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OPTOMETRIC CLINICAL PRACTICE GUIDELINE

Care of the Patient with

Accommodative and Vergence Dysfunction

American Optometric Association

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American Optometric Association

CARE OF THE PATIENT WITH ACCOMMODATIVE AND VERGENCE DYSFUNCTION

Reference Guide for Clinicians

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Approved by the AOA Board of Trustees March 20, 1998

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Printed in U.S.A.

NOTE: Clinicians should not rely on this Clinical Guideline alone for patient care and management. Refer to the listed references and other sources for a more detailed analysis and discussion of research and patient care information. The information in the Guideline is current as of the date of publication. It will be reviewed periodically and revised as needed.

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INTRODUCTION

Optometrists, through their clinical education, training, experience, and broad geographic distribution, provide primary eye and vision care for a significant portion of the American public. Optometrists are often the first health care practitioners to diagnose patients with accommodative or vergence dysfunction.

This Optometric Clinical Practice Guideline on Care of the Patient with Accommodative and Vergence Dysfunction describes appropriate examination and treatment procedures to reduce the risk of visual disability from these binocular vision anomalies through timely diagnosis, treatment, and, when necessary, referral for consultation with or treatment by another health care provider. This Guideline will assist optometrists in achieving the following goals:

- Identify patients at risk for developing accommodative or vergence dysfunction
- Accurately diagnose accommodative and vergence anomalies
- Improve the quality of care rendered to patients with accommodative or vergence dysfunction
- Minimize the adverse effects of accommodative or vergence dysfunction and enhance the quality of life of patients having these disorders
- Inform and educate other health care practitioners, including primary care physicians, teachers, parents, and patients about the visual complications of accommodative or vergence dysfunction and the availability of treatment.

The term "vision therapy" denotes an approach to management and rehabilitation of the accommodative and vergence systems. The descriptions of this approach found in the literature have identified vision therapy by various terminology, such as "vision training" or "orthoptics," depending upon the preference of the author.

I. STATEMENT OF THE PROBLEM

In previous generations, when survival depended on the ability to hunt, fish, and farm, the visual system had to respond to constantly changing, distant stimuli. Good distance visual acuity and stereoscopic vision were of paramount importance. Today, the emphasis has shifted from distance to two-dimensional near vision tasks such as reading, desk work, and computer viewing. In some persons, the visual system is incapable of performing these types of activities efficiently either because these tasks lack the stereoscopic cues required for accurate vergence responses or because the tasks require accommodative and vergence functioning that is accurate and sustained without fatigue. When persons who lack appropriate vergence or accommodative abilities try to accomplish near vision tasks, they may develop ocular discomfort or become fatigued, further reducing visual performance.

Accommodative and vergence dysfunctions are diverse visual anomalies. Any of these dysfunctions can interfere with a child's school performance, prevent an athlete from performing at his or her highest level of ability, or impair one's ability to function efficiently at work. Those persons who perform considerable amounts of close work or reading, or who use computers extensively, are more prone to develop signs and symptoms related to accommodative or vergence dysfunction. Symptoms commonly associated with accommodative and vergence anomalies include blurred vision, headache, ocular discomfort, ocular or systemic fatigue, diplopia, motion sickness, and loss of concentration during a task performance. The prevalence of accommodative and vergence disorders, combined with their impact on everyday activities, makes this a significant area of concern.

An accommodative or vergence dysfunction can have a negative effect on a child's school performance, especially after third grade when the child must read smaller print and reading demands increase. Due to discomfort, the child may not be able to complete reading or homework assignments and may be easily distracted or inattentive. Such children may not report symptoms of asthenopia because they do not realize that they should be able to read comfortably. The clinician should suspect a binocular or accommodative problem in any child whose school performance drops around third grade or who is described as inattentive.[1]

Many children who have reading problems or who are learning disabled or dyslexic have accommodative and vergence problems.[2-4] Even if one of these ocular conditions is not the primary factor in poor academic performance, it can contribute to a child's difficulty with school work; therefore, any child who is having academic problems should have a comprehensive optometric examination. If indicated by signs or symptoms, optometric vision therapy to improve accommodative and binocular skills may enable the child to perform near tasks more comfortably and benefit more effectively from educational remediation.

Good binocular skills contribute to better athletic performance. Sports such as basketball, baseball, and tennis require accurate depth perception, which in turn depends upon good binocularity. Studies show that tennis players have significantly lower amounts of and more stable heterophoria than nonathletes[5] and that varsity college athletes have better depth perception than nonathletes.[6]

The increased use of computers in the workplace, and in schools, has focused attention on the impact of binocular vision dysfunction on both performance and comfort. A high percentage of symptomatic computer workers have binocular vision problems[7] and ocular discomfort increases with the extent of computer use.[8-10] Similar findings are reported for other populations who perform sustained near work, such as students, accountants, and lawyers. Asthenopia associated with sustained near work can usually be eliminated with proper lens correction or vision therapy to improve accommodative-convergence function.

A. Description and Classification of Accommodative and Vergence Dysfunction

Although clinicians attempt to classify their vision problems, many patients do not fit perfectly into specific diagnostic categories. Most symptomatic patients have defects in more than one area of binocular vision. For example, the patient with vergence dysfunction may have a secondary accommodative problem, while one with an accommodative problem may have a secondary vergence problem, because the accommodative and vergence systems are controlled by an interactive negative feedback loop,[11] as depicted in Appendix Figure 1. Blur and unresolved disparity vergence errors are used to activate the system to eliminate residual blur and disparity vergence errors. The ICD-9-CM classification of accommodative and vergence dysfunction is shown in Appendix Figure 6.

1. Accommodative Dysfunction

This Guideline uses the Duke-Elder classification of accommodative dysfunction.[12]

a. Accommodative Insufficiency

Accommodative insufficiency occurs when the amplitude of accommodation (AA) is lower than expected for the patient's age and is not due to sclerosis of the crystalline lens.[12,13] Patients with accommodative insufficiency usually demonstrate poor accommodative sustaining ability.

b. Ill-Sustained Accommodation

Ill-sustained accommodation is a condition in which the AA is normal, but fatigue occurs with repeated accommodative stimulation.[12,13]

c. Accommodative Infacility

Accommodative infacility or accommodative inertia occurs when the accommodative system is slow in making a change, or when there is a considerable lag between the stimulus to accommodation and the accommodative response.[13] The patient often reports blurred distance vision immediately following sustained near work. Some have considered this infacility to be a precursor to myopia.[14]

d. Paralysis of Accommodation

Paralysis of accommodation is a rare condition in which the accommodative system fails to respond to any stimulus. It can be caused by the use of cycloplegic drugs, or by trauma, ocular or systemic disease, toxicity, or poisoning.[13] The condition, which can be unilateral or bilateral, may be associated with a fixed, dilated pupil.

e. Spasm of Accommodation

The result of overstimulation of the parasympathetic nervous system, spasm of accommodation may be associated with fatigue. It is sometimes part of a triad (overaccommodation, overconvergence, and miotic pupils) known as spasm of the near reflex (SNR).[15] This condition may also result from other causes, such as the use of either systemic or topical cholinergic drugs, trauma, brain tumor, or myasthenia gravis.

2. Vergence Dysfunction

The classification of vergence dysfunction is based on a system originally developed by Duane for application to strabismus.[16] The system has been modified for the classification of heterophoria and intermittent strabismus (Table 1).

a. Convergence Insufficiency

Classic convergence insufficiency (CI) consists of a receded near point of convergence (NPC), exophoria at near, reduced positive fusional convergence (PFC), and deficiencies in negative relative accommodation (NRA).[16] However, not all patients with CI have all of these clinical findings. CI can be described as a deficiency of PFC relative to the demand and/or a deficiency of total convergence, as measured by the NPC.[17]

b. Divergence Excess

Divergence excess (DE) can be described clinically as exophoria or exotropia at far greater than the near deviation by at least 10 prism diopters (PD).[18]

c. Basic Exophoria

The patient with basic exophoria has a deviation of similar magnitude at both distance and near.[19,20]

Table 1
Modified Duane Classification System*

Convergence insufficiency
- X < X'
- Low AC/A ratio
- Receded near point of convergence, reduced fusional convergence

Divergence excess
- X > X'
- High AC/A ratio
- High tonic exo
- Large exophoria/tropia at distance

Basic exo
- X = X'
- Normal AC/A ratio

Convergence excess
- E < E'
- High AC/A ratio

Divergence insufficiency
- E > E'
- Low AC/A ratio
- High tonic eso

Basic eso
- E = E'
- Normal AC/A ratio

Vergence insufficiency
- Normal AC/A ratio
- Restricted fusional vergence amplitudes
- Steep fixation disparity curve

Vertical phorias
- Comitant deviations
- Noncomitant deviations
- Old decompensated 4th nerve palsies
- Newly acquired 4th nerve palsies

Legend: X = exophoria at distance; E = esophoria at distance; X' = exophoria at near; E' = esophoria at near

* Modified from Duane A. A new classification of the motor anomalies of the eye, based on physiologic principles. Part 2. Pathology. Ann Ophthalmol Otolaryngol 1897; 6:247-60.

d. Convergence Excess

The patient with convergence excess (CE) has a near deviation at least 3 PD more esophoric than the distance deviation.[21] The etiology of the higher eso deviation at near most commonly is indicated by a high accommodative convergence/accommodation (AC/A) ratio.

e. Divergence Insufficiency

In a patient with divergence insufficiency (DI) tonic esophoria is high when measured at distance but less at near.[22] Symptomatic patients usually have low fusional divergence amplitudes at distance and low AC/A ratios.

f. Basic Esophoria

The patient with basic esophoria has high tonic esophoria at distance, a similar degree of esophoria at near, and a normal AC/A ratio.[16]

g. Fusional Vergence Dysfunction

Patients with fusional vergence dysfunction (vergence insufficiency) often have normal phorias and AC/A ratios but reduced fusional vergence amplitudes.[23] Their zone of clear single binocular vision (CSBV) is small.

h. Vertical Phorias

Vertical phorias may be either comitant and idiopathic or noncomitant, due to muscle paresis or other mechanical cause.[24] One of the most common causes of newly acquired vertical diplopia or asthenopia with vertical deviation is longstanding, decompensated, fourth nerve palsy, which results in superior oblique paresis. These patients demonstrate a hyperphoria in primary gaze that is initially greatest during depression and adduction of the affected eye. Over time, secondary overaction and contracture of the inferior oblique muscle may overshadow the initial fourth nerve palsy. Thus, the deviation may be largest during elevation and adduction of the affected eye.

B. Epidemiology of Accommodative and Vergence Dysfunction

1. Accommodative Dysfunction

a. Prevalence

Accommodative dysfunction has been reported to occur in 60 to 80 percent of patients with binocular vision problems;[25,26] however, few studies have been conducted to determine the prevalence of accommodative dysfunction in the general population. An investigation of the prevalence of symptomatic accommodative dysfunction in nonpresbyopic patients examined in an optometry clinic found that 9.2 percent of these patients had accommodative insufficiency, 5.1 percent had accommodative infacility, and 2.5 percent had accommodative spasm.[25]

b. Risk Factors

Most nonpresbyopic accommodative disorders originate from the need to sustain the increased accommodation required for viewing two-dimensional targets at near. Sustaining accommodation can fatigue the accommodative system. One theory suggests that the cause of accommodative fatigue is accommodative adaptation or slow accommodation.[27]

Accommodation can be affected by a number of drugs and by diseases (e.g., diabetes mellitus, myasthenia gravis).

2. Vergence Dysfunction

a. Prevalence

There are conflicting estimates of the exact prevalence of vergence anomalies because clinicians and researchers use different definitions of these conditions and different methods of analysis.

- **Convergence insufficiency.** CI is the most common vergence anomaly. The reported prevalence of CI is 1 to 25 percent of clinic patients.[16,17,28,29] The median prevalence of CI in the population is 7 percent, and it is similar for adults and children.[17]

A report that 5 percent of a school-age population have reduced NPC and 6 percent fail a cover test used the following criteria for failure: at near, more than 5 PD esophoria, 9 PD exophoria, or 1 PD vertical phoria; at far, more than 5 PD esophoria, 5 PD exophoria, or 2 PD vertical phoria.[14] The findings were similar in the young adult population. The ratio of females to males with CI is 3:2.[30]

- **Divergence excess.** The prevalence of DE is approximately 0.03 percent of the population, and it is more common in women and blacks.[18] DE strabismus has a strong hereditary predisposition.[18]

- **Convergence excess.** One study of an urban population reported that 5.9 percent of patients seeking optometric care had CE,[25] and another found a 7.1 percent prevalence in a pediatric population.[31]

- **Divergence insufficiency.** DI is probably the least common vergence dysfunction. The only report on its prevalence came from a study of urban pediatric patients seeking optometric care, which showed a prevalence of 0.10 percent.[31]

- **Basic exophoria and esophoria.** One study of 179 patients with exo deviation found that 62 percent had CI and 27 percent had basic exophoria.[32] Based on the prevalence of CI (approximately 7 percent), the interpolated prevalence of basic exophoria is 2.8 percent of the population.

- **Fusional vergence dysfunction.** One report ranks the prevalence of this condition just below those of CI and CE.[33]

- **Vertical phorias.** Early estimates of the prevalence of vertical deviations ranged from 7 percent[34] to 52 percent.[35] A recent estimate of the prevalence of vertical phorias is about 20 percent of the population.[36] The reported prevalence differs on the basis of criteria used to diagnose a

clinically significant vertical phoria. Only about 9 percent of vertical phorias are clinically significant.[24]

b. Risk Factors

Many patients with vergence anomalies are asymptomatic. Symptoms usually occur when the visual environment is altered, specifically, when near work is increased in situations such as school, work, and computer use. Patients with low pain thresholds tend to be more symptomatic, while patients who suppress an eye tend to be less symptomatic.

Defects in vergence may also be the result of trauma and certain systemic diseases. For example, CI and fourth nerve palsy are common after closed head trauma, especially in the presence of a concussion.[37-39] CI is the most common vergence dysfunction found with Graves disease.[40] Myasthenia gravis may present as a CI or any other fusional vergence disorder. Fusional vergence disorders are often associated with Parkinson disease and Alzheimer disease.[41,42]

C. Clinical Background of Accommodative and Vergence Dysfunction

1. Accommodative Dysfunction

a. Natural History

Accommodation, which provides the retina with a clear, sharp image, develops by 4 months of age.[13] The primary stimulus for accommodation is blur, with lesser roles played by apparent perceived distance, chromatic aberration, and spherical aberration. During accommodation, the ciliary muscle contracts, relaxing the tension on the zonular fibers.[43] This relaxation increases the convexity of the anterior surface of the lens. If the system does not respond accurately, a negative feedback loop repeats the process and reduces the error. This process continues until the error is reduced to as near zero as possible. With age, the lens fibers and lens capsule lose their elasticity and the size and shape of the lens increase.[44] This sclerosis of the lens causes presbyopia and a reduction in AA.

The accommodative response is the actual amount of accommodation by the lens for a given stimulus. It is usually the

least accommodation required to obtain a clear image. It is limited by the depth of focus (which is dependent on pupil size) and the inability to detect small amounts of blur.[45] At distance, the system usually overaccommodates, while at near the system usually underaccommodates, creating a lag in accommodation. The resting state of accommodation is not at infinity but at an intermediate distance that varies from individual to individual within a range of 0.75 to 1.50 diopters (D). The resting state is similar to the accommodation measured in night myopia or empty field myopia.[46,47]

Sustained accommodative effort has been reported to cause accommodative fatigue and asthenopia. In some individuals, the punctum proximum recedes after repeated push-up stimulation of accommodation.[48] One study showed that the amplitude of accommodation increased in 29 percent of the subjects after sustained push-ups, while in 31 percent there was a decrease in amplitude and an associated blur.[49] Repeated near-far stimulation does not affect the AA in most subjects.[50] The few subjects who demonstrated fatigue also reported asthenopia that was not age dependent.[50] From these studies it can be concluded that the accommodative system is resistant to fatigue in most individuals. However, in patients who demonstrate fatigue, asthenopia usually ensues.

b. Common Signs, Symptoms, and Complications

- **Accommodative insufficiency.** Patients with accommodative insufficiency often complain of blurred vision, difficulty reading, irritability, poor concentration, and/or headaches. Attempting to accommodate, some patients may stimulate excessive convergence by the AC/A crosslink and be incorrectly classified as having CE.

  In accommodative insufficiency, the AA is less than expected for the patient's age. Patients with accommodative insufficiency usually fail the +/- 2.00 D flipper test and have positive relative accommodation (PRA) under -1.50 D. These patients may be able to make appropriate accommodative responses, but they expend so much effort that asthenopia ensues. They may complain about blur after

sustained reading or at the end of the day. The fast accommodative mechanism becomes fatigued and the slow adaptive accommodative mechanism takes over, resulting in blur.

- **Ill-sustained accommodation.** The most common sign or symptom of ill-sustained accommodation is blurred vision after prolonged near work. It occurs because the accommodative system fails to sustain long-term accommodative effort. In ill-sustained accommodation which is similar to accommodative insufficiency, except that the AA is normal, the patient generally fails the +/-2.00 D flipper test and has a decreased PRA. In addition, such patients often have asthenopia.

- **Accommodative infacility.** Patients with accommodative infacility report that after prolonged near focusing, their distance vision is blurred and/or that, after prolonged distance viewing, reading material is blurred. These patients invariably fail the +/- 2.00 D accommodative facility test monocularly and binocularly. They have normal AAs, but they may have abnormal relative accommodative findings, PRA or NRA.

- **Paralysis of accommodation.** Paralysis of accommodation results when a nonpresbyopic patient loses the ability to accommodate either monocularly or binocularly. The chief complaint is blur due to failure to accommodate, and there may be associated micropsia. Paralysis can be the result of trauma, toxicity, Adie's pupil, neuropathy, and/or drugs, such as cycloplegic agents. The etiology of the paralysis should be identified if possible.

- **Spasm of accommodation.** Spasm of accommodation occurs when the accommodative system inappropriately overaccommodates for a stimulus. It is most often secondary to constant parasympathetic innervation as part of the SNR but its origin is usually not associated with serious organic disease. Spasms as great as 25 D have been reported, and distance vision is usually impaired. One study reported that for most patients with this disorder, the

etiology is probably psychogenic. Some clinicians use the term "accommodative excess" interchangeably with "accommodative spasm."[15]

c. Early Detection and Prevention

Although early detection and treatment are ideal, there is no evidence that early treatment affects the long-term use or disuse of the accommodative system. However, early detection is important when the AC/A ratio is high and accommodation results in an esotropia at near. Early examination of children is important to detect and eliminate both accommodative and vergence dysfunction because these anomalies may affect future school performance adversely. The child's first eye and vision examination should be scheduled just after 6 months of age. When no abnormalities are detected at this age, the next examinations should be scheduled at age 3 and before the first grade (age 6).*

2. Vergence Dysfunction

a. Natural History

Rapid, accurate eye movements are necessary to fixate and stabilize a retinal image. It is imperative to maintain a fixed retinal image to stabilize the visual world during body movement. The eyes and the neck work together to localize and stabilize an image by optokinetic and vestibular reflexes. These reflexes provide a platform from which voluntary eye movements are executed.[51] Several components are required to maintain fixation and to shift the line of sight to a new point of interest: an accurate, efficient, smooth pursuit system to hold a moving target on the fovea; a saccadic system to bring the fovea to the object of regard; and a vergence system to place the object of regard on both foveas while looking from near to far.

To maintain exact alignment, the eyes must incorporate disjunctive movements into the scheme of normal conjugate movements. These movements must be extremely accurate to

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* Refer to the Optometric Clinical Practice Guideline for Pediatric Eye and Vision Examination.

avoid diplopia and facilitate a unified perception. Two different types of stimuli initiate these disjunctive movements: retinal disparity for vergence movements and defocused (blurred) objects for accommodative responses.[52]

Two different types of fusional vergence have been described: (1) a fast, reflexive vergence system driven by retinal disparity and (2) a slow, adaptive system which receives its input from the fast system.[11] The slow system is also known as vergence adaptation. Theoretically, heterophoria is a vergence error that is eliminated by fusional or disparity vergence. Slow vergence reduces the stress or load placed on the fast vergence system by heterophoria during binocular viewing. Total fusional vergence is equal to the sum of the fast and slow systems.

The initial response to a new vergence demand is initiated by the fast, disparity-driven vergence system. Upon attainment of fusion, the output from the fast fusional system decreases; the output from the slow vergence system increases proportionally. Once adaptation has occurred, total fusional vergence is supplied by the slow vergence system and the residual fast vergence. The residual error from the initiation of a new disparity vergence response is the fixation disparity (FD). Thus, the slow vergence system is responsible for sustaining CSBV during prolonged reading or other near tasks. It is failure of the slow vergence system that results in asthenopia.

- **Convergence insufficiency.** The etiology of CI is controversial. It probably results from a breakdown in the accommodative-convergence relationship.[17,53-55] It is likely that a genetic predisposition for CI exists because the parents of children with CI often have the condition. Symptoms tend to occur when persons use their eyes in a two-dimensional reading environment for extended periods of time. The symptoms tend to increase during the teenage years and continue to increase during the early twenties. Symptoms commonly occur with computer use or in a visually demanding work environment.[8-10,17,56,57]

Most patients with CI have normal stereopsis but may exhibit suppression when viewing first-degree fusion targets. It is not uncommon for the CI patient to manifest an exotropia during near point testing without reporting diplopia. When an eye deviates, the patient may report blurred vision or suppress the eye. Suppression provides a mechanism of eliminating diplopia or asthenopia.

Patients with CI generally have poor fusional convergence ability, compared with the magnitude of their exophoria. Typically, they do not meet Sheard's criterion (i.e., a fusional vergence reserve at least twice the magnitude of the heterophoria).[17,58,59] Many patients with CI also have poor accommodative facility.[17,60] In some instances, CI results from the accommodative system's failure to accommodate accurately at near. The inability to obtain an appropriate accommodative response results in an exodeviation at near because of a low AC/A ratio. Patients experiencing this phenomenon have been called "pseudo-CI patients."

- **Divergence excess.** The most widely accepted theory of the etiology of DE involves innervation and is based upon the use of the eyes. According to this theory, divergence is active and purposeful, and it occurs in the absence of stereoscopic cues.[18] The deviation may present as a heterophoria or a strabismus. It has been suggested that the deviation extends the peripheral field of view when the patient manifests a strabismus.[18] The deviation is often first noticed in children under 18 months of age.[61] Progression may occur throughout life, but at about 6 years of age, the deviation becomes more noticeable because of an increase in both the frequency and extent of the deviation.

- **Basic exophoria.** The clinical findings of the patient with basic exophoria are similar to those of the DE patient. Basic exophoria is thought to occur in a patient with DE who develops secondary CI. The extent of the deviation tends to increase with age at both distance and near.

- **Convergence excess**. CE is due to a high AC/A ratio.[62] The angle of deviation is usually stable until school age, when it tends to increase.

- **Divergence insufficiency**. This condition is due to high tonic esophoria and tends not to change with time.

- **Basic esophoria**. Little is known about the natural history of basic esophoria. The condition is presumed to be due to tonic vergence errors, such as DI which develops early in life (at about 6-9 months of age). Deficits related to an abnormal accommodative vergence system first occur at about 2 years of age. Basic esophoria is probably due to an abnormal gain in output from the neuromuscular system (i.e., high AC/A ratio). A genetic predisposition for basic esophoria seems to exist in a significant proportion of those who have it.

- **Fusional vergence dysfunction.** The etiology of fusional vergence dysfunction is uncertain. The patient often first notices it when asthenopia occurs.

- **Vertical Phorias**. Vertical deviations have three different origins; therefore, patients can present with three different histories. Congenital or early acquired comitant hyperdeviations are usually small in magnitude and nonprogressive over time. Congenital fourth nerve palsies, which decompensate over time, may be first noted after an insult, such as a high fever or trauma. Newly acquired fourth nerve palsies occur after vascular, infectious, traumatic, or neoplastic incidents.[63] Depending on the etiology of the vertical deviation, its course may change. Deviations that occur secondary to vascular or ischemic involvement tend to improve with time; those caused by trauma may remain stable; and those of neoplastic origin usually worsen.

b. Common Signs, Symptoms, and Complications

Most patients report symptoms of vergence dysfunction during their second through fourth decades of life, when they have the greatest amount of near work. Eliciting symptoms from

patients can sometimes be difficult, especially when the patients are very young children. Many patients with chronic problems have learned to live with their condition and may not voluntarily reveal their symptoms. Children may have fewer near vision needs; more importantly, many are unable to describe their symptoms. Young children may not report symptoms because they consider diplopia and asthenopia normal. During the formative school years, the additional load on the visual system may result in avoidance of near tasks, such as reading. The relationship between asthenopia and school performance is governed, to some extent, by pain thresholds. The increase in symptoms reported by young adults is probably related to increased severity of chronic symptoms that have been present most of their academic lives.

Presbyopic patients may demonstrate vergence dysfunction due to the loss of accommodative convergence or due to prism induced through their bifocals. Those who are symptomatic generally have poor fusional convergence and poor slow (adaptive) vergence abilities. Patients with vergence anomalies may have the following symptoms: asthenopia, headaches, pulling sensation, blurred vision, intermittent diplopia, inability to sustain concentration, pulling of the eyes, and burning or tearing of the eyes. Symptoms tend to increase by the end of the day and are related to the use of the eyes.

- **Convergence insufficiency.** The most common symptoms associated with CI are blurred vision, diplopia, a gritty sensation of the eyes, discomfort associated with near work, frontal headaches, pulling sensation, heavy eyelids, sleepiness, loss of concentration, nausea, dull ocular discomfort, and general fatigue. Some patients with CI report decreased depth perception. A significant number of patients with CI complain of motion sickness or car sickness.[17] A high percentage of patients with CI have emotional problems and anxiety reactions, and it has been suggested that all symptomatic CI results from psychosis and emotional problems.[64,65] However, there is no evidence to substantiate this theory, although it is possible that CI may cause nervousness, tension, and anxiety.[17]

Most patients with CI have a low PFC amplitude (10 PD or less).[17] One study reported that 79 percent of all patients with CI have an exophoria at near, while 18 percent are orthophoric and 3 percent are esophoric.[56] Another study found that 63 percent of patients with CI have an exophoria.[66]

Symptomatic CI patients have poor prism adaptation and slow vergence ability. Recovery values, which represent voluntary convergence, also may be below normal. The NPC, which is receded in most CI patients, represents the most consistent finding.[55,67] Other clinical findings include low AC/A ratio, low NRA, and failure with plus lenses or the +/-2.00 D accommodative facility test.

- **Divergence excess**. The patient with DE may be asymptomatic. When the deviation occurs with either deep suppression or anomalous correspondence, asthenopia is not usually present. However, if either suppression or anomalous correspondence has failed to develop, diplopia or asthenopia generally ensues. The closing of an eye in bright sunlight may be pathognomonic of DE. Some DE patients complain of distance blur because they overaccommodate to keep their eyes aligned. Common clinical findings associated with DE include normal NPC, adequate PFC at near, equal vision in each eye, and normal stereopsis at near.[68]

  When the eyes of a patient with DE deviate, any of a variety of sequelae—e.g., suppression, diplopia with normal retinal correspondence (NRC), anomalous retinal correspondence (ARC) with single vision—may occur.[69] If ARC occurs when the eye deviates, the DE patient has an extension of the binocular field known as panoramic viewing.[69] Retinal projection shifts to match the objective angle (harmonious ARC). There may be little or no foveal suppression during deviation because each fovea has its own unique visual direction.

- **Basic exophoria.** The most common symptoms of basic exophoria are related to asthenopia. The clinical findings of basic exophoria are similar to those of DE because the basic exophoric patient is considered to be a DE patient who acquires CI. Thus, like the DE patient, the patient with basic exophoria may have no symptoms.

- **Convergence excess.** Symptoms of CE include blurred vision, diplopia, headaches, and difficulty concentrating on near tasks. Symptomatic patients with CE have low fusional divergence amplitudes and PRAs in relationship to their near point demands. Not all patients with CE present with symptoms. Some patients with CE suppress, some have strong vergence adaptation, and some have a high pain threshold, while others have no symptoms because they avoid near work.[70]

- **Divergence insufficiency.** Symptomatic patients with DI usually have reduced fusional divergence amplitudes at distance. They also have low AC/A ratios. Such patients often report diplopia or blur at distance.

- **Basic esophoria.** Patients with basic esophoria are symptomatic only when their fusional divergence amplitudes are not large enough to compensate for the esophoria. Moreover, symptoms may not occur in the patient who suppresses. Because the deviation is present at all distances, the symptoms are generally the same with either far viewing or near viewing.

- **Fusional vergence dysfunction.** Some patients with vergence anomalies do not have significant heterophorias present at any distance; instead, like patients who have CI, they present with asthenopia. If appropriately questioned, these patients generally report asthenopia during vergence testing. They usually have reduced fusional vergence amplitudes (fast vergence) in both convergence and divergence directions. In addition, these patients usually have accompanying accommodative problems. Typically, the fixation disparity curve (FDC) is very narrow, with a small flat zone indicating poor vergence adaptation.

- **Vertical phorias.** Diplopia is the typical presenting sign of the patient who has a significant vertical deviation. The patient may also have a head tilt and/or asthenopia as a result of trying to maintain single, binocular vision. The patient with a recent-onset vertical deviation has a normal break and recovery (approximately ±3 D of vertical fusional amplitude, as measured from the heterophoria), while those with longstanding vertical deviations usually have abnormally large opposing vertical fusion ranges. The high opposing vertical fusional vergence amplitudes are associated with a robust, slow vergence system.

c. Early Detection and Prevention

Early detection of clinically significant nonstrabismic vergence anomalies is important. Without treatment, some of these deviations may decompensate and become strabismic, resulting in the loss of stereopsis and the development of suppression. This risk is greatest during the critical period of visual development (0-2 years of age)[71] because ocular alignment is a prerequisite for the development of normal binocularity.[72]

Treatment of nonstrabismic vergence anomalies is not age restricted. Treatment can be performed in a motivated 60-year-old patient as well as a 10-year-old patient. However, vergence dysfunction in a child should be detected and treated as early as possible to provide the best opportunity for academic success.

Although vergence dysfunction does not cause learning disabilities, it may be a contributing factor.[2,73,74] Because elimination of certain vergence anomalies can improve reading scores,[75] it is critical to evaluate both accommodative and vergence functioning in the school-age population.

II. CARE PROCESS

A. Diagnosis of Accommodative and Vergence Dysfunction

The evaluation of a patient with accommodative and vergence dysfunction may include, but is not limited to, the following areas. The examination components described are not intended to be all inclusive. Professional judgment and the individual patient's symptoms and findings have a significant impact on the nature, extent, and course of the services provided. Some components of care may be delegated (See Appendix Figure 2).

1. Patient History

The patient history is the initial component of the examination and an important part of making an appropriate diagnosis. A good history should lead to a tentative diagnosis, which the examination will either confirm or disprove. A suggested history to investigate accommodative and vergence problems is shown in Table 2.

2. Ocular Examination

The simplest way to evaluate the relationship of accommodation and vergence to asthenopia is to place stress on the visual system during the examination in an attempt to produce asthenopia. The clinician should be as concerned with the patient's reaction to testing as with the absolute values obtained. Accommodative and vergence measurements may be more revealing at the end of the day when fatigue is more likely to occur. Futhermore, even with normal fusional vergence amplitudes, some patients complain of asthenopia when tested with lenses and prisms. Because this finding is diagnostic of an accommodative-vergence anomaly, one goal of testing is to create asthenopia similar to that which occurs during normal day-to-day activities.

Normally, all components of vergence and accommodation are synergistic; accommodation, convergence, and pupillary miosis occur in synchrony. Procedures that isolate these individual

Table 2
Suggested Questions for Patient History

1. Do your eyes bother you?
   If yes, how often and under what circumstances?

2. How do your eyes bother you?
   Do you experience eyestrain, fatigue, headaches, sleepiness, etc., associated with near tasks?

3. Do you ever get headaches?
   If yes, explore further (e.g., frequency, location, type, and associated activities).

4. How long can you read comfortably?
   Have the patient specify an actual time.

5. When you read, does the print ever blur, double, or move around?

6. Do you experience car or motion sickness?

functions by holding one function constant actually measure the plasticity or flexibility of the system. Patients who demonstrate poor plasticity or flexibility often are those who experience symptoms. Measurements are influenced by the size of the target, illumination, speed of measurement, and the effort exerted by the patient.[76] When taking any clinical measurement, the optometrist should encourage the patient to exert maximum effort. The clinician should record any asthenopic complaints induced by the measurements. Patients who become uncomfortable or fatigued by testing are usually symptomatic in everyday life.

a. Visual Acuity

The best corrected visual acuity should be measured for each eye individually and for both eyes together, at distance and near. Variability between distance and near visual acuity may

indicate an accommodative anomaly. Some patients with accommodative dysfunction report that their vision fluctuates, especially after prolonged near tasks. When visual acuity is better monocularly than binocularly, the clinician should suspect vergence dysfunction.

b. Refraction

The patient's refractive status should be evaluated. Patients with uncorrected hyperopia—especially latent hyperopia—often have accommodative dysfunction because accommodation compensates for the hyperopia. Cycloplegic refraction is advised for the patient who could have an excessive accommodative response that could affect the measurement of refractive error.

c. Ocular Motility and Alignment

Cover testing should be performed with a small target to control accommodation.[77] The eye should be occluded for a minimum of 2 seconds to elicit any existing deviation. During unilateral testing, the clinician should pay careful attention to the movement of the fellow eye and, upon alternate cover testing, to the movement of the uncovered eye. Both the extent of the deviation and the quality of fusion should be noted. Any significant deviation seen upon alternate cover testing should be neutralized with prisms. When the patient has poor fixation, a muscle light (penlight or transilluminator) can be substituted for an accommodative target.

In the evaluation of ocular motor function, versions should be performed to rule out paresis, paralysis, overaction, or underaction of a muscle. Careful attention should be given to lateral fields of gaze especially during elevation and adduction. Defects associated with overaction of the inferior oblique muscles, superior oblique palsy, Brown's syndrome, and V syndromes are apparent in these fields of gaze. When the clinician has difficulty evaluating motor response in a particular field of gaze, the alternate cover test with prism neutralization should be performed in that field.

The heterophoria may also be measured using Risley prisms in a phoropter, or in free space at both distance and near, using

an accommodative target. When a torsional component is suspected, the patient can be asked whether the two test targets are parallel. Other methods that can be used to measure heterophoria include the Maddox rod and stereoscopic devices.

d. Near Point of Convergence

The NPC test is important for assessment of binocular function. It is best performed using a small accommodative target.[78] The break and recovery, as well as any discomfort evoked by testing, should be recorded. The patient who grimaces, moves away from the target, or is bothered by the test is usually symptomatic. The test should be repeated several times if necessary. If the patient cannot provide good verbal responses or demonstrates suppression (denoted by not reporting diplopia upon deviation), the clinician should use a penlight to observe the corneal reflexes. Placing a red lens over one eye and repeating the NPC measurement 4 or 5 times will often cause a fragile binocular system to break down and the NPC to recede.[79]

e. Near Fusional Vergence Amplitudes

Positive and negative fusional vergence amplitudes are measures of the amount of prism that can be placed in front of the eyes before the patient reports a sustained blur. Once blur is reported, the patient is no longer using only fusional vergence to maintain single binocular vision, but is also employing accommodative vergence. The measurements may be made with a Risley prism or prism bar. It is advantageous to use a prism bar to observe the eyes of young children or verbally uncooperative patients.

The order in which fusional vergence tests are administered may affect subsequent measurement of vergence functions.[80,81] If base-out (BO) fusional vergence amplitudes are measured before base-in (BI) amplitudes, the BI fusional amplitudes will be reduced and vice versa. In addition, the position of the heterophoria may be influenced by the test that precedes its measurement. Measurement of convergence amplitudes before heterophorias may cause the heterophoria to appear more esophoric or less exophoric. Thus, the heterophoria should be measured first, followed by divergence amplitudes, and then convergence amplitudes.

Divergence and convergence fusional amplitudes should be measured using an accommodative target.[82] The patient should be instructed to keep the target single and clear and to report whether the test bothers his or her eyes. This is important because many patients experience fatigue associated with the exertion of maximum effort to keep the target single and clear. In this regard it is extremely important to note the patient's subjective symptoms. These tests should be repeated if the patient's responses are equivocal.

f. Relative Accommodation Measurements

Positive relative accommodation and negative relative accommodation are indirect assessments of the fusional vergence system. In the measurement of relative accommodation, plus or minus lenses are added binocularly over the lenses that fully correct any refractive error until the patient reports either blur or diplopia. The end point is the amount of accommodation (clinically, the stimulus to accommodation) that can be increased or decreased with a fixed amount of convergence. When minus lenses are placed in front of the eyes, accommodation occurs, clearing the image. The eyes converge by the AC/A crosslink. In order to maintain CSBV, the eyes must neutralize this accommodative convergence by fusional divergence. At the limit of PRA, fusional divergence is exhausted, and accommodation must be inhibited to reduce convergence, resulting in blur. An analogous response occurs when plus lenses are substituted for minus lenses in these assessments.

g. Accommodative Amplitude and Facility

AA may be measured monocularly, using either the push-up or the minus lens method. Generally, the optometrist uses a 20/20 to 20/30 target and notes the first sustained blur.[83]

Accommodative facility testing can be performed using a +/-2.00 D lens flipper or a phoropter. The patient should be able to clear these lenses monocularly within 11 cycles per minute without evidence of fatigue.[84]

Patients with accommodative infacility frequently report intermittent blurred vision and asthenopia after near work. Symptomatic patients demonstrate reduced accommodative facility on the +/-2.00 D flipper test.[13,85,86]

h. Stereopsis

Stereopsis can be assessed and quantified using measures such as the Randot or Titmus Stereo tests. Contour or line stereograms can be used to measure stereoacuity. Appreciation of a random dot stereogram requires both fusion and bifoveal fixation,[87] thus, confirming that the patient was not strabismic at the time of testing.

i. Ocular Health Assessment and Systemic Health Screening

Gross inspection of the eyelids and adnexa is important to rule out abnormalities such as exophthalmos associated with Graves disease, facial and orbital asymmetry, and ptosis. Biomicroscopy may also be performed to rule out media abnormalities that may cause decreased visual acuity. A dilated fundus examination may be needed to rule out retinal and vitreal abnormalities. Certain systemic diseases (e.g., multiple sclerosis, diabetes mellitus, Graves disease, and myasthenia gravis) can cause accommodative-vergence anomalies.[88] Many medications (e.g., tranquilizers, antidepressants, antispasmodics, and motion sickness medications)[89] can also cause accommodative dysfunction.

3. Supplemental Tests

When the comprehensive examination does not identify a cause for asthenopia, the following tests may be helpful:

a. Accommodative Convergence/Accommodation Ratio

The AC/A ratio is a measure of the convergence induced by accommodation per unit of accommodation. In a perfect physiological system, accommodative convergence supplies all the necessary convergence for near viewing. The normal AC/A ratio is 4:1.

Both high and low AC/A ratios have been implicated in binocular vision problems. The two most popular methods of

calculating the AC/A ratio are the calculated distance-near deviation method and the gradient method.

Distance-near method. Many clinicians advocate using the calculated distance-near method of determining the AC/A ratio because it takes into account the actual position of the eyes during distance and near fixation. Clinically, however, the calculation method suffers from the noncalculated effects of the effort of accommodation, depth of field, proximal accommodation and convergence, and blur interpretation. Moreover, the calculation varies with fixation distance and interpupillary distance (IPD). The AC/A ratio may be calculated by the following formula:

$$\text{AC/A ratio} = \frac{\text{convergence demand of near target - Hd + Hn}}{\text{stimulus to accommodation of near target}}$$

Where: Hd = Distance heterophoria
Hn = Near heterophoria

With this formula, an esophoria is a plus value, while an exophoria is a minus value. Convergence demand is calculated by dividing the IPD by 4 (e.g., 60/4 = 15).[90]

Alternatively,

$$\text{AC/A ratio} = \text{IPD (cm)} + \text{N (Hn-Hd)}$$

Where N is the near fixation distance in meters.

Gradient method. The gradient method of calculating the AC/A ratio uses the change in vergence angle at a given distance in association with a change in the stimulus to accommodation produced by ophthalmic lenses. Either plus (+1.00 D or +2.00 D) or minus (-1.00 D or -2.00 D) lenses are placed in front of each eye. The heterophoria is remeasured while the patient views the same target through the lens and the ratio is calculated thus:

$$\text{AC/A ratio} = \frac{\text{heterophoria 1 - heterophoria 2}}{\text{lens power (D)}}$$

The AC/A is thought to be innate and stable until the beginning of presbyopia;[91] however, the stimulus and response to accom-

modation differ. Theoretically, the response AC/A ratio may be estimated by multiplying the stimulus AC/A ratio by 1.08.[92]

b. Fixation Disparity/Associated Phoria

Fixation disparity is the small misalignment of the eyes that occurs while single binocular vision is maintained for the point of fixation. FD is a direct measurement of this misalignment, and the associated phoria is the amount of prism needed to neutralize the FD. Measurements of FD may be obtained to determine the forced FDC, the associated phoria, and the FDC. The chief advantage of the FD method over methods that interrupt fusion is that it permits evaluation of the vergence system under binocular conditions.

c. Distance Fusional Vergence Amplitudes

Distance fusional vergence amplitudes are determined in the same manner as near vergence amplitudes, except that the targets are placed at 20 feet. The testing should be performed when the patient experiences asthenopia or when a significant heterophoria is present with distance fixation.

d. Vergence Facility

Prism flippers may be used to test vergence facility. Normative values have been established for 16 PD BO and 8 PD BI prisms.[93] Mean values are 8 cycles per minute for children ages 5-8 years and 13 cycles per minute for children ages 7-14 years.[93] Prism flippers may be used when standard testing does not elicit a clearly defined reason for asthenopia.

e. Accommodative Lag

The lag of accommodation is the difference between the stimulus of accommodation and the response. It may be measured using binocular cross-cylinders or near point retinoscopy, such as the monocular estimated method (MEM).

MEM retinoscopy is performed by having the patient read grade-level words at his or her habitual near working distance while the clinician performs retinoscopy. The clinician rapidly interposes a lens in front of the eye being evaluated and estimates the motion of the light reflex. Lenses of various

power are briefly interposed in this manner until neutrality is found. Each lens is removed before an accommodative response occurs. For most patients, the lag is between approximately +0.25 D and +0.75 D. A lag of greater than +1.00 D is often found in individuals with accommodative insufficiency or infacility, suggesting the using of plus lenses at near. A lead of -0.25 D or more usually indicates accommodative excess.

The fused cross-cylinder test is a subjective means of determining the lag of accommodation. It is not as accurate as the MEM test and is often difficult to perform in children under the age of 8 years.

4. Assessment and Diagnosis

The clinician can use the history and clinical findings to make the diagnosis, assess the need for treatment, and determine the plan of treatment. Clinical assessment has used the following protocols:

a. Graphical Analysis

Graphical analysis is not a method of analyzing binocular function; rather, it involves plotting test results to form a visual representation of accommodation and vergence, and their interaction.[94,95] The relationship between accommodation and convergence can be demonstrated by plotting five findings: distance and near heterophorias, AC/A ratio, PFC, negative fusional vergence (NFV), and AA. The outer boundaries of these measurements define the zone of CSBV.

b. Zones of Comfort

Several attempts have been made to develop clinical rules for the prediction of asthenopia.[59,96,97] One approach, suggested by Sheard,[59] takes the heterophoria into account and specifies that the fusional vergence reserve should be twice the demand (i.e., heterophoria) for sustained comfort. For example, for a patient with 10 PD of exophoria, the base-out to blur measurement should be at least 20 PD. A base-out to blur measuring only 8 PD would not meet Sheard's criterion.

c. Comparison to Expected Values

Accommodation and vergence findings can be statistically analyzed and compared with normative values. The assumption is that any finding that deviates from the norm by 2 standard deviations may indicate an anomaly. Although this type of statistical analysis does not provide correlative information with regard to asthenopia, it can alert the clinician to a potential problem. Table 3 shows the most commonly used norms for accommodation and vergence testing.

d. Fixation Disparity and Vergence Adaptation

Small errors in vergence often occur during normal binocular fixation, in which the eyes do not align exactly on the target. As long as the vergence error does not exceed Panum's fusional area and the patient does not report diplopia, this error is called FD.[98] Controversy exists regarding whether FD provides a purposeful error to stimulate the vergence system, or whether it is an error-related indicator of a malfunction of the vergence system.[11,99] Proponents of the latter theory have used FD measurements to determine the need for and amount of prism to prescribe.

Although heterophoria and FD measures are often correlated, they often differ as well. For example, some patients require only a small amount of prism to neutralize a large horizontal FD, while others may require a large amount of prism for neutralization of a small FD. Proponents of FD methods have suggested that clinicians should prescribe the amount of prism that neutralizes or eliminates the FD.[100] FD neutralization methods are probably more useful in measuring and prescribing for vertical imbalances than for horizontal deviations. The prism prescribed should be the least required to neutralize the horizontal and vertical components of the FDC for 10 minutes.[101]

e. Comparison of Methods of Analysis

Evaluation of these methods of measurement of heterophoria, vergences, and FDCs in symptomatic and asymptomatic patients has been accomplished with the aid of discriminant analysis.[102,103] The application of Sheard's criterion was found

Table 3
Expected Values*

Measurements	Mean	S.D.	Range
Distance			
Phoria	1 X	2 X	0-2 X
Base-in blur	-	-	-
Base-in break	7	3	5-9
Base-in recovery	4	2	3-5
Base-out blur	9	4	7-11
Base-out break	19	8	15-23
Base-out recovery	10	4	8-12
Near			
Phoria	3 X'	5 X'	0-6 X
Base-in blur	13	4	11-15
Base-in break	21	4	19-23
Base-in recovery	13	5	10-16
Base-out blur	17	5	14-20
Base-out break	21	6	18-24
Base-out recovery	11	7	7-15
PRA	-2.25	.50	-1.75-+2.25
NRA	+2.00	1.1	+1.75-+2.25
Gradient AC/A	4/1	2	3-5
AA	16-(0.25 x age)	±2.00	±1.0

Legend: AA = Amplitude of accommodation;
AC/A = Accommodative convergence/ accommodation ratio;
NRA = Negative relative accommodation;
PRA = Positive relative accommodation;
X = exophoria at distance; X' = exophoria at near

* Modified from Morgan MW. Analysis of Clinical Data. Am J Optom 1944; 21:477-91.

to be any means of identifying symptomatic exophoric patients. When the use of Sheard's criterion does not differentiate asthenopic from nonasthenopic exophoric patients, the angular measurement of FD has been found to be effective. The absolute magnitude of esophoria was found to be most predictive of asthenopia for esophoric patients; the second best measure of esophoria is the NFV recovery value.

B. Management of Accommodative and Vergence Dysfunction

Management of the patient with an accommodative or vergence dysfunction is based on such interpretation and analysis of the examination results. Appendix Figures 3 and 4 provide an overview of patient management strategies for accommodative and vergence dysfunction, respectively.

1. Basis for Treatment

The general goals for treating accommodative and/or vergence dysfunction are:

- To assist the patient to function efficiently in school performance, at work, and/or in athletic activities
- To relieve ocular, physical, and psychological symptoms associated with these disorders.

a. Vision Therapy

- **Accommodative Therapy**. The purpose of accommodative therapy is to increase the amplitude, speed, accuracy, and ease of accommodative response. At the end of therapy the patient should be able to make rapid accommodative responses without evidence of fatigue. Studies of the effectiveness of vision therapy for types of accommodative dysfunction are summarized in Table 4.

  Several studies have reported that accommodation can be modified with therapy.[13,49,104,105] Repeated accommodative testing itself improves accommodative responses.[49] Studies have also shown that voluntary accommodation can be taught[106] and that accommodation skills developed by biofeedback can transfer from one task to another.[107]

Accommodative therapy has demonstrated effectiveness in eliminating decreased accommodative amplitude and facility.[105,108] In one study, 87 percent of the patients with accommodative anomalies eliminated their asthenopia and normalized their accommodative findings after approximately 26 therapy sessions.[26]

Therapy to improve AA can result in a concurrent improvement of PFC, NFV, and stereopsis.[109] Vision therapy is the method of choice in eliminating asthenopic symptoms associated with accommodative anomalies.[110] For those patients who cannot participate in vision therapy, plus lenses may successfully decrease symptoms.

In a double-blind prospective study to determine the effects of monocular AA therapy on asthenopia[110] the patients in the experimental group had dramatically improved AA, reduced accommodative time constants, and significantly reduced symptoms. None of these changes was evident in the control group. When the control group underwent therapy identical to that received by the experimental group, a similar reduction in symptoms and normalization of accommodative function was achieved.[110]

These studies demonstrate that vision therapy can alter accommodation, with a resultant change in the amplitude and facility and a decrease in symptoms. Therapy can also result in positive changes in the magnitude, velocity, and gain of the accommodative response.[111,112] Accommodative therapy not only eliminates symptoms but is associated with objective changes in the velocity of the accommodative response and a concurrent decrease in recorded time constants.[112] Therapy improves the time characteristics, including both latency and velocity, of the accommodative response.[113]

Table 4
Effectiveness of Vision Therapy for Accommodative Dysfunction Research Results*

Accommodative Dysfunction	Study Authors	Summary/Interpretation
Accommodative insufficiency Accommodative infacility	Cooper,[13] Berens & Stark[49] Carr & Allen,[104] Sisson[105]	Accommodation can be modified with training.
	Berens & Stark[49]	Repeated accommodative testing has been shown to improve accommodative responses.
	Marg[106]	Voluntary accommodation can be taught.
	Cornsweet & Crane[106]	Accommodation developed by biofeedback can transfer from one task to another.
	Sisson[105] Morris[108]	Accommodative therapy has been shown to be effective in eliminating decreased accommodative amplitude and facility.
	Hoffman et al[26]	In 87% of patients with accommodative anomalies, asthenopia was eliminated and accommodative findings were normalized with approximately 26 therapy sessions.
	Daum[109]	Therapy to improve accommodative amplitudes can result in a concurrent improvement of positive and negative fusional amplitudes and stereopsis.
	Cooper et al[110]	Vision therapy is the method of choice in eliminating asthenopic symptoms associated with accommodative anomalies. For patients who cannot participate in vision therapy, plus lenses are often successful in decreasing symptoms. Monocular accommodative amplitude therapy for asthenopia patients effected dramatic improvement in accommodative amplitudes, a reduction in accommodative time constants, and a significant reduction in symptoms.
	Randle & Murphy,[111] Liu et al[112]	Vision therapy may result in positive changes in the magnitude, velocity, and gain of accommodative response.
	Liu et al[112]	Accommodative therapy not only eliminates symptoms but shows objective changes in velocity of the accommodative response and a concurrent decrease in recorded time constants.
	Bobier & Sivak[113]	Vision therapy improves the time characteristics of the accommodative response, including the latency and velocity.

*Table 4 extends horizontally on page 35.

- **Vergence Therapy**. Fusional vergence therapy improves slow vergence (vergence adaptation); thus it reduces the apparent vergence error. This reduction in the residual vergence error apparently causes a change in the AC/A ratio.[114] Other important functions of slow vergence include maintenance of fusion following blinking, reduction of the fusional demand with the onset of presbyopia, and maintenance of binocularity with the alteration of orbital contents that occurs with age and diseases such as hyperthyroidism. If the vergence and accommodative systems are functioning properly when a steady-state level of accommodation or vergence is reached, the slow accommodation and vergence systems maintain accommodation and vergence without effort. The fast and slow vergence and accommodative systems also use proximal, tonic, and voluntary vergence and accommodation to reduce their loads. Defects in any one of these systems alone may not result in asthenopia or strabismus, owing to overlap with components in other systems.

  Numerous studies have evaluated the effectiveness of vergence therapy in eliminating subjective and objective findings associated with binocular anomalies.[87,115-119] These studies demonstrate that vergence therapy improves vergence ability, and that the effects persist over time (Table 5). It should be noted that all of the studies demonstrating the efficacy of vision therapy used in-office therapy regimens.

  Vision therapy for vergence dysfunctions has a high success rate. Pooled data for patients with CI indicate that 72 percent of patients have been cured, 19 percent improved significantly, and only 9 percent failed.[17,54] Vision therapy has a lasting effect when a complete cure is achieved.[115] Moreover, age is not a deterrent in the successful treatment of binocular anomalies.[120]

  A controlled, prospective, double blind, A-B reversal study to evaluate experimental treatment versus placebo treatment for a group of patients diagnosed with CI used automated therapy with random dot stereograms in an operant conditioning paradigm to improve vergence amplitudes. The experimental group had dramatic improvement in vergence amplitudes and concurrent decrease in symptoms. When the control group crossed over to become the experimental group, the findings were similar.[121]

The pooled success rates of different treatment regimens for intermittent exotropia have been reported as: 59 percent for vision therapy, 46 percent for surgery, and 28 percent for passive therapy (minus lenses, occlusion, and/or prisms).[122] These data suggest that vision therapy is more effective than surgery in patients with intermittent exotropia.[122]

A study evaluating the use of vision therapy in 31 intermittent exotropia patients reported that 64.5 percent were classified as cured; 9.7 percent, improved; and 9 percent, fair.[123] A followup study found that after 5 years, 52 percent of these patients remained cured, while 32 percent were in the improved group.[124] Similar findings have been reported by other studies.[20,125-129] One study reported that the highest success rate occurred when office therapy was supplemented with home vision therapy.[130]

The latest of recent studies demonstrating the effectiveness of vision therapy for CE,[131] treated 68 patients diagnosed with CE. Total elimination of symptoms occurred in 80 percent of the patients. Among the improvements achieved with vision therapy were an increase in mean divergence amplitude from 8 PD to 16 PD, an increase in recovery value from 2 PD to 10 PD, and increased accommodative facility from 1.5 to 8 cycles per minute. Prior to therapy, some subjects had spectacles prescribed to eliminate the esophoria; others did not. When the results for the patients receiving vision therapy alone were compared with the results for those patients initially receiving reading spectacles and then undergoing vision therapy, there was no difference in the post-vision therapy results, suggesting that vision therapy alone is highly effective in eliminating abnormal vergence findings associated with CE.[131]

Table 5
Effectiveness of Vision Therapy for Vergence Dysfunction Research Results*

Vergence Dysfunction	Study Authors	Summary/Interpretation
Convergence insufficiency	Cooper & Duckman[17], Grisham[54]	72% of patients reported cured, 19% reported improved, 9% reported failed
	Grisham, et al[115]	Vision therapy has a lasting effect when a complete cure is achieved.
	Wick[120]	Age is not a deterrent to successful treatment.
	Cooper, et al[121]	Results demonstrated a dramatic improvement in vergence amplitudes with a concurrent decrease in symptoms.
Intermittent exotropia	Coffey, Wick, Cotter, et al[122]	Pooled success rates of different treatment regimens (59% for vision therapy, 46% for surgery, and 28% for passive therapy [e.g., minus lenses, occlusion, and/or prisms]) suggest that vision therapy is more effective than surgery.
	Sanfilippo & Clahane[123] Sanfilippo & Clahane[124]	64.5% reported cured, 9.7% reported improved, 9% reported fair Subsequently after 5 years, 52% remained cured, 32% remained improved.
	Mann[125], Durran[126], Cooper & Leyman,[127] Altzier,[20] Chryssanthau[128] Daum[129]	Similar rates of success for vision therapy have been reported by these studies.
	Goldrich[130]	Highest success rate occurred when office therapy was supplemented with home vision therapy.
Convergence excess	Gallaway & Scheiman[131]	Total elimination of symptoms in 80% of patients with the following improvements: mean divergence amplitude from 8 PD to 16 PD, recovery value from 2 PD to 10 PD, and accommodative facility from 1.5 cpm to 8 cpm. Vision therapy alone is highly effective in eliminating abnormal vergence findings associated with CE.
Vertical deviations	Cooper[132] Robertson & Kuhn[133]	Vision therapy is effective with patients having small vertical deviations and for older decompensated vertical deviations. Vision therapy may be used to decrease prism adaptation and the need for future increases in prism correction.
	Cooper[132]	Vision therapy is a better option for patients with noncomitant deviations, patients who wish to wear contact lenses, patients in whom the size of the vertical deviation is different at distance vs. near, and patients who adapt to prism.

*Table 5 extends horizontally on page 39.

Table 5 Continued . . .

Vertical prism is usually the treatment of choice for vertical deviation. However, vision therapy has been shown to be effective in a small sample of patients with vertical deviations and in patients with longstanding decompensated vertical deviations. Vision therapy may be used to decrease prism adaptation as well as to reduce the need for future increases in prism correction.[132,133] Vision therapy may be a better option for a range of patients who have noncomitant deviations, who wish to wear contact lenses, whose vertical deviation differs in magnitude at distance and near, and who adapt to prism.[132]

Patients with closed head injuries often develop accommodative dysfunction and CI secondary to trauma. Studies comparing therapeutic options for these patients[37,38,134-136] have concluded that patients with closed head injuries who have associated accommodative and/or vergence anomalies have a higher success rate with vision therapy than with surgery and/or lens therapy. However, head-injured patients may need prisms or surgery to supplement vision therapy treatment.

b. Lens and Prism Therapy

- **Horizontal Prisms**. Clinicians often prescribe prism to eliminate symptoms of asthenopia and to reduce the fusional vergence demand in patients with vergence dysfunction. Two common methods of determining the amount of prism to prescribe are (1) to satisfy Sheard's criteria and (2) to eliminate the FD.[137] One study evaluated the effect of prescribing prism using the associated heterophoria to eliminate the FD in three groups of patients: symptomatic exophoric patients, symptomatic esophoric patients, and a control group. All patients were given two pairs of spectacles to be worn for 2 weeks, one pair with a prismatic correction that eliminated the associated phoria and the second pair with no prism. While 73 percent of the symptomatic exophoric patients and 90 percent of the symptomatic esophoric patients preferred the prismatic glasses, 86 percent of the asymptomatic patients rejected the prismatic glasses.[138]

Prism may be the only viable treatment for CI in patients who are unable to participate in a vision therapy program because of time, cognitive, or financial constraints. Patients with symptomatic vergence anomalies may be treated with prisms. Unfortunately, some patients' adaptation to prismatic correction limits its effectiveness. Slow vergence (prism or vergence adaptation) varies from patient to patient. It also varies with the amount of time spent wearing the prism, the power or strength of the prism, and the direction of prism placement (e.g., base-out, base-up). When prism adaptation occurs, prism therapy is contraindicated for two reasons: (1) the prism will not permanently neutralize the deviation, and (2) strong vergence adaptation will not be able to handle the stress placed on the vergence system by the heterophoria. Only when there is a significant deviation with minimal vergence adaptation can prism compensation be effective.

Adaptation to base-out and base-in prisms differs. As expected, most people adapt faster and more completely to base-out prism than to base-in prism.[139,140] Prolonged wearing of prisms not only alters the heterophoria position, but also results in a readjustment of horizontal fusional amplitudes.[140] Once adaptation has occurred, measurements of the fusional vergence amplitudes, with the prism in place, are almost identical to the measurements prior to wearing the prism. Most of this change occurs within the first 15 minutes of wearing the prism.

Vergence adaptation also occurs with noncomitant deviations.[141-143] The phenomenon of adaptation, a continuous process that can occur over the entire oculomotor field, explains why patients who wear incorrectly centered ophthalmic lenses or anisometropic prescriptions may not complain. Many patients adapt to a newly introduced prism and its abrupt removal may result in diplopia and/or asthenopia. Symptomatic patients who do not adapt to prisms usually report a reduction in asthenopia once they wear a prism prescription.

- **Vertical Prisms.** Vertical deviations may be divided into three different categories: small-angle comitant deviations; large-angle, newly acquired paretic deviations; and large-angle, decompensated, older deviations. Studies have shown that patients with these deviations differ in their adaptation responses to vertical prism.[139,144] Although the adaptation process varies from individual to individual, in general, the larger the prism, the less complete the adaptation process. The longer the prism is worn, the more complete the adaptation process and the longer the recovery when the prism is removed. Patients who do not show significant adaptation may benefit from prism correction.

  Clinically, adaptation can be determined by having the patient wear a vertical prism for as little as 1-2 hours. Adaptation can be predicted to occur whenever a heterophoria increases dramatically after repeated, prolonged cover testing.

  The effectiveness of prism is limited by torsional deviations, noncomitancies, and anisometropia. Surgery or vision therapy may be needed to supplement prismatic correction.

- **Plus Lenses.** The purpose of plus lenses is to decrease the demand on the accommodation system and/or to reduce the amount of the esodeviation by manipulating the crosslink AC/A ratio. Adaptation does seem to play a significant role in the prescription of plus lenses. The effectiveness is limited in patients who demonstrate accommodative dysfunction with asthenopia in the absence of a large heterophoria, and in those whose accommodative and fusional amplitudes are constricted but balanced.

- **Minus Lenses.** Minus lenses may be used to change the motor demand of the vergence system to reduce the amount of exodeviation.

- **Surgery.** The purpose of extraocular surgery is to decrease the size of the deviation; therefore, it is rarely indicated for nonstrabismic binocular vision disorders. One study

advocates surgical intervention for CI when vision therapy fails;[145] however, this study did not have a large enough sample to support the author's conclusion concerning the use of surgery as a primary mode of treatment for CI. Surgery may be considered in noncomitant vertical deviations which have a significant torsional component. Newly acquired large-angle vertical deviations that cannot be resolved within 6 months may require surgery.[24] As a general rule, vision therapy alone is ineffective in treating newly acquired large-angle vertical deviations. If the patient is satisfied with prismatic correction or vision therapy, surgical intervention is not necessary.

2. Available Treatment Options

Treatment of accommodative and vergence anomalies is designed to eliminate signs and symptoms such as headaches, asthenopia, poor academic performance, poor job performance, loss of concentration, and ocular and systemic fatigue. Because it also eliminates other symptoms such as diplopia, reduced stereopsis, and motion sickness, treatment generally improves the patient's quality of life.

Treatment options can be divided into the following broad categories: optical correction including added lens power and prism; vision therapy; pharmaceutical agents; and extraocular muscle surgery. Therapeutic results can vary due to differences in the application of the specific treatment regimen.

a. Optical Correction

- **Ophthalmic lenses.** Appropriate spectacle lens correction of any existing refractive error is the first consideration in treating persons with vergence or accommodative anomalies. Plus lenses are often effective in eliminating symptoms in the patient who has an accommodative insufficiency or imbalanced positive and negative relative accommodative values. In addition, plus lenses may positively affect abnormal esophorias according to the AC/A ratio.

  Plus additions at near may be used for patients diagnosed with an accommodative anomaly, or for those with an abnormally high AC/A ratio. The lens power may be

determined by many different methods: balancing the PRA and NRA values; cross-cylinder; near point retinoscopy; or calculation of the AC/A ratio to determine the minimum lens power that can significantly reduce the near deviation.

- **Prisms.** Prisms are often effective in eliminating vergence disorders symptoms that involve a significant motor deviation (tonic vergence anomaly).

  Horizontal Prisms — Sheard's criterion can be used to calculate the amount of prism required to alleviate symptoms using the following formula:

$$\text{prism power} = \frac{2 \times \text{heterophoria} - \text{opposing vergence}}{3}$$

  Other methods of prescribing prism include using Percival's criterion, in which the clinician prescribes prism to place Donder's line in the middle third of the graph in graphical analysis, and FD methods, in which the clinician prescribes the amount of prism that eliminates the FD (i.e., the associated phoria).

  Vertical Prisms — There are three types of vertical deviations: (1) longstanding, asymptomatic deviations that have very strong vergence adaptation; (2) longstanding deviations that decompensate and have moderate vergence adaptation; and (3) recent, small deviations with minimal vergence adaptation. Each of these vertical deviations requires a different prismatic correction. Patients with old deviations that decompensate usually present with minimal symptoms in relationship to the size of the deviation. The prismatic correction needed to eliminate or reduce symptoms is usually minimal compared with the magnitude of the deviation. On the other hand, the patient who has a newly acquired hyperdeviation with minimal vergence adaptation may require full prism correction, which is defined as the amount of prism needed to correct either the heterophoria or the recovery value. Patients who have strong vergence adaptation and are asymptomatic usually should not be treated with prism.

b. Vision Therapy

Three general phases of vision therapy will be discussed in this section: accommodation, vergence, and accommodative/vergence interaction. The first phase of therapy is to normalize accommodative and vergence amplitudes. Most clinicians use large targets in which convergence and divergence demand is slowly changed. The patient is encouraged to exert maximum effort to increase his or her vergence amplitudes. Accommodative facility exercises are performed concurrently.

The second phase of accommodative and vergence therapy is designed to increase the speed of response to accommodative and vergence stimuli. During this phase, it is beneficial to use targets that gradually become smaller and to use different stimuli to obtain generalization. After the amplitudes reach normal levels, the patient is encouraged to repeat the task enough times to make the response become automatic and effortless. Once monocular accommodative facility has improved, binocular accommodative facility procedures can be performed. Suppression controls may be needed with the binocular accommodative techniques. In general, the power of the binocular accommodative flippers is increased until the patient can successfully clear +/-2.50 D, according to a specified criterion.[13]

The third phase of vision therapy uses jump or step vergence stimuli. Instead of responding to incrementally increasing stimuli, the patient is required to make large-jump accommodative and vergence movements. Finally, accommodation and vergence are integrated through techniques that stimulate accommodation while holding vergence stable and vice versa. This final phase of vision therapy is designed to automate both accommodative and vergence reflexes.

Vision therapy increases the magnitude and the velocity of the fast fusion system. In addition, there is a concurrent increase in both the magnitude and velocity of the slow vergence system (vergence adaptation). In a study to evaluate the effect of vision therapy on vergence adaptation, individuals who

underwent 8 weeks of vision therapy that consisted of push-ups and fusional amplitude therapy had improved vergence adaptation and fusional amplitudes.[144] Subsequent studies have demonstrated that vision therapy alters the FDC, specifically, flattening the FDC and concurrently reducing the symptoms.[11]

The success of vision therapy lies in the improvement of both the accommodative and vergence adaptation systems because these systems are the most important for a person's long-term comfort.[146] Although the patient may have a normal fast vergence system, he or she may have an abnormal slow vergence system, with the resulting symptoms. Thus, therapy is first aimed at improving reflex-fast fusional vergence, then at expanding slow vergence responses. In the process, accommodative flexibility is also restored. The last stage of therapy enhances the flexibility between accommodation and vergence. The goal of vision therapy is to re-establish automated, effortless accommodative and vergence responses under any stimulus condition. Improvement of amplitudes alone is not sufficient.

There is a paucity of data demonstrating the efficacy of using home-based vision therapy alone. Home-based vision therapy may be less effective than in-office therapy because no therapist is available to correct inappropriate procedures or to motivate the patient. Thus, preferred clinical management consists of in-office vision therapy supplemented with home therapy.

c. Medical (Pharmaceutical) Treatment

Pharmacological agents are of minimal use in the treatment of accommodative and vergence anomalies, except in the rare case of myasthenia gravis and CE. With myasthenia gravis, trial use of Mestinon 60 mg (1-4 times) may be appropriate.*

* Every effort has been made to ensure that the drug dosage recommendations are accurate at the time of publication of this Guideline. However, because treatment recommendations change, due to continuing research and clinical experience, clinicians should verify drug dosage schedules with product information sheets.

CE patients may benefit occasionally from the judicious use of phospholine iodine 0.06% in 2.5% neosynephrine at bedtime.[147]

d. Surgery

Extraocular muscle surgery is rarely advocated to treat nonstrabismic vergence defects. As a general rule, it should be considered only when optical correction or vision therapy methods have failed and a significant heterophoria continues to produce symptoms. There is no surgery available for accommodative dysfunction.

3. Management Strategy for Accommodative Dysfunction

a. Accommodative Insufficiency

The most effective treatment for accommodative dysfunction is vision therapy to build AA and accommodative facility.[112] Therapy should focus on increasing accommodative amplitudes. Alternatively, plus lenses may be prescribed at near,[148] if the patient is not interested in or is unable to meet the time requirements for vision therapy.

b. Ill-Sustained Accommodation

Plus lenses and vision therapy are effective in treating ill-sustained accommodation.[148] Vision therapy is used to improve the speed of the accommodative response, and generally is the treatment of choice.

c. Accommodative Infacility

Plus lenses may be prescribed initially, but vision therapy is highly effective in correcting accommodative infacility.[110] The goal of therapy is to improve the speed and flexibility of accommodation.

d. Paralysis of Accommodation

The treatment of paralysis of accommodation is directed at determining its underlying cause and correcting it when necessary. Paralysis of accommodation may be treated with a progressive addition lens in front of the affected eye.[149] Vision therapy is not effective in treating this condition.

e. Spasm of Accommodation

The initial treatment of spasm of accommodation consists of plus lenses. Because, in most cases, lenses alone are not sufficient to eliminate an accommodative spasm, the clinician should also prescribe vision therapy to relax accommodation.[150] If vision therapy fails, short-term use of a cycloplegic agent may be prescribed. The ultimate goal is elimination of the spasm (and the need for cycloplegia and/or plus lenses). In addition to these treatments, the clinician should reinforce the importance of visual hygiene in the form of proper working distance, lighting, and appropriate rest periods.

4. Management Strategy for Vergence Dysfunction

a. Convergence Insufficiency

Patients with CI can be treated by various strategies, depending on the severity of symptoms. Numerous studies have shown that vision therapy is the treatment of choice for CI (Table 6).[26,29,56,57,64,66,120,151-159] The recommended treatment includes in-office therapy and supplemental home therapy. Home therapy alone, which is less effective, may be prescribed when in-office therapy is not possible. To ensure its success, home therapy should be closely monitored for patient compliance and to make adjustments when needed. For the patient who cannot participate in vision therapy, prescribed prisms may reduce the load on the vergence system; however, prisms do not always alleviate the patient's symptoms.

b. Divergence Excess

Among the variety of treatments for DE are occlusion, over-minus lenses, base-in prism, active vision therapy, and, if necessary, surgery. Therapy combining diplopia awareness with operant-conditioning technique to reinforce alignment in the absence of visual cues has been advocated for DE.[18] When active vision therapy is not successful or the deviation is too large, surgery may improve the outcome. For the noncommunicative patient, passive therapy that includes part-time occlusion, base-in prism, and over-minus lenses may be effective.

Table 6
Vision Therapy Success Rate for Convergence Insufficiency Patients in Large Studies[a]

Author	Number	% Cured	% Improved	% Failed
Mayou[154]	87	92	6	2
Lyle & Jackson[151]	300	83	10	7
Mann[64]	142	68	30	3
Cushman & Burri[66]	80	66	30	4
Duthie[155]	123	88	6	6
Mayou[153]	420[b]	72	7	5
Mayou[153]	100	93	5	2
Mellick[152]	88	77	10	12
Hirsch[57]	48	77	12	10
Passmore & MacLean[56]	100	82	18	0
Norn[29]	65	10	60	30
Hoffman et al[26]	17	94	6	0
Wick[120]	134	93	4	3
Daziel[157]	100	84	9	7
Pantano[158]	207	79	6	5
Daum[156]	110	41	56	3
Cohen & Soden[159]	28	96	4	0
Total	2149	78[c]	15	5

[a] Adapted from Cooper J, Duckman R. Convergence insufficiency: incidence, diagnosis, and treatment. J Am Optom Assoc 1978; 49:673-80.

[b] The author reported that data were incomplete for 16% of the study population.

[c] Mean weighted cure rate; 2% did not complete orthoptics.

c. Basic Exophoria

Most patients with a basic exophoria may be treated like CI patients for near problems and like DE patients for distance problems. Vision therapy is usually the initial treatment of choice, and the general goal of treatment is to improve convergence amplitudes. Therapy usually starts with near targets; distance targets are added later. Prism treatment is also an option.

d. Convergence Excess

Most patients with CE are emmetropic. When hyperopia is present, it should be corrected. The best treatment options for CE are plus lenses at near, vision therapy, or both.[147] A plus lens addition at near may be part of the initial treatment for these patients. The prescription can be determined by calculating the AC/A ratio and prescribing the amount of plus lens power that significantly reduces or eliminates the near esophoria. Vision therapy can be successful in meeting its primary goal to alleviate the symptoms associated with CE. This therapy should incorporate divergence training and minus lenses. A secondary goal of therapy for CE is to increase plus lens acceptance to make the spectacle correction more comfortable and uncover any latent hyperopia, if present.

e. Divergence Insufficiency

Many patients with DI present with minimal symptoms because they suppress at distance and have normal binocular vision at near. Symptomatic patients usually complain of diplopia and asthenopia during night-time driving, when there are fewer fusion cues. Because patients with DI usually have low hyperopia or emmetropia and low AC/A ratios, plus lenses have minimal effect. Prism should be prescribed for distance only, because wearing the prism at near can cause asthenopia.

Vision therapy is usually successful in patients with DI. If vision therapy does not provide the needed therapeutic effect, a prismatic correction at distance should be considered. Vision therapy may be used in conjunction with prism correction to decrease the possibility of adaptation to the prism. When the patient is young, it is important to differentiate functional DI from acquired DI. Because a sudden-onset DI in a child is sometimes the first sign of a brain tumor or other serious

neurological condition, the child should have an appropriate neurological evaluation.

f. Basic Esophoria

Patients with basic esophoria often have uncorrected hyperopia, and correcting the hyperopia may eliminate the deviation. If not, prismatic correction may be prescribed. Generally, the patient should be given the least amount of prism needed to eliminate all of the symptoms. When the patient has residual asthenopia or wishes to avoid prismatic correction, a program of vision therapy may be helpful. The goal is to eliminate the prism through vergence adaptation, which can be achieved by increasing the fusional divergence amplitude and decreasing the prismatic correction by approximately 2 PD every month or so. After the patient overcomes both the accommodative and vergence deficits for suppression, he or she should be re-evaluated. If suppression is present, it should be eliminated.

g. Fusional Vergence Dysfunction

Patients with fusional vergence dysfunction have no significant heterophoria at distance or near; therefore, lenses and prisms are generally ineffective. The only treatment for this common binocular problem is vision therapy focusing on both convergence and divergence amplitudes. The patient with fusional vergence dysfunction usually has an abnormal accommodative system, which should also be treated.

h. Vertical Phorias

Treatment of vertical phorias generally consists of correcting the vertical deviation with prism. The prism prescribed should be the least required to eliminate the symptoms. If the symptoms remain, the patient may have a vergence dysfunction, for which horizontal vergence therapy should be prescribed.[132] The vertical prism may be decreased slowly over time, concurrent with the extension of horizontal amplitudes. Vision therapy to increase the ability to control vertical vergence is also an option, but it is more difficult to train the patient to control vertical vergence than to control horizontal vergence.[160]

5. Patient Education

Patients should be advised that many accommodative and

vergence anomalies are neuromuscular problems and not refractive problems. Thus, the most effective treatment relies on not only spectacles, but active vision therapy to eliminate neuromuscular dysfunction. The patient should also be told that treatment improves accommodative and vergence reflexes. Proper treatment usually results in a permanent cure, due to changes in the slow vergence system.

6. Prognosis and Followup

When the patient is cooperative, the prognosis for the elimination of accommodative and vergence dysfunction is excellent (See Appendix Figure 5). The most effective treatment appears to be in-office vision therapy, supplemented by home therapy. Prisms and lenses may be less effective in eliminating some vergence dysfunction. The difficulty with lenses is that they do not affect either the fast vergence or slow vergence systems. Futhermore, the effectiveness of prism and lenses may be reduced by adaptation.[140] These options will only be effective if there is significant heterophoria or an inability to sustain accommodation.

Patients with accommodative and convergence problems who have been treated successfully should be seen twice a year for the first year, then annually thereafter. Patients for whom spectacles are prescribed to eliminate symptoms of asthenopia should be followed up as necessary. Many practitioners schedule a followup after the patient has worn his/her prescribed spectacles for one month and again 3-6 months later.

CONCLUSION

Accommodative and vergence dysfunction is a collection of neuromuscular disorders that may occur at any time after the normal development of binocular vision (6 months of age). These anomalies may cause a host of symptoms, including, but not limited to, blurred vision, headaches, asthenopia, diplopia, loss of concentration, motion sickness, and fatigue. Such symptoms may interfere with school or work performance and thus decrease a patient's quality of life. Most accommodative and vergence dysfunction responds to the appropriate use of lenses, prisms, or vision therapy. It is medically necessary for the optometrist to diagnose the condition accurately, discuss the diagnosis and the risks and potential benefits of existing treatment options with the patient, and initiate treatment when appropriate. Treatment, including lenses, prisms and vision therapy, is not age restricted. Vision therapy can be given at any age. In some cases, the best treatment includes a combination of lenses, prisms, and/or vision therapy. Proper treatment usually results in rapid, cost-effective, and permanent improvement in visual skills.

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IV. APPENDIX

Figure 1
Control Theory of Accommodative and Vergence Interactions*

* Adapted from Schor CM, Kotulak JC. Dynamic interactions between accommodation and convergence are velocity sensitive. Vision Res 1986; 26:940.

Figure 2
Potential Components of the Diagnostic Evaluation for Accommodative and Vergence Dysfunction

A. Patient history

B. Ocular examination

C. Visual acuity

D. Refraction

E. Ocular motility and alignment

F. Near point of convergence

G. Near fusional vergence amplitudes

H. Relative accommodation measurements

I. Accommodative amplitude and facility

J. Stereopsis

K. Ocular health assessment and systemic health screening

L. Supplemental tests
   1. AC/A ratio
   2. Fixation disparity
   3. Distance fusional vergence ranges
   4. Vergence facility
   5. Accommodative lag

Figure 3
Optometric Management of the Patient with Accommodative Dysfunction: A Brief Flowchart

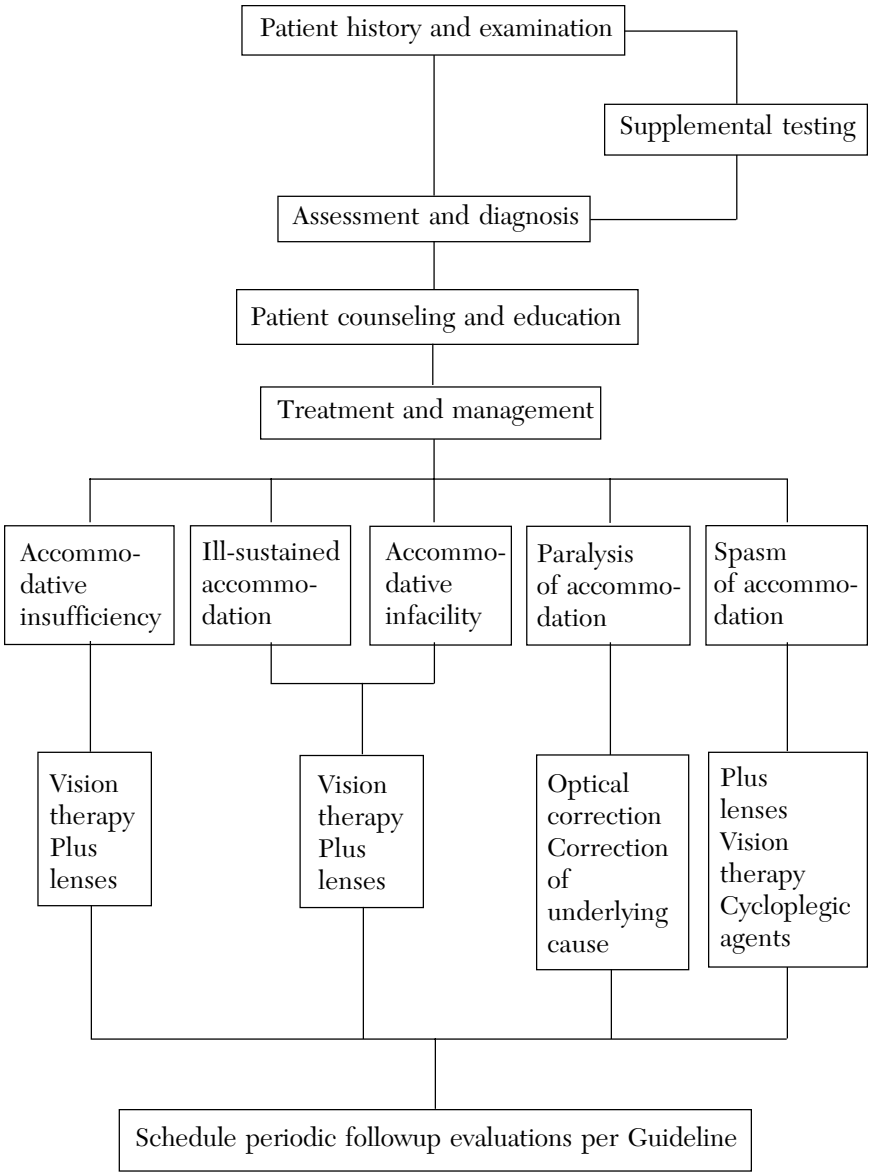

Figure 4
Optometric Management of the Patient with Vergence Dysfunction: A Brief Flowchart

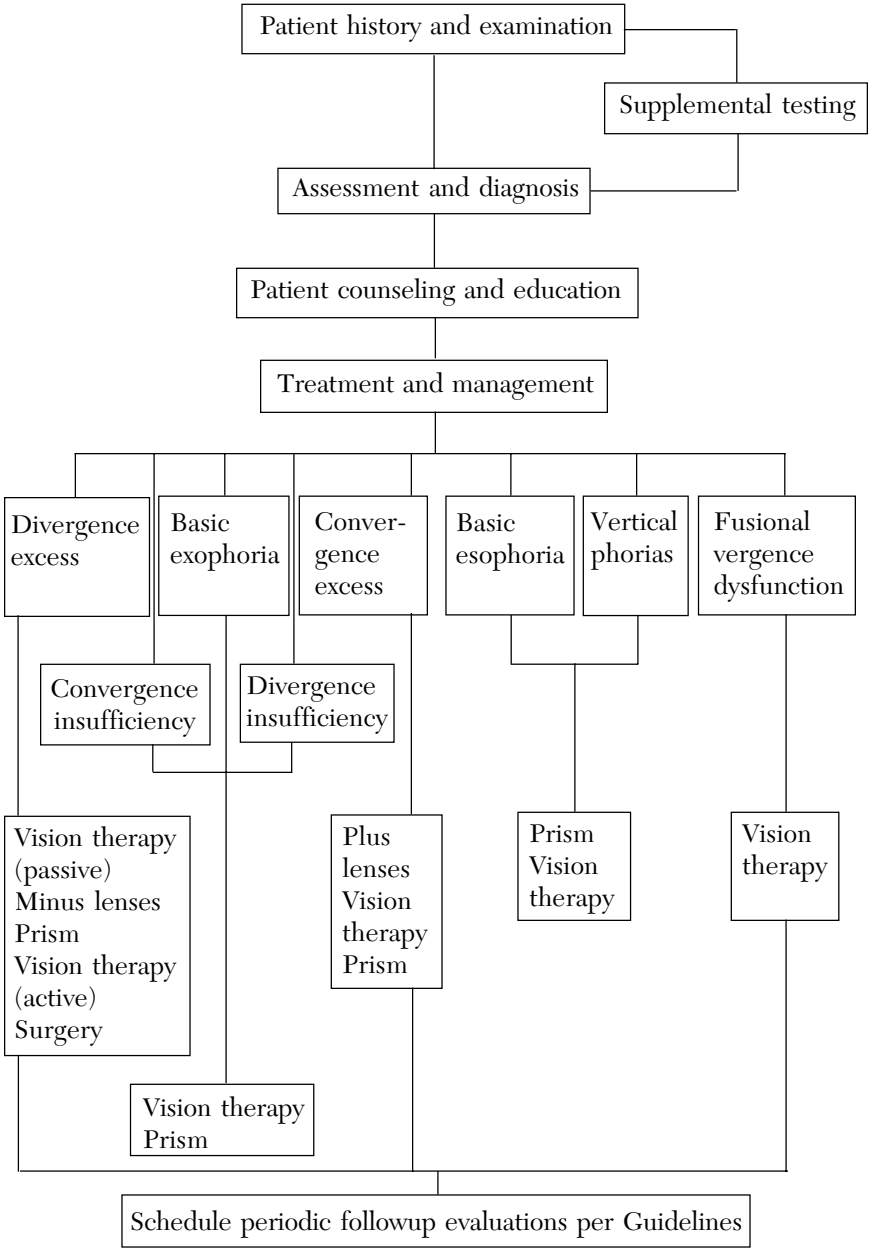

Figure 5
Frequency and Composition of Evaluation and Management Visits for Accommodative or Vergence Dysfunction

Dysfunction	Number of Evaluation Visits	Treatment Options	Prognosis
Convergence insufficiency	1	Vision therapy; prisms	Excellent
Divergence excess	2	Vision therapy; prism; minus lenses; surgery	Good
Basic exophoria	1	Prism; vision therapy	Good
Convergence excess	1	Plus lenses; vision therapy; prism	Excellent
Divergence insufficiency	1-2	Vision therapy; prism	Fair
Basic esophoria	1	Prism; vision therapy	Good
Fusional vergence dysfunction	1	Vision therapy	Excellent
Vertical phorias	1-2	Prism; vision therapy	Good
Accommodative insufficiency	1	Vision therapy; plus lenses	Excellent
Ill-sustained accommodation	1	Vision therapy; plus lenses	Excellent
Accommodative infacility	1	Plus lenses; vision therapy	Excellent
Paralysis of accommodations	1	Optical correction	Poor
Spasm of accommodation	1-2	Plus lenses; vision therapy; cycloplegic drug	Fair

Note: VT = vision therapy; MRI = magnetic resonance imaging.

*Figure 5 extends horizontally on page 75.

Figure 5 Continued . . .

Number of Followup Visits (VT)	Management Plan*
15-20	Provide in-office VT with supplemental home VT; use prisms if patient is not able to participate in VT; educate patient
30	Provide active VT; use passive VT including occlusion, base-in prism, and minus lenses for noncommunicative patient; surgery if VT is not successful or the deviation is too large; educate patient
30	Treat near problems like CI; treat distance problems like DE; educate patient
15-25	Prescribe plus lens addition at near; provide VT for residual symptoms; increase plus acceptance; use prism for the nonresponsive patient; educate patient
15-25	Differentiate functional DI from acquired DI in children; refer patient for MRI if neurological; treat with VT or prismatic correction at distance; educate patient
20	Eliminate deviation by correcting hyperopia; prescribe prismatic correction; provide VT for residual asthenopia and to eliminate prism; educate patient
15-20	Provide VT balanced between convergence and divergence; treat abnormal accommodative system; educate patient
20	Correct vertical deviation with prism; if vergence dysfunction, proceed with horizontal vergence VT; educate patient
10	Provide VT to build accommodative amplitudes and accommodative facility; prescribe plus lenses at near; educate patient
10	Treat with VT or plus lenses; educate patient
10	Improve speed of accommodation with plus lenses initially; proceed with vision therapy; educate patient
—	Determine underlying cause; correct with progressive lens when necessary; educate patient
10	Begin with plus lenses and VT; if VT fails, use cycloplegic agent temporarily; educate patient

* See Guideline for other management strategies

Figure 6
ICD-9-CM Classification of Accommodative and Vergence Dysfunction

Presbyopia	**367.4**
Disorders of accommodation	**367.5**
Paresis of accommodation Cycloplegia	**367.51**
Total or complete internal ophthalmoplegia	**367.52**
Spasm of accommodation	**367.53**
Other disorders of refraction and accommodation	**367.8**
Transient refractive change	**367.81**
Other Drug-induced disorders of refraction and accommodation Toxic disorders of refraction and accommodation	**367.89**
Unspecified disorder of refraction and accommodation	**367.9**
Visual disturbances *Excludes: electrophysiological disturbances (794.11-794.14)*	**368**
Subjective visual disturbances	**368.1**
Subjective visual disturbance, unspecified	**368.10**
Visual discomfort Asthenopia Photophobia Eye strain	**368.13**
Other visual distortions and entoptic phenomena Photopsia Visual halos Refractive: diplopia polyopia	**368.15**
Diplopia Double vision	**368.2**
Other disorders of binocular vision	**368.3**

Figure 6 Continued . . .

Binocular vision disorder, unspecified	**368.30**
Suppression of binocular vision	**368.31**
Simultaneous visual perception without fusion	**368.32**
Fusion with defective stereopsis	**368.33**
Abnormal retinal correspondence	**368.34**
Other specified visual disturbances Blurred vision NOS	**368.8**
Unspecified visual disturbance	**368.9**
Heterophoria	**378.4**
Heterophoria, unspecified	**378.40**
Esophoria	**378.41**
Exophoria	**378.42**
Vertical heterophoria	**378.43**
Cyclophoria	**378.44**
Alternating hyperphoria	**378.45**
Other disorders of binocular eye movements *Excludes: nystagmus (379.50-379.56)*	**378.8**
Palsy of conjugate gaze	**378.81**
Spasm of conjugate gaze	**378.82**
Convergence insufficiency or palsy	**378.83**
Convergence excess or spasm	**378.84**
Anomalies of divergence	**378.85**
Internuclear ophthalmoplegia	**378.86**
Other dissociated deviation of eye movements Skew deviation	**378.87**

Abbreviations of Commonly Used Terms

AA ..... Amplitude of accommodation

AC/A ..... Accommodative convergence/accommodation ratio

ARC ..... Anomalous retinal correspondence

BI ..... Base-in

BO ..... Base-out

CE ..... Convergence excess

CI ..... Convergence insufficiency

CSBV ..... Clear, single binocular vision

D ..... Diopter

DE ..... Divergence excess

DI ..... Divergence insufficiency

FD ..... Fixation disparity

FDC ..... Fixation disparity curve

IPD ..... Interpupillary distance

MEM ..... Monocular estimated method

NFV ..... Negative fusional vergence

NPC ..... Near point of convergence

NRA ..... Negative relative accommodation

NRC ..... Normal retinal correspondence

PD ..... Prism diopter

PFC ..... Positive fusional convergence

PRA ..... Positive relative accommodation

SNR ..... Spasm of the near reflex

Glossary

Accommodation The ability of the eyes to focus clearly on objects at various distances.

Accommodative convergence/accommodation (AC/A) ratio The convergence response of an individual to a unit stimulus of accommodation.

Accommodative infacility Slow or difficult accommodative response to dioptric change in stimulus; accommodative inertia.

Accommodative insufficiency Less accommodative amplitude than expected for the patient's age.

Accommodative vergence Vergence as a result of accommodation.

Amplitude of accommodation (AA) The difference between the farthest point and the nearest point of maximum accommodation denoted by first sustained blur with respect to the spectacle plane, the entrance pupil, or some other reference point of the eye, expressed in diopters.

Anomalous retinal correspondence (ARC) A type of retinal projection, occurring frequently in strabismus, in which the foveae of the two eyes do not facilitate a common visual direction; a condition in which the fovea of one eye has the same functional direction with an extrafoveal area of the other eye; anomalous correspondence.

Asthenopia Subjective symptoms or distress arising from use of the eyes; eyestrain.

Convergence The turning inward of the primary lines of sight toward each other.

Convergence excess (CE) Vergence condition characterized by orthophoria or near-normal phoria at distance and esophoria at near.

Convergence insufficiency (CI) Vergence condition characterized by an inability to maintain effortless convergence at near distances. CI is often accompanied by reduced near point of convergence, exophoria or exotropia at near greater than the distance measurement, and/or reduced convergence amplitude in relationship to the demand.

Cover test A clinical test to determine the ocular alignment of the eyes.

Diplopia A condition in which a single object is perceived as two rather than one; double vision.

Divergence excess (DE) A vergence anomaly characterized by exotropia or high exophoria at distance greater than the near deviation.

Divergence insufficiency (DI) A vergence anomaly characterized by esotropia or high esophoria at distance greater than the near deviation.

Esophoria, basic Vergence position of the eyes in which the two eyes' lines of sight cross closer to the patient than the object of regard when binocular fusion is disrupted, the magnitude of the deviation being the same at both far and near fixation distances.

Exophoria, basic Vergence position of the eyes in which the two eyes' lines of sight cross further than the object of regard when binocular fusion is disrupted, the magnitude of the deviation being the same at both far and near fixation distances.

Fixation disparity (FD) Overconvergence or underconvergence, or vertical misalignment of the eyes under binocular (both eyes) viewing conditions small enough in magnitude so that fusion is present.

Fusion The process by which stimuli seen separately by the two eyes are combined, synthesized, or integrated into a single perception.

Fusional vergence Vergence (convergence or divergence) stimulated by retinal disparity resulting in the avoidance of diplopia. Synonyms: reflex vergence, disparity vergence.

Fusional vergence amplitude The angle between the maximum convergence and the maximum divergence of the eyes that can be elicited in response to change in convergence while the accommodation response remains constant.

Ill-sustained accommodation A condition similar to accommodative insufficiency but lesser in extent.

Near point of convergence (NPC) The maximum extent the eyes can be converged.

Negative fusional vergence (NFV) A measure of fusional convergence from the phoria position of the eyes to the prism base-in limit of clear, single binocular vision; fusional divergence.

Negative relative accommodation (NRA) A measure of the maximum ability to relax accommodation while maintaining clear, single binocular vision.

Negative relative convergence The base-in prism range of clear, single binocular vision as measured from Donder's line.

Normal retinal correspondence (NRC) Retinal projection in which the two foveae (and/or other binocularly paired extrafoveal receptor areas) have common lines of direction or a common local sign.

Orthophoria Condition in which, in the absence of an adequate fusion stimulus, the lines of sight intersect at a given point of reference, usually the point of binocular fixation; absence of heterophoria.

Orthoptics The treatment process for the improvement of visual perception and coordination of the two eyes for efficient and comfortable binocular vision. Synonyms: vision training, vision therapy.

Paralysis of accommodation Absence of accommodation due to paralysis of the ciliary muscle.

Positive fusional convergence (PFC) Fusional convergence measured in a positive or increasing direction from the phoria position of the eyes to the base-out prism limit of clear, single binocular vision. Synonym: positive fusional vergence (PFV).

Positive relative accommodation (PRA) A measure of the maximum ability to stimulate accommodation while maintaining clear, single binocular vision.

Positive relative convergence The base-out prism range of clear, single binocular vision as measured from Donder's line.

Proximal convergence Convergence due to the awareness of nearness. Synonyms: psychic convergence, voluntary convergence.

Proximal vergence Convergence response attributed to the awareness of, or, the impression of nearness of an object of fixation.

Sensory fusion The ability of the brain to bring together two sensations with the end result of a single percept.

Spasm of accommodation A ciliary muscle spasm that produces excess accommodation.

Stereopsis The ability to perceive three-dimensional or relative depth due to retinal disparity.

Tonic vergence Convergence due to the basic tonicity of the extraocular muscles, which are responsible, in part, for the distance phoria.

Vergence The disjunctive movements of the eyes in which the visual axes move toward each other (convergence) or away from each other (divergence).

Vergence insufficiency See convergence or divergence insufficiency.

Version A conjugate movement in which the two eyes move in the same direction.

Vertical phoria Deviations in the direction of gaze that are perpendicular to the plane of fixation.

Vision therapy Treatment process for the improvement of visual perception and coordination of the two eyes for efficient and comfortable binocular vision. Synonyms: orthoptics, visual training.

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