, they tested accommodative vergence, in which the viewing eye remained relatively stationary while the fellow occluded eye varied in position due to blur-driven accommodative vergence. From these experiments, they concluded that the “version and vergence commands are summed centrally with the net result appearing as more global neural

activity in a shared final common path. That is, during asymmetric vergence, the vergence and version neural commands obey reciprocal innervation and simply cancel in each motor nucleus of the horizontal recti of the non-moving eye" (see Fig. 11.7). Thus, their conclusion supports Hering's law.

Mays (1984), and Judge and Cummings (1986), found that premotor neurons in the mesencephalic reticular formation, 1-2 mm dorsal and dorsolateral to the oculomotor nucleus, were involved specifically in the control of vergence and discharged in relation to convergence angle. Also, Mays et al (Mays & Porter, 1984; Mays et al, 1986) found motoneurons that displayed signals related to both position and velocity of the movement. In addition, there were a small number of divergence burst neurons, and burst-tonic cells that combined vergence velocity and position, respectively, in their output (Zee & Levi, 1989). Anatomical studies of the oculomotor nucleus revealed three aggregates of cells: subgroup A was located in the ventral and rostral region, subgroup B was located in the dorsal and caudal region, and subgroup C located in the dorso-medial and rostral region. Zee and Levi (1989) speculated that subgroup C, comprising of the smallest cell bodies that were traced using radioactive label to the small outer layer fibers of the medial rectus muscle, was perhaps selective for vergence. Moreover, in comparing the vergence and pursuit subsystems, the position and velocity signals from these different subsystems were found to be generated independently and were distributed to motoneurons in a way that was not highly correlated between the subsystems (Gamlin & Mays, 1992). Finally, Gamlin and Clarke (1995) found cells in the medial nucleus reticularis tegmenti pontis (NRTP) that increased their activity for far responses and other cells that increased their activity for near responses. Although these cells were often encountered close to neurons that displayed saccade-related activity, their activity did not increase with conjugate eye movements. Therefore, there was anatomical and neurophysiological evidence for separate control of vergence and version, although it may be speculated that the physical proximity of neurons for these two systems allowed for interactive influences. Nevertheless, as further support for separation of control, Keller and Robinson (1972) showed that although the control signals for saccade and vergence travelled along the same motoneuron in the final common pathway, there was no evidence of (nonlinear) interaction between saccade and vergence signals. Therefore, these neurophysiological findings also support Hering's law of equal central innervation to the two eyes.

11.2.8 Models of Saccade-Vergence Interactions

Zee et al (1992) proposed three models for saccade-vergence interactions. The first model hypothesized the existence of a separate class of saccade-related vergence burst neurons (SVBN) (see Fig. 11.9), which were gated by the omnidirectional pause neurons (OPN) and generated premotor horizontal vergence commands but only during saccades. The second model hypothesized separate right and left eye saccade burst neurons that received not only conjugate, but also equal and oppositely-directed vergence error signals. The difference between the right and left eye burst neurons was used to produce a saccade-related vergence command. The third model proposed that the facilitation of vergence during saccades was a result of an increase in gain of premotor vergence velocity neurons caused by a lifting of the inhibition by the OPN during saccades. Their model simulation results favored the first and third models. They also proposed that disconjugate adaptation to, for example, wearing anisometric spectacle correction, which creates unequal and also continually different amounts of prism vergence demand through different parts of the spectacle lens in each eye (thus resulting in asymmetrical saccades), could be accounted for in their model by modification of the SVBN to link a change

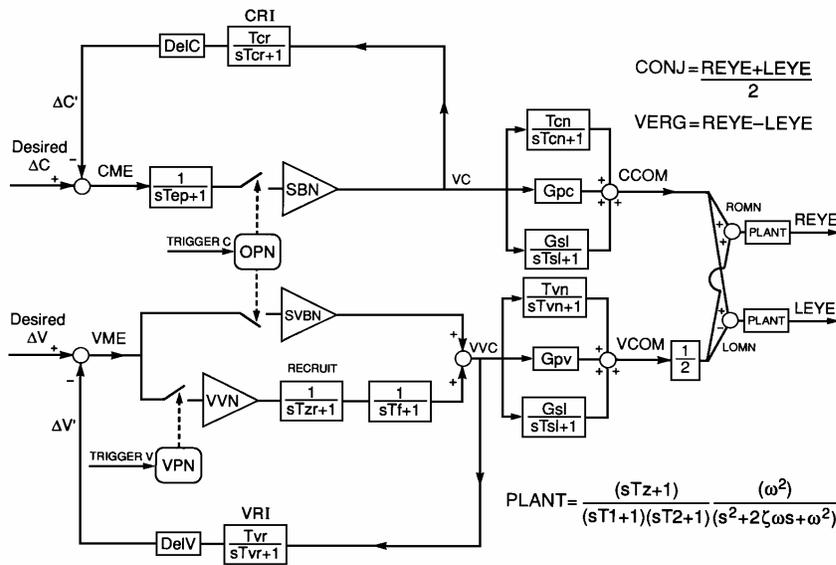


Figure 11.9. Saccade-Related Vergence Burst Neuron (SVBN) model of Zee et al (1992). Reprinted from Zee et al (1992), pg 1638, Fig. 19, with permission of The American Physiological Society.

in ocular alignment automatically with saccades, even in the absence of disparity cues. However, it should be noted that such linkage would require the SVBN to be continually changing and responding nearly instantaneously to each change of gaze. Such a mechanism seems to be physiologically unrealistic.

Mays and Gamlin (1995) presented a similar model in which the inhibition by the OPN is shared by the saccadic and vergence systems. They proposed that there is normally a “weak” inhibition of the vergence burst cells by the OPN to allow them to fire in the absence of saccades. The inhibition is released during a saccade in a mixed vergence-saccade movement to result in unequal saccades.

Both of the above models involve modification of neural signal processing of vergence during the saccade. However, since saccadic latency is approximately 200 msec, it would require 200 msec before the saccade can appropriately modify the vergence movement. That is, the modification would be for the stimulus disconjugacy that occurred 200 msec earlier. Thus, such a rapid-modification mechanism also appears to be physiologically unrealistic.

Moreover, it may be argued that the saccade and vergence signals are pre-planned based on the target positions. Such a saccadic modification of vergence would appear to be purposeful, and therefore should assist vergence in arriving at its final position. However, it can be shown (see Hung, 1998a,b; Collewijn et al, 1997) that in a number of experimental conditions when a convergence movement is required, the transient movement is that of divergence, which is the opposite of that required for a purposeful assisted movement. Thus, neither timing nor directionality are consistent with these models.

This leads to a dilemma as to why the visual system would process and modify responses that do not lead to a benefit in responsiveness. Indeed, to modify the response, processing would be required to determine whether a saccade and vergence are about to occur simultaneously, and then energy expended to provide the inhibitory signals, but without any apparent benefit. This would not be consistent with parsimony of neural signal processing exhibited in the nervous system (Glassman, 1999). The answer may lie in the fact the transient vergence during saccades is simply a result of neuronal latency difference between the two eyes, and thus there was no intended purposeful, higher-level, modification of vergence during saccades. Such a mechanism has been proposed by Hung (1998a,b) in the Differential Latency Theory, and is described in Section 11.3.

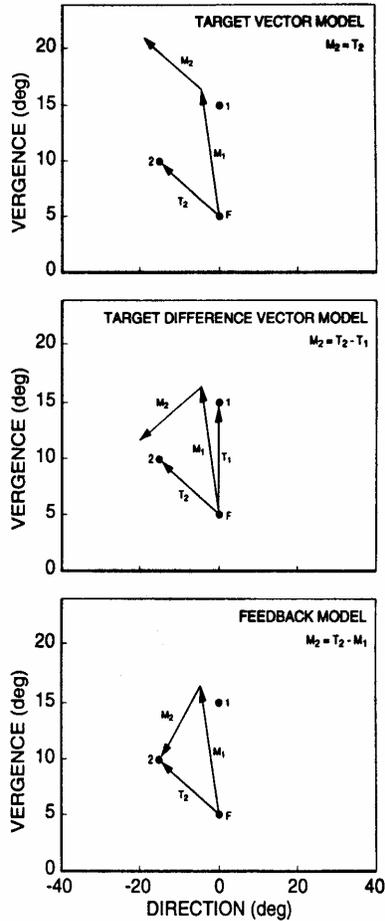


Figure 11.10. Models tested by Krommenhoek and van Gisbergen (1994). The three models have different predictions for the second movement, as illustrated by an example where the first movement was not precisely directed at the first target (1). Note that only the feedback model can correct the second movement to the final target (2) for an error in the first movement (T_1 first target position, T_2 second target position, M_1 first movement, M_2 second movement, F fixation position during target presentation). Reprinted from Krommenhoek and van Gisbergen (1994), pg. 96, Fig. 1, with permission of Springer-Verlag.

Krommenhoek and van Gisbergen (1994) examined eye movement responses in 5 subjects to test three hypothesized models regarding the control of version and vergence (see Fig. 11.10). They used a paradigm in which two target positions in direction and depth were presented in sequence; after the second target was extinguished, the subject was instructed to direct his fixation in the dark to the imagined position of the two targets in the same sequence. In all three models, the

eyes would initially make a movement to the first target, T_1 . For Model 1 (Target Vector), it is predicted that the eyes would then make a movement to the second target, T_2 , based on the vector direction from the subject to T_2 prior to the initiation of the movement sequence. For Model 2 (Target Difference), it is predicted that the eyes would move based on the vector difference between T_1 and T_2 estimated prior to initiation of the movement sequence. Finally, for Model 3 (Feedback), it is predicted that the eyes would move based on non-retinal local feedback loops (Scudder, 1988) derived from the efference copy signals. They found that the Feedback Model provided the best fit to the experimental data. Moreover, they

proposed that a higher center, such as the lateral intraparietal (LIP) area in the parietal cortex, was involved in the control of saccade and vergence eye movements, where the commands for version and vergence components of binocular gaze shifts may be represented by a single control center.

Recently, Chaturvedi and van Gisbergen (1999) produced brief-duration electrical microstimulation of the deep layers of the right superior colliculus in the monkey to elicit a nearly pure leftward conjugate movement. The microstimulation was then applied during eye movements to asymmetrical targets in space to determine whether such stimulation influenced the timing and metric (amplitude and direction) of the vergence portion of the resultant response. Experimental results showed that whereas early electrical stimulation resulted in a clear vergence component, perturbation during midflight “markedly curtailed the ongoing vergence component.” Thus, instead of facilitating the vergence response, electrical stimulation that should have mimicked a saccadic assist signal actually reduced the vergence response. They proposed a model in which stimulation of the superior colliculus elicited not only a conjugate signal, but also a “zero vergence-change” signal, which competed with the visually-driven convergence signal to result in a compound (step-slide-step shaped) time course. This simulated the diminution of the vergence response, but was not able to model any proposed facilitation.

The source of saccadic disconjugacy has also been described by Averbuch-Heller et al (1998). They ascribed it as being primarily due to faster “abducting saccades ... than adducting saccades”, and is thus based on difference in saccadic speeds rather than latencies, as in Hung’s theory (Hung, 1998a,b). However, in the reference that they cited, Abel et al (1979) actually found “no overall trend” regarding abducting and adducting saccades; moreover, they found that for both eyes, centering saccades were faster than eccentrically-directed saccades. Thus, this could not serve as a basis for disconjugacy between the two eyes during a saccade.

11.3 SACCADE-VERGENCE DYNAMIC MODEL: HUNG’S DIFFERENTIAL LATENCY THEORY

The Differential Latency Theory states that the transient divergence seen during saccade-vergence responses can be accounted for by a small difference in the latencies between the contralateral and ipsilateral neural pathways driving the conjugate eye movement (Hung, 1998a,b) (Fig. 11.11). The efferent pathway from the deep layers of the contralateral superior colliculus crosses the predorsal bundle (or tectospinal tract) to arrive at the ipsilateral abducens nucleus (Sparks, 1986), which in turn sends axons to the

ipsilateral lateral rectus muscles as well as the contralateral medial rectus muscle via the medial longitudinal fasciculus and the oculomotor nucleus (Leigh & Zee, 1991). Due to the brief latency difference (~ 6 msec) between these two neuronal pathways (Smith et al, 1970), there is a transient difference in the movement in the two eyes, which is exhibited as transient divergence during conjugate eye movement in the ipsilateral direction (see Fig. 11.11).

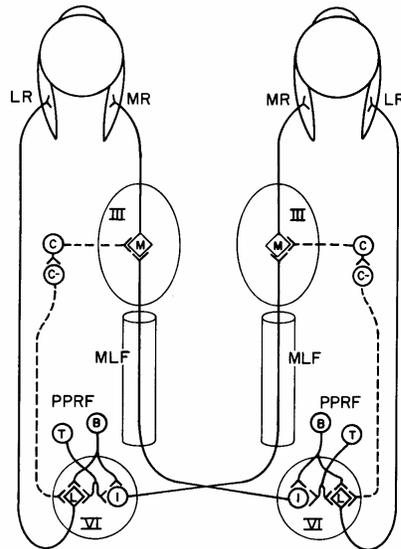


Figure 11.11. Neural pathways for saccade and vergence. For saccades, horizontal burst neurons (B) and tonic eye position neurons (T) in the paramedian pontine reticular formation (PPRF) provides similar input signals to both lateral rectus motoneurons (LR) and internuclear neurons (I) in the abducens nucleus (VI). The abducens internuclear neurons cross the midline and ascend in the medial longitudinal fasciculus (MLF) to drive the medial rectus motoneuron. Also, for vergence, the presumed complementary vergence signals, c and c-, innervate the ipsilateral MR and LR to drive the response (Reprinted from Mays (1983), pg. 656, Fig. 20.4, with permission of K. J. Ciuffreda, the copyright holder). Hung's Differential Latency Theory is based on a difference in latency between ipsilateral and contralateral neural pathways. For example, a signal from the right (contralateral) superior colliculus (not shown) crosses over to innervate the left (ipsilateral) VI nucleus. From here, neuronal signal are sent to the left LR as well as to the right MR via the MLF and the oculomotor nucleus (III). For a leftward saccade, there is a slightly longer latency (by about 6 msec; Smith et al, 1970) through the MLF to the right MR than the left LR, resulting in a brief transient divergence (Hung, 1998a,b).

In daily life, target changes are almost always asymmetrical in nature, and therefore must involve both saccade and vergence movements occurring in an interactive manner. Accommodation has a much longer latency (~350 msec) than those for saccades (~200 msec) and vergence (~180 msec), and therefore plays a relatively minor role in these early-occurring dynamic interactions. Saccadic and vergence eye movements direct gaze laterally and in depth, respectively. Indeed, the interactions between saccade and vergence have been of considerable interest in recent years, because of the erroneous hypothesis put forth by some investigators that saccades facilitated vergence dynamics, and that the extent of the facilitation was based on the configuration and richness of the visual scene (Erkelens et al, 1989; Collewijn et al, 1995; Enright, 1986; Zee et al, 1992). The following is a discussion of experimental and modeling development based on the Differential Latency Theory (Hung, 1998a,b):

Experiments were conducted to examine the trajectories of the bifixation point in space (called top-view trajectories) during combined saccade-vergence movements under both instrument-space (IS) and free-space (FS) conditions (Hung, 1998a,b) (Fig. 11.12). Then, a saccade-vergence model was constructed and simulations performed to test the Differential Latency Theory to determine whether the latency difference between neuronal pathways can result in the same top-view trajectories seen in the experiments (Hung, 1998a,b) (Fig. 11.13).

The experimental apparatus used under the instrument-space (IS) (Zhu, 1995; Hung et al, 1994, 1997) and free-space (FS) (Collewijn et al, 1997) environments are shown in Figs. 11.12A, B, respectively. The FS environment corresponds to the natural viewing of objects in a scene that consists of all the usual cues such as blur, disparity, size, perspective and overlap, etc., whereas the IS environment corresponds to a more restricted viewing of targets in an optical assembly that consists only of retinal disparity cues to the two eyes (Hung et al, 1994). It was found that under both IS and FS viewing environments, oculomotor responses to asymmetrical target displacements exhibited transient divergence, mainly for far-to-near target displacement, with the occurrence of large divergence transients being greater under the FS (Fig. 11.14A) (Enright, 1984; Erkelens et al, 1989; Zee et al, 1992) than the IS (Fig. 11.14B) environment (Hung and Ciuffreda, 1996; Hung, 1998a,b). These transient divergent movements were seen as large loops in the dynamic binocular fixation two-dimensional top-view plot (see Fig. 11.14A, bottom subplot, loop portion of the trajectory), which deviated from the classical iso-vergence curves (Ono and Nakamizo, 1977). The movements were generally followed by a pure vergence response, which is seen as radially-directed (i.e., towards the midpoint between the eyes) traces (Figs. 11.14 A,B). A variety of targets at

different distances and directions were presented, the subjects' responses were recorded, and the trajectories plotted as top-view plots (Hung, 1998a,b).

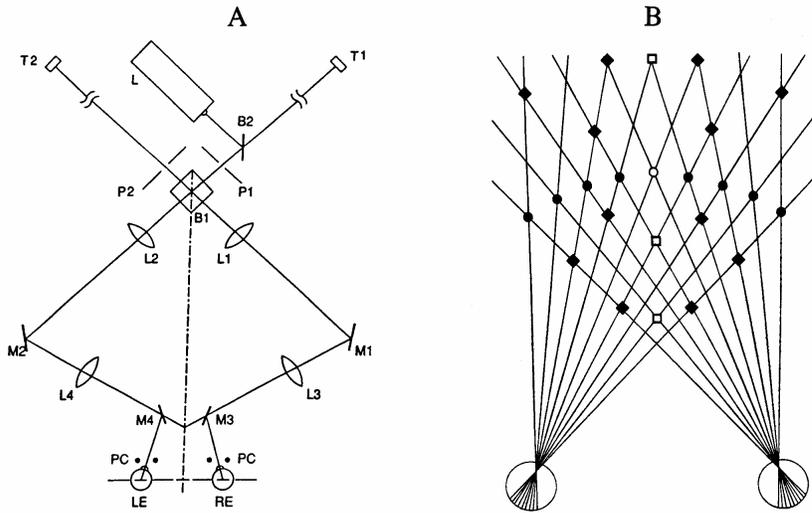


Figure 11.12. Experimental apparatus for the (A) Instrument-space environment: Dynamic Binocular Stimulator (DBS) (Semmlow and Venkiteswaren, 1976); Pinhole apertures P1 and P2 are optically conjugate to the pupils of the left and right eye, respectively. When the aperture of P1 and P2 are reduced to < 1 mm, blur stimulation is eliminated and cannot directly or indirectly (through accommodative feedback) influence the eye movement response. Since T1 and T2 are located 28.5 cm from their respective pinhole apertures, a horizontal target movement on an oscilloscope of 1 cm equals 2 deg of angular change in the eye. The target in each oscilloscope consists of a thin vertical line 0.25 deg wide and 8 deg high, presented as bright bars against a totally dark background. The relatively long bar targets assist in horizontal binocular fusion of the targets. The beam splitter B1 is used for alignment only and is removed during the experiments. Adapted from Hung et al (1994), pg. 3488, Fig. 2, with permission of Invest. Ophthal. Vis. Sci. (B) Free-space environment: Schematic diagram of the target positions used in the free-space experiments. The initial fixation point is indicated by the open circle (O) at 28.5 cm from the subject's eyes. Isovergence points are represented by the filled circles (●). Pure vergence points are represented by open squares (□), while asymmetrical target positions are represented by filled diamonds (◆). Not shown is a symmetric target at optical infinity (20 ft away). The separation between the lines is 4 deg (drawing not to scale).

To simulate the various experimental top-view trajectories, a saccade-vergence model was constructed based on Hung's Differential Latency Theory (Fig. 11.13). The two main characteristics of the model were: there was no central computation for assisting the vergence movement; and there was included in the pathway from the ipsilateral abducens nucleus to the

contralateral medial rectus muscle an increased latency of 6 msec. It turned out that this latency was crucial in producing the transient divergence (which was reflected in the loops seen in the top-view trajectories) during asymmetrical eye movements. The dynamic saccade-vergence model consists of both conjugate pulse-step and disjunctive step controllers. Conjugate and disjunctive transfer functions are used to provide appropriate overall dynamics for saccade and vergence, respectively. Each conjugate (C1 to C4) and disjunctive (D1 to D4) gain has a nominal value of 0.5. The extraocular muscle plant is based on that by Robinson (1973) and Zuber and Stark (1968) and is given by $1/(0.064s + 1)(0.007s + 1)$. The conjugate signal to the MR of the contralateral adducting eye is set to be 6 msec longer than that for the LR of the ipsilateral abducting eye. For example, for a rightward saccade, the delay at C2 is 6 msec longer than that at C4 (see Fig. 11.13). A 1st-order filter with a time constant of 20 msec is placed following the delay stage to provide smooth transients. The model simulation results for conditions similar to the experimental trials seen in Figs. 11.14A and B are shown in Figs. 11.15A and B, respectively. The correspondence between model and experimental results was shown for other target conditions as well, thus demonstrating the accuracy of the model in simulating experimental results under a variety of asymmetrical stimulus conditions (Hung, 1998a,b). Therefore, this relatively simple model was able to account for the previously unexplained vergence behavior during combined saccade-vergence movements.

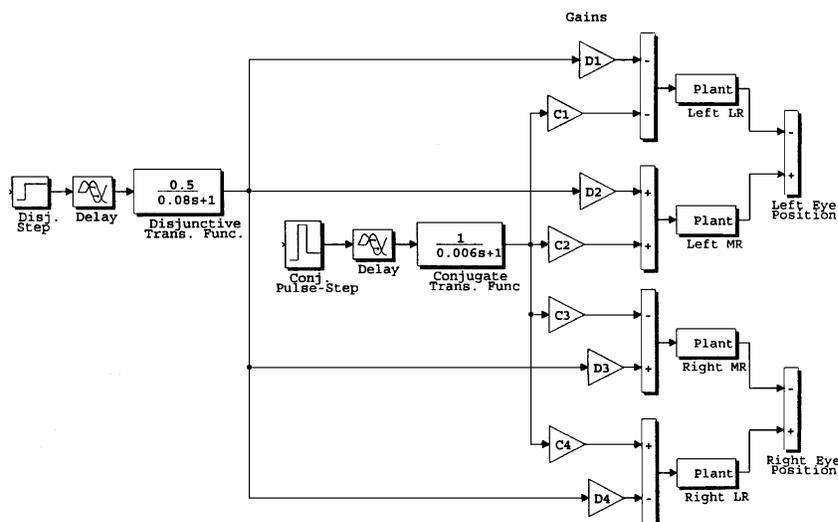


Figure 11.13. Dynamic saccade-vergence model. See text for details. Reprinted from Hung (1998a), pg. 10, Fig. 1, with permission of Med. Sci. Res.

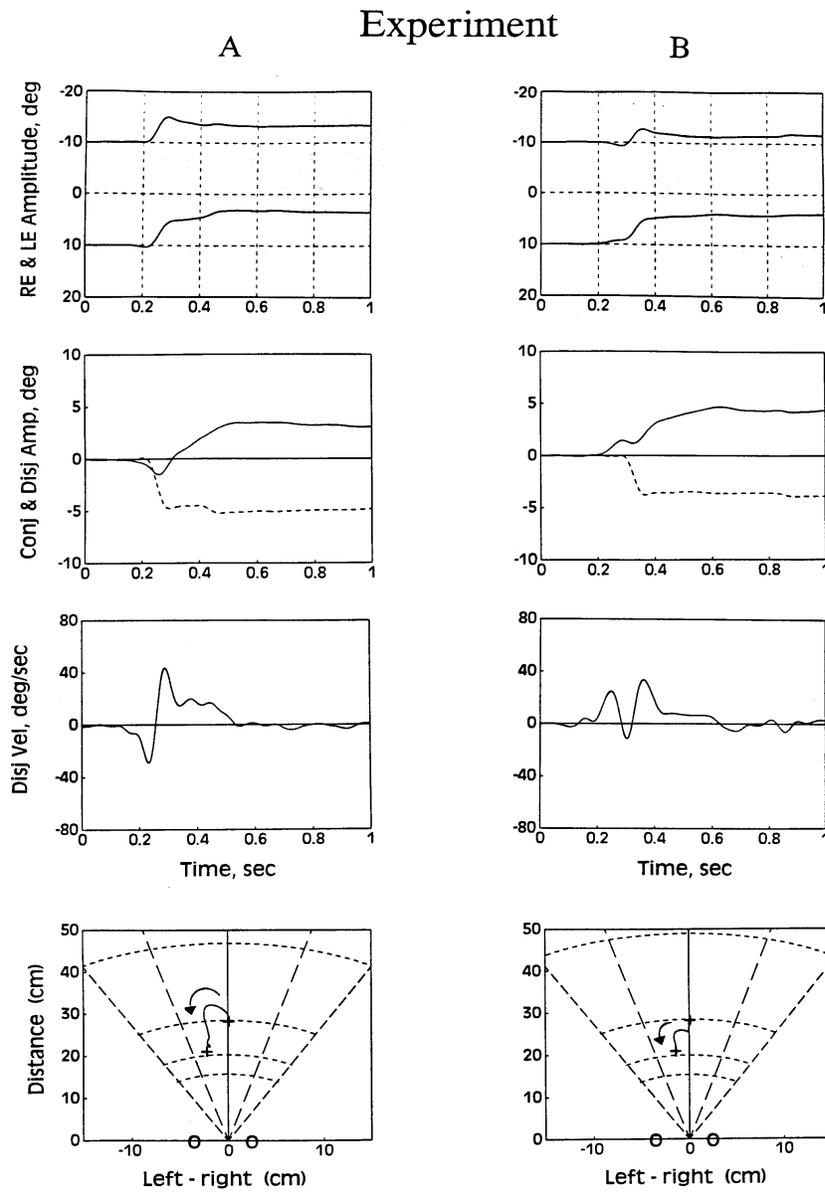


Figure 11.14 - See figure legend two pages down.

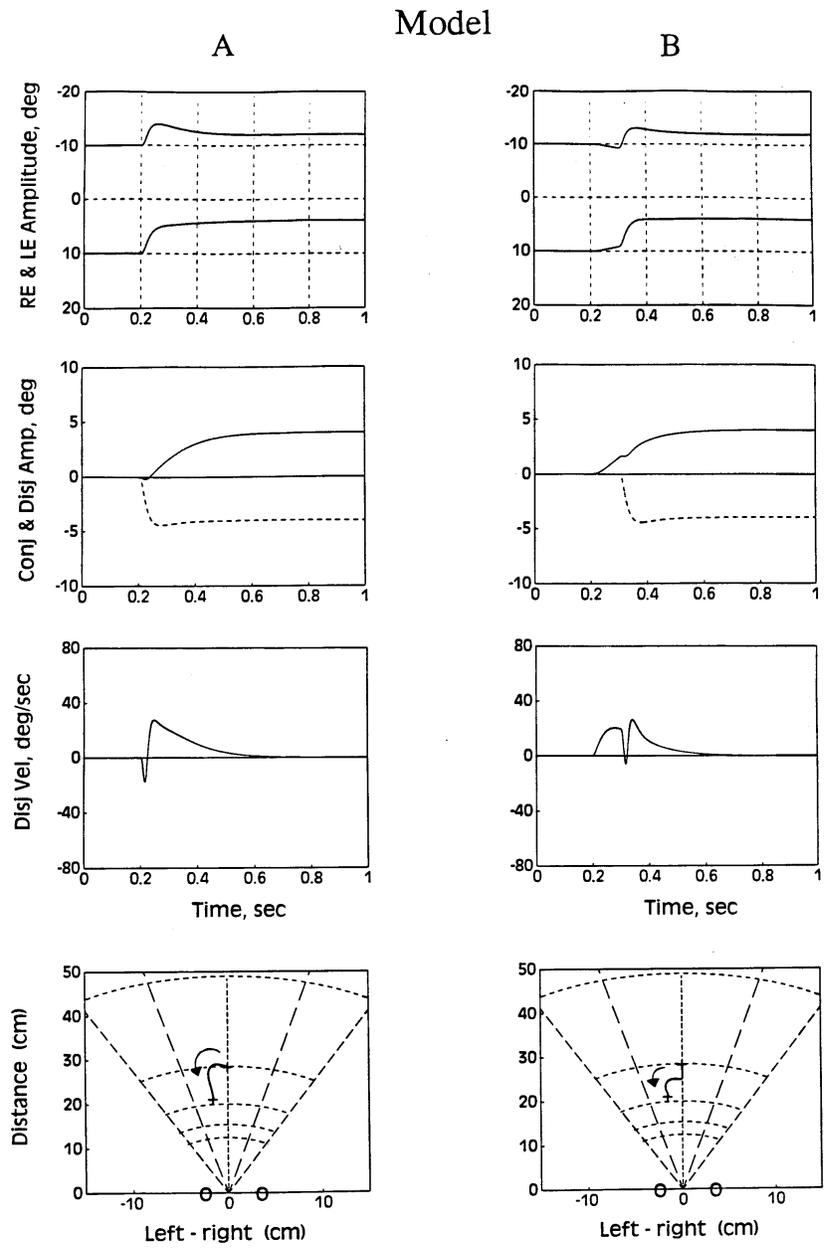


Figure 11.15 - See figure legend next page.

11.4 SUMMARY

In summary, the higher neural centers are concerned primarily with the generation of the appropriate vergence and version controller signals. The overall goal is to direct the eyes from the initial fixation point to the new target position. The various trajectory shapes can be accounted for by the difference in peripheral neural delays between the two eyes, as well as the timing between saccade and vergence controller signals. Nevertheless, all the different trajectories arrive accurately at the target. Thus, the parsimony and simplicity offered by the present Differential Latency Theory model (Hung, 1998a,b) reflects the essence of Hering's law (Hering, 1977), which states that the two eyes act as one, so that the separate conjugate and disjunctive controllers work together to drive the eyes toward the target in space.

Figure 11.14. (See 2 pages back). (A) Representative experimental time traces under the free-space (FS) environment for stimulus requiring a response of -4 deg in the LE and -8 deg in the RE (Positive and negative numbers represents rightward and leftward target displacement, respectively), corresponding to 4 deg of convergence and 6 deg of leftward versional movement (Subj. SW). (B) Representative experimental time traces under the instrument-space (IS) environment for stimulus requiring a response of -2 deg in the LE and -6 deg in the RE, corresponding to 4 deg of convergence and 4 deg of leftward versional movement (Subj. GH). Top Graph - Left eye (LE, upper) and right eye (RE, lower) time traces. Second Graph - Conjugate (dotted) and disjunctive (solid) amplitude time courses. Third Graph - Disjunctive velocity time course. Bottom graph - Top-view binocular fixation trajectories corresponding to the movements shown in top graph. The initial central fixation point and the target are shown as "+" symbols. The circular-shaped iso-vergence arcs (dotted) are separated at 5 deg intervals, whereas the radial lines (dashed) are separated at 10 deg intervals. Note that for the bottom graph under the FS environment (A), the trajectory, starting from a position indicated by the central fixation cross, consists of an overshoot loop followed by a radially-directed vergence movement towards the target. On the other hand, under the IS environment (B), the trajectory consists of an initial convergence (along the central radial line), followed by a saccadic trajectory, which is then followed by a final convergence movement (along another radial line). Reprinted from Hung (1998b), pg. 12, Fig. 2, with permission of Swets and Zeitlinger.

Figure 11.15. (See previous page). Model simulation responses for a target displacement requiring -2 in the left eye and -6 deg in the left eye, corresponding to 4 deg of convergence and 4 deg of leftward saccadic response for the conditions of (A) simultaneous (latency=200 msec) and (B) sequential (latency: disjunctive=200 msec; and conjugate=300 msec) onset of controller signals. The description of the traces are the same as those for Fig. 11.14. Reprinted from Hung (1998b), pg. 13, Fig. 3, with permission of Swets and Zeitlinger.

11.5 REFERENCES

- Abel, L., Dell'Osso, L., Daroff, R.B. and Parker, L., 1979, Saccades in extremes of lateral gaze, *Invest. Ophthalm. Vis. Sci.* **18**: 324-327.
- Alpern, M., 1953, Metacontrast, *J. Opt. Soc. Am.* **29**: 631-646.
- Alpern, M. and Ellen, P., 1956, A quantitative analysis of the horizontal movements of the eyes in the experiment of Johannes Mueller. I. Methods and results, *Am. J. Ophthalmol.* **42**: 289-303.
- Aslin, R. N. and Shea, S. L., 1987, The amplitude and angle of saccades to double-step target displacements, *Vis. Res.* **27**: 1925-1942.
- Averbauch-Heller, L., Lewis, R. F., and Zee, D. S., 1999, Disconjugate adaptation of saccades: contribution of binocular and monocular mechanisms. *Vis. Res.* **39**: 341-352.
- Bahill, A. T. and Stark, L., 1979, The trajectory of saccadic eye movements, *Sci. Am.* **240**: 108-117.
- Bahill, A. T., 1981, *Bioengineering: Biomedical, Medical, and Clinical Engineering*, Prentice-Hall, Englewood Cliffs, NJ, pg. 146.
- Becker, W. and Jurgens, R., 1979, An analysis of the saccadic system by means of double step stimuli, *Vis. Res.* **19**: 967-982.
- Bucci, M. P., Kapoula, Z., and Eggert, T., 1999, Saccade amplitude disconjugacy induced by aniseikonia: role of monocular depth cues, *Vis. Res.* **39**: 3109-3122.
- Campbell F. W., and Wurtz, R. H., 1978, Saccadic omission: Why we do not see a grey-out during a saccadic eye movement, *Vis. Res.* **18**: 1297-1303.
- Chaturvedi, V. and van Gisbergen, J. A. M., 1999, Perturbation of combined saccade-vergence movements by microstimulation in monkey superior colliculus, *J. Neurophysiol.* **81**: 2279-2296.
- Ciuffreda K.J. and Stark, L., 1975, Descartes' law of reciprocal innervation, *Amer. J. Optom. Physiol. Opt.* **52**: 662-673.
- Ciuffreda, K.J. and Tannen, B., 1995, *Eye Movement Basics for the Clinician*, Mosby, St. Louis, MO, pp. 36-71, 127-160
- Clark, M. R. and Crane, H. D., 1978, Dynamic interactions in binocular vision, in: *Eye Movements and the Higher Psychological Functions*, J. W. Senders, D. F. Fisher, and R. A. Monty, eds., Erlbaum, Hillsdale, NJ, pp.77-88.
- Collewijn, H., Erkelens, C. J., and Steinman, R. M., 1988, Binocular co-ordination of human saccadic eye movements, *J. Physiol.* **404**: 157-182.
- Collewijn, H., Erkelens, C. J., and Steinman, R. M., 1995, Voluntary binocular gaze-shifts in the plane of regard: dynamics of version and vergence, *Vis. Res.* **35**: 3335-3358.
- Collewijn, H., Erkelens, C. J., and Steinman, R. M., 1997, Trajectories of the human binocular fixation point during conjugate and non-conjugate gaze-shifts, *Vis. Res.* **37**: 1049-1069.
- Eggert, T. and Kapoula, Z., 1995, Position dependency of rapidly induced saccade disconjugacy, *Vis. Res.* **35**: 3493-3503.
- Enright, J. T., 1984, Changes in vergence mediated by saccades, *J. Physiol.* **350**: 9-31.
- Enright, J. T., 1986, Facilitation of vergence changes by saccades: influences of misfocused images and of disparity stimuli in man, *J. Physiol.* **371**: 69-87.

- Enright, J. T., 1992, The remarkable saccades of asymmetrical vergence, *Vis. Res.* **32**: 2261-2276.
- Erkelens, C. J., Steinman, R. M., and Collewijn, H., 1989, Ocular vergence under natural conditions: II: Gaze shifts between real targets differing in distance and direction, *Proc. R. Soc. London.(B)*. **236**: 444-465.
- Findlay, J. M., and Harris, L. R., 1993, Horizontal saccades to dichoptically presented targets of differing disparities, *Vis. Res.*, **33**: 1001-1010.
- Gamlin, P. D. R. and Clarke, R. J., 1995, Single-unit activity in the primate nucleus reticularis tegmenti pontis related to vergence and ocular accommodation, *J. Neurophysiol.* **73**: 2135-2119.
- Gamlin, P. D. R. and Mays, L. E., 1992, Dynamic properties of medial rectus motoneurons during vergence eye movements, *J. Neurophysiol.* **67**: 64-74.
- Glassman, R. B., 1999, A working memory "theory of relativity": Elasticity in temporal, spatial, and modality dimensions conserves item capacity in radial maze, verbal tasks, and other cognition, *Brain Res. Bull.* **48**: 475-489.
- Hering, E., 1977 (originally 1868), *The Theory of Binocular Vision*, B. Bridgeman and L. Stark L, eds. trans., Plenum Press, New York.
- Hou, R. L. and Fender, D. H., 1979, Processing of direction and magnitude by the saccadic eye movement system, *Vis. Res.* **19**: 1421-1426.
- Hung, G. K., 1998a, Dynamic model of saccade-vergence interactions, *Med. Sci. Res.* **26**: 9-14.
- Hung, G. K., 1998b, Saccade-vergence trajectories under free- and instrument-space environments, *Curr. Eye Res.* **17**: 159-164.
- Hung, G. K. and Ciuffreda, K. J., 1996, Schematic model of saccade-vergence interactions, *Med. Sci. Res.* **24**: 813-816.
- Hung, G. K., Ciuffreda, K. J., Semmlow, J. L., and Horng, J.-L., 1994, Vergence eye movements under natural viewing conditions, *Invest. Ophthalm. Vis. Sci.* **35**: 3486-3492.
- Hung, G. K., Li, S., Semmlow, J. L., and Ciuffreda K. J., 1990, Suppression of sensitivity to change in target disparity during vergence eye movements, *Exp. Neurol.* **110**: 291-297.
- Hung, G. K., Semmlow, J. L., and Ciuffreda, K. J., 1983, Identification of accommodative vergence contribution to the near response using response variance., *Invest. Ophthalm. Vis. Sci.*, **24**: 772-777.
- Hung, G. K., Semmlow, J. L., and Ciuffreda, K. J., 1984, The near response: modeling, instrumentation, and clinical applications, *IEEE Trans. Biomed. Engin.* **31**: 910-919.
- Hung, G. K., Semmlow, J. L., and Ciuffreda, K. J., 1986, A dual-mode dynamic model of the vergence eye movement system, *IEEE Trans. Biomed. Engin.* **33**: 1021-1028.
- Hung, G. K., Wang, T. J., Ciuffreda, K. J., and Semmlow, J. L., 1989, Suppression of sensitivity to surround displacement during vergence eye movements. *Exp. Neurol.* **105**: 300-305.
- Hung, G. K., Zhu, H. M. and Ciuffreda, K. J., 1997, Convergence and divergence exhibit different response characteristics to symmetric stimuli, *Vis. Res.* **37**: 1197-1205.
- Judge, S. J. and Cumming, B. G., 1986, Neurons in the monkey midbrain with activity related to vergence eye movement and accommodation, *J. Neurophysiol.* **55**: 915-930.

- Jurgens, R., Becker, W., and Kornhuber, H. H., 1981, Natural and drug-induced variations of velocity and duration of human saccadic eye movements: evidence for a control of the neural pulse generator by local feedback, *Biol. Cyber.* **39**: 87-96.
- Kapoula, Z., Eggert, T., and Bucci, M. P., 1995, Immediate saccade amplitude disconjugacy induced by unequal images, *Vis. Res.* **35**: 3505-3516.
- Keller, E. L., 1977, Control of saccadic eye movements by midline brain stem neurons, in: *Control of Gaze by Brain Stem Neurons*, Baker R and Berthoz A, eds., Elsevier-North Holland, Amsterdam, pp. 327-336.
- Keller, E. L. and Robinson, D. A., 1972, Abducens unit behavior in the monkey during vergence movements, *Vis. Res.* **12**: 369-382.
- Kenyon, R. V. and Ciuffreda, K. J., 1978, Unequal saccades during vergence, *Am. J. Optom. Physiol. Opt.* **57**: 586-594.
- Kenyon, R. V., Ciuffreda, K. J., and Stark, L., 1980, An unexpected role for normal accommodative vergence in strabismus and amblyopia, *Am. J. Optom. Physiol. Opt.* **57**: 655-577.
- King, W. M. and Fuchs, A. F., 1977, Neuronal activity in the mesencephalon related to vertical eye movements, in: *Control of Gaze by Brain Stem Neurons*, R. Baker and A. Berthoz, eds., Elsevier-North Holland, Amsterdam, pp. 319-326.
- Krishnan, V. V. and Stark, L., 1977, A heuristic model for the human vergence eye movement system. *IEEE Trans. Biomed. Engin.* **24**: 44-48.
- Krommenhoek, K. P. and van Gisbergen, J. A. M., 1994, Evidence for nonretinal feedback in combined version-vergence eye movements, *Exp. Brain Res.* **102**: 95-109.
- Leigh, R. J. and Zee, D. S., 1991, *The Neurology of Eye Movements*, 2nd Ed., F.A. Davis Company, Philadelphia, PA.
- Manning, K. A. and Riggs, L. A., 1984, Vergence eye movements and visual suppression, *Vis. Res.* **24**: 521-526.
- Maxwell, J. S. and King, W. M., 1992, Dynamics and efficacy of saccade-facilitated vergence eye movements in monkeys, *J. Neurophysiol.* **68**: 1248-1260.
- Mays, L. E., 1983, Neurophysiological correlates of vergence eye movements, in: C. M. Schor and K. J. Ciuffreda, eds. *Vergence Eye Movements: Basic and Clinical Aspects*. Butterworths, Boston, pp. 649-670.
- Mays, L. E., 1984, Neural control of vergence eye movements: convergence and divergence neurons in the midbrain, *J. Neurophysiol.* **51**: 1091-1108.
- Mays, L. E. and Gamlin, P. D. R., 1995, A neural mechanism subserving saccade-vergence interactions, in: J. M. Findlay et al, eds., *Eye Movement Research*, Elsevier Science, Oxford, pp. 215-223.
- Mays, L. E. and Porter, J. D., 1984, Neural control of vergence eye movements: activity of abducens and oculomotor neurons, *J. Neurophysiol.* **52**: 743-761.
- Mays, L. E., Porter, J. D., Gamlin, P. D. R., and Tello, C. A., 1986, Neural control of vergence eye movements: neurons encoding vergence velocity, *J. Neurophysiol.* **56**: 1007-1021.
- Miller, J. M., Ono, H., and Steinbach, M. J., 1980, Additivity of fusional vergence and pursuit eye movements, *Vis. Res.* **20**: 43-47.

- Ono, H. 1983, The combination of version and vergence, in: *Vergence Eye Movements: Basic and Clinical Aspects*, C. Schor and K. J. Ciuffreda, eds., Butterworths, Boston, pp. 373-400.
- Ono, H. and Nakamizo, S., 1977, Saccadic eye movements during changes in fixation to stimuli at different distances, *Vis. Res.* **17**: 233-238.
- Panum, P. L. 1858. Physiologische Untersuchungen über das Sehen mit zwei Augen. Schwesche Buchhandlung, Kiel, Germany.
- Rashbass, C. and Westheimer, G., 1961a, The relationship between saccadic and smooth tracking eye movements, *J. Physiol.* **159**: 326-338.
- Rashbass, C. and Westheimer, G., 1961b, Disjunctive eye movements, *J. Physiol.* **159**: 339-360.
- Riggs, L. A. and Niehls, E. W., 1960, Eye movements recorded during convergence and divergence, *J. Opt. Soc. Am.* **50**: 913-920.
- Robinson, D. A., 1973, Models of the saccadic eye movement control system, *Kybernetik.* **14**: 71-83.
- Schor, C. M., 1979, The influence of rapid prism adaptation upon fixation disparity, *Vis. Res.* **19**: 757-765.
- Schor C. M., Robertson K. M., and Wesson, M., 1986, Disparity vergence dynamics and fixation disparity, *Am. J. Optom. Physiol. Opt.* **63**: 611- 616.
- Scudder, C. A., 1988, A new local feedback model of the saccadic burst generator, *J. Neurophysiol.* **59**: 1455-1475.
- Semmlow, J.L., Hung, G. K., and Ciuffreda, K. J., 1986, Quantitative assessment of disparity vergence components, *Invest. Ophthal. Vis. Sci.* **27**: 558-564.
- Semmlow, J. L., Hung, G. K., Horng, J. L., and Ciuffreda, K. J. 1993, The initial control component in disparity vergence eye movements, *Ophthal. Physiol. Optics*, **13**: 48-55.
- Semmlow, J. L., Hung, G. K., Horng, J. L., and Ciuffreda, K. J. 1994, Disparity vergence eye movements exhibit preprogrammed motor control, *Vis. Res.* **34**: 1335-1343.
- Semmlow, J. L. and Venkiteswaren, N., 1976, Dynamic accommodative vergence in binocular vision, *Vis. Res.* **16**: 403-411.
- Smith, K. U., Schmidt, J., and Putz V., 1970, Binocular coordination: feedback synchronization of eye movements for space perception, *Am. J. Optom. Arch. Am. Acad. Optom.* **47**: 679-689.
- Sparks, D. L., 1986, Translation of sensory signals into commands for control of saccadic eye movements: role of primate superior colliculus, *Physiol. Rev.* **66**: 118-171.
- Stark, L., 1968, *Neurological Control Systems: Studies in Bioengineering*. Plenum Press, New York.
- Steinman, R. M. and Collewijn, H., 1981, Binocular retinal image motion during active head rotation, *Vis. Res.* **20**: 415-429.
- Tamler, E., Jampolsky, A., and Marg, E., 1958, An electromyographic study of asymmetric convergence, *Am. J. Ophthalmol.* **46**: 174-182.
- van der Steen, J. and Bruno, P., 1995, Unequal amplitude saccades produced by aniseikonic patterns: effect of viewing distance, *Vis. Res.* **35**: 3459-3471.
- van Gisbergen, J. A., Robinson, D. A., and Gielen, S., 1981, A quantitative analysis of generation of saccadic eye movements by burst neurons, *J. Neurophysiol.* **45**: 417-442.

- Volkman, F. C., Riggs, L. A., Ellicott, A. G., and Moore, R. K., 1982, Measurements of visual suppression during opening and closing, and blinking of the eyes, *Vis. Res.* **22**: 991-996.
- Westheimer, G. and Mitchell, A. M., 1956, Eye movement responses to convergence stimuli., *Arch Ophthalmol.* **55**: 848-856.
- Wheeler, L., Boynton, R., and Cohen, G., 1966, Eye movement responses to step and pulse-step stimuli, *J. Opt. Soc. Am.* **56**: 956-960.
- Yarbus, A. L., 1967 (originally 1956), *Eye Movements and Vision*, B. Haigh, trans. and L. A. Riggs, ed., Plenum Press, New York.
- Young, L. R. and Stark, L., 1962. A sampled-data model for eye tracking movements, *Quart. Prog. Rept., Res. Lab. Electronics*, M.I.T. **66**: 370-383.
- Zee, D. S., Fitzgibbon, E. J., and Optican, L. M., 1992, Saccade-vergence interactions in humans, *J. Neurophysiol.* **68**: 1624-1641.
- Zee, D. S. and Levi, L., 1989, Neurological aspects of vergence eye movements., *Rev. Neurol. (Paris)*. **145**: 613-620.
- Zhu, H. M., 1995., *Investigation of interaction between vergence and saccadic eye movements*. M.S. Thesis, Biomedical Engineering, Rutgers University, pp. 35-38.
- Zuber, B. L. and Stark, L. 1966. Saccadic suppression: elevation of visual threshold associated with saccadic eye movements, *Expt. Neurol.* **16**: 65-79.
- Zuber, B. L. and Stark, L. 1968, Dynamical characteristics of fusional vergence eye movement system., *IEEE Trans. Syst. Sci. Cybern.* **4**: 72-79.