Chapter 17

Model-Based Understanding of Clinical Vergence Testing

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17.1 INTRODUCTION

Both symmetric and asymmetric disparity vergence have been investigated extensively over the past 50 years to gain a more complete understanding of the sensory, motor, and perceptual processes subserving and controlling binocular *fusion*, which is the primary goal of the vergence system (Schor and Ciuffreda, 1983; Ciuffreda and Tannen, 1995). Pure symmetric vergence can be elicited when a target is displaced in depth precisely along one's egocentric midline. In this case, the *retinal disparity* is equally distributed between the two eyes (Fig. 17.1A). The response consists of a saccade-free, relatively slow and smooth, symmetric disparity vergence tracking movement in depth. On the other hand, asymmetric vergence can be elicited when a target is displaced in depth anywhere except precisely along the egocentric midline, as is true under most naturalistic conditions. In this case, the initial retinal disparity is unequally distributed between the two eyes (Fig. 17.1B). However, now the response consists of a rapid saccade and a relatively slower disparity vergence movement, with the conjugate saccade functioning to shift the eyes laterally such that the retinal disparity is once again symmetrically distributed between the two eyes for symmetric disparity vergence to correct the residual bifixation error within foveal Panum's fusional areas (PFA). Furthermore, there is the special case

of line-of-sight asymmetric vergence, in which the target is moved along the line-of-sight of one eye (Fig. 17.1C). Here too both a saccade and a disparity vergence movement are executed, as described earlier for the more general case of asymmetric vergence (Fig. 17.1B).

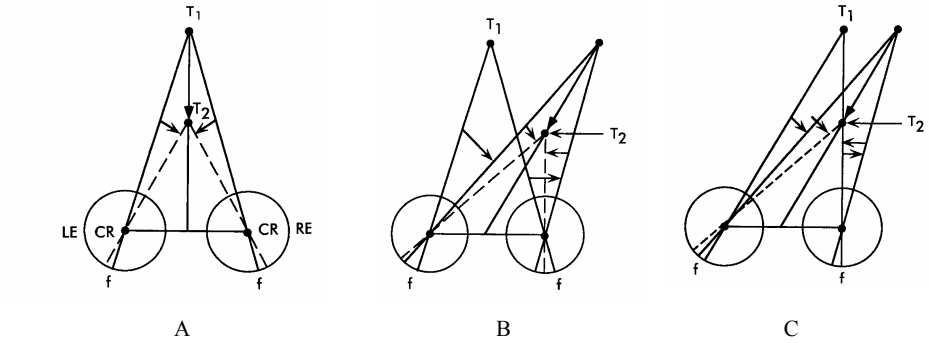


Figure 17.1. Symmetrical versus asymmetrical disparity stimuli and overall response patterns. A. Symmetric vergence. B. Asymmetric vergence. C. Line-of-sight asymmetric vergence. T_1, initially fixated target; T_2, subsequently fixated target; f, fovea; *CR*, center of rotation of the eye; *LE*, left eye; *RE*, right eye. Reprinted from Ciuffreda and Tannen (1995) with permission of Harcourt Health Sciences.

In this chapter, the static model-based aspects of clinical disparity vergence testing and the underlying saccade-vergence eye movements will be considered. Although much of the clinical testing to be discussed incorporates symmetric disparity stimuli to drive the vergence system directly (Benjamin, 1998), and the accommodative system indirectly (Ciuffreda, 1991, 1998), once fusion is disrupted and the disparity vergence system is rendered open-loop, asymmetric vergence becomes dominant. (Also see Chapter 11 in this volume). An understanding of the mechanisms involved requires conceptualization of the components that comprise the vergence response. This will be conducted in a systematic manner by first discussing and detailing a quantitative model of the system, then describing the clinical diagnostic testing, and concurrently relating these vergence tests and the clinical vergence deficits they may uncover to the underlying oculomotor model control structure. In essence, oculomotor-related clinical signs and symptoms of binocular vergence disorders may now be understood in terms of abnormality of specific disparity vergence and accommodative components in the static model.

To assist the reader in understanding the clinical terms denoted by italics, a glossary is provided at the end of this chapter.

17.2 BACKGROUND

17.2.1 Static Model of the Vergence and Accommodative Systems

A comprehensive and homeomorphic static, or steady-state, quantitative model of the vergence and accommodative systems and its motor interactions has been developed by Hung, Ciuffreda, and Rosenfield (1996). It incorporates disparity, blur, proximal, and tonic inputs to each system. This model and its earlier versions have been useful for furthering our understanding of a wide range of basic normal mechanisms and abnormal clinical conditions (Hung, 2001).

The latest version of the model is presented in Fig. 17.2, with model parameter values presented in Table 17.1. Progressing from left to right in Fig 17.2, it may be seen that the accommodative (upper) and vergence (lower) negative feedback control loops have similar component control structures.

Table 17.1

Static Model Values for Accommodation and Vergence

Accommodation	Vergence
DOF = ± 0.15 D	PFA = ± 5.0 min of arc
ACG = 10	VCG = 150
AC = 0.80 D/MA	CA = 0.37 MA/D
ABIAS = 0.61 D	VBIAS = -0.29 MA
ADAPTA = 4	ADAPTV = 9
APG = 2.10	VPG = 0.067
PDG = 0.212	PDG = 0.2.12

Input. The input or stimulus change for accommodation (AS; target distance in *diopters*, D) and disparity vergence (VS; target distance in meter angles, MA, or *prism diopters*, PD) sum with the negative feedback response of the respective system at that moment. The resultant difference represents the initial system error. The input for the proximal branch is perceived target distance, with such perceptually-derived information not having a separate feedback loop but rather inputting directly and simultaneously into both the accommodative and vergence negative feedback loops.

Under normal binocular, closed-loop viewing conditions (i.e., with blur and retinal disparity feedback present), blur and disparity provide the primary motor drives to their respective systems, with their crosslinks providing a secondary drive to the fellow system. The proximal drive adds to the final steady-state vergence (VR) and accommodative (AR) responses by only 0.4% and 4%, respectively. However, this concordant tertiary proximal information is still quite important, as it influences overall responsivity by providing perceptual cue reinforcement derived from the perceived depth information. Lastly, the tonic inputs to each system have little motor-response effect in

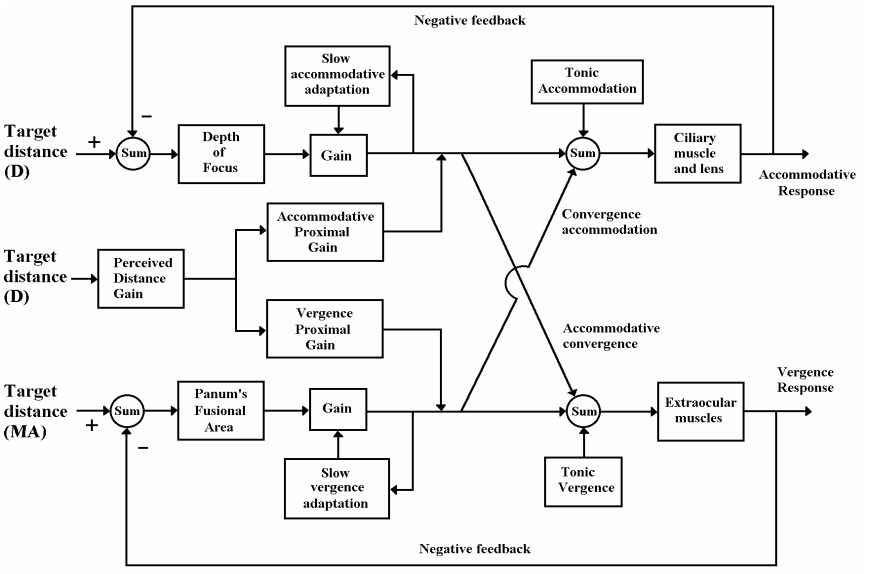


Figure 17.2. Conceptual version of Hung, Ciuffreda, and Rosenfield (1996) interactive static model of accommodation and vergence. Reprinted from Ong and Ciuffreda (1997) with permission of Optom. Extension Program Foundation Press, Santa Ana, CA.

visually-normal individuals, especially at near, under naturalistic binocular viewing conditions.

Threshold "Deadspace" Operator (DSP). This represents the depth-of-focus for accommodation (in diopters) and Panum's fusional areas for disparity vergence (in minutes of arc). DSP allows small neurosensory-based accommodative (AE) and vergence (VE) system errors (i.e., retinal defocus and retinal disparity, respectively) to be tolerated without adverse perceptual consequences (i.e., blur and diplopia, respectively). If the input error exceeds its threshold level, this error information proceeds to drive the respective system.

Gain. The accommodative (ACG=AR-ABIAS/AE-DSP) and vergence (VCG=VR-VBIAS/VE-DSP) controller gains represent the experimentally-derived, open-loop, internal neurological controller gains of the respective systems. The final system error signal, which equals the initial system error minus the deadspace threshold value, is multiplied by this gain element. Its output provides the primary neurological control signal and drive to formulate the final steady-state motor response. The output of the controller gain is then input to three other components (see next three components below).

Adaptive Gain. Although typically regarded as a dynamic model element, following sustained nearwork it may bias either the final static open-loop or closed-loop system response. However, under non-sustained viewing conditions, its value is zero. Hence, it will not be further considered in this chapter.

Cross-Link Gain. The cross-link gain (AC for accommodation and CA for vergence) multiplies the output of the direct ACG or VCG pathway, respectively. It provides a secondary drive to the fellow system, as mentioned earlier. For accommodation, this represents the effective accommodative-convergence to accommodation (AC/A) ratio, whereas for *convergence* it represents the effective convergence-accommodation to convergence (CA/C) ratio.

Tonic Input. Tonic input for accommodation (ABIAS; in diopters) and vergence (VBIAS; in meter angles) reflects midbrain baseline neural innervation. Although tonic terms may have substantial effects on the response amplitude when both systems are rendered open-loop (i.e., with visual feedback rendered ineffective), they have negligible influence on the overall closed-loop near response and only modest influence on the closed-loop far response (Hung & Semmlow, 1980). This is shown in Eq. 17.1 with respect to monocular blur-driven accommodation, where

$$AR = (AS - DSP) * \frac{ACG}{1 + ACG} + ABIAS * \frac{1}{1 + ACG} \qquad (17.1)$$

For a typical value of ABIAS = 1 diopter and ACG = 9, the effect of ABIAS on AR would only be 0.1 diopter. This relative lack of effect is even more dramatic for disparity vergence, with its much higher controller gain value, as shown in Eq. 17.2 with accommodation open-loop, where

$$VR = (VS - DSP) * \frac{VCG}{1 + VCG} + VBIAS * \frac{1}{1 + VCG} \qquad (17.2)$$

For a typical value of VBIAS = 1 MA and VCG = 149, the effect of VBIAS on VR would only be 0.007 MA.

Summing Junction. The direct controller gain output is also sent to the summing junction, where it adds with the cross-link and tonic inputs, both of which have only modest influence on the fellow system, to formulate the final combined signal to drive the respective system.

Peripheral Apparatus. The output of the summing junction proceeds to cortical and subcortical centers related to accommodation and to vergence to formulate the respective aggregate neural signals (Ciuffreda, 1991, 1998; Ciuffreda and Tannen, 1995). It then advances to innervate the appropriate peripheral apparatus, that is the ciliary muscle and crystalline lens complex for accommodation and the extraocular muscles for vergence.

Output. These motor changes are then fed back to the initial summing junction via their respective negative feedback pathways. If a relatively large residual error remains, the cycle is repeated, until an acceptably small and stable steady-state error for both systems is attained.

17.2.2 Clinical Vergence Testing

In the early part of the 20th century, the development of clinical test procedures to assess sensory and motor aspects of the vergence system began in optometric clinical and academic facilities (Sheard, 1917). Concurrently, test values were ascertained in large groups of asymptomatic and symptomatic clinical patients, especially as related to nearwork activities (Sheard, 1917; Skeffington, 1928). Normative values evolved for each test, which segregated these two clinic populations: patients having symptoms typically fell outside the range of values established for the asymptomatic individuals. Thus, such normative test values could henceforth be used as a valuable diagnostic tool (Benjamin, 1998). Furthermore, with the development of oculomotor-based *vision therapy* (Skeffington, 1928; Peckham, 1928), these norms also possessed therapeutic value because vision therapy could now be justified on a sound clinical and motor learning basis (Ciuffreda, in press). Therapy would be continued, until the established norms were attained, and then maintained by

Table 17.2

Relationship of Clinical Vergence Test, Vergence Model Component, and Clinical Vergence Deficit

Test	Component	Deficit
Four base-out test	Disparity vergence (central field open-loop)	Suppression scotoma in strabismus
Cover test	Disparity vergence (central and peripheral field open-loop) Accommodative vergence crosslink gain (AC)	Large phoria /strabismus/paresis
Distance phoria	Tonic vergence (VBIAS) Accommodative vergence crosslink gain (AC) Disparity vergence (central and peripheral field open-loop)	Symptomatic large phoria / intermittent strabismus/paresis
Near phoria	Accommodative vergence crosslink gain (AC) Disparity vergence (central and peripheral field open-loop) Perceived / proximal vergence gain	Symptomatic large phoria / intermittent strabismus/paresis
Prism vergence ranges	Vergence controller gain (VCG) Extraocular muscles (PLANT)	Convergence insufficiency Convergence excess Large phoria Extraocular muscle paresis
Near point of convergence	Vergence controller gain (VCG) Extraocular muscles (PLANT)	Convergence insufficiency Large exophoria at near Extraocular muscle paresis

the patient for a sufficient period of time. Such clinical findings first demonstrated the remarkable degree of neural plasticity in the vergence oculomotor system, and, in turn, justified the use of specific interactive vergence and accommodative therapeutic regimens designed to attain normal static and dynamic symmetric and asymmetric vergence function (Ciuffreda, in press). Furthermore, with the development of quantitative models, the individual abnormal vergence and accommodative model components could now be ascertained, and vision therapy directed specifically towards each component's normalization (Hung et al, 1986; Ciuffreda, in press).

Gross abnormalities of vergence are typically found in patients with *strabismus* (Figs. 17.2 and 17.3; Table 17.2). This abnormal sensorimotor ocular condition is associated with either an anomalous central field, open-loop, disparity vergence response or an anomalous closed-loop, full-field disparity vergence response (Ciuffreda and Tannen, 1995). Binocular *suppression* is commonly found in conjunction with strabismus (Fig. 17.3). It is a key factor in understanding the anomalous asymmetric fusional vergence responses observed in some strabismic patients due to presence of a binocular *suppression scotoma* (Ciuffreda and Tannen, 1995).

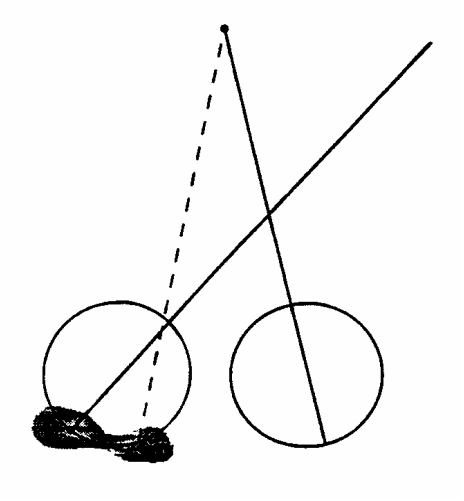


Figure 17.3. Binocular suppression scotoma (stippled region) in the deviated eye. Left esotropia. Solid lines project from the fovea of each eye. Small solid circle represents the test target. Reprinted from Ciuffreda and Tannen (1995) with permission of Harcourt Health Sciences.

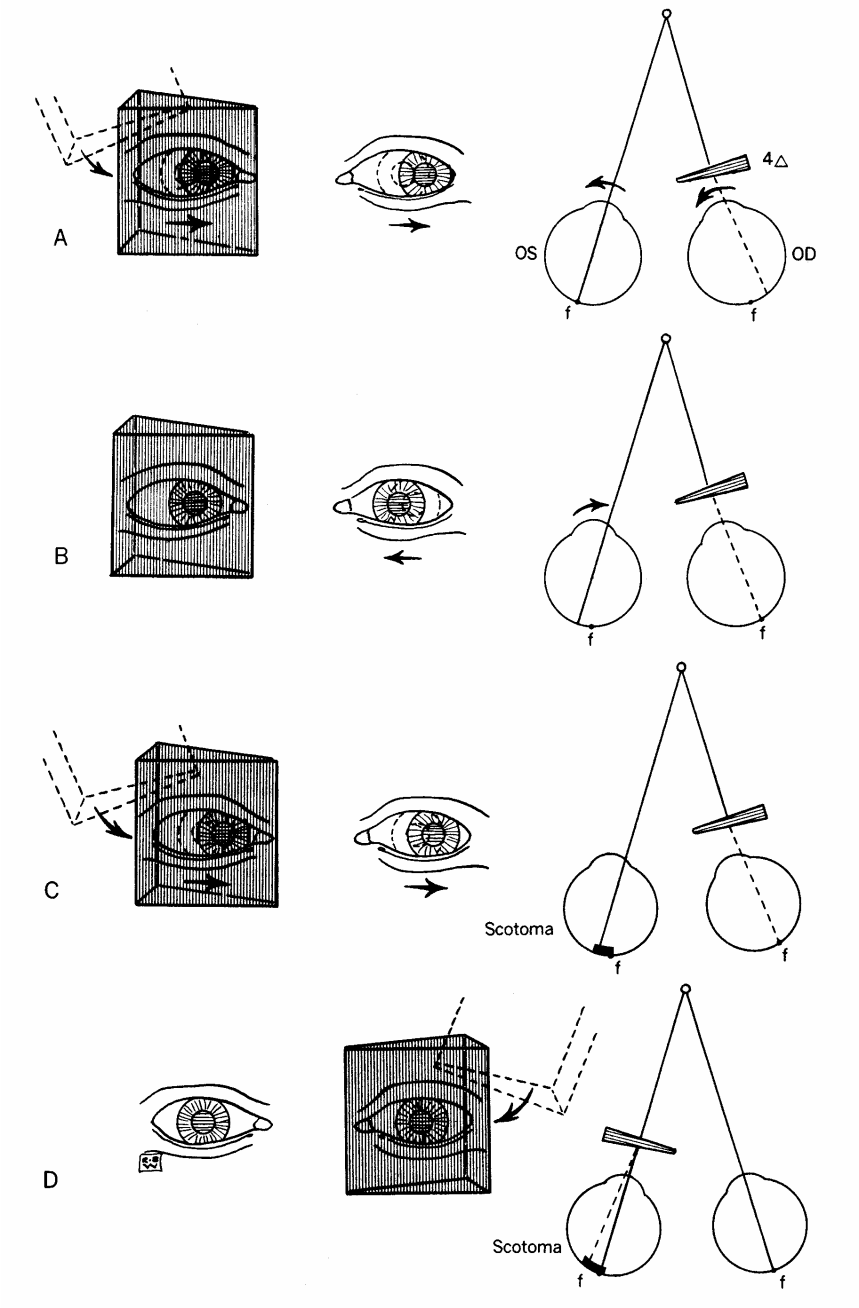
17.3 CLINICAL DIAGNOSTIC VERGENCE TESTING

17.3.1 Four Base-Out Test

This test directly involves careful detection for the presence of an asymmetric vergence response to assess the neurosensory binocular status of the strabismic patient. It is used diagnostically to detect for the presence of a central binocular suppression scotoma in the deviated eye of *esotropic* patients (Fig. 17.3) (von Noorden and Maumenee, 1967). This sensory-based, binocular suppression scotoma is adaptive in nature at the cortical level (Blake and Lehmkule, 1976) to prevent the occurrence of visually-debilitating *diplopia* and *visual confusion* (Ciuffreda and Tannen, 1995). It effectively acts to open-loop the disparity vergence system over the central field by cortically suppressing the retinal disparity information over that region in the deviated eye, with this occurring in the model representation between the summing junction and Panum's fusional areas (Figs. 17.2 and 17.3, and Table 17.2).

In essence, the test involves careful scrutiny of the two eyes immediately upon interposition of a 4 prism diopter base-out ophthalmic prism before one eye, which deviates the incoming light (approximately 2 degrees) towards the prism base and projects the deviated image in the direction of the prism apex. This procedure is repeated in the fellow eye. Since the prism does not distribute the retinal disparity equally interocularly (i.e., a 4 prism diopter change in the eye with the prism, and zero in the fellow eye), the vergence stimulus and hence vergence response are asymmetric in nature in a normal individual with the prism placed before either eye (Figs. 17.4A and B). In contrast, in a strabismic patient with abnormal binocular vision and presence of a central binocular suppression scotoma, either only a conjugate versional movement (i.e., a saccade) (Fig. 17.4C) or no response (Fig. 17.4D) will be found, depending upon which eye the prism is interposed.

Figure 17.4. (See next page). The Four Base-Out Test. (A) On placing the prism over the right eye, a rapid leftward movement occurs during refixation with the right eye. This indicates absence of foveal suppression in the right eye. (B) A subsequent very slow fusional movement of the left eye is observed to correct for the image displacement. This indicates absence of foveal suppression in the left eye. The combined movements of (A) and (B) represent asymmetric vergence. (C) In another patient, the left eye remains turned out after a prism is placed over the right eye. Absence of the secondary asymmetric vergence fusional movement of the left eye indicates a foveal suppression scotoma of the left eye; the image has been shifted within a binocularly nonfunctioning retinal area. (D) To confirm this diagnosis, the prism is placed over the left eye. Neither eye will move under these circumstances, since the prism has merely displaced the image within the binocular suppression scotoma, and hence no stimulus for asymmetric vergence exists for refixation. Reprinted from von Noorden and Maumenee (1967) with permission of Harcourt Health Sciences.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17.3.2 Cover Test Measurement and Fusional Recovery

One of the most common tests performed in ophthalmic clinical practice is the "unilateral cover test", which is used to detect the presence of an oculomotor deviation when binocular sensory fusion is disrupted (Table 17.2) (Benjamin, 1998; von Noorden and Maumenee, 1967). This test is critical in the diagnosis and treatment of binocular vision disorders, such as a large *phoria, extraocular muscle paresis,* or intermittent strabismus.

The cover test assesses the position of the eyes when disparity vergence is open-loop both centrally and peripherally, and hence the main drive to the vergence system now comes from the accommodative system via its crosslink, AC; perceived distance and proximal vergence gain may influence the near response in a secondary manner by introducing a constant bias effect; and, the influence of tonic vergence (VBIAS) is tertiary at near, but may be of somewhat more importance at distance in assessment of the phoria (Table 17.2). In essence, while having the patient binocularly view an object placed along the midline, an occluder is first placed fully over one eye for up to 15 seconds to allow for dissipation and decay of the fusional vergence response adaptive component. Furthermore, this effectively renders the disparity vergence system open-loop, with the final eye position dictated by the accommodative vergence drive (i.e., AC/A ratio) via the AC model crosslink component. Then the occluder is quickly removed to restore binocular viewing and provide closed-loop retinal disparity feedback. The initial direction and magnitude of the movement in the uncovered eye is denoted (Fig. 17.5). This procedure is then repeated with the fellow eye.

Three possible motor responses may be found. (1) If the patient manifests *orthophoria*, and hence the occluded eye remains stationary, then no movement will be observed when the occluder is removed. (2) However, if the patient exhibits a phoria such that the eye under cover shifts in an exponentially decelerating manner to the fusion-free, phoria position (Fig. 17.2), then eye movements will be observed when the occluder is removed. Since an asymmetric vergence stimulus condition is present immediately upon removal of the occluder, as all of the retinal disparity is present in the deviated eye rather than being symmetrically distributed between the two eyes, an asymmetric vergence, "fusional recovery" motor response will be executed. At distance, the phoria position will approximate the tonic vergence level, or VBIAS (O'Shea et al, 1988) (Fig. 17.2). (3) And, if the patient has an intermittent strabismus, so that when the occluder is introduced, the eye under cover again exponentially shifts in approximately 10 to 15 seconds to the fusion-free strabismic position, then two responses are possible when the occluder is initially removed: (a) if the patient does not either reflexively or volitionally regain binocular motor fusion, the eye will remain in the deviated

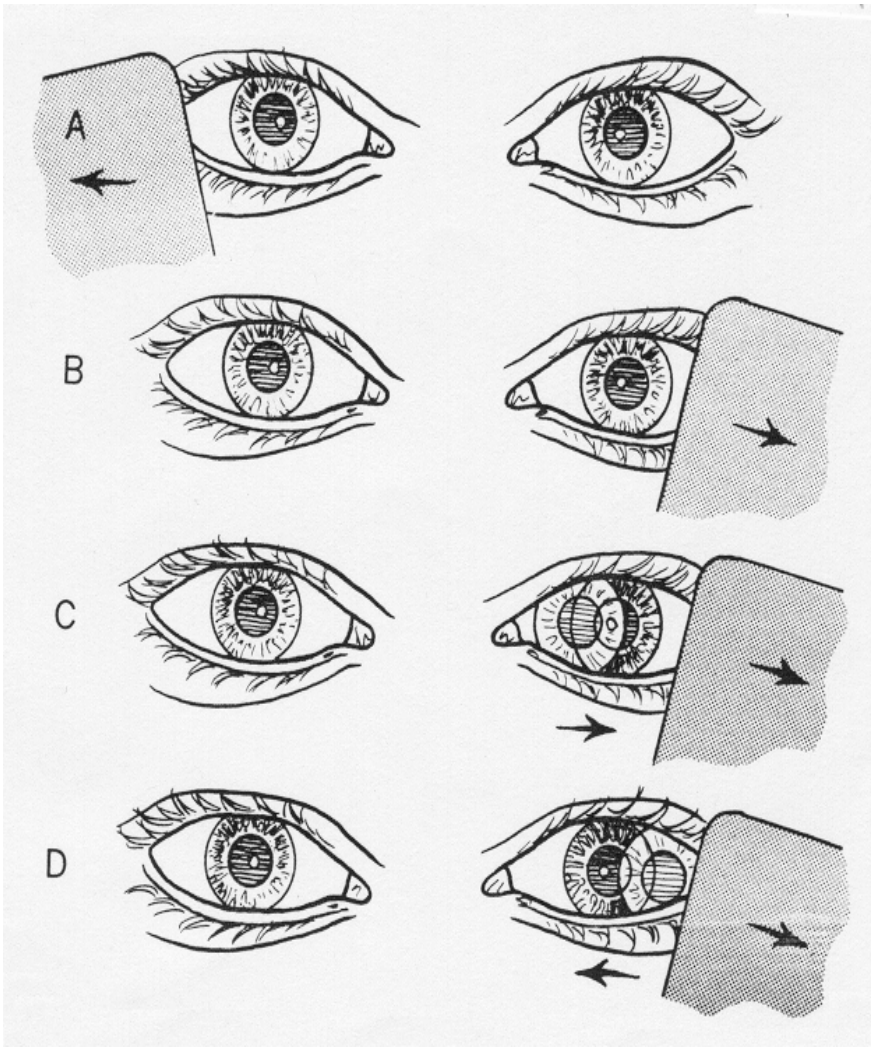

Figure 17.5. The Unilateral Cover Test. (A) The cover has been removed from the right eye, and no movement of the right eye can be detected. (B) The cover has been removed from the left eye, and no movement of the left eye can be detected. (C) When uncovered, the left eye moves outward to fixate. *Esophoria.* (D) When uncovered, the left eye moves inward to fixate. *Exophoria.* Reprinted from von Noorden and Maumenee (1967) with permission of Harcourt Health Sciences.

strabismic position, or (b) if the patient does regain binocular motor fusion, an asymmetric vergence "fusional recovery" motor response will take place, as all of the retinal disparity is present in the deviated eye rather than being symmetrically distributed between the two eyes (Ciuffreda et al, 2001).

17.3.3 Prism Disparity Vergence Ranges

Another important clinical test used in the diagnosis and treatment of binocular vision disorders is the measurement of prism vergence ranges at both distance and near. This assesses the motor limits of crossed and uncrossed disparity vergence, with the accommodative stimulus level remaining fixed (Ciuffreda, 1992). These tests examine the integrity of the vergence controller gain (VCG), and secondarily the vergence plant (i.e., extraocular muscles) (Table 17.2). A higher controller gain results in greater accuracy in the vergence response; however, abnormally-high gain results in *convergence excess,* and abnormally-low gain results in *convergence insufficiency*. A functional plant is needed to provide a full range of vergence movements, and hence a mild extraocular muscle paresis would produce restriction.

Basically, a pair of variable power ophthalmic prisms with bases in opposite directions (e.g., bases out, or templeward) is placed before the eyes and increasing amounts of fusional vergence stimulus demand via the prisms (i.e., crossed convergent or uncrossed divergent retinal disparity; horizontal, vertical, or cyclorotary in nature) (Ciuffreda and Tannen, 1995) is introduced with respect to a fixed target positioned along the midline. Hence, the initial stimulus and response are symmetric in nature. And, the magnitude of the vergence response is dictated by the vergence controller gain, or VCG, as well as the integrity of the plant (see Fig. 17.2 and Table 17.2). As the disparity vergence demand increases, the patient will eventually note some target blur due to the concurrently increased vergence accommodation from the crosslink gain (CA) as it exceeds the depth-of-focus of the eye (Benjamin, 1998) (Fig. 17.2); and with yet further increases in prism vergence demand will subsequently note the onset of diplopia as fusion can no longer be maintained. Once diplopia is consistently present, and hence the disparity vergence system is now rendered open-loop, the non-fixating eye will begin to shift slowly and exponentially to the fusion-free phoria position. After a few seconds, the vergence demand is then gradually reduced optically via the prisms, until binocular fusion is regained. The "fusional recovery" movement is accomplished with asymmetric vergence, as once again the retinal disparity is markedly asymmetric.

17.3.4 Near Point of Convergence

The symmetric and asymmetric responses as described above are also found in this important diagnostic test (Benjamin, 1998; Ciuffreda, 1992). And, as was true for the prism vergence ranges described above, this test reflects vergence controller gain (VCG) and extraocular muscle (i.e., plant) integrity (Table 17.2). However, now a physical (versus optical) target is moved in space slowly along the midline towards the patient, thus producing a symmetric vergence stimulus and response, to determine the maximum convergence ability. Once this point in physical space is exceeded, fusion will no longer be possible and hence become disrupted, and the non-dominant eye will shift exponentially to the disparity vergence, open-loop, phoria position (generally outward). Diplopia may or may not be reported. As the target is then moved away from the patient, at a certain point an asymmetric vergence movement will be executed to regain fusion, as the retinal disparity is markedly asymmetric.

17.4 CONCLUSIONS

Clearly, our interactive vergence and accommodation model, with its specific subcomponents, can be used successfully to describe and understand the diagnostic tests used by optometrists and others in their overall clinical armamentarium for a variety of binocular vision dysfunctions (Table 17.2). While such model-based conceptualization of these tests represents a major step forward as compared with their early origins over 75 years ago, such a notion must have been in the pioneering minds of Sheard (1917), Skeffington (1928), Peckham (1928) and others, as only if one could understand the basic normal and abnormal control structure could progress and advances take place in the equally important areas of prevention and treatment. Future directions are two-fold: to develop a comprehensive and quantitative interactive oculomotor model of binocular vision system dysfunctions and, (2) to perform computer simulations on the above to develop more effective vision therapy, and perhaps even surgical, paradigms that would "target" specific abnormal model component structures.

17.5 REFERENCES

Benjamin, W.J. (ed.), 1998, *Borish's Clinical Refraction*, W.B. Saunders Company, Philadel., PA.

Blake, R., and Lehmkule, S.W., 1976, On the site of strabismic suppression, *Invest. Ophthal. Vis. Sci.* **15:** 660-663.

Ciuffreda, K.J., 1991, Accommodation and its anomalies, in *Vision and Visual Dysfunction: Visual Optics and Instrumentation*, Vol. 1, W.N. Charman ed., Macmillan, London, pp. 231-279.

Ciuffreda, K.J., 1992, Components of clinical near vergence testing, *J. Behav. Optom.* **3:** 3-13.

Ciuffreda, K.J., 1998, Accommodation, the pupil, and presbyopia, in *Borish's Clinical Refraction*, W.J. Benjamin ed., W.B. Saunders Company, Philadel., PA, pp. 77-120.

Ciuffreda, K.J, The scientific basis for and efficacy of optometric vision therapy in non-strabismic accommodative and vergence disorders, *Optometry*, in press.

Ciuffreda, K.J., Suchoff, I.B., Kapoor, N., Jackowski, M.M., and Wainapel, S.F., 2001, Normal vision function, in *Downey and Darling's Physiological Basis of Rehabilitation Medicine*, Gonzalez, E.G. et al, eds., Butterworth-Heinemann, Boston, pp. 241-261.

Ciuffreda, K.J., and Tannen, B., 1995, *Eye Movement Basics for the Clinician*, Mosby Yearbook, St. Louis.

Hung, G.K., 2001, *Models of Oculomotor Control*, Singapore, World Scientific Publishing Co. Inc.

Hung, G.K., Ciuffreda, K.J., and Rosenfield, M., 1996, Proximal contribution to a linear static model of accommodation and vergence, *Ophthal. Physiol. Opt.* **16:** 31-41.

Hung, G.K., Ciuffreda, K.J., and Semmlow, J.L., 1986, Static vergence and accommodation: population norms and orthoptic effects, *Doc. Ophthal.* **62:** 165-179.

Hung, G.K., and Semmlow, J.L., 1980, Static behavior of accommodation and vergence: computer simulation of an interactive dual-feedback system, IEEE Trans. Biomed. Eng. **27:** 439-447.

Ong, E., and Ciuffreda, K.J., 1997, *Accommodation, Nearwork, and Myopia*, OEP Foundation Press, Santa Ana, CA.

O'Shea, W.F., Ciuffreda, K.J., Fisher, S.K., Tannen, B., and Super, P., 1988, The relationship between the distance heterophoria and tonic vergence, *Amer. J. Optom. Physiol. Opt.* **65:** 787-793.

Peckham, R.H., 1928, *The Modern Treatment of Binocular Imbalance*, Shur-On Standard Optical, New York.

Schor, C.M., and Ciuffreda, K.J., (eds.), 1983, *Vergence Eye Movements: Basic and Clinical Aspects*, Butterworth, Boston.

Sheard, C., 1917, *Dynamic Ocular Tests*, Lawrence Press, Columbus, Ohio.

Skeffington, A.M., 1928, *Procedure in Ocular Examination*, A.J. Fox Co., Chicago.

von Noorden, G.K., and Maumenee, A.E., 1967, *Atlas of Strabismus*, Mosby, St. Louis.

Glossary of Clinical Terms	
Convergence	The inward-directed turning of the lines-of-sight toward each other; this is in contrast to divergence, the outward turning of the lines-of-sight away from each other.
Convergence excess	Abnormal increase in convergence at near relative to that at distance under fusion-free conditions.
Convergence insufficiency	Abnormal decrease in convergence at near relative to that at distance under fusion-free conditions.
Diopter	A unit of ophthamic lens power; one diopter focuses light from infinity at a distance of one meter.
Diplopia	Similar images falling on non-corresponding retinal points, and hence projecting to different visual directions; "double" vision; non-fused images.
Esophoria	An inward lateral deviation of the eye in the fusion-free state accomplished either by prismatic dissociation or occlusion of one eye.
Esotropia	A manifest inward lateral deviation of one eye.
Exophoria	An outward lateral deviation of the eye in the fusion-free state accomplished either by prismatic dissociation or occlusion of one eye.
Fusion	Higher-order cortical integration of the left and right eyes' images; haplopia.
Orthophoria	Lack of deviation of the eye in the fusion-free state.
Paresis	Partially-paralyzed extraocular muscle.
Phoria	A deviaiton of the eye (inward, outward, upward, downward, or cyclorotatory in nature) in the fusion-free state (i.e., typically either with one eye occluded or with prismatic dissociation).
Prism diopter	A unit of ophthalmic prism power; one prism diopter deviates light from infinity 1 cm at 1m; 1.745 prism diopters equal 1 degree.
Retinal disparity	The geometric angular difference at the eyes between the bifixation target and any other object in the visual field.
Scotoma	A relative or absolute blind area of the visual field.
Strabismus	An anomaly of binocular vision in which the visual axis of one eye fails to intersect the object of interest.

Glossary of Clinical Terms (con't)	
Suppression	An anomaly of binocular vision in which part of the ocular image of the strabismic, deviated eye is prevented from contributing to the fused binocular percept.
Vision therapy	Highly specific, sequential, sensory-motor-perceptual stimulation paradigms and regimens aimed at normalizing binocular vision. It incorporates the use of blur (via lenses and target distance), disparity (via prisms and target distance), and proximity (via perceived target distance) stimuli; also referred to as v.t., vision training, visual training, visual therapy, "eye exercises", and orthoptics.
Visual confusion	Dissimilar images falling on foveal corresponding retinal points, and hence projecting to identical visual directions.