

The scientific basis for and efficacy of optometric vision therapy in nonstrabismic accommodative and vergence disorders

Kenneth J. Ciuffreda, O.D., Ph.D.

State University of New York, State College of Optometry, Department of Vision Sciences, New York, New York

Background: For nearly 75 years, optometric vision therapy has been an important mode of therapy for both children and adults who manifested a range of nonstrabismic accommodative and vergence disorders.

Methods: In this article, the scientific basis for, and efficacy of, optometric vision therapy in such patients will be discussed. Using bio-engineering models of the oculomotor system as the conceptual framework, emphasis will be focused on studies that used objective recording techniques to directly assess therapeutically related changes in oculomotor responsivity.

Results and Conclusions: The findings clearly support the validity of optometric vision therapy. Furthermore, the results are consistent with the tenets of general motor learning.

Key Words: Accommodation, behavior modification, motor learning, oculomotor plasticity, oculomotor responsivity, vergence, vision therapy

Optometric vision therapy for nonstrabismic accommodative and vergence disorders involves highly specific, sequential, sensory-motor-perceptual stimulation paradigms and regimens. It incorporates purposeful, controlled, and scientifically based manipulations of target blur, disparity, and proximity, with the aim of normalizing the accommodative system, the vergence system, and their mutual interactions.¹⁻³ In addition, other sources of sensory information, such as kinesthesia⁴ (e.g., touching the near test object) and audition^{5,6} (e.g., oculomotor auditory biofeedback) correlated to the accommodative and vergence states (e.g., position, innervation, effort, etc.) can provide cue reinforcement. Inclusion of related behavioral modification paradigms,⁷⁻⁹ such as general relaxation,⁵ visual imagery,^{10,11} (e.g., “think far or near”), and attentional shaping¹² may help one learn to initiate (i.e., provide a “trigger” mechanism) and/or enhance the appropriate motor responses. However, the ultimate goal of optometric vision therapy is not simply to impact positively on various aspects of the oculomotor system *per se*, in isolation, but to attain clear and comfortable binocular vision at all times. It involves oculomotor integration with the head (i.e., eye-head coordination),¹³ neck (i.e., proprioceptive information),¹⁴ limbs,¹⁵ and overall body,^{16,17} with information from the other sensory modalities, producing temporally efficient, coordinated behavior^{18,19} within a context of harmonious spatial sense^{16,17,20} under a variety of external and internal conditions and states.

Prevalence of nonstrabismic accommodative and vergence disorders

Nonstrabismic accommodative and vergence disorders of a non-organic, nonpathological nature (i.e., “functional” in origin) are the most-common ophthalmic vision conditions (other than refractive error) that present in the general opto-

Ciuffreda KJ. The scientific basis for and efficacy of optometric vision therapy in nonstrabismic accommodative and vergence disorders. *Optometry* 2002;73:735-62.

metric clinical practice. The specifically related signs and symptoms may also initially be reported to, or uncovered by, the orthoptist, ophthalmologist, neurologist, internist, or general primary care medical practitioner, as well as others in the allied health and educational professions (e.g., school nurses, remedial reading teachers, etc.). Failure to detect and diagnose these problems may have grave consequences to some patients and, hence, legal consequences.²¹

In symptomatic, nonpresbyopic clinic patients, the prevalence of accommodative dysfunction and correlated symptoms is estimated at: accommodative insufficiency, 9.2%; accommodative infacility, 5.1%; and accommodative spasm, 2.5%.²² However, as one might expect, in clinic patients who are receiving treatment for manifest binocular dysfunctions, the prevalence is much higher (60% to 80%).^{22,23} *Thus, accommodative disorders are common in the general optometric clinic population.*

With respect to the prevalence of fusional vergence dysfunction, the most-common type is convergence insufficiency. The median prevalence is 7%, in both children and adults.²⁴ Other relatively frequent vergence dysfunctions include: convergence excess, 5.9% to 7.1%;^{22,25} basic exophoria, 2.8%;²⁶ fusional vergence dysfunction, < 7%;²⁷ and clinically significant vertical phoria, 9%.²⁸ *Thus, as was true for accommodative disorders, vergence disorders are also common in the general optometric clinic population.*

Symptoms reported in patients who manifest nonstrabismic accommodative and vergence disorders

There are a wide range of symptoms reported in patients who manifest nonstrabismic accommodative and vergence disorders.²⁶ With respect to accommodation, these include: blurred vision at distance and/or near during or immediately following nearwork, headaches, poor concentration, and difficulty reading. With respect to vergence, these include: blurred vision, diplopia, ocular discomfort during or immediately following nearwork, frontal headaches, nausea, sleepiness, loss of concentration, heavy lid sensation, general fatigue, and “pulling” sensation of the eyes. Of interest, the symptom preventing tactic of task “avoidance” may be used by some, thus negatively impacting on overall quality of life—especially with respect to school and work performance.

Cure rates in patients who manifest nonstrabismic accommodative and vergence disorders

The cure rates in symptomatic patients who manifest nonstrabismic accommodative and fusional vergence disorders are very high (also see ‘Accommodative Therapy’ and ‘Vergence Therapy’ sections). Cure rates for accommodative disorders generally ranged from 80% to 100%,²⁶ and cure rates for vergence disorders generally ranged from 70% to 100%.²⁶ *Thus, both the accommodative and vergence systems are remarkably remediable and exhibit considerable oculomotor plasticity, with concurrent marked reduction of symptoms.*

The clinical practice of optometric vision therapy has had nearly 75 years to evolve to its present level within the optometric community.^{29,30} Before that, however, its more narrowly focused counterpart of orthoptics was founded in France by the ophthalmologist Javal in the mid-nineteenth century (1858) and is still practiced widely—especially in Europe and the United Kingdom—in ophthalmological clinics.³ In 1915, in the United States, Duane³¹ reported that 10% of his ophthalmological clinic patients manifested nonpathological, functional, accommodative disorders that he believed could be remediated by accommodative “exercises” he proposed. Both optometrically based vision therapy and ophthalmologically based orthoptics have had a long history, and are currently actively involved in the successful management and treatment of a wide range of oculomotor dysfunctions, including those with nonstrabismic accommodative and vergence disorders. From this rich background, well-developed and scientifically based treatment plans with common elements have evolved—especially in optometry—to efficiently and cost-effectively remediate disorders of both of these oculomotor systems²⁶ (see Figures 1 and 2). Clinical guidelines and important conceptual notions regarding these specific nonstrabismic accommodative and vergence disorders have evolved in optometry based on a wide range of laboratory, clinical, and epidemiologic investigations over the years.^{26,32-36} All of these factors have contributed to keen insights and increased understanding of functionally based, nonstrabismic, accommodative and vergence disorders in clinical practice, resulting in the high success rates found following therapeutic intervention.

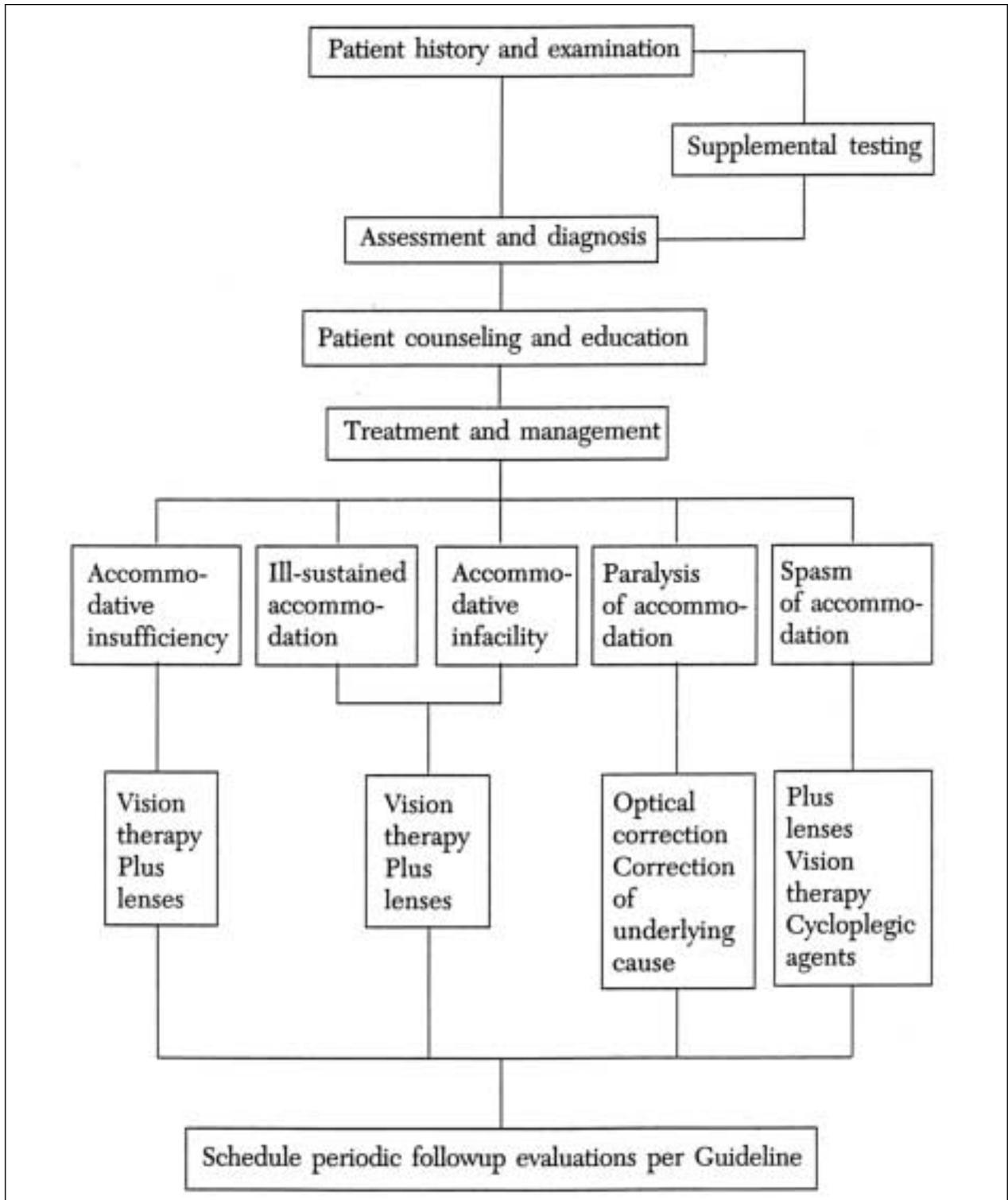


Figure 1 Optometric management of the patient with accommodative dysfunction: a brief flowchart (reprinted with permission from the American Optometric Association, 1998).

The balance of this article will establish the scientific basis for, and efficacy of, optometric vision therapy. It will have the following organizational structure. *First*, using bio-engineering models of

the oculomotor system as the conceptual framework, a detailed quantitative overview of various static and dynamic models of accommodation and/or vergence having a direct bearing on opto-

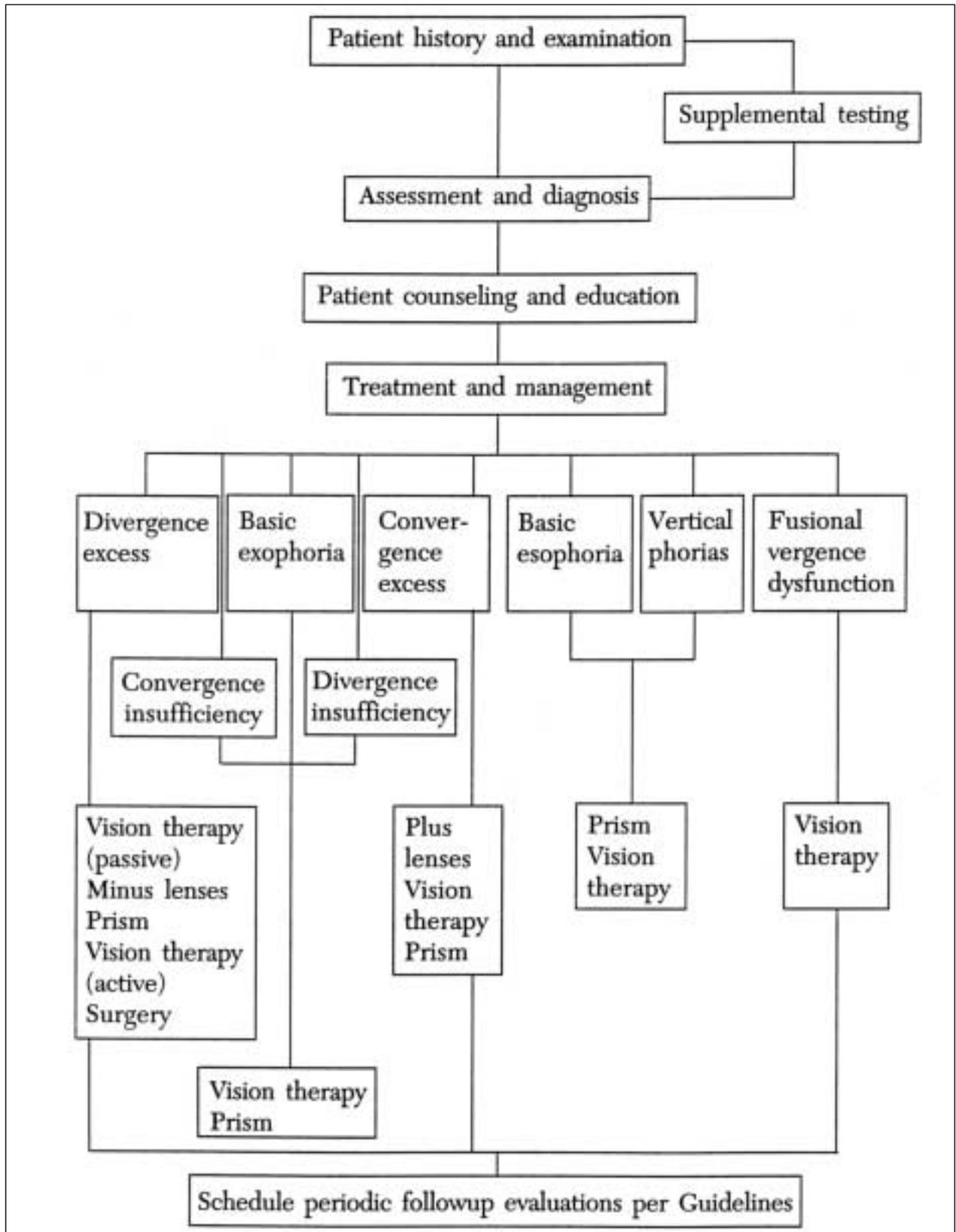


Figure 2 Optometric management of the patient with vergence dysfunction: a brief flowchart (reprinted with permission from the American Optometric Association, 1998).

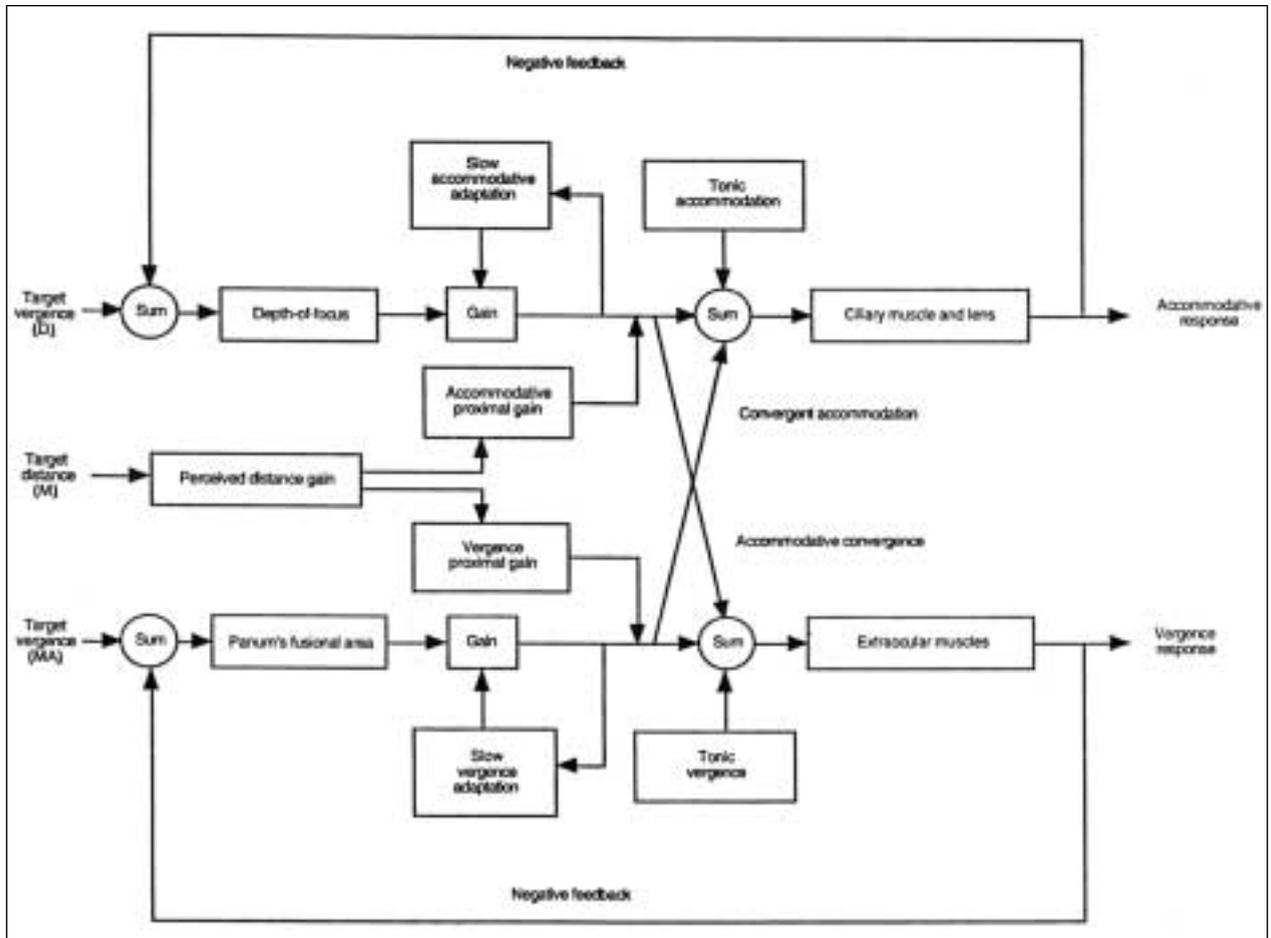


Figure 3 Simplified, conceptual model of Hung et al., 1996 (reprinted with permission, from Ong and Ciuffreda, 1997).

metric vision therapy will be provided. In addition to specifying and describing these direct applications, the importance of models will be further developed in subsequent sections. *Second*, selected research studies that provide support for the scientific basis for and efficacy of optometric vision therapy will be reviewed. The emphasis will focus on objective findings, although the results of other carefully conducted clinical investigations will be considered. And *third*, more global aspects will be considered, including the relationship between vision therapy, general motor learning, and oculomotor plasticity.

Models of the accommodative and vergence systems

Various static and dynamic models of the accommodative and vergence systems have been proposed over the past 50 years.³⁷⁻⁵³ Models provide a comprehensive, organizational framework for logical thinking and conceptual understanding of a system's elemental components within the con-

text of its overall structural framework, especially as the body of knowledge increases. By considering individual components, one can understand when specific system aspects are abnormal prior to vision therapy, which aspects normalize subsequent to vision therapy, and how vision therapy itself may be specifically tailored based on the overall model structure. All of these points will be addressed to some extent in subsequent sections of this article. Models also allow non-invasive, "dry dissection" and testing of a system and its underlying neurological control structure by performing computer simulations incorporating a variety of mathematical techniques. For example, sensitivity analysis⁴⁹ allows one to ascertain the likelihood of a specific oculomotor system component being responsible for the observed anomalous response pattern. As an illustration, it was found that the oculomotor gain control components were most sensitive to variation in model parameter values, as gain terms effectively multiply the incoming system error information.

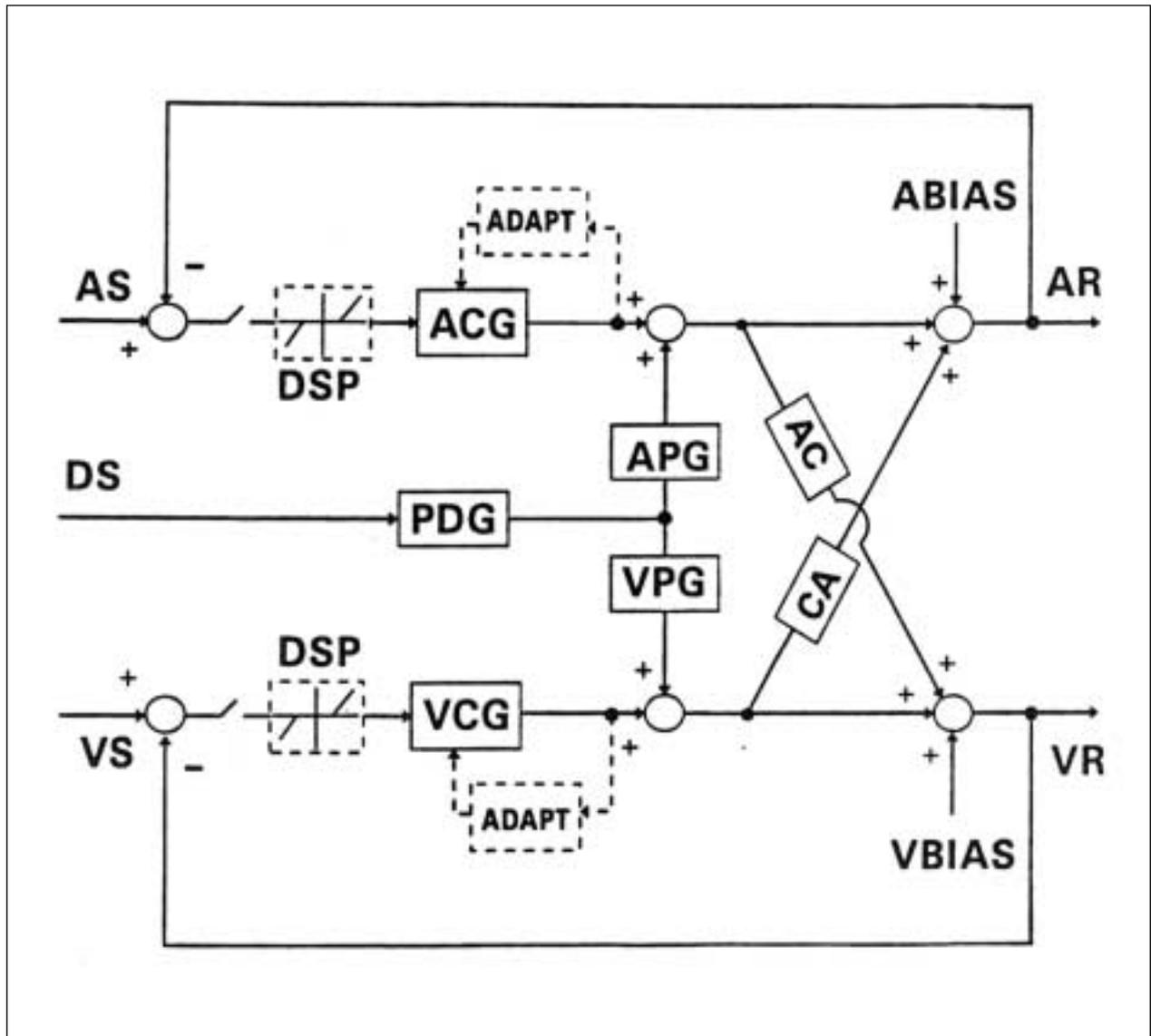


Figure 4 Complete nonlinear static interactive dual-feedback model of the accommodative and vergence systems. For the *accommodative* system, the switch controls feedback to accommodation. With the switch closed, the input to the accommodative dead-space operator (*DSP*, which represents the depth-of-focus) is effectively zero. On the other hand, with the switch open, the difference between accommodative stimulus (*AS*) and accommodative response (*AR*), or accommodative error, is input to *DSP*. The output of *DSP* is multiplied with the accommodative controller gain (*ACG*) to give the accommodative controller output. The controller output is input to an adaptive element (*ADAPT*), which in turn controls the time constant of the accommodative controller. The distance stimulus (*DS*), or the distance of the target from the viewing subject, is input to the perceived distance gain (*PDG*) element, which represents the subjective apparent distance estimate. The *PDG* output then goes through the accommodative proximal gain (*APG*) element, which represents the contribution from target proximity. The outputs from *ACG* and *APG* are summed at the summing junction and are also crosslinked to the vergence system via gain *AC*. The accommodative bias (*ABIAS*), or tonic accommodation, is summed at the next summing junction, along with the crosslink signal from the vergence controller output via *CA*. These four signals are added together to give the overall accommodative response. Analogous descriptions of the parameters are applicable for the *vergence* system. (Reprinted with permission from Hung et al., 1996).

Thus, small variations in, or deviations from, the norm would be predicted to have large effects on system response amplitude—i.e., either being excessive or insufficient.

However, few models have met three basic requirements:

1. model parameter values that agree with empirically derived physiologic data,
2. computer-simulated responses—especially dynamically—to a variety of inputs (such as pulses, steps, ramps, and sinusoids) that agree with the empirically derived physiologic data, and

Table 1. Static model values for accommodation and vergence

Accommodation	Vergence
DSP = ± 0.15 D	PFA = ± 5.0 min arc
ACG = 10.0	VCG = 150
AC = 0.80 D/MA	CA = 0.37 MA/D
ABIAS = 0.61 D	VBIAS = - 0.29 MA
ADAPT = 4.0	ADAPT = 9
APG = 2.10	VPG = 0.067
PDG = 0.212	PDG = 0.212

(See text for explanation and description of abbreviations. Also see related figures/figure legends.)

- homeomorphic model structure that therefore reflects the underlying anatomy and physiology. All of the models to be discussed fulfill these basic requirements.

Static model of the accommodative and vergence systems

A useful static, or steady-state, model of the accommodative and vergence systems and its motor interactions by Hung and his colleagues^{46,47,53} has evolved over the past 20 years. This model and its variations have been useful for furthering our understanding of a wide range of basic mechanisms⁵³ (e.g., vergence and accommodative adaptation), as well as their application to a range of important clinical conditions (i.e., amblyopia,^{54,55} strabismus,⁵⁶ nystagmus,⁵⁷ myopia,⁵⁸ and vergence and accommodative dysfunction⁴⁹). For example, after completion of a dynamic vergence response from far to near, the static model specifies the resultant steady-state error for accommodation (e.g., lag of accommodation as assessed clinically with the retinoscope) and vergence (e.g., fixation disparity as assessed clinically with a disparometer), and allows the modeller to vary the internal, neurologically based control parameters to predict and assess the impact on system errors. A conceptual version of their latest model is presented in Figure 3.⁵⁹

This latest version, transformed into the bio-engineering domain, is presented in Figure 4,⁴⁷ with parameter values presented in Table 1. Progressing from left to right in the figure, it may be seen that the accommodative (*upper*) and vergence (*lower*) negative feedback control loops have similar component structures.

Input

The input or stimulus change for accommodation (AS) (target distance in diopters) and disparity

vergence (VS) (target distance in meter angles, MA) sum algebraically with the negative feedback response of the respective system at that moment. The resultant difference represents the system's initial error (AE or VE, respectively). The input for the proximal branch is perceived target distance, with such perceptually derived proximal information not having a separate feedback loop, but rather inputting directly and

simultaneously into both the accommodative and vergence feedback loops. Under normal binocular, closed-loop (i.e., with blur and retinal disparity feedback present) viewing conditions, the proximal drive only adds 0.4% and 4% to the final steady-state vergence and accommodative responses, respectively.⁴⁷ Hence, *blur and disparity are the primary stimuli in their respective systems. Thus, appropriate manipulation of the blur (via lenses and target distance) and retinal disparity (via prisms, mirrors, and target distance) used in vision therapy will maximize the potential improvements in system responsivity. Proximal information enhances overall responsivity by providing important cue reinforcement derived from the perceived depth information. However, without the constraints imposed by feedback control (i.e., under open-loop conditions), the effect of proximal information on the motor response is substantial.*⁴⁷

Threshold "deadspace" operator (DSP)

This represents the depth-of-focus for accommodation and Panum's fusional areas for disparity vergence. This component allows small neurosensory-based system error (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. However, even if the vergence error does *not* exceed the deadspace, a large residual fixation disparity may adversely affect visual performance or cause a small foveal binocular suppression scotoma to develop. *Therapeutic manipulations involving the development of increased sensitivity to blur and disparity via lens and prism sorting procedures,⁵⁵ respectively, will improve neurosensory sensitivity and increase response amplitude in cases in which this has been reduced.*

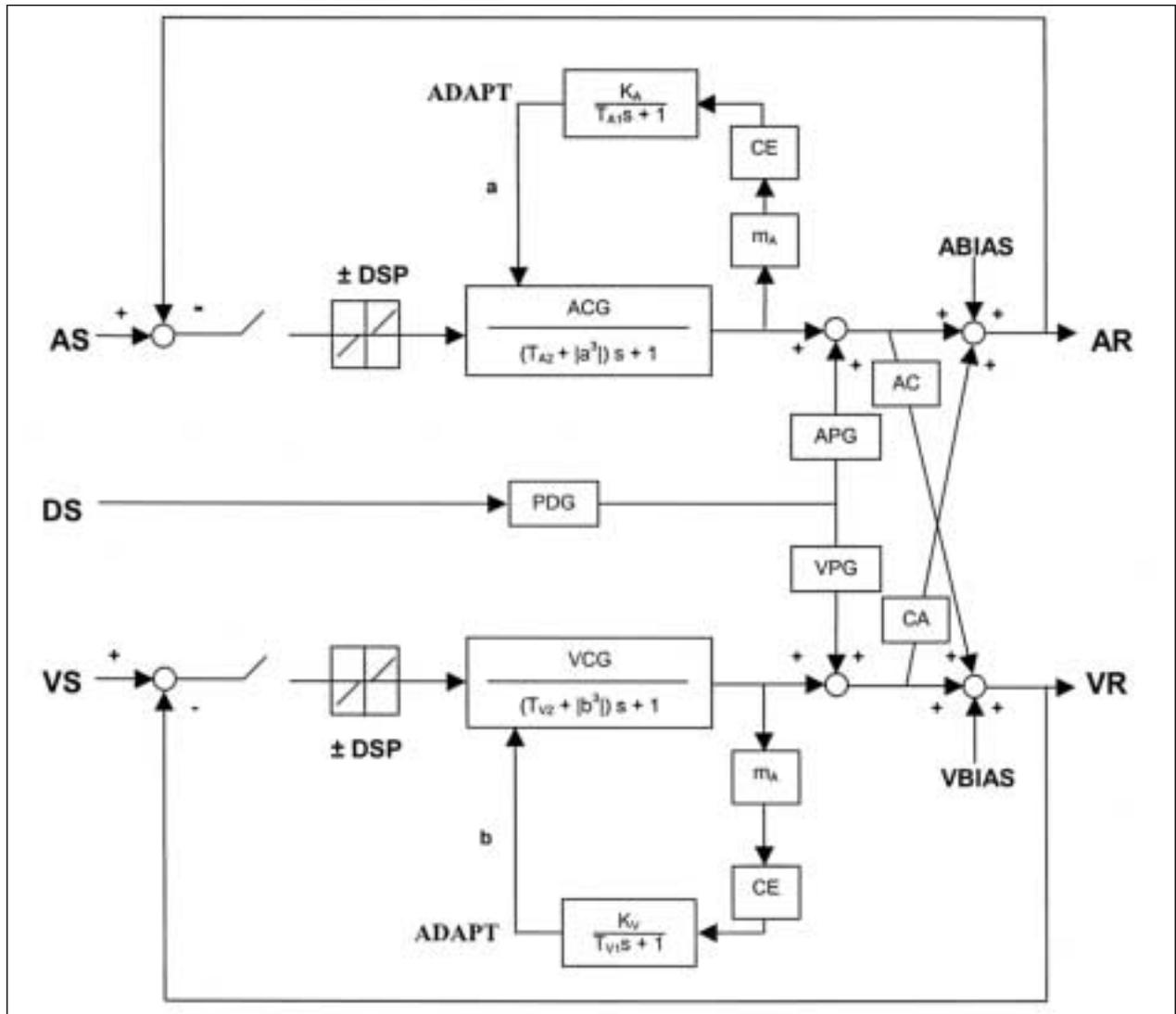


Figure 5 Comprehensive, dual-interactive, static model of the accommodative and vergence systems, with inclusion of dynamic adaptive components. Consider first the accommodative loop. The deadspace element ($\pm DSP$) represents the neuro-optical DOF. The controller output is multiplied by factor m_A and input to a tanh function which serves as a compression element (CE). The factor m_A is used to provide an appropriate range on the abscissa of the tanh function. The CE reduces the controller output for large magnitude inputs so that the adaptation effect is not drastically different at various adapting stimulus levels. The adaptive component is represented by the first-order dynamic element $\frac{1}{T_{A1}s + 1}$

in which T_{A1} is the accommodative adaptation time constant. The accommodative adaptation gain, K_A , controls the magnitude of the adaptive component output level. The adaptive element output, a , modifies the time constant of the accommodative controller via the term, $T_{A2} + |a|^3$, in which T_{A2} is the fixed portion of the time constant. The cubic relationship was obtained empirically to provide a negligible increase in time constant for a small amount of adaptation, but very long time constant for a large amount of adaptation. A similar configuration applies to the vergence system, where the deadspace element ($\pm DSP$) represents PFA. The vergence adaptive components consist of multiplier m_V , compression element CE, adaptive gain K_V , adaptive time constant T_{V1} , adaptive element output b , and controller time constant $T_{V2} + |b|^3$. (Reprinted with permission from Hung and Ciuffreda, 1999).

Gain

The accommodative ($ACG = \frac{AR - ABIAS}{AE - DSP}$) and vergence ($VCG = \frac{VR - VBIAS}{VE - DSP}$) 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 majority of the neurological control signal to formulate the final steady-state motor response. For example, an abnormally high accommodative controller gain would result in accommodative excess, whereas

an abnormally low gain would result in accommodative insufficiency. Thus, therapeutic manipulations that normalize gain will markedly improve response accuracy. The output of the controller gain is then input to three other components (see next three components below).

Adaptive gain loop

Although typically regarded to be a dynamic model element, following intense and prolonged nearwork, it may bias the final, static open-loop or closed-loop system response. However, under nonsustained viewing conditions, its value is zero (see later dynamic model section).

Crosslink gain

The crosslink gain (AC for accommodation and CA for convergence) multiplies the output of the direct ACG or VCG pathway, respectively. For accommodation, this new value represents the effective accommodative-convergence to accommodation ($\frac{AC}{A}$) ratio, whereas for convergence it

represents the effective convergence-accommodation to convergence ($\frac{CA}{C}$) ratio. These gains have

been found to be highly sensitive to parameter value variations, and thus responsible for producing a range of clinical oculomotor imbalances.⁴⁹ For example, an abnormally low AC crosslink gain value would result in convergence insufficiency, whereas an abnormally high value would result in convergence excess (see Figures 3 and 4).

Tonic input

Tonic input for accommodation (ABIAS) and vergence (VBIAS) has been speculated to reflect low-level, stable midbrain baseline neural innervation, although other brain regions may be involved. Although both the tonic vergence and accommodative terms have substantial effects on the response amplitude with both systems rendered open-loop (i.e., with their 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.⁴⁶ Thus, any abnormality of or therapeutic effect on system responsivity under normal viewing conditions would be minor. This is shown in the following equation and example with respect to monocular blur-driven accommodation, in which:

$$AR = \frac{ACG}{1+ACG} \times AS + \left[-DSP \times \frac{ACG}{1+ACG} + ABIAS \times \frac{1}{1+ACG} \right]$$

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

$$VR = \frac{VCG}{1+VCG} \times VS + \left[-DSP \times \frac{VCG}{1+VCG} + VBIAS \times \frac{1}{1+VCG} \right]$$

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 gain output is also sent to the neurological summing junction, where it adds with the crosslink output and the tonic input, both of which have only modest-to-moderate influence on the fellow system, to formulate the final combined signal to drive the respective system. This summing junction may reside in the midbrain.

Peripheral apparatus

The output of the summing junction proceeds to cortical and subcortical centers related to accommodation^{60,61} and vergence⁵¹ to formulate the aggregate neural signal. It then advances to innervate the appropriate peripheral apparatus—the ciliary muscle and lens complex for accommodation and the extraocular muscles for vergence.

Output

These motor changes are then fed back to the initial summing junction via the 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. If the error cannot be reduced for whatever reason, such as low vergence controller gain, however, then diplopia would result; for accommodation, the outcome would be sustained blur.

Incorporation of adaptive dynamic components into the model

In addition to the basic dual-interactive static components described above, adaptive dynamic gain components were more-recently incorporated into the accommodative and vergence controllers for

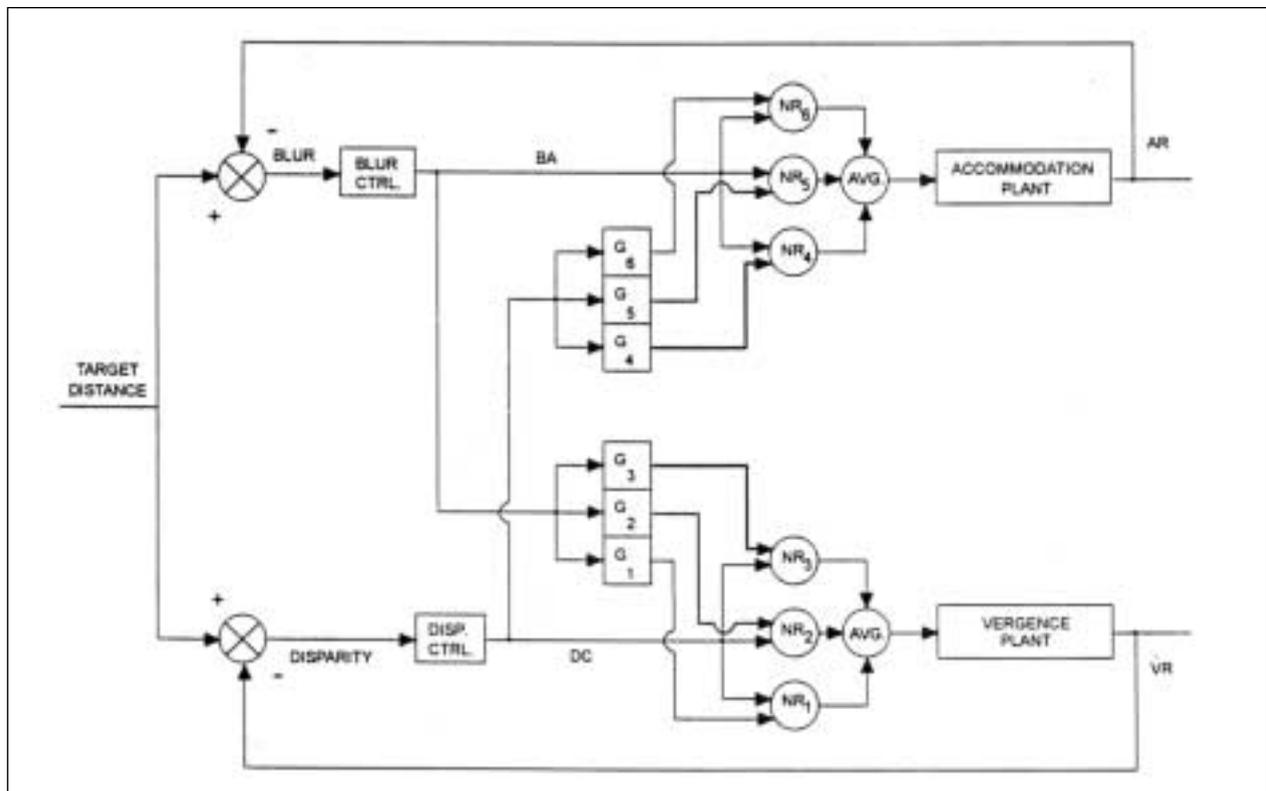


Figure 6 Neurophysiological version of static accommodative and vergence model of Hung and colleagues. Symbols: *BA* = blur-driven accommodation, *DC* = disparity-driven convergence, *NR* = midbrain near response cells, *G* = midbrain near response cell gains, *AR* = accommodative response, and *VR* = vergence response. (Reprinted with permission from Mays and Gamlin, 2000). For Figures 3, 4, 5, and 6, see text for explanation of symbols.

establishment of a new dynamic, time-dependent, nearwork oculomotor model,⁵³ which was specifically developed to gain insight into the clinical entities of nearwork-induced transient myopia and permanent myopia⁵⁹ (see Figure 5). The adaptive loops function to sustain the motor response for a prolonged period (i.e., several seconds or minutes); for example, during reading. The adaptive gain element in each feedback loop receives its input signal from the controller (ACG or VCG) output, with the adaptive loop output in turn modifying the time constant of the controller via terms 'a' and 'b' for the accommodative and vergence loops, respectively. For example, the accommodative controller output (ACG) is input to a multiplier (m_A) and compression element (CE) to drive the adaptive element having gain (K_A) and time constant (T_{A1}). Of considerable clinical importance was the finding that this accommodative adaptive gain, K_A , was the primary model component related to nearwork-induced transient myopia (NITM):⁵⁹ high adaptive gain, as found in myopes, produced significantly slowed decay of accommodation to pre-task baseline distance levels. And accommodative vision therapy normalized this model parameter value⁶² (see 'Accommodative Ther-

744

apy' section). The multiplier and compression elements are necessary to provide a saturation effect for large inputs that are found in the various adaptation experiments. The adaptive element output, 'a', is incorporated into and modifies the overall time constant of the accommodative controller via the term, $T_{A2} + |a^3|$, in which T_{A2} is the fixed portion of the time constant. The cubic relationship was obtained empirically to provide negligible increase in the time constant for smaller amounts of adaptation, but a larger increase in the time constant for greater amounts of adaptation. Similar to the accommodative adaptive element, the vergence adaptive component consists of multiplier, m_V , compression element, CE, adaptive gain, K_V , adaptive time constant, T_{V1} , adaptive element output, 'b', and controller time constant, $T_{V2} + |b^3|$.

Neurophysiological version of static model of the accommodative and vergence systems

Recently, a neurophysiologically based configuration of the basic Hung and et al.'s^{45,47} static model of the accommodative and vergence system has been proposed⁶³ (see Figure 6; compare

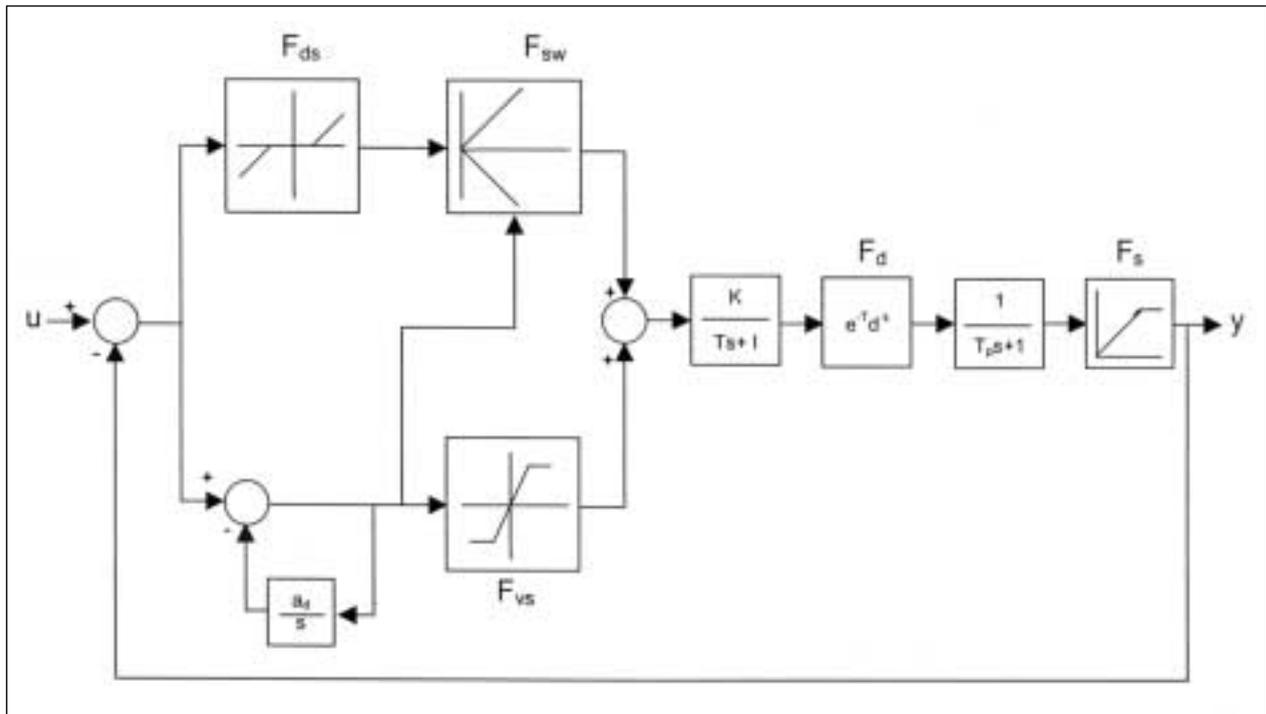


Figure 7 Simplified version of Krishnan and Stark's (1975) dynamic model of accommodation. Symbols: F_{ds} , depth of focus; F_{sw} , switching component (even-error component); F_{vs} , velocity-sensitive saturation, or velocity operator, which limits the velocity change; u , input; $\frac{a_d}{s}$, lead/lag term

(quasi-derivative controller, or velocity operator; $a_d = 10$ and involves dynamics and stability); F_s , accommodative amplitude, or "plant" saturation; K , gain; T_p , time constant or decay for the accommodative peripheral apparatus ("plant") = 0.4 sec; F_d , time delay = $e^{-t}d^s$, in which T_d = accommodative latency = 0.38 sec; y , output; $\frac{1}{s}$, integrator; $\frac{1}{1+Ts}$, leaky

integrator; t , neural time constant, or accommodative decay = 10 sec (reprinted with permission from Krishnan and Stark, 1975).

with Figure 4). All of the intermediary block elements of the earlier static control system models described have been replaced by their neurological analogs—namely, gains (G), near response cells (NR), and neural averagers (AVG). Hence, the bioengineering models described earlier are indeed homeomorphic, and thus have true neurophysiological representation, albeit somewhat spartan at the present time. However, no simulations have been performed using this model.

Dynamic models of accommodation

Figure 7 presents a dynamic model of the accommodative system. This model is adapted from Krishnan and Stark (1975).⁴³ *It has tremendous potential for future use in understanding the component contribution to a range of clinical dynamic accommodative disorders, including the dynamic accommodative dysfunctions discussed in the next section.* The various model elements—progressing from left to right in the figure—are discussed below (also see the legend for symbols and additional information).

Input (u)

The input is target accommodative stimulus level; that is, the target distance in diopters. It sums with the instantaneous accommodative level of the system via the negative-feedback loop. The difference represents the resultant system error.

Threshold "deadspace" operator (F_{ds})

This represents the depth-of-focus, which allows for a small neurosensory-based system error and resultant retinal defocus to be tolerated without the perception of blur. *As discussed earlier for the static model, therapeutic manipulation of this component will improve accommodative accuracy.* If the input error exceeds this threshold level, it proceeds to drive the system.

Nonlinear switching element (F_{sw})

Because blur is an even-error signal (i.e., it lacks directional information),³⁹ this element uses the sign information from the derivative operator to determine its direction. It generates a signal that is directionally-correct and proportional to the magnitude of blur.

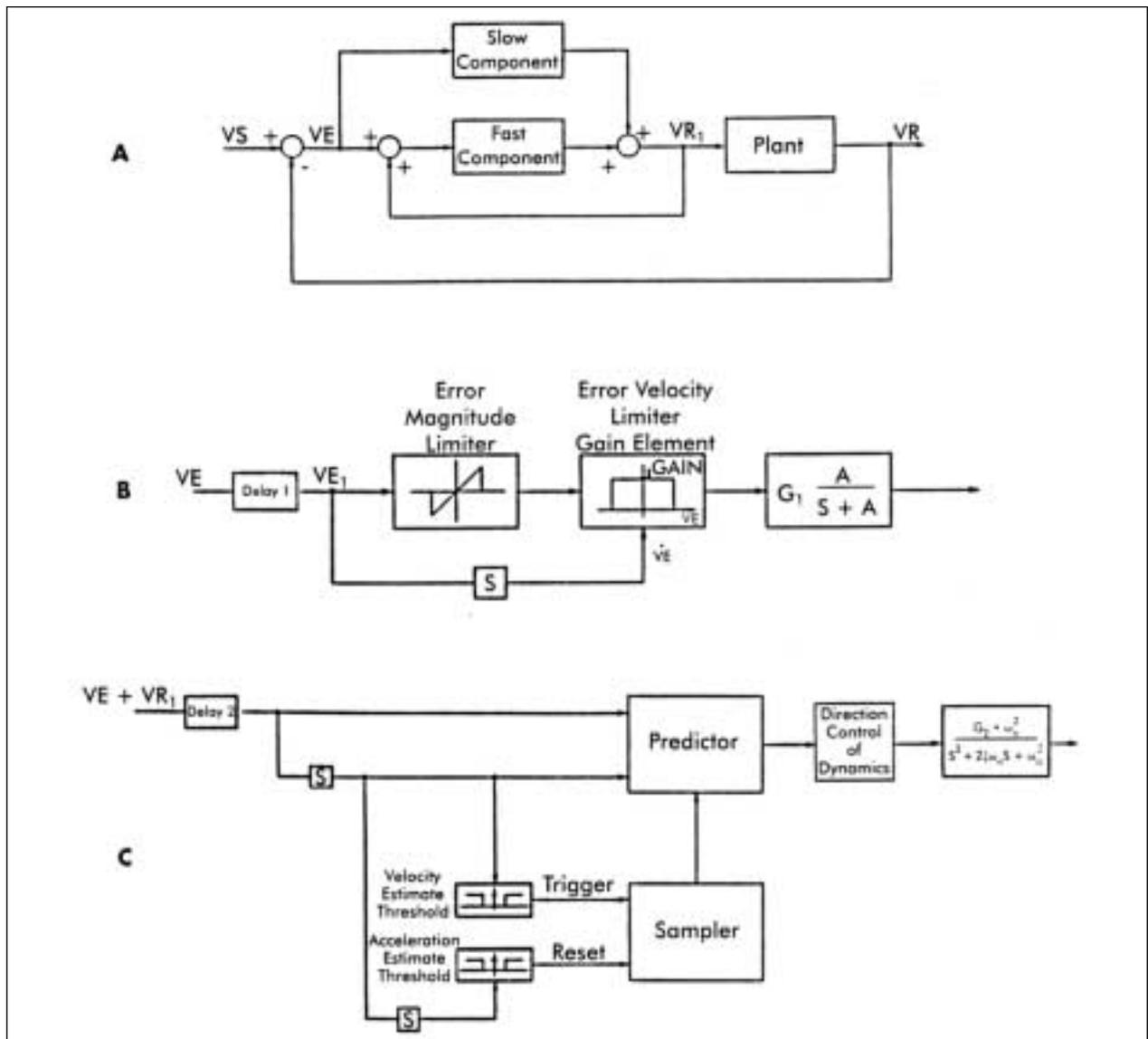


Figure 8 **A**, Overall dynamic model of disparity vergence system shows slow and fast components in the forward loop. Slow and fast component responses are summed to give VR₁. Internal positive feedback from VR₁ is summed with vergence error (VE) to give an estimate of target position. **Plant** represents mechanical properties of eyeball and musculature and is assumed to have unity gain (= 1) for the vergence simulation. Vergence response (VR) is subtracted from vergence stimulus (VS) to give vergence error (VE). **B**, Slow component in forward loop: delayed vergence error (VE₁) is vergence error (VE) delayed by 200 msec (delay 1). Error magnitude limiter (up to 1 degree) and error velocity limiter gain element (up to 2 degrees/sec) simulate range of slow component dynamics. Time constant 1/A is 10 seconds. Gain (G₁) was determined via simulation to be 30. VE, velocity; **A**, reciprocal of the time constant; **S**, Laplace operator; $\frac{1}{s}$, differentiator. **C**, Fast component in forward loop: vergence error (VE) is summed with VR₁ to give an estimate of target position. Delay element (delay 2) represents effective delay throughout fast component. Estimated target velocity above a threshold of 1.7 degree/sec is used to trigger sampler. Sampler enables predictor to use estimated target position and velocity to predict future position of, for example, a ramp stimulus. After triggering, threshold increases slightly to 2.1 degrees/sec. This accounts for initial step but subsequent smooth following seen in response to a 1.8 degree/sec ramp stimulus. If estimated stimulus velocity remains constant, sampler repeats every 0.5 second. This accounts for staircase, steplike responses to ramp stimuli. Sudden large changes in velocity will reset sampler. This accounts for ramp-pulse data. Predictor also reduces its calculation time, thus reducing delay 2, for repetitive stimuli, such as sinusoids. This accounts for relatively small phase-lag found in sinusoidal responses (reprinted with permission from Hung et al., 1986).

Derivative controller ($\frac{a_d}{s}$)
 This parallel, pseudo-derivative (i.e., a mathematical approximation to a true derivative) controller component is a velocity operator. It generates the derivative of the error signal (i.e., the instantaneous velocity) for use by its control process. Such a controller improves the transient stability, as well as the speed, of the response. *Specific therapy (e.g., low-power lens flippers creating*

relatively small blur magnitudes) directed to sensitizing this component will produce more-rapid, oscillation-free, and time-optimal responsiveness.

Nonlinear saturation element (F_{vs})

This element is a velocity-sensitive component that prevents the resultant response velocity from exceeding a specified limit. *This, too, facilitates dynamic response stability and limits the amplitude of instability oscillations of the accommodative response.*

"Leaky" integrator ($\frac{1}{1 + Ts}$)

The "leaky" integrator is a "charge/discharge" element. It represents a central neurological integrating circuit that is rapidly activated ("charged" like a capacitor) by the visual input which then "stores" this information, thus providing steady-state maintenance of the response. "Discharge" of this element is reflected in the decay rate, or reciprocal of the time constant, of the system dynamics.⁶⁴ *Abnormally slow decay may result in transient blur and diplopia for the accommodative and vergences systems, respectively.*

Time delay (F_d)

This represents the combined neural and biomechanical transmission time delays, or latency/reaction time. *As discussed in the next section, accommodative vision therapy has been demonstrated to reduce this delay by a modest amount in cases in which it is abnormally long, with this perhaps also involving attentional and/or predictive aspects.*

Ciliary muscle/lens dynamics ($\frac{1}{T_p s + 1}$)

This represents the biomechanical response characteristics of the combined ciliary muscle/lens zonules/lens/lens capsule complex, or "plant".

Saturation element (F_s)

The saturation element limits the accommodative response imposed by the lens elasticity and related structures. *In effect, it represents the amplitude of accommodation.*

Output (y)

This represents the accommodative system's final output. It is transmitted back to the input via the negative feedback loop, where it is subtracted from the current input level. If a relatively large

residual error remains, the cycle is repeated until an acceptable error is attained.

A different dynamic model, one in which the dual-mode control characteristics were emphasized, was developed by Hung and Ciuffreda (1988).⁴⁸ The rationale for a system having "dual-mode" control—i.e., having "fast" and "slow" dynamic control components—is to provide overall system stability in a feedback loop that has a relatively long latency (i.e., equal to or longer than the dynamic step response latency). *This model provides the scientific basis and justification for use of both step and ramp stimuli in accommodative vision therapy paradigms.*

Based on monocular accommodative tracking responses to ramps (i.e., constant velocity blur-only stimuli), as well as computer simulations, it was concluded that the accommodative system operated in two basic modes, "fast" and "slow", and hence the term *dual-mode* control. The "fast" component is used to track rapidly moving (> 2.0 D/sec), constant-velocity ramp stimuli, as well as instantaneous steps of blur. This "fast" component is preprogrammed and thus open-loop in nature, and does not use visual feedback related to blur of the retinal image during its initial dynamic trajectory. By contrast, the "slow" component is used to track slowing-moving (≤ 0.5 D/sec) ramp stimuli, as well as to correct small, residual errors remaining in the step tracking response. This "slow" component is closed-loop in nature, and thus uses visual feedback related to blur of the retinal image during its dynamic trajectory. And for intermediate velocity ramps of blur stimuli, a mixture of ramp-like and step-like motor responses are intermingled.

Thus, for a typical blur-driven, accommodative step response, the "fast" component is responsible for the initial (300 to 500 msec) and large open-loop, exponentially characterized portion of the motor response, whereas the "slow", visual feedback-driven, closed-loop component is responsible for completion of the response (500 to 700 msec) to attain clear retinal imagery lying within the depth-of-focus of the eye.

Dynamic model of disparity (fusional) vergence

A dynamic model⁴⁵ of disparity vergence is presented in Figure 8. *This model provides the scientific basis and justification for use of both step and ramp stimuli in the vergence vision therapy para-*

digms. The current model has two major subdivisions or components analogous to those proposed earlier for accommodation:

1. The “fast component” is used to track perceived target velocity (with a latency/reaction time or delay of 200 msec) of rapidly moving targets ($> \sim 2$ degrees/sec), such as occurs with rapid ramps, fast sinusoids, pulses, and the more-common and naturalistic steps of disparity input. Rather than using continuous visual feedback—as earlier simple servomechanism-based models had assumed—the fast component is preprogrammed or open-loop, and it is triggered by, and then samples, the rapidly moving target. Its direct feed-forward pathway dynamically represents the “fast” subsystem. That is, this complex is responsible for generating the initial 300 to 500 msec or so of the response to a disparity input and accounts for most of the overall response amplitude. A predictor operator then predicts future target position, such as where it will be 500 msec later based on estimated target position and velocity at the time of sampling, and subsequently sends a command to make such a motor response. The fast component’s motor response approximates an exponential.
2. The “slow component” is used to track slowly-moving targets ($< \sim 2$ degrees/sec), as well as small residual errors remaining in the step tracking response. It is driven by vergence error (with a delay of 50 msec for predictable stimuli and 200 msec for nonpredictable stimuli). Thus, in contrast to the open-loop, nonvisual feedback neurological control structure and response of the initial fast component described above, the slow component incorporates a closed-loop response. Since the slow component uses continuous visual feedback, it functions to correct any slow and small residual or accumulating vergence error (< 1 degree amplitude, < 1.8 degrees/sec velocity), especially

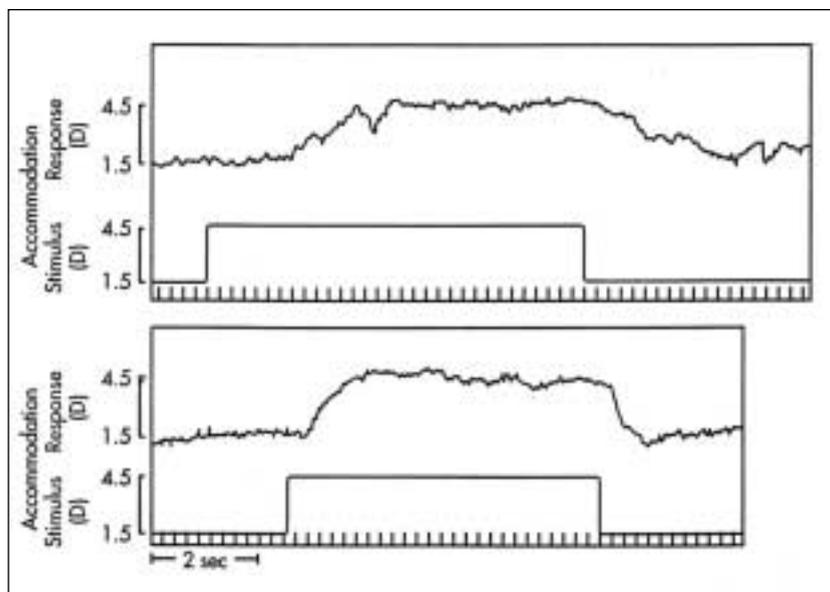


Figure 9 Accommodation responses of subject. *Upper* records show slow response dynamics for positive accommodation and slow, multiphasic response dynamics for relaxation of accommodation before orthoptic training. *Bottom* records show the patient’s improvement after training with faster velocities in both directions of accommodation. Note two discontinuous spikes in the upper record when the patient blinked; stimuli for each are unpredictable step changes between targets set at 1.5 and at 4.5 D (reprinted with permission from Liu et al., 1979).

following slightly inaccurate and rapid open-loop fast component step responses, or also fast-ramp responses. Note that this slow component is *not* to be confused with the “slow” adaptive dynamic gain element discussed earlier, which is responsible for long-term decay following prolonged nearwork.

Thus, in a typical step disparity vergence response, the “fast”, preprogrammed, open-loop component is responsible for the initial and large portion of the response, whereas the “slow,” small, visual feedback-driven, closed-loop component is responsible for completion of the response to attain bi-fixation and fusion within foveal Panum’s fusional areas.

Accommodative therapy

Primary studies

There have been three primary studies conducted over the past 20 years or so that have clearly demonstrated the efficacy of accommodative therapy in patients who manifested combined accommodative insufficiency (i.e., reduced response amplitude) and infacility (i.e., slowed dynamics). These investigations used objective recording techniques to assess therapeutically

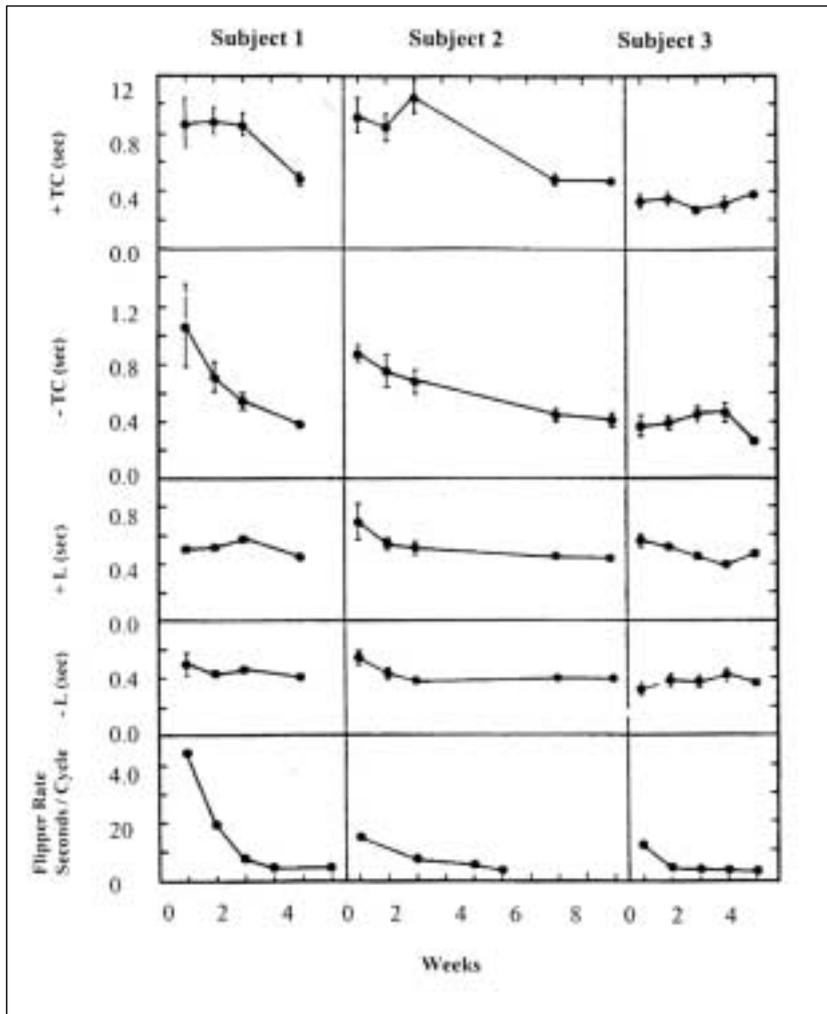


Figure 10 Change of accommodative characteristics in the three subjects as measured weekly through changes in time constants (TC), latencies (L), and flipper rates during their accommodative therapy program. Mean values are plotted for time constant and latency graphs, with standard errors denoted by the error bars. Flipper rates are self-reported by each subject (reprinted with permission from Liu et al., 1979).

related anatomical and physiological changes in the accommodative system, which reflect system neuroplasticity, as well as appropriate statistical analyses whenever possible.

The earliest study was performed by Liu et al.⁶⁵ in 1979. Three young adult patients with symptoms of blur during and/or immediately following short periods of nearwork were tested. A dynamic optometer integrated within a clinical slit-lamp, which directly measured anatomical crystalline lens movement and physiologically-related luminous flux changes in the central anterior region of the crystalline lens and anterior chamber, was used in the laboratory environment to assess accommodative dynamics pre- and post-accommodative vision therapy. Conventional

home therapy was instituted over periods that ranged from 4 to 7 weeks for 20 minutes per day. The therapy consisted of step dioptric blur stimuli (i.e., jump focus and lens flippers) and ramp dioptric blur stimuli (i.e., pencil push-ups),¹⁻³ which is consistent with the dynamic accommodative model and dual-mode accommodative control described earlier. Direct changes in crystalline lens response dynamics pre- and post-therapy are presented in Figures 9 and 10. *Clearly, the overall lens dynamics became more-rapid and normalized following the accommodative therapy.* The primary change was in the innervational and biomechanical aspect of the crystalline lens response time. This can be described and quantified using the bioengineering measure of “time constant,” which refers to the time for an exponential response to attain 63% of its final steady-state amplitude.⁶⁴ Clearly, the time constant for both increasing and decreasing accommodation reduced and normalized (see Figure 10). The reduction in time constant suggested revision and improvement in the neuromotor control program,⁶⁶ thereby leading to a more-efficient and time-

optimal response. Furthermore, a reduction in time constant means a greater rate of change of the anterior curvature of the lens with increasing or decreasing accommodation. That in turn implies either a greater rate of force output produced by the neuromuscular system of the ciliary muscle⁶⁷ and/or more-synchronized innervation. The accommodative latency, or reaction time—the time from stimulus onset to crystalline lens response onset—also normalized (see Figure 10), although this parameter was only slightly abnormal (i.e., prolonged) initially. Reduction of latency means a shorter reaction time of the system, which may indicate more-efficient signal processing at the cortical level. Specifically, with respect to the dynamic accommodative model (see Figure 7), these findings reflect changes in the

threshold deadspace operator, derivative controller, time delay, and ciliary muscle/lens dynamics components. These objectively based measures, as well as the clinical lens flipper accommodative facility measure¹⁻³—which are correlated with objective measures of overall accommodative dynamic responsiveness (i.e., dynamic facility)⁶⁸—were themselves positively correlated with marked reduction in the patients' nearwork symptoms. *Thus, for the first time, it was clearly and convincingly demonstrated that accommodative optometric vision therapy produced true physiological alterations in the accommodative system, reflecting the underlying oculomotor plasticity, with correlated reduction in symptoms.*

In tandem with the above study, control experiments were independently performed.⁶⁹ Subjects included optometry students with accommodative insufficiency, patients with accommodative insufficiency who elected not to receive vision therapy, and visually normal individuals who underwent accommodative therapy. None exhibited any change in accommodative dynamics.

Four years later, the landmark Liu et al. (1979) investigation⁶⁵ was confirmed and extended by Bobier and Sivak⁷⁰ (1983) in five young adult patients who manifested nearwork-related blur symptoms and accommodative infacility. They used the objective laboratory technique of *photorefraction*, which assesses refractive-related, optical changes in the retinal reflection as observed in the plane of the pupil, similar to the standard clinical technique of *retinoscopy*. Patients received 20 minutes of daily home therapy for 3 to 6 weeks, which consisted of monocular and binocular accommodative therapy (step dioptric blur stimuli). In addition to confirming the types of objective changes reported by Liu et al.⁶⁵ (as described earlier), they also found lack of regression of the positive therapy effect over the 4.5-month follow-up test period. Over the same period, no changes in accommodative dynamics were found in their control subject. *Clearly, the positive physiologically based therapeutic effects persisted.*

Three years later (1986), Hung, Ciuffreda, and Semmlow⁴⁴ assessed static (i.e., steady-state changes rather than the dynamic changes assessed in both the Liu et al.,⁶⁵ and Bobier and Sivak,⁷⁰ investigations described above) in 21 college students who reported visual symptoms of

blur and intermittent diplopia after short periods of nearwork. They used a Hartinger coincidence refractometer to measure accommodation objectively via the Scheiner principle of optical vergence at the retina, and used a bioengineering model approach (see Figures 3, 4, 5, and 6) to assess the individual component contribution changes *versus* overall global changes in accommodative responsiveness resulting from the accommodative vision therapy. Accommodative therapy was performed both daily in the home (15 minutes/day; step dioptric blur stimuli) and weekly in the laboratory (30 minutes/session; ramp dioptric blur stimuli) for an average of 12 weeks. *All of the model-based accommodative components tested normalized immediately following the accommodative therapy in 15 of the subjects, with 2 of the 3 components normalizing in the others.* These included tonic accommodation (system bias), slope of the accommodative stimulus/response function (the closed-loop system gain = $\frac{ACG}{1 + ACG}$) and the convergence-accommodation to convergence ratio ($\frac{CA}{C}$ ratio; related to crosslink system gain CA).

All of the above changes improved accuracy of the steady-state accommodative responses at all distances. *Furthermore, there was long-term (i.e., 6 to 9 months) maintenance of the positive accommodative training effects. There was also maintenance of the markedly reduced nearwork symptoms based on a rating-scale questionnaire, as well as clinical lens flipper accommodative facility improvement, which has been found to correlate well with overall lens dynamic responsiveness determined objectively in both young adults⁶⁵ and children.⁶⁸ Thus, on a larger population scale, this study confirmed and extended the two earlier investigations^{65,70} using objective measures of accommodation, statistical analyses, and, for the first time, a mathematically based bioengineering control systems model.*

Other studies

Many patients with accommodative dysfunction not only have symptoms at near, but also transient (5 seconds to 5 minutes) blur at distance following relatively short periods (≤ 15 minutes) of nearwork.⁵⁹ This has been termed *nearwork-induced transient myopia* (NITM).^{53,59} This anomalous accommodative after effect results from an inability to relax accommodation fully and rapidly in the distance in a time-optimal and efficient manner. This appears to reflect physiologically an

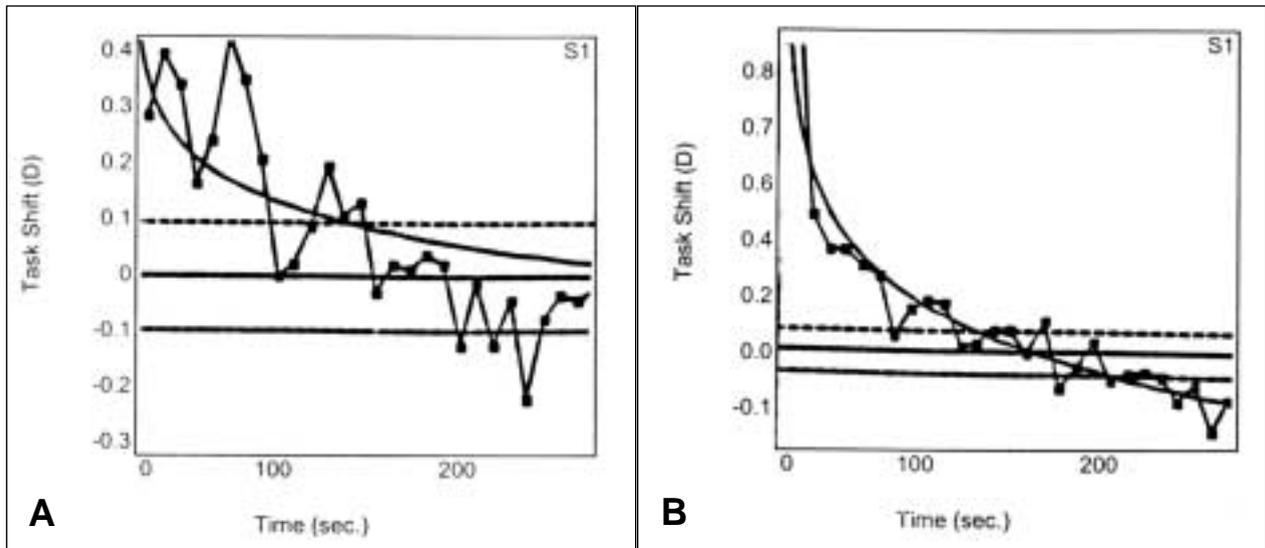


Figure 11 NITM decay curves in a symptomatic patient, S1; **A**, Pre-therapy and **B**, Post-therapy. Exponential curve fit. Dashed lines represent ± 1 SD about the normalized distance refraction (solid line). (From Ciuffreda and Ordonez, unpublished results).

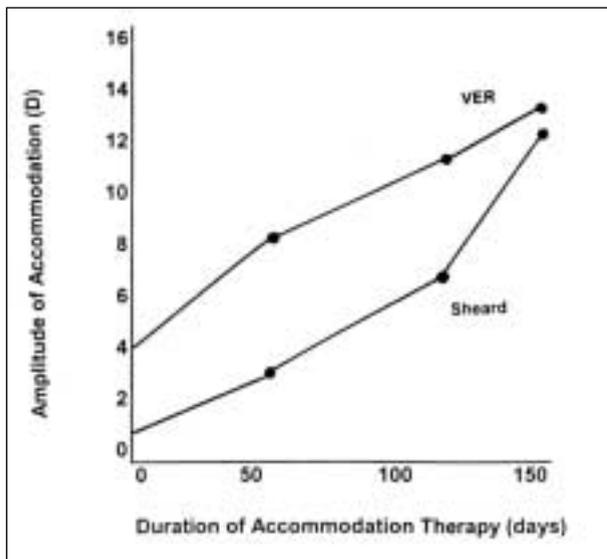


Figure 12 Changes in the amplitude of accommodation measured both objectively (VER) and subjectively (Sheard's technique of minus lens to blur) as a function of the duration of accommodation therapy. Note that while both techniques show a similar progressive increase in the amplitude of accommodation with therapy, the VER nearly always predicted a higher amplitude and showed good concordance with the Sheard value at the latest measurement (reprinted with permission from Lovasik and Wiggins, 1984).

abnormality of the sympathetic system^{59,71} and neurologically increased gain (K_A) of the adaptive loop.⁵³ This transient myopia has been linked to permanent myopia.^{53,59} NITM can be conceptualized in clinical terms as reflecting a very mild accommodative spasm,⁵⁹ and in bioengineering terms as reflecting nonlinear accommodative

dynamics and a hysteresis (i.e., an accommodative after effect) phenomenon.³⁹ Patients with NITM have responded favorably to conventional daily accommodative home therapy (20 minutes/day, 3 to 5 weeks; step dioptric blur-only stimuli and combined blur/disparity/proximal stimuli) and similar weekly laboratory therapy (10 minutes per session).⁶² Using an objective, infrared Canon R-1 autorefractor, including the grating focus principle, dynamic accommodative responses from near-to-far immediately after near-work were found to normalize following this relatively short period of accommodative vision therapy, with the responses becoming more rapid and less variable. Complete results for one subject are presented in Figure 11. This objective finding again correlated with marked reduction in symptoms. Thus, these new objective findings extended the earlier work of others to include an easily remediable accommodative disorder with possible long-term refractive error consequences if left untreated.

Taking a very different objective neurophysiological approach, Lovasik and Wiggins⁷² (1984) measured and compared changes in accommodative amplitude with a standard subjective clinical technique (i.e., minus lens to blur),⁷³ as well as an objective laboratory technique (i.e., visually evoked cortical response),⁷³ which reflects the summed electrophysiological activity in the primary visual cortex. This was assessed in one patient with nearwork symptoms during the course of conventional accommodative ther-

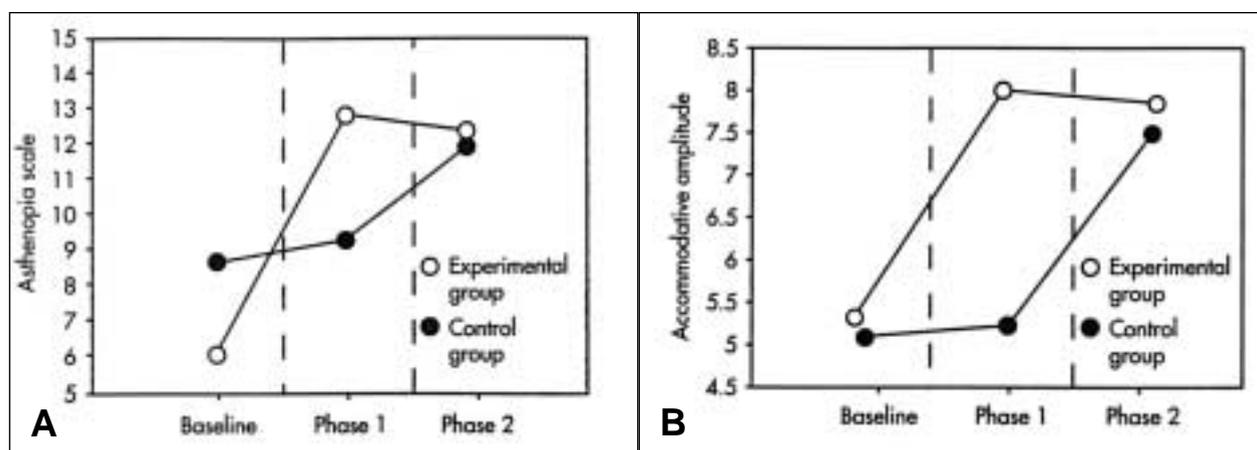


Figure 13 **A**, Mean asthenopia scores are presented on the ordinate, whereas phases of testing are presented on the abscissa. *Open circles (○)* represent patients who received experimental therapy first; *closed circles (●)* represent those patients who received placebo therapy first (reprinted with permission from Cooper et al, 1983). **B**, The abscissa depicts the three phases of testing, i.e., baseline, phase 1, and phase 2. Mean accommodative amplitude for all patients in each phase (determined by minus lens to blur) is plotted on the ordinate. *Open circles (○)* represent patients who received experimental, accommodative training during phase 1 and placebo during phase 2. *Closed circles (●)* represent patients who received the opposite condition, i.e., phase 1, control (placebo); phase 2, accommodative training (reprinted with permission from Cooper et al., 1983).

apy. There was a large and progressive correlated increase in *both* measures over the 4-month vision therapy period (see Figure 12). Thus, *direct and objective measures of visual cortical activity revealed markedly increased responsivity concurrent with the improvement of accommodation, both during and immediately following the course of accommodative vision therapy.*

An investigation by Cooper et al.⁷⁴ focused on changes in nearwork-related asthenopia with conventional accommodative vision therapy.¹⁻³ Eight young adult patients with combined accommodative insufficiency and infacility underwent computer-automated, monocular accommodative facility therapy in the clinic (6 weeks, 2 times/week for 30 minutes; steps of dioptric blur stimuli) using a matched-subjects, cross-over experimental design to control for placebo effects. All patients exhibited a marked reduction in nearwork-related asthenopia as assessed by a 5-point symptom rating scale questionnaire (see Figure 13, A), as well as correlated increases in both clinical static accommodative amplitude (see Figure 13, B) and dynamic accommodative facility. Hence, this relatively short period of accommodative vision therapy was sufficient to remedy both the patients' nearwork-related signs and symptoms. Statistically significant results were found that demonstrated true performance improvement related to the accommodative therapy. Furthermore, there was no such change during the cross-over control phase. Thus, those

who received the control phase first exhibited no change, while those who received the control phase second exhibited maintenance of the initial positive therapeutic effect. *Attaining statistically significant changes with such a relatively small sample size and short therapy duration attests to the robustness of the accommodative therapeutic paradigm.*

In a recent clinical study conducted in a Swedish ophthalmology department, Sterner et al.⁷⁵ tested and trained school-age children ($n = 38$; ages 9 to 13 years) who manifested accommodative dysfunction. Symptoms included asthenopia, headaches, blurred vision, and avoidance of nearwork. Accommodative facility therapy (i.e., lens flippers) was performed at home (15 minutes/day, typically for 8 weeks or less). Relative accommodation improved in all children, and all were now asymptomatic. In a two-year followup, 20 of the original 38 children agreed to participate in a telephone interview. All remained asymptomatic. Thus, *both short- and long-term efficacy of accommodative vision therapy was once again demonstrated, but now in younger school-age children.*

In summary, the above findings clearly and scientifically demonstrate the efficacy of accommodative vision therapy. This is reflected in the objective measures of accommodation and related cortical control structures, formal quantitative and informal symptom surveys, and standard clinical optometric/ophthalmologic test

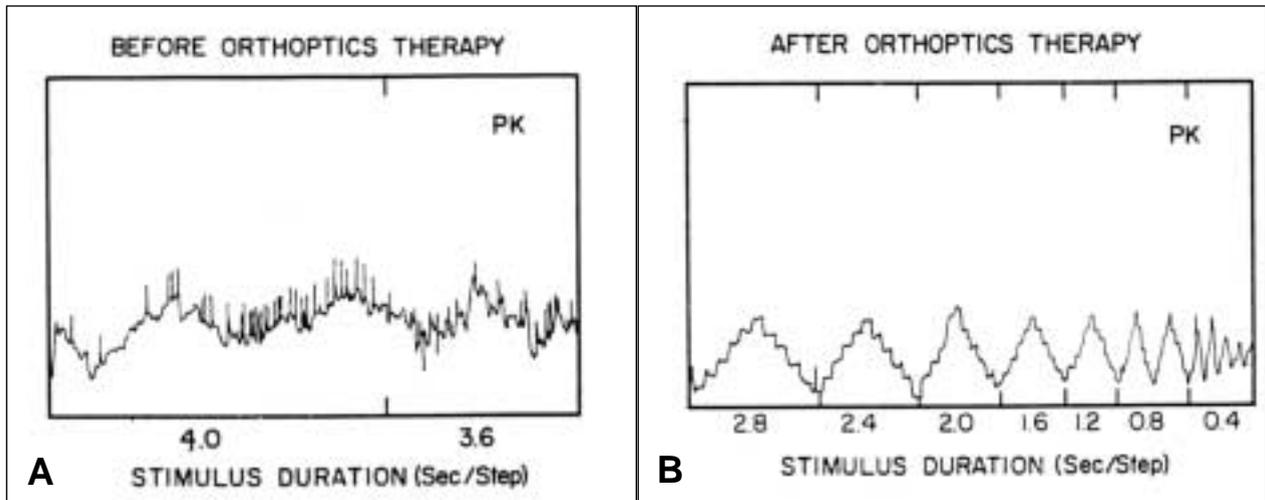


Figure 14 Step vergence tracking recordings before and after orthoptics therapy. **A**, Before orthoptics therapy, subject PK could not adequately track a step vergence staircase stimulus changing at the rate of 4 sec/step. **B**, After 8 weeks of home orthoptics therapy, PK successfully tracked steps changing at the rate of 0.8 sec/step (reprinted with permission from Grisham et al., 1991).

findings. The results are in agreement with a constellation of clinical case reports and case series, as well as retrospective and prospective clinical studies, found in the optometric, ophthalmologic, and orthoptic literatures over the past several decades demonstrating modifiability (i.e., visual system neurosensory and neuromotor plasticity) and normalization of accommodative responsivity, subsequent to relatively short durations of accommodative vision therapy.^{26,32-36}

Fusional vergence therapy

Therapy for fusional (i.e., disparity) vergence disorders has been recognized clinically for decades by both optometrists and ophthalmologists:

“Most recognized textbooks in the treatment of binocular vision disorders, including those of Duke-Elder, von Noorden and Burian, Hugonnier and Hugonnier, Lyle and Wybar, Dale, and Griffin recommend vision therapy, or orthoptics, as the preferred treatment option in cases of symptomatic convergence insufficiency. This unanimity of opinion is justified on the basis of over 1900 reported clinical cases assimilated during the last 47 years in which the average cure rate is 72%.”⁷⁶

Primary studies

The seminal work in this area based on objective documentation was conducted by Grisham et al.^{77,78} over the past two decades in two primary investigations. In both studies, objective recordings of horizontal fusional vergence eye

movements were made that reflected the actual oculomotor physiology related directly to the underlying neurological control structure. In addition, appropriate statistical analyses were incorporated whenever possible.

In the first study (1980), Grisham⁷⁷ differentiated between asymptomatic normal patients ($n = 4$) and those with symptoms related to fusional vergence dysfunction ($n = 4$ to 14, depending on the experiment) based on statistically significant differences in their objectively based, dynamic fusional vergence oculomotor parameters. Test and therapy stimuli consisted of small steps (2 prism diopters) of convergent (10 prism diopter range) and divergent (10 prism diopter range) disparity centered about the heterophoria position with a variable rate of step input change, thus producing variable frequency “staircases” of disparate stimuli. Overall vergence tracking rate (analogous to clinical prism flipper vergence facility¹), percent completion of step responses, response velocity, and divergence response latency discriminated statistically between the two groups; only convergence response latency did not. Thus, this investigation clearly demonstrated that most of the objectively measured dynamic fusional vergence response parameters were abnormal in young adult patients with symptomatic convergence insufficiency.

Given the above critical information, the key question remained to be answered in the laboratory environment using objective measures:

“Does the fusional vergence system have sufficient plasticity to alter and normalize its dynamic neuromotor response characteristics?” The second study (1991) by Grisham et al.⁷⁸ clearly provided a positive answer to this important question, which had its origins in the pioneering work of Javal (1858), an ophthalmologist and the “father of orthoptics.”

Using a stimulus system and objective infra-red eye movement system similar to that used in the aforementioned study, they first tested patients ($n = 3$ to 6, depending on the experiment) with nearwork-related visual discomfort and clinical signs of convergence insufficiency. A variety of standard clinical vergence and accommodative therapy techniques¹⁻³ that involved both step and ramp disparity stimuli were assigned to each patient (8 weeks, 30 minute/day at home). Post-therapeutic objective changes in their fusional vergence eye movements were striking. Figure 14 shows overall fusional vergence tracking to small steps of disparity before and after therapy. Before fusional vergence therapy, only a very slow stimulus rate of change could be followed, and that was poorly executed using small and variable amplitude responses. By contrast, following therapy, the fusional vergence responses were full, and tracking rate was remarkably increased (about 10-fold). These improvements reflected changes in dynamic vergence model parameters analogous to those suggested earlier with respect to accommodation dynamics. The striking vergence improvements were correlated with marked reduction in symptoms, as well as normalization of related clinical findings. Positive therapy results did not show evidence of regression over the 6- to 9-month follow-up period in most cases (see Figure 15). And there were no changes in the control group monitored over a similar time course (see Figure 16).

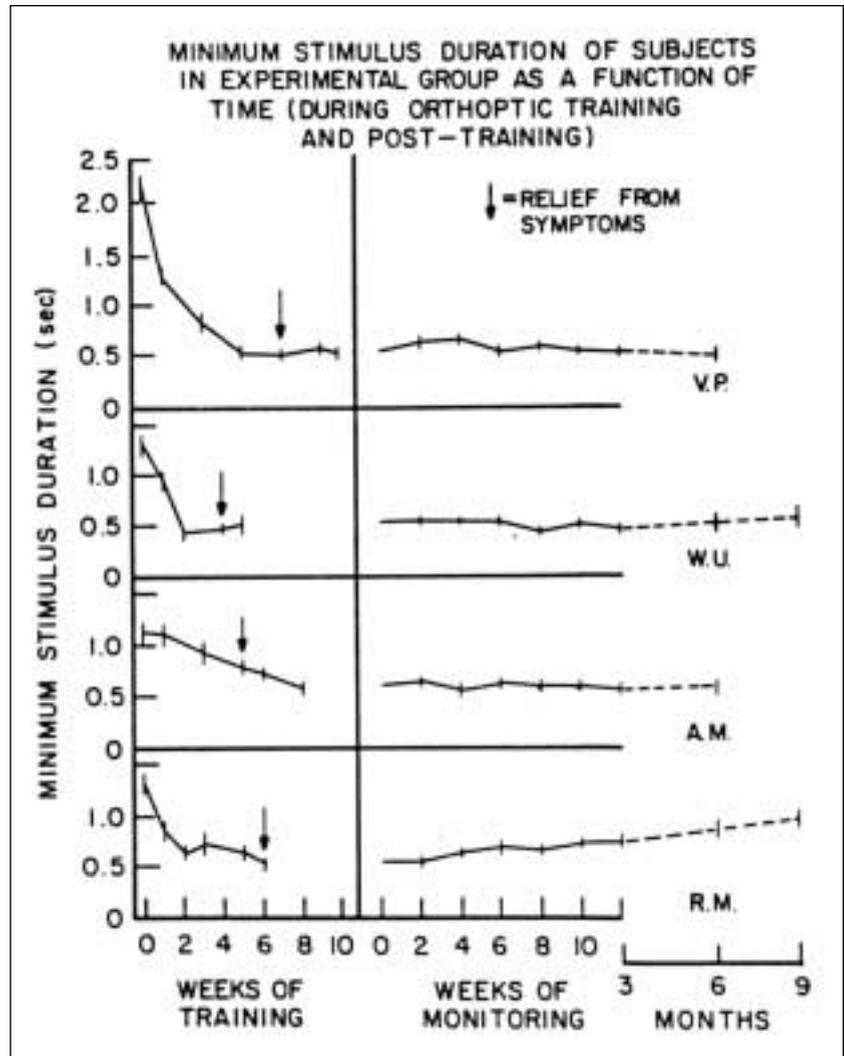


Figure 15 The tracking rate of vergence-deficient subjects as a function of weeks of training and post-training monitoring. All subjects initially showed slow tracking rates that increased to maximum levels in 2 to 8 weeks of orthoptics therapy. All subjects, except for RM, maintained their maximum tracking rate over the post-therapy period of monitoring. The bar (|) indicates the measurement standard error (reprinted with permission from Grisham et al., 1991).

Hence, based on objective measurements and statistical analyses, the above two investigations clearly demonstrated deficient dynamic fusional vergence oculomotor parameters in patients with symptomatic convergence dysfunction, considerable vergence oculomotor plasticity, and relatively long-term retention of the fusional vergence therapeutic effects, along with correlated reduction of symptoms.

Other studies

Another model-based fusional vergence parameter is “vergence adaptation”⁷⁹ (see Figures 4 and 5). It is believed to be critical for a range of vergence functions,⁷⁹ (e.g., maintaining a stable phoria posi-

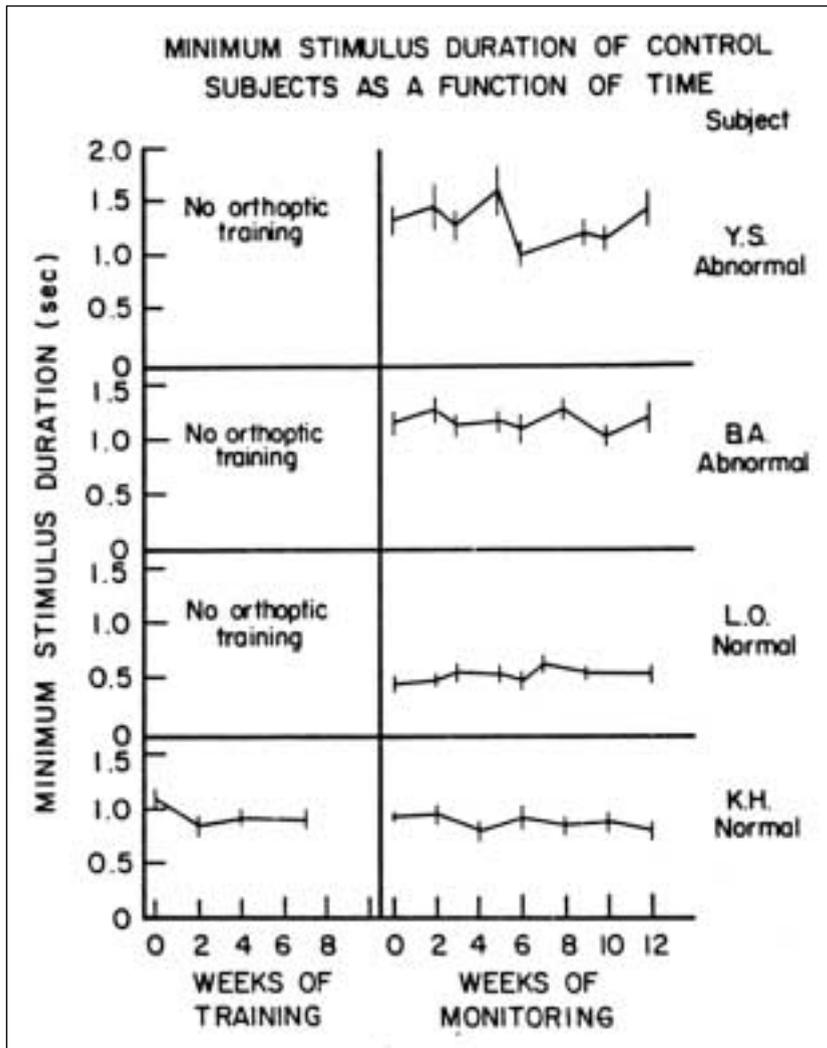


Figure 16 The vergence tracking rate of control subjects as a function of weeks of training and/or monitoring. Two vergence-deficient subjects and one normal subject were monitored for 12 weeks and showed no overall change in their vergence tracking rates. One normal subject, who was trained, showed a small but significant increase in tracking rate that persisted during the monitoring period. The bar (|) indicates the measurement standard error (reprinted with permission from Grisham et al., 1991).

tion⁸⁰ in the presence of changes in disparity stimulation, fatigue effects, illness, etc.). Such changes may occur either transiently or over more-sustained periods of time, such as during prolonged nearwork. Using oculomotor model-driven investigations that incorporated psychophysical test procedures, North and Henson⁸¹ found that vergence adaptation discriminated well between symptomatic and asymptomatic patients who manifested nonstrabismic vergence dysfunctions. In a later study, they—like Grisham et al.^{77,78}—assessed vision therapy-related plasticity of this specific oculomotor component.^{82,83} Seven young adult patients with symptomatic convergence insufficiency and abnormal vergence

adaptation received daily vergence optometric vision therapy at home for 8 weeks (push-ups, physiological diplopia awareness, and relative positive fusional vergence disparity stimulation). Before therapy, responses were poor and outside normal limits; by contrast, after therapy, the overall vergence adaptation response pattern normalized, showing a +163% improvement. Furthermore, symptoms markedly reduced and related clinical findings normalized. In contrast, in the normal control group ($n = 6$), there was no statistically significant change in vergence adaptation (+4.8%).

Lastly, as they did for accommodative optometric vision therapy described earlier, Cooper et al.⁸⁴ formally investigated therapeutically related changes in asthenopia in seven young adult patients with symptomatic convergence insufficiency using a matched-subjects, control group, cross-over experimental design to minimize placebo effects. Following clinical testing and the diagnosis of convergence insufficiency, the patients initially underwent computer-automated (random-dot stereograms with controlled, variable vergence demands), and then conventional (accommodative, fusional, and

stereographic procedures) fusional vergence therapy (100 trials per session; 1 session per week for at least 8 weeks per phase). The vergence stimulus demand was progressively increased automatically by the computer program, based on the patient's rate of improved performance. In the experimental phase, there were statistically significant improvements in the fusible stereogram stimulus range, as well as asthenopia reduction as assessed with their 5-point symptom-rating questionnaire. Furthermore, other abnormal clinical vergence-related findings (such as fixation disparity) normalized with vision therapy. Again, as was true for their accommodative vision therapy asthenopia study described earlier (see 'Accom-

modative Therapy' section), attaining statistically significant changes with such a relatively small sample size attests to the robustness of the fusional vergence therapeutic paradigm.

In summary, the findings clearly demonstrate the scientific efficacy of fusional vergence vision therapy. This is reflected in the objective measures of fusional vergence, formal quantitative and informal symptom surveys, and standard clinical optometric/ophthalmological test findings. The results are in agreement with a constellation of clinical case reports and case series, as well as retrospective and prospective clinical studies, in the optometric, ophthalmologic, and orthoptic literatures over the past several decades demonstrating modifiability (i.e., neuroplasticity) and normalization of vergence responsivity, subsequent to relatively short durations of fusional vergence vision therapy.^{26,32-36}

Discussion

In this article, selected literature has been reviewed that provides supportive evidence of the scientific basis for and efficacy of optometric vision therapy in the areas of nonstrabismic accommodative and vergence disorders. The supporting evidence is manifold, including objective physiological measures, symptom rating-scale questionnaire confirmation, statistical verification, oculomotor model-based bioengineering quantitative approaches, cross-over experimental designs in clinical studies, and correlated and positive clinical test findings. Furthermore, when the above information is combined with the numerous case reports/case studies, and retrospective/prospective investigations summarized and reviewed in the optometric, ophthalmologic, and orthoptic literatures,^{26,32-36} and if one adopts a more global meta-analysis approach,⁸⁵ the evidence in support of optometric vision therapy in patients who manifest symptomatic, nonstrabismic vergence and accommodative disorders is even stronger.

How models of the accommodative and vergence systems can be helpful to the clinician

Numerous examples of how these models have been used to provide new insights regarding diag-

Table 2. Neurophysiological analogs of static and dynamic model components of accommodation and vergence

Model component	Probable neural sites
Depth-of-focus (DF)	Areas V1, V2 visual cortex (sensory; contrast detectors)
Panum's fusional areas (PFA)	Areas V1, V2 visual cortex (sensory; disparity detectors)
Controller gain (ACG; VCG)	Midbrain (motor near response cells)
Crosslink gain (AC; CA)	Midbrain
Adaptive gain (ADAPT)	Cerebellum; neuroreceptors of ciliary body
Tonic innervation (TA; TV)	Midbrain

nostic aspects of static and dynamic accommodative and vergence oculomotor anomalies and their specific abnormal model-based subcomponents, as well as the effectiveness of specific therapeutic subcomponent targeting, have been enumerated and detailed.

Let us reinforce this notion using a specific case example. Suppose an adolescent patient exhibits reduced (by 1 D) steady-state accommodation at near under binocular viewing conditions as assessed by dynamic retinoscopy.⁷³ Several possible individual static model components may be involved (see Figures 3, 4, and 5), such as the depth-of-focus, accommodative controller gain, adaptive gain, tonic accommodation, proximal accommodation, and convergence accommodation. *First*, the magnitude of vergence accommodation is typically relatively minor, as most of a system's response output is derived from the specific stimulus drive (i.e., blur) within its own negative feedback control loop. *Second*, under normal binocular viewing conditions—with both the accommodative and vergence negative feedback control loops in their closed-loop mode, such that blur and disparity information, respectively, are effective—the proximal and tonic terms are negligible, especially at near. *Third*, since one is not dealing with sustained (i.e., minutes) accommodation at near, the adaptive loop would not be activated. Thus, one is left with two components; namely, depth-of-focus and accommodative controller gain. It can be difficult to disentangle their individual component effects on steady-state accommodation, as their typical abnormal dynamic accommodative retinoscopy response would independently result in reduced static

accommodative levels. Assuming a high state of attention and motivation during the diagnostic testing—and in the absence of more-severe ocular conditions such as amblyopia and nystagmus—the anticipated increased amount of depth-of-focus might only be 0.25 D or so, which is considerably less than the 1.0 D lag of accommodation uncovered at near clinically. Hence, the accommodative controller gain component would be responsible for contributing the residual amount. Thus, vision therapy would incorporate blur discrimination techniques (e.g., sequential lens sorting⁵⁵) to sensitize and normalize the neurosensory aspects of the depth-of-focus. However—and perhaps even more importantly—monocular and binocular lens flipper therapy using slightly-above-threshold visual acuity letter targets (or perhaps low-contrast gratings) would also be used to force more-accurate accommodation (and thus larger response amplitudes), which translates into normalization of the accommodative controller gain parameter. While additional therapeutic procedures would still be used, only specific ones would be emphasized, perhaps with various degrees of “weighting.” *Such model-based clinical logic and specific component therapeutic targeting may result in even-better final outcomes.*

Homeomorphic nature of the models

As mentioned at the beginning of the model section, a good model should have neuroanatomic and neurophysiologic under-pinnings. Thus, it should be homeomorphic in nature. At this time, some of this information is not fully understood. With recent advances in brain-imaging technology, however, answers to these critical questions should be forthcoming. Within these constraints, an attempt has been made to relate the static and dynamic model components to possible sensory and motor neural sites of involvement (see Table 2). However, additional brain areas are probably involved in dissemination of this information in a more-complex and comprehensive neural network.^{51,61,62}

A primary thrust of this article has been on those studies that have used objective measures of accommodative and vergence responsivity before and after therapy, as well as in some cases during specific therapeutic phases. *In the future, an additional and important objective measure should include neural imaging: first, to document the affected site and its cerebral pervasiveness and second, to*

assess directly the neurological therapeutic changes and effectiveness.

Motor learning and motor planning

Motor learning (i.e., perceptual-motor skill acquisition) involves describing and explaining changes in motor performance and motor control that occur with specific practice paradigms.¹⁹ Acquiring new (or altering old) motor skills takes place in three well-defined phases.

1. **Verbal-cognitive phase:** This primarily involves conscious thinking and planning of movement strategies; hence, one either learns new movement patterns, or reshapes old ones, via a trial-and-error approach. Initial performance varies considerably as a range of movement strategies is attempted, with most being discarded in favor of the most-effective and efficient one.
2. **Associative phase:** This single, new movement pattern is practiced repeatedly and “fine-tuned.” When the movement pattern is learned reasonably well, increases in task complexity and changes in prevailing conditions are instituted to ensure task success and systematic continuation of motor skill development.
3. **Autonomous phase:** The highly practiced movement pattern, or motor skill, has become automatic and below the level of consciousness. Motor performance is consistent, precise, efficient, “time-optimal,” and accurate. Hence, the motor pattern becomes “pre-programmed” and, in essence, “open-loop” (i.e., without the need to consciously monitor its feedback). This is in contrast to the earlier two phases, in which feedback is essential and continuously monitored (i.e., closed-loop) to improve motor performance.

The above description of motor skill acquisition must be learned for each new motor skill. There is relatively little transfer. *Thus, if this basic tenet of motor learning is related specifically to optometric vision therapy for nonstrabismic accommodative and vergence disorders, it provides justification for training of a series of oculomotor learning skills encompassing a wide range of stimuli and test conditions to ensure development of the full complement of motor skills that can then be used in one’s everyday environment.*

These principles of motor learning can be conceptualized mechanistically in a global manner (as shown in Figure 17, which presents the classical

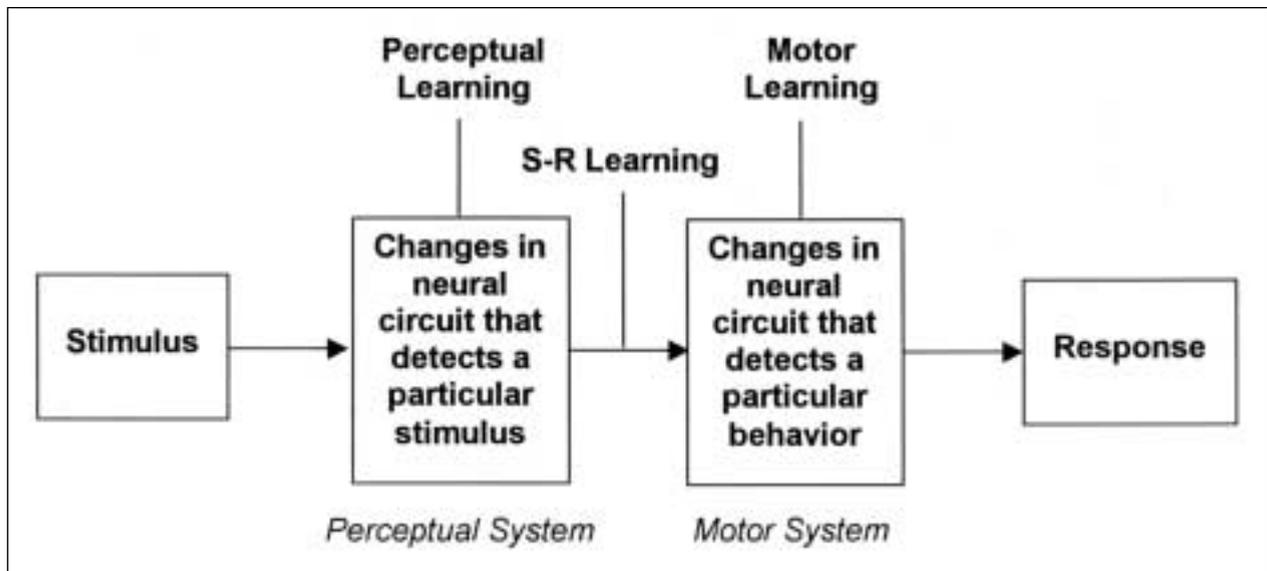


Figure 17 Schematic view of the different types of learning examined by neurophysiologists (Reprinted with permission from Carlson, 1994).

psychology “stimulus-response” paradigm).⁸⁶ It can be further appreciated in the dynamic motor response patterns (response acceleration as a function of time) comparing a novice versus expert squash player (see Figure 18).⁸⁷ What is referred to as greater “consistency” for the expert player can be translated into the more-general neurological principle of neural signal “synchronization.”^{19,88-90} With appropriate practice and repetition, there is more synchronous firing in the group of motor neurons controlling the muscles involved in a specific movement. Greater mass neuronal synchrony translates into greater motor response consistency and time optimality.⁶⁷ Clearly, this idea can be transferred to optometric vision therapy for nonstrabismic accommodative and vergence disorders. One would use monocular lens flipper techniques, wherein only monocular accommodative neural control is conditioned and shaped, then fusional vergence in isolation using prism flippers and a blur-free DOG (difference of Gaussians) stimulus,^{60,61} followed by a similar process involving binocular interactive closed-loop aspects of the accommodative and vergence systems in the dynamic free-space environment. *Thus, in such therapy, synchronization of motor neurons **within** each system would first be conditioned, followed by synchronization of motor neurons **between** each system.*

Lastly, one can proceed to the synaptic and molecular level of motor learning. This was first enunciated by Hebb in 1949⁹¹ within the context of his

now-classic notion of “cell assemblies,” the predecessor to contemporary “neural networks.”⁹² He stated, “...when an axon of Cell A is near enough to Cell B and repeatedly or persistently takes part in firing it, some growth processes or metabolic changes take place in one or both cells such that A’s efficiency, as one of the cells firing B, is increased.” Hence, by repetition, patterns of neural activation of specific neurons and their interconnections are enhanced, whereas those without such correlated patterns of activity are weakened. This neural enhancement, in effect, produces relatively long-term neural “potentiation,” such that a certain level of neural activation now produces increased cell responsivity, which suggests the efficiency of synapses has been enhanced via the repetitive motor learning process. Current research is focused on determining which neuroreceptor is involved in such learning, with the N-methyl-D-aspartate (NMDA) receptor appearing to be a likely candidate.⁹² *Clearly, such synaptic plasticity and conditioning could account for the motor improvements found during the course of optometric vision therapy for nonstrabismic accommodative and vergence disorders.*

Future directions

While there is considerable evidence for the scientific basis and efficacy of optometric vision therapy, as in any clinical discipline, more research and advances are welcome to understand the basic mechanisms more comprehensively, and

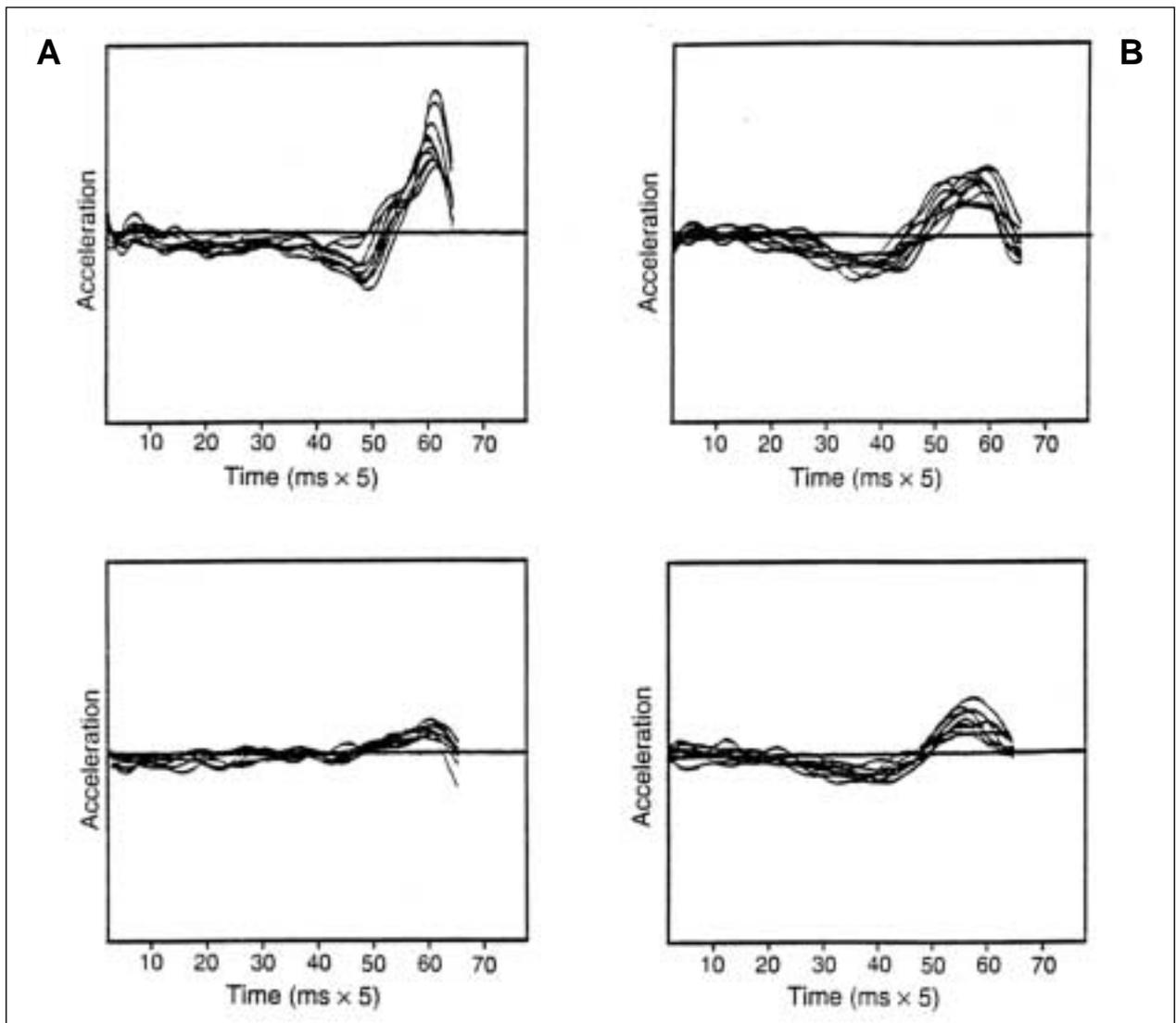


Figure 18 Acceleration-time curves from an expert (A) and a novice (B) squash player executing 10 fast strokes (top panels) and 10 slow strokes (bottom panels). The greater consistency of the expert is particularly evident for the slow strokes. (Reprinted with permission from Wollstein and Abernethy, 1988).

to further enhance the results of clinical interventions. This includes:

1. *Clinical trials with appropriately large sample sizes to investigate and confirm overall group trends, as well as subgroup effects.* Not all patients will respond similarly to the same vision therapy paradigm. It will be important to determine how the subgroups differ, and why, and then to develop more-specific therapeutic paradigms, resulting in greater success levels. Clearly, given the high prevalence of these nonstrabismic accommodative and vergence disorders and their related symptoms in the general optometric clinic, as well as their apparent ease of remediation via optometric intervention, the
- public health impact is enormous. Furthermore, it behooves all parties involved in providing and managing vision health care to lend support for such endeavors.
2. *Objective recording techniques of oculomotor responsivity before, during, and after optometric vision therapy to determine and document specific static and dynamic parameter changes correlated with the therapy.* Such information will not only provide critical insights into the basic neural mechanisms involved, but may also help develop more-specific and “targeted” therapeutic paradigms.
3. *Objective recordings of neural events related to the changes in oculomotor responsivity enumerated above (see item 2).* This would

include functional MRI,⁹³ magnetoencephalography (MEG),⁶⁷ and positron emission tomography (PET)^{94,95} non-invasive brain-imaging techniques to localize the basic neural sites involved in the clinical abnormality, as well as those that contribute to the oculomotor therapeutic changes reflecting oculomotor learning and plasticity.

4. *Investigations into the synaptic and molecular basis of vision therapy and cortical plasticity*,^{86,92,96,97} as pioneered by the classic Hebbian notion.⁹¹
5. *Development of new instrumentation and clinical procedures to assess oculomotor performance in infants and very young children who are "at risk" for development of a binocular vision disorder.*
6. *Further development of computerized technology to standardize training procedures, as well as to provide more controlled home therapy.*^{98,99}
7. *Expansion of the oculomotor models to incorporate such important aspects as attention and proprioception, as well as visual stress-related components and correlated neuropharmacological mechanisms.*³⁶

Acknowledgments

This document was funded in part by a grant from the Mountain States Congress of Optometry, in association with the American Optometric Association.

I wish to thank Drs. Jeffrey Cooper, Sue Cotter, Esther Han, George Hung, Neera Kapoor, Leonard Press, Michael Rouse, Mitchell Scheiman, Irwin Suchoff, Barry Tannen, and Gary Williams for their helpful and insightful discussions and comments.

References

1. Griffin JR, Grisham JD. *Binocular anomalies*, 3rd ed. Boston: Butterworth-Heinemann, 1995.
2. Rutstein RP, Daum KM. *Anomalies of binocular vision: diagnosis and management*. St. Louis: Mosby, 1998.
3. Press LJ, ed. *Applied concepts in vision therapy*. St. Louis: Mosby, 1997.
4. Cornell E, Mitchell R. An evaluation of proximal convergence by the use of infra-red photography. *Aust Orthopt J* 1979;17:24-9.
5. Ciuffreda KJ, Goldrich SG. Oculomotor biofeedback therapy. *Int Rehab Med* 1983;5:111-7.
6. Scheiman MM, Peli E, Libassi D. Auditory biofeedback used to enhance convergence insufficiency. *J AM OPTOM ASSOC* 1983;54:1001-3.
7. Groffman S. Operant conditioning and vision training. *Am J Optom Arch Am Acad Optom* 1969;46:583-94.
8. Letourneau JE. Application of biofeedback and behavior modification technique in visual training. *Am J Optom Physiol Optics* 1976;53:187-90.
9. Feldman J. Behavior modification in vision training: facilitating prerequisite behaviors and skills. *J AM OPTOM ASSOC* 1981;52:329-40.
10. Malmstrom FV, Randle RJ. Effects of visual imagery on the accommodative response. *Percept Psychophys* 1976;19:450-3.
11. Forrest EB. *Visual imagery: an optometric approach*. Duncan, Okla.: OEP Foundation, 1981.
12. Richman JE. The influence of visual attention and automaticity on the diagnosis and treatment of clinical oculomotor, accommodative, and vergence dysfunctions. *J Optom Vis Dev* 1999;30:132-41.
13. Lie I, Watten R, Fostervold KI. Accommodation/vergence/fixation disparity and synergism of head, neck and shoulders. In: Franzen O, Richter H, Stark L, eds. *Accommodation and vergence mechanisms in the visual system*. Basel, Switzerland: Birkhauser Verlag, 2000:303-12.
14. Han Y, Lennerstrand G. Effect of neck muscle proprioceptive activation on the dynamics of monocularly-driven horizontal vergence movements. *Acta Ophthalmol Scand* 1998;76:283-8.
15. Servos P, Goodale MA, Jakobson LS. The role of binocular vision in prehension: a kinematic analysis. *Vision Res* 1992;32:1513-21.
16. Howard IP, Templeton WB. *Human spatial orientation*. London: Wiley and Sons, 1966.
17. Suchoff IB. *Visual-spatial development in the child: an optometric theoretical and clinical approach*. New York: SUNY/State College of Optometry, 1974.
18. Kelso JAS, ed. *Human motor behavior*. Hillsdale, N.J.: Lawrence Erlbaum Assoc. Pub., 1982.
19. Abernathy B, Kipper V, Mackinnon LT, et al. *The biophysical foundations of human movement*. Champaign, Ill.: Human Kinetics Pub., 1997:269-352.
20. Kephart NC. *The slow learner in the classroom*. Columbus, Ohio: C.E. Merrill Books, 1960.
21. Classé JG, Rutstein RP. Binocular vision anomalies: an emerging cause of malpractice claims. *J AM OPTOM ASSOC* 1995;66:305-9.
22. Hokoda SC. General binocular dysfunction in an urban optometry clinic. *J AM OPTOM ASSOC* 1985;56:560-2.
23. Hoffman L, Cohen AH, Feuer G. Effectiveness of optometric vision therapy/orthoptics in a private practice. *Am J Optom Arch Am Acad Optom* 1973;50:813-6.
24. Cooper J, Duckman R. Convergence insufficiency; incidence, diagnosis, and treatment. *J AM OPTOM ASSOC* 1978;49:673-80.
25. Scheiman M, Galloway M, Goutler M, et al. Prevalence of vision and ocular disease conditions in a clinical pediatric practice. *J AM OPTOM ASSOC* 1996;67:193-202.
26. American Optometric Association Concensus Panel with Accommodative and Vergence Dysfunction. Care of the Patient with Accommodative and Vergence Dysfunction. Optometric Clinical Practice Guidelines. St. Louis: AOA, 1998.
27. Scheiman MM, Wick B. *Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders*. Philadelphia: JB Lippincott, 1994.
28. Amos JF, Rutstein RP. Vertical deviations. In: Amos JF, ed. *Diagnosis and management of vision care*. Boston: Butterworth, 1987:515-83.
29. Skeffington AM. *Procedure in ocular examination*. Chicago: AJ Fox Co., 1928.
30. Peckham RH. *The modern treatment of binocular imbalance*. New York: Shur-On Standard Optical, 1928.
31. Duane A. Anomalies of the accommodation, considered clinically. *Trans Am Ophthalmol Soc* 1915;14:386-402.

32. American Optometric Association. Position statement on vision therapy. *J AM OPTOM ASSOC* 1985;56:782-3.
33. The 1986/87 Future of Visual Development/Performance Task Force. The efficacy of optometric vision therapy. *J AM OPTOM ASSOC* 1988;59:95-105.
34. Joint Organizational Policy Statement of the American Academy of Optometry and the American Optometric Association. Vision therapy. *J Behav Optom* 1999;10:109-10.
35. Suchoff IB, Petito GT. The efficacy of visual therapy: accommodative disorders and nonstrabismic anomalies of binocular vision. *J AM OPTOM ASSOC* 1986;57:119-25.
36. Birnbaum MH. *Optometric management of nearpoint vision disorders*. Boston: Butterworth-Heinemann, 1993:381-93.
37. Westheimer G. Amphetamine, barbiturates, and accommodative vergence. *Arch Ophthalmol* 1963;70:830-6.
38. Toates FM. Accommodation and convergence in the human eye. *Measurement and Control* 1969;3:29-33.
39. Stark L. *Neurological control systems*. New York: Plenum Press, 1968.
40. Saladin JJ. Interpretation of divergent oculomotor imbalance through control systems analysis. *Am J Optom Physiol Optics* 1988;65:439-47.
41. Jiang BC. A modified control model for steady-state accommodation. In: Franzen O, Richter H, Stark L, eds. *Accommodative and vergence mechanisms in the visual system*. Basel, Switzerland: Birkhauser Verlag, 2000:235-43.
42. Krishnan VV, Stark L. A heuristic model of the human vergence eye movement system. *IEEE Trans Biomed Engn* 1977;24:44-9.
43. Krishnan VV, Stark L. Integral control in accommodation. *Comp Prog Biomed* 1975;4:237-45.
44. Hung GK, Ciuffreda KJ, Semmlow JL. Static vergence and accommodation: population norms and orthoptics effect. *Doc Ophthalmol* 1986;62:165-79.
45. Hung GK, Semmlow JL, Ciuffreda KJ. A dual-mode dynamic model of the vergence eye movement system. *IEEE Trans Biomed Engn* 1986;33:1021-8.
46. Hung GK, Semmlow JL. Static behavior of accommodation and vergence: computer simulation of an interactive dual feedback system. *IEEE Trans Biomed Engn* 1980;27:439-47.
47. Hung GK, Ciuffreda KJ, Rosenfield M. Proximal contribution to a linear static model of accommodation and vergence. *Ophthalmic Physiol Opt* 1996;16:31-41.
48. Hung GK, Ciuffreda KJ. Dual-mode behavior in the human accommodation system. *Ophthalmic Physiol Opt* 1988;8:327-32.
49. Hung GK, Ciuffreda KJ. Sensitivity analysis of relative accommodation and vergence. *IEEE Trans Biomed Engn* 1994;41:241-8.
50. Schor C, Horner D. Adaptation disorders of accommodation and vergence in binocular dysfunction. *Ophthalmic Physiol Opt* 1989;9:264-8.
51. Schor C, Ciuffreda KJ, eds. *Vergence eye movements: basic and clinical aspects*. Boston: Butterworth, 1983.
52. Schor CM. The relationship between fusional vergence eye movements and fixation disparity. *Vision Res* 1979;19:1359-67.
53. Hung GK, Ciuffreda KJ. Adaptation model of nearwork-induced transient myopia. *Ophthalmic Physiol Opt* 1999;19:151-8.
54. Hung GK, Ciuffreda KJ, Semmlow JL, et al. Model of static accommodative behavior in human amblyopia. *IEEE Trans Biomed Engn* 1983;30:665-72.
55. Ciuffreda KJ, Levi D, Selenow A. *Amblyopia: basic and clinical aspects*. Boston: Butterworth, 1991.
56. Ciuffreda KJ, Hokoda SC, Hung GK, et al. Accommodative stimulus/response function in human amblyopia. *Doc Ophthalmol* 1984;56:303-26.
57. Ong E, Ciuffreda KJ, Tannen B. Static accommodation in congenital nystagmus. *Invest Ophthalmol Vis Sci* 1993;34:194-204.
58. Hung GK, Ciuffreda KJ. A unifying theory of refractive error development. *Bull Math Biol* 2000;62:1087-108.
59. Ong EO, Ciuffreda KJ. *Accommodation, nearwork, and myopia*. Santa Ana, Calif.: OEP Foundation Press, 1997.
60. Ciuffreda KJ. Accommodation and its anomalies. In: Charman WN, ed. *Vision and visual dysfunction: visual optics and instrumentation*. New York: MacMillan, 1991:231-79.
61. Ciuffreda KJ. Accommodation, the pupil, and presbyopia. In: Benjamin WJ, ed. *Borish's clinical refraction*. Philadelphia: WB Saunders Co., 1998:77-120.
62. Ciuffreda KJ, Ordonez X. Vision therapy to reduce abnormal nearwork-induced transient myopia. *Optom Vis Sci* 1998;75:311-5.
63. Mays LE, Gamlin PDR. Neuronal circuits for accommodation and vergence in the primate. In: Franzen O, Richter H, Stark L, eds. *Accommodation and vergence mechanisms in the visual system*. Basel, Switzerland: Birkhauser Verlag, 2000:1-9.
64. Bogart TF Jr. *Laplace transforms and control systems theory and technology*. New York: John Wiley and Sons, 1982:73-4.
65. Liu JS, Lee M, Jang J, et al. Objective assessment of accommodation orthoptics: dynamic insufficiency. *Am J Optom Physiol Optics* 1979;56:285-94.
66. Gottlieb GL, Corcos DM, Jarie S, et al. Practice improves even the simplest movements. *Exp Brain Res* 1988;73:436-40.
67. Takeda T, Endo H, Hashimoto K. Research on dynamic accommodation using TDO III (three dimensional optometer III) and MEG (magnetoencephalography). In: Franzen O, Richter H, Stark L, eds. *Accommodative and vergence mechanisms in the visual system*. Basel, Switzerland: Birkhauser Verlag, 2000:51-66.
68. Mah MM, Pope RS, Wong JH. *Testing of accommodation facility in school age children*. University of California (Berkeley): School of Optometry, O.D. thesis, 1981.
69. Liu J, Lee M, Jang J. *An objective assessment of accommodation orthoptics*. University of California (Berkeley): School of Optometry, O.D. thesis, 1978.
70. Bobier WR, Sivak JG. Orthoptic treatment of subjects showing slow accommodative response. *Am J Optom Physiol Optics* 1983;60:678-87.
71. Rosenfield M, Gilmartin B. Temporal aspects of accommodative adaptation. *Optom Vis Sci* 1989;66:229-34.
72. Lovasik JV, Wiggins R. Cortical indices of impaired ocular accommodation and associated convergence mechanisms. *Am J Optom Physiol Optics* 1984;61:150-9.
73. Benjamin WJ, ed. *Borish's clinical refraction*. Philadelphia, WB Saunders Co., 1998.
74. Cooper J, Feldman J, Selenow A, et al. Reduction of asthenopia after accommodative facility training. *Am J Optom Physiol Opt* 1987;64:430-6.
75. Sterner B, Abrahamsson M, Anders S. Accommodative facility training with a long-term followup in a sample of school-aged children showing accommodative dysfunction. *Doc Ophthalmol* 1999;99:93-101.

76. Grisham JD. Visual therapy results for convergence insufficiency: a literature review. *Am J Optom Physiol Optics* 1988;65:448-54.
77. Grisham JD. The dynamics of fusional vergence eye movements in binocular dysfunction. *Am J Optom Physiol Optics* 1980;57:645-55.
78. Grisham JD, Bowman MC, Owyang LA, et al. Vergence orthoptics: validity and persistence of the training effect. *Optom Vis Sci* 1991;68:441-51.
79. Cooper J. Clinical implications of vergence adaptation. *Optom Vis Sci* 1992;69:300-7.
80. McCormack GL. Vergence adaptation maintains heterophoria in normal binocular vision. *Am J Optom Physiol Opt* 1985;62:555-61.
81. North R, Henson DB. Adaptation to prism-induced heterophoria in subjects with abnormal binocular vision or asthenopia. *Am J Optom Physiol Opt* 1981;58:746-52.
82. North R, Henson DB. The effect of orthoptic treatment upon the ability of patients to adapt to prism-induced heterophoria. *Am J Optom Physiol Opt* 1982;59:983-6.
83. North R, Henson DB. The effect of orthoptic treatment upon the vergence adaptation mechanism. *Optom Vis Sci* 1992;69:294-9.
84. Cooper J, Selenow A, Ciuffreda KJ, et al. Reduction of asthenopia in patients with convergence insufficiency after fusional vergence training. *Am J Optom Physiol Opt* 1983;60:982-9.
85. Glass GV. Primary, secondary, and meta-analysis of research. *Educational Researcher* 1976;5:3-8.
86. Carlson NR. *Physiology of behavior*. Boston: Allyn and Bacon Inc., 1997:576-608.
87. Wollstein JJ, Abernethy B. Timing structure in squash strokes: further evidence for the operational timing hypothesis. *J Hum Mov Studies* 1988;15:70-1.
88. Smith KU, Schmidt J, Putz V. Binocular coordination: feedback synchronization of eye movements for space perception. *Am J Optom Arch Am Acad Optom* 1970;47:679-89.
89. Singer W. Synchronization of cortical activity and its putative role in information processing and learning. *Ann Rev Physiol* 1993;55:349-74.
90. Singer W. Time as coding in neocortical processing. In: Buzsaki G, Llinas R, Singer W, et al., eds. *Temporal coding in the brain*. Berlin: Springer Verlag, 1994:51-79.
91. Hebb DO. *The organization of behavior*. New York: John Wiley and Sons Inc., 1949.
92. Rauschecker JP. Mechanisms of visual plasticity: Hebb synapses, NMDA receptors, and beyond. *Physiol Rev* 1991;71:587-615.
93. Ellermann JM, Siegal JD, Strupp JP, et al. Activation of visuomotor systems during visually-guided movements: a functional MRI study. *J Magn Reson* 1998;131:272-85.
94. Richter H, Lee JT, Pardo JV. Neuroanatomical correlates of the near response: voluntary modulation of accommodation in the human visual system. In: Franzen O, Richter H, Stark L, eds. *Accommodative and vergence mechanisms in the visual system*. Basel, Switzerland: Birkhauser Verlag, 2000:67-78.
95. Pardo JV, Sponheim SR, Costello P, et al. Functional neuroanatomy of the human near/far response of the visual system to blur cues: fixation to point targets at different viewing distances. In: Franzen O, Richter H, Stark L, eds. *Accommodative and vergence mechanisms in the visual system*. Basel, Switzerland: Birkhauser Verlag, 2000:79-90.
96. Daw NW. *Visual development*. New York, Plenum Press 1995:155-92.
97. Windhorst U. Specific networks of the cerebral cortex: functional organization and plasticity. In: Greger R, Windhorst U, eds. *Comprehensive human physiology*, Vol. 1. Berlin: Springer-Verlag, 1996:1105-36.
98. Cooper J. Review of computerized orthoptics with specific regard to convergence insufficiency. *Am J Optom Physiol Opt* 1988;65:455-63.
99. Cooper J. Diagnosis and treatment of accommodative and vergence anomalies using computerized vision therapy. *Practical Optometry* 1998;9(1):6-11.

Corresponding author:

Kenneth J. Ciuffreda, O.D., Ph.D.
State University of New York
State College of Optometry
33 West 42nd Street
New York, New York 10036

kciuffreda@sunyopt.edu