

Care of the Patient with
**Accommodative and
Vergence
Dysfunction**

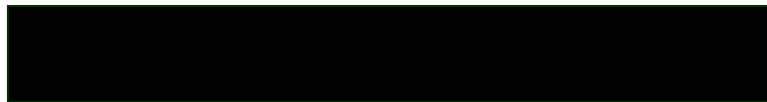


**OPTOMETRY:
THE PRIMARY EYE CARE PROFESSION**

Doctors of optometry are independent primary health care providers who examine, diagnose, treat, and manage diseases and disorders of the visual system, the eye, and associated structures as well as diagnose related systemic conditions.

Optometrists provide more than two-thirds of the primary eye care services in the United States. They are more widely distributed geographically than other eye care providers and are readily accessible for the delivery of eye and vision care services. There are approximately 36,000 full-time-equivalent doctors of optometry currently in practice in the United States. Optometrists practice in more than 6,500 communities across the United States, serving as the sole primary eye care providers in more than 3,500 communities.

The mission of the profession of optometry is to fulfill the vision and eye care needs of the public through clinical care, research, and education, all of which enhance the quality of life.



**OPTOMETRIC CLINICAL PRACTICE GUIDELINE
CARE OF THE PATIENT WITH ACCOMMODATIVE
AND VERGENCE DYSFUNCTION**

Reference Guide for Clinicians

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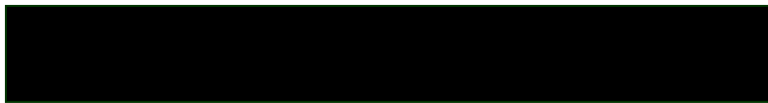
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NOTE: Clinicians should not rely on the Clinical Guideline alone for patient care and management. Refer to the listed references and other sources for a more detailed analysis and discussion of research and patient care information. The information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



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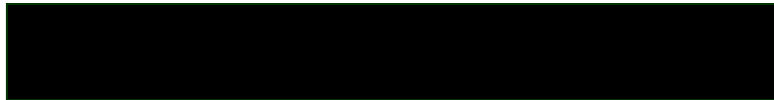
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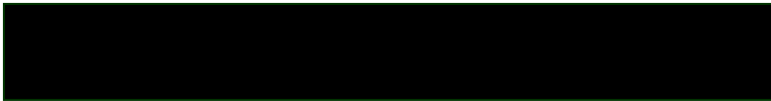
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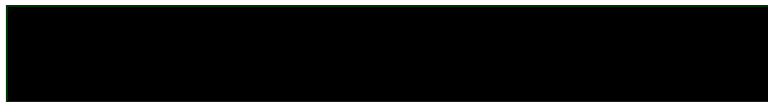
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INTRODUCTION

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, diagnosis, treatment, and management to reduce the risk of visual disability from these binocular vision anomalies through timely care. 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 and management.



2 Accommodative and Vergence Dysfunction



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



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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, 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 non-athletes⁵ and that varsity college athletes have better depth perception than non-athletes.^{6,7,8}

The use of computers at home, 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 problems⁹ and ocular discomfort increases with the extent of computer use.¹⁰⁻¹² 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 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,¹³ 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-10-CM classification of accommodative and vergence dysfunction is shown in Appendix Figure 2.

1. Accommodative Dysfunction

This Guideline uses the Duke-Elder classification of accommodative dysfunction.¹⁴

a. Accommodative Insufficiency

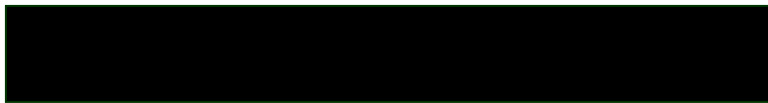
Accommodative insufficiency occurs when the amplitude of accommodation (AA) is lower than expected for the patient's age and is not due to sclerosis of the crystalline lens.^{14,15} Patients with accommodative insufficiency usually demonstrate poor accommodative sustaining ability.

b. Ill-Sustained Accommodation

Ill-sustained accommodation is a condition in which the AA is normal, but fatigue occurs with repeated accommodative stimulation.^{14,15}

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.¹⁵ The patient often reports blurred distance vision immediately following sustained near work. Some have considered this infacility to be a precursor to myopia.¹⁶



6 Accommodative and Vergence Dysfunction

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.¹⁵ 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).¹⁶ 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.¹⁷ 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).¹⁷ 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; it has been called "common CI."¹⁸

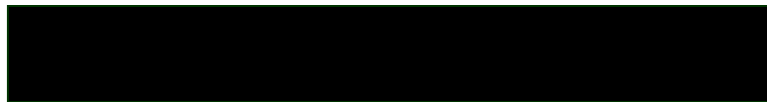


Table 1
Modified Duane Classification System*

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 exophoria Large exophoria/tropia at distance
Basic exophoria	$X = X'$ Normal AC/A ratio
Convergence excess	$E < E'$ High AC/A ratio
Divergence insufficiency	$E > E'$ Low AC/A ratio High tonic esophoria
Basic esophoria	$E = E'$ Normal AC/A ratio
Vergence insufficiency	Normal AC/A ratio Restricted fusional vergence amplitudes Steep fixation disparity curve
Vertical phorias	Comitant deviations Noncomitant deviations <ul style="list-style-type: none">• Old decompensated 4th nerve palsies• Newly acquired 4th nerve palsies

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.



8 Accommodative and Vergence Dysfunction

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).¹⁹ Divergence excess can be further divided into true or simulated DE on the basis of responses to occlusion. In simulated DE, occlusion dramatically affects slow vergence, increasing the angle of deviation slightly at distance and significantly at near. Occlusion does not affect true DE.

c. Basic Exophoria

The patient with basic exophoria has a deviation of similar magnitude at both distance and near.^{20, 21}

d. Convergence Excess

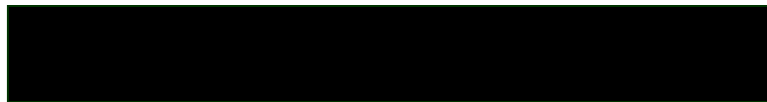
The patient with convergence excess (CE) has a near deviation at least 3 PD more esophoric than the distance deviation.²² The etiology of the higher esodeviation at near most commonly is indicated by a high accommodative convergence/accommodation (AC/A) ratio.

e. Divergence Insufficiency

In a patient with divergence insufficiency (DI), tonic esophoria is high when measured at distance but less at near.²³ 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.¹⁷



g. Fusional Vergence Dysfunction

Patients with fusional vergence dysfunction (vergence insufficiency) often have normal phorias and AC/A ratios but reduced fusional vergence amplitudes.²⁴ Their zone of clear, single binocular vision (CSBV) is small.

h. Vertical Heterophorias

Vertical heterophorias may be either comitant and idiopathic or noncomitant, due to muscle paresis or other mechanical cause.²⁵ 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^{26,27}; 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.²⁶



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.²⁸

Accommodation can be affected by a number of drugs and diseases.²⁹

2. Vergence Dysfunction

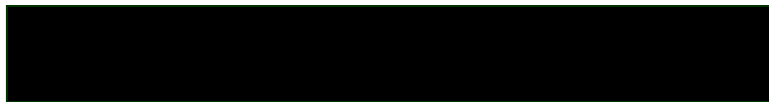
a. Prevalence

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 in adults is between 1 and 25 percent of clinic patients.^{17,18,30,31} The median prevalence of CI in the population is 7 percent, a prevalence that is similar for adults and children.¹⁸ The ratio of females to males with CI is 3:2.³²

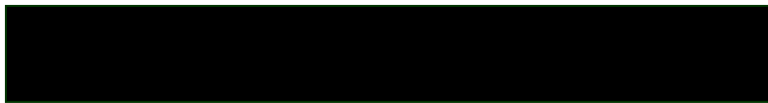
The authors of some early studies defined CI as including symptoms (reduced NPC, exophoria, and reduced convergence amplitudes), while others specified the inclusion of anyone with asthenopia associated with convergence.

Recent studies have investigated the relationship of various signs of CI in school and clinic populations. A study to establish the frequency of CI in 5th and 6th graders ages 9-13 years defined high suspect CI as "exophoric at near and two clinical signs" and definite CI as "exophoric at near and three clinical signs." Among the 453 subjects for whom all CI measurement data were collected, 8.8 percent were classified as high suspect and 4.2 percent as



definite CI. Of those with CI, 89 percent also had AI.³³ In a second study by the same group, testing of 392 elementary school children 8–15 years of age revealed 18 (4.6%) with three signs of CI and 50 (12.7 %) with two signs of CI; 41 (10.5%) of the children were classified as having pure accommodative insufficiency (AI), i.e., no signs of CI.³⁴

- **Divergence excess.** The prevalence of DE is approximately 0.03 percent of the population, and it is more common in women and African Americans.¹⁹ DE strabismus has a strong hereditary predisposition.¹⁹
- **Convergence excess.** One study of an urban population reported that 5.9 percent of patients seeking optometric care had CE,²⁶ and another found a 7.1 percent prevalence in a pediatric population.³⁵
- **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.³⁵
- **Basic exophoria and esophoria.** One study of 179 patients with exodeviation found that 62 percent had CI and 27 percent had basic exophoria.³⁶ 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.³⁷
- **Vertical heterophoria.** Early estimates of the prevalence of vertical deviations ranged from 7 percent³⁸ to 52 percent.³⁹ An estimate of the prevalence of vertical heterophorias is about 20 percent of the population.⁴⁰ The reported prevalence differs on the basis of criteria used to diagnose a clinically significant vertical heterophoria. Only about 9 percent of vertical heterophorias are clinically significant.⁴⁰



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.⁴¹⁻⁴³ CI is the most common vergence dysfunction found with Graves disease.⁴⁴ 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.^{45,46}

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.¹⁵ 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.⁴⁷ 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, resulting in reduction of accommodative amplitude and the onset of presbyopia.⁴⁸

The accommodative response is the actual amount of accommodation produced 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.⁴⁹ 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.^{50,51}

Sustained accommodative effort has been reported to cause accommodative fatigue and asthenopia. In some individuals, the punctum proximum (PP) recedes after repeated push-up stimulation of accommodation.⁵² 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.⁵³ Repeated near-far stimulation does not affect the AA in most subjects.⁵⁴ The few subjects who demonstrated fatigue also reported asthenopia that was not age dependent.⁵⁴ 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

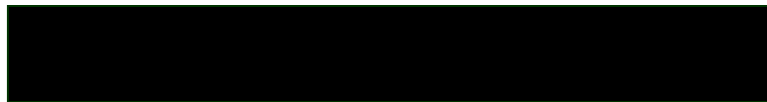
- **Accommodative insufficiency.** Patients with accommodative insufficiency often complain of blurred vision, difficulty reading, irritability, poor concentration, and/or headaches. Attempting to accommodate, some patients may stimulate excessive convergence by the AC/A crosslink and be incorrectly 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. There have been cases associated with LASIK,^{55,56} multiple sclerosis,⁵⁷ and closed head injury.⁵⁸⁻⁶⁰ Spasms as great as 25 D have been reported, and distance vision is usually impaired.⁶¹ For most patients with this disorder, the etiology is probably psychogenic.⁶²



Some clinicians use the term "accommodative excess" interchangeably with "accommodative spasm."¹⁶

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 the age of 3 years 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.⁶² 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

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



16 Accommodative and Vergence Dysfunction

movements must be extremely accurate to 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.⁶³

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.¹³ 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.

This feedback system analysis has been expanded to demonstrate that there is not only neurological adaptation but also muscular adaptation. Over time tonic muscle position will result in either shortening or lengthening the muscles to take a load off the vergence system. According to the model described by Guyton, based upon Schor's work: the eyes initially make a movement to eliminate vergence disparity; followed by slow vergence to eliminate the fast vergence error; fine tuning is provided by fixation disparity; with long-term changes taking place in alteration in muscle length by either addition or subtraction of sacromeres.⁶⁴

- **Convergence insufficiency.** The etiology of CI is controversial. It probably results from a breakdown in the accommodative-

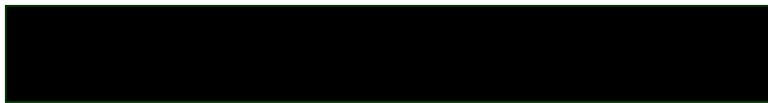


convergence relationship.^{18,65-67} It is possible that a genetic predisposition for CI exists, because the parents of children with CI often have the condition. Symptoms tend to occur when persons use their eyes in a two-dimensional reading environment for extended periods of time. The symptoms tend 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.^{10-12,18,68,69}

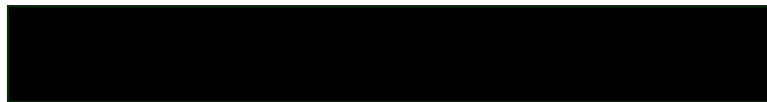
Most patients with CI have normal stereopsis but may exhibit suppression when viewing first-degree fusion targets. It is not uncommon for the CI patient to manifest an exotropia during near point testing without reporting diplopia. When an eye deviates, the patient may report blurred vision or suppress 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).^{18,70,71} Many patients with CI also have poor accommodative facility.^{18,72,73} 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.¹⁹ 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.¹⁹ The deviation is often first noticed in children under 18 months of age.⁷⁴ 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.²² 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 this problem when asthenopia occurs.
- **Vertical heterophoria.** 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 will decompensate over time, may be first noted after insult such as high fever or trauma. A newly acquired fourth nerve palsy occurs after a vascular, infectious, traumatic, or neoplastic incident.⁷⁵ 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. Young children may not report symptoms because they consider diplopia and asthenopia normal. During the formative school years, the additional demand 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 discomfort. 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.

The development of scaled questionnaires has improved the ability to detect and quantify symptoms associated with either accommodative or vergence anomalies.

The Convergence Insufficiency Symptom Survey (CISS) has 15 questions designed to quantify symptoms associated with reading and near work. The survey has been used for prospective assessment of symptoms in school-age children (8-13 years)^{1,6} and adults (19-30 years).⁹ The results of these studies demonstrate that the CI Symptom Survey is a valid and reliable instrument that can be used clinically or as an outcome measure for research studies for individuals with CI. However, the questionnaire does not help detect the patient whose symptoms are few but dramatic (e.g., constant diplopia without other symptoms).

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.¹⁸ In addition, CI may be associated with emotional problems and anxiety.¹⁸

Studies have shown that children with attention deficit hyperactivity disorder (ADHD) have a higher percentage of CI than normal children.^{76,77}

Most patients with CI have a low PFC amplitude (10 PD or less).¹⁸ One study reported that, at near, 79 percent of all patients with CI have an exophoria, 18 percent are orthophoric, and 3 percent are esophoric.⁶⁸ Thus, if CI is defined as reduction in PFC and a receded NPC, it is possible for a patient to have either orthophoria or esophoria and still be classified as having CI.

Symptomatic CI patients have poor prism adaptation and slow vergence ability.^{78,79} 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.^{67,80} 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.⁸¹

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.⁸² If ARC occurs when the eye deviates, the DE patient has an extension of the binocular field known as panoramic viewing.⁸² 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.

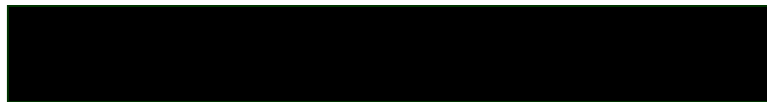
- **Basic exophoria.** The most common symptoms of basic exophoria are related to asthenopia. The clinical findings of basic exophoria are similar to those of DE because the basic exophoric patient is considered to be a DE patient who acquires CI. Thus, like the DE patient, the patient with basic exophoria 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.⁸³
- **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.⁸⁴



- **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) for both convergence and divergence measurements. 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 heterophoria.** 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 PD 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)⁸⁵ because ocular alignment is a prerequisite for the development of normal binocularity.⁸⁶



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 complicating factor.^{2,87,88} Because elimination of certain vergence anomalies can improve reading scores,⁸⁹ 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 3).

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.

Alternatively, for patients suspected of convergence insufficiency, the Convergence Insufficiency Symptom Survey (CISS) can be used to quantify symptoms using a standardized symptom questionnaire (Appendix Figure 4).^{32,90}

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. Furthermore, 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.

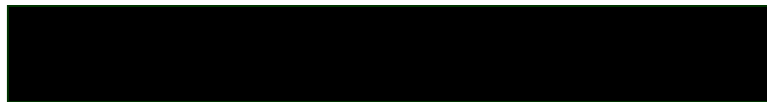
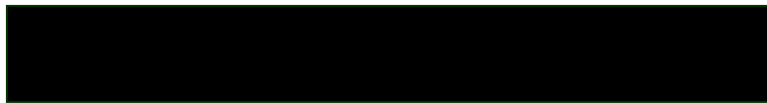


Table 2
Suggested Questions for Patient History

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?
-

Normally, all components of vergence and accommodation are synergistic; accommodation, convergence, and pupillary miosis occur in synchrony. Procedures that isolate these individual 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.⁹¹ When taking any clinical measurement, the optometrist should encourage the patient to exert maximum effort. The clinician should record any symptoms 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 accommodative and/or 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 whose excessive accommodative response could affect the measurement of refractive error.

c. Ocular Motility and Alignment

Cover testing should be performed with a small target to control accommodation.⁹² 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 with Risley prisms in a phoropter, or using a handheld Risley prism or loose prisms 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 Near Point of Convergence (NPC) test is important for assessment of binocular function. It is best performed using a small accommodative target.^{93,94} 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.^{95,96}

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.

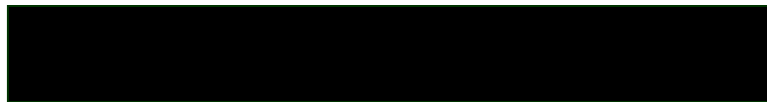


The order in which fusional vergence tests are administered may affect subsequent measurement of vergence functions.^{97,98} If base-out (BO) fusional vergence amplitudes are measured before base-in (BI) amplitudes, the BI fusional amplitudes will be reduced and vice versa. In addition, the position 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.⁹⁹ The fusional amplitudes are dependent on the size of the target and method of presentation.¹⁰⁰ Patients should be instructed to keep the target single and clear, and to report whether the test bothers their 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 measurement of relative accommodation in the non-presbyopic patient, 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. 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

Accommodative Amplitude (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.¹⁰¹

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.¹⁰² Some clinicians advocate using a ± 1.50 D lens test, because it can be done easily in the phoropter and normal patients are less likely to fail it.¹⁵

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.^{15, 103, 104}

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 (RDS) requires both fusion and bifoveal fixation,¹⁰⁵ thus confirming that the patient is not strabismic at the time of testing. A large-disparity RDS is easily appreciated by any patient demonstrating bifoveal fixation and normal stereopsis.¹⁰⁶ Thus, it makes an effective screening device.¹⁰⁷

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 should 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.¹⁰⁸ Many medications can also cause accommodative dysfunction.²⁹



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 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 non-calculated 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 distance–near method gives the clinician a rapid method for determining the appropriate lens power to eliminate the deviation without regard to the specific influence of proximal convergence. The AC/A ratio may be calculated by this formula:

$$\text{AC/A ratio} = \frac{\text{convergence demand of near target} - \text{Hd} + \text{Hn}^1}{\text{stimulus to accommodation of near target}}$$

Where Hd = distance heterophoria

Hn¹ = 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).¹⁰⁹



Alternatively,

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

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:

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

The AC/A is thought to be innate and stable until the beginning of presbyopia¹¹⁰; however, the stimulus and response to accommodation differ. Theoretically, the response AC/A ratio may be estimated by multiplying the stimulus AC/A ratio by 1.08.¹¹¹

b. Fixation Disparity/Associated Phoria

Fixation disparity (FD) 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 Fixation Disparity Curve (FDC) and the associated phoria. 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.¹¹² Mean values are 8 cycles per minute (cpm) for children ages 5-8 years and 13 cpm for children ages 7-14 years¹¹²; the mean value for adults is approximately 7 cpm.¹¹³ Gall and associates reported that single-flip prism 3 PD BI/12 PD BO differentiates optimally between normal adults and patients with asthenopia at distance and near.^{114,115} 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 Estimate Method (MEM).

MEM retinoscopy is performed while having patients read grade-level words at their habitual near working distance. 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 use of plus lenses at near. A lag 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.^{116,117}



4. Assessment and Diagnosis

The clinician may use the history and clinical findings to make the diagnosis and assess the need for treatment and management. Clinical assessment has used the following protocols:

a. Graphic Analysis

Graphic 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.^{118,119} 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.^{71,120,121} One approach, suggested by Sheard,⁷¹ 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.¹²² 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.^{13, 123} 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.¹²⁴ 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.¹²⁵

e. *Comparison of Methods of Analysis*

Discriminant analysis enables evaluation of these methods of measuring heterophoria, vergences, and FDCs in symptomatic and asymptomatic patients.^{126,127} The application of Sheard's criterion is another means of identifying symptomatic exophoric patients.¹²⁶ When the use of Sheard's criterion does not differentiate asthenopic from nonasthenopic exophoric patients, the angular measurement of FD has been effective. The absolute magnitude of esophoria is most predictive of asthenopia for esophoric patients; the second best measure of esophoria is the Negative Fusional Vergence (NFV) recovery value.¹²⁶ The clinician should use these principles as a guide in determining the need for and amount of prismatic correction.

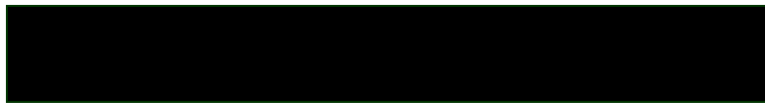


Table 3
Expected Values for Accommodation and Vergence Testing*

Measurement	Mean	S.D.	Range
Distance:			
Phoria	1X	2X	0-2X
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	3X'	5X'	0-6X
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	0.50	-1.75 - +2.25
NRA	+2.00	1.1	+1.75 - 2.25
Gradient AC/A	4/1	2	3-5
AA	$16 - (0.25 \times \text{age})$	± 2.00	± 1.00

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

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.



B. Management of Accommodative and Vergence Dysfunction

Management of the patient with an accommodative or vergence dysfunction is based on interpretation and analysis of the examination results. Appendix Figures 5 and 6 provide an overview of patient management strategies for accommodative and vergence dysfunction.

1. Basis for Treatment

The general goals for the treatment and management of 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.^{15,53,128,129} Repeated accommodative testing itself improves accommodative responses.⁵³ Studies have also shown that voluntary accommodation can be taught¹³⁰ and that accommodation skills developed by biofeedback can transfer from one task to another.¹³¹

Research has demonstrated the effectiveness of accommodative therapy in eliminating decreased accommodative amplitude and facility.^{129,132,133} In one study, 87 percent of the patients with



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

Accommodative Dysfunction	Study Authors	Summary/Interpretation
Accommodative insufficiency	Cooper ¹⁵	Accommodation can be modified with training.
	Berens and Stark ⁵³	
	Carr and Allen ¹²⁸	
	Sisson ¹²⁹	
Accommodative infacility	Berens and Stark ⁵³	Repeated accommodative testing has been shown to improve accommodative responses.
	Marg ¹³⁰	Voluntary accommodation can be taught.
	Cornsweet and Crane ¹³¹	Accommodation developed by biofeedback can transfer from one task to another.
	Sisson ¹²⁹	Accommodative therapy has been shown to be effective in eliminating decreased accommodative amplitude and facility.
	Morris ¹³²	
Hoffman et al. ²⁷	In 87% of patients with accommodative anomalies, asthenopia was eliminated and accommodative findings were normalized with approximately 26 therapy sessions.	
Daum ¹³⁴	Therapy to improve accommodative amplitudes can result in a concurrent improvement of positive and negative fusional amplitudes and stereopsis.	



Table 4 (cont.)

Accommodative Dysfunction	Study Authors	Summary/Interpretation
	Cooper et al. ¹³⁵	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 and Murphy ¹³⁶ Liu et al. ¹³⁷	Vision therapy may result in positive changes in the magnitude, velocity, and gain of accommodative responses.
	Liu et al. ¹³⁷	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 and Sivak ¹³⁸	Vision therapy improves the time characteristics of the accommodative response, including the latency and velocity.

accommodative anomalies had eliminated their asthenopia and normalized their accommodative findings after approximately 26 therapy sessions.²⁷



Therapy to improve AA can result in a concurrent improvement of PFC, NFV, and stereopsis.¹³⁴ Vision therapy is the method of choice in eliminating asthenopic symptoms associated with accommodative anomalies.¹³⁵ 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,¹³⁵ 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.¹³⁵

These studies suggest that vision therapy is effective in altering accommodation, with a resultant change in 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.^{136,137} 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.¹³⁷ Therapy improves the time characteristics, including both latency and velocity of the accommodative response.¹³⁸

- **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.¹³⁹ 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 when age and diseases such as hyperthyroidism have altered orbital contents. 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.^{105,140-144} These studies demonstrate that vergence therapy improves vergence ability, and that the effects persist over time (Table 5). All of the studies demonstrating the efficacy of vision therapy used in-office therapy regimens.

Vision therapy for vergence dysfunction has a high success rate. Pooled data for patients with CI indicate that 72 percent of patients have been successfully managed, while 19 percent improved significantly and only 9 percent failed.^{18,66} Vision therapy has a lasting effect when a complete cure is achieved.¹⁴⁰ Moreover, age is not a deterrent in the successful management of binocular anomalies.^{145,146}

A controlled, prospective, double-blind, A-B reversal study compared vergence treatment and placebo treatment in a group of patients diagnosed with CI. Random dot stereograms were presented 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.¹⁴⁷

The Convergence Insufficiency Treatment Trial (CITT) comprised two pilot studies and one large clinical trial.^{96,148,149} In the first clinical trial, 47 children 9–17 years of age with symptomatic CI were randomly assigned to office-based vision therapy, office-

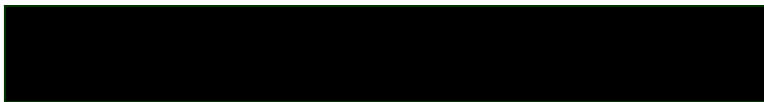


Table 5
Effectiveness of Vision Therapy for Vergence Dysfunction:
Research Results

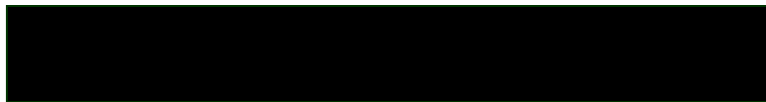
Vergence Dysfunction	Study Authors	Summary/Interpretation
Convergence insufficiency	Cooper and Duckman ¹⁸ Grisham ⁶⁶	72% of patients reported cured, 19% reported improved, 9% reported failed.
	Grisham et al. ¹⁴⁰	Vision therapy has a lasting effect when a complete cure is achieved.
	Wick ¹⁴⁵	Age is not a deterrent to successful treatment.
	Cooper et al. ¹⁴⁷	Results demonstrated a dramatic improvement in vergence amplitudes with a concurrent decrease in symptoms.
Intermittent exotropia	Coffey, Wick, Cotter, et al. ¹⁵²	Pooled success rates of different treatment regimens (59% for vision therapy, 46% for exotropia surgery, and 28% for passive therapy [e.g., minus lenses, occlusion, and/or prisms]) suggest that vision therapy is more effective than surgery.
	Sanfilippo and Clahane ¹⁵³	64.5% reported cured, 9.7% reported improved, 9% reported fair.
	Sanfilippo and Clahane ¹⁵⁴	Subsequently after 5 years, 52% remained cured, 32% remained improved.
	Mann ¹⁵⁵ Durrant ¹⁵⁶	Similar rates of success for vision therapy have been reported by these studies.
	Cooper and Leyman ¹⁵⁷ Altizer ²¹	
	Chryssanthau ¹⁵⁸ Daum ¹⁵⁹	



Table 5 (cont.)

Vergence Dysfunction	Study Authors	Summary/Interpretation
	Goldrich ¹⁶⁰	Highest success rate occurred when office therapy was supplemented with home vision therapy.
Convergence excess	Gallaway and Scheiman ¹⁶¹	Total elimination of symptoms in 80% of patients with the following improvements: mean divergence amplitude increased 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 ¹⁶² Robertson and Kuhn ¹⁶³	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 ¹⁶²	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.

based placebo vision therapy, or home-based pencil push-up therapy. Symptoms were significantly reduced in the vision therapy group but not in the pencil push-up or placebo vision therapy groups. Only patients in the vision therapy group



demonstrated both statistically and clinically significant changes in near point of convergence and positive fusional vergence at near. Neither pencil push-ups nor placebo vision therapy was effective in improving either symptoms or signs associated with convergence insufficiency.¹⁴⁸

Adults ages 19–34 years were evaluated using an identical protocol to that of the children’s study. At the end of therapy, the CI Symptom Survey showed a significant reduction in symptoms for patients in each of the three treatment groups. However, patients in the vision therapy group had the greatest reduction in symptoms. Patients treated with pencil push-ups and placebo vision therapy did not achieve normal symptom scores.⁹⁶

Two hundred and twenty-one 9- to 17-year-olds with symptomatic CI participated in the full CITT clinical trial.¹⁴⁹ The four management strategies included active vision therapy, computerized home therapy, placebo vision therapy, and push-ups. At the end of 12 weeks of therapy, there was a reduction in symptoms for patients in each of the four treatment groups. Patients in the vision therapy group had the greatest reduction in symptoms. Those in the pencil push-up, computerized home therapy, and placebo vision therapy groups did not achieve normal symptom scores. Over time, trends showed a reduction in symptoms for all groups. These studies indicate that vision therapy results in a clinical and statistical reduction in symptoms that is not due to placebo effects.¹⁴⁹

All CITT groups showed an improvement in the NPC. Over time, the office-based vision therapy group showed the greatest changes, followed by the home computerized vision therapy, pencil push-ups, and placebo therapy groups. Follow-up at 6 months and 1 year after 12 weeks of therapy showed no regression from the post-treatment results for any of the four management strategies.¹⁵⁰ Thus, post-therapy effects persist for at least 1 year.

A retrospective study evaluated 43 prepresbyopic patients with presumed symptomatic vergence anomalies who completed a

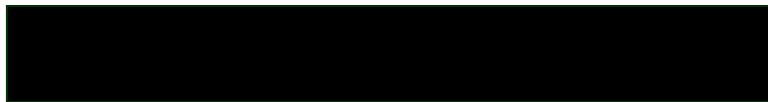


home computerized vision therapy program. The authors concluded that when in-office vision therapy is not practical, patients who demonstrate symptoms associated with an accommodative/vergence anomaly may use a home computerized therapy system.¹⁵¹

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).¹⁵² These data suggest that vision therapy is more effective than surgery in patients with intermittent exotropia.¹⁵²

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.¹⁵³ A follow-up study found that after 5 years, 52 percent of these patients remained cured, while 32 percent were in the improved group.¹⁵⁴ Similar findings have been reported by other studies.^{21,155-159} One study reported that the highest success rate occurred when in-office therapy was supplemented with home vision therapy.¹⁶⁰

An additional study to demonstrate the effectiveness of vision therapy for CE,¹⁶¹ 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 been prescribed spectacles to eliminate the esophoria; others had not. Comparison of the results for the patients receiving vision therapy alone with the results for patients initially receiving reading spectacles and then undergoing vision therapy showed no difference in the post-vision therapy results, suggesting that vision therapy alone is highly effective in eliminating abnormal vergence findings associated with CE.¹⁶¹

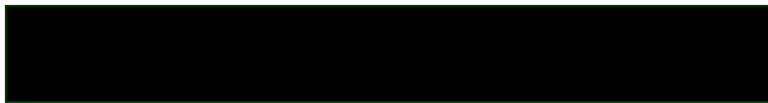


Vertical prism is the usual treatment of choice for vertical deviation. However, vision therapy was effective in a small sample of patients with vertical deviations and in patients with longstanding decompensated vertical deviations. Vision therapy may be used to decrease prism adaptation as well as to reduce the need for future increases in prism correction.^{162,163} 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.¹⁶²

Patients with closed head injuries often develop accommodative dysfunction and CI secondary to trauma. Studies comparing therapeutic options for these patients^{41,42,58,164,165} 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.

b. Lens and Prism Therapy

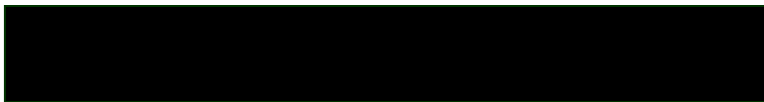
- **Horizontal prisms.** Clinicians often prescribe prism to eliminate symptoms of asthenopia and to reduce the fusional vergence demand in patients with vergence dysfunction. Two common methods of determining the amount of prism to prescribe are (1) to satisfy Sheard's criteria and (2) to eliminate the FD.¹⁶⁶ 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. Each patient received 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.¹⁶⁷



In a prospective double-masked, multicenter, randomized clinical trial, 72 children aged 9 through 17 years with symptomatic CI were randomly assigned to base-in prism glasses or glasses without prism. They were instructed to wear the glasses for all near tasks. The CISS was given at the baseline examination and after 6 weeks of wearing glasses. The BI-prism reading glasses were found to be no more effective than placebo reading glasses in alleviating symptoms or improving the near point of convergence or positive fusional vergence at near in the children with symptomatic CI. These results demonstrate the potentially powerful placebo effect of prescribing any eyeglasses for children 9 through 17 years old.¹⁶⁸

A prospective study of symptomatic CI subjects aged 45–68 years was performed; whereby each subject was assigned two pairs of progressive addition glasses, one with BI prism and one without prism. The subjects wore each pair of glasses for 3 weeks and then completed the CISS. The mean CISS score of 30 at baseline decreased to 13 with the BI-prism glasses and to 24 with glasses without prism. Progressive addition glasses with BI prism were found to be effective in reducing symptoms of presbyopes with symptomatic CI, at least for the short term.¹⁶⁹

Prism may be the only viable treatment for CI in patients who are unable to participate in a vision therapy program because of time, or cognitive or financial constraints; however, it should be used with caution. Although patients with symptomatic vergence anomalies may be treated with prisms, 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



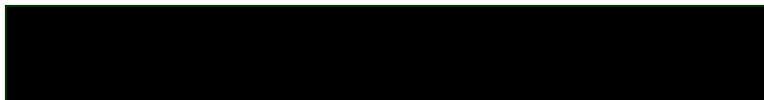
significant deviation with minimal vergence adaptation can prism compensation be effective.¹⁶⁹

Adaptation to base-out and base-in prisms differs. As expected, most people adapt faster and more completely to BO prism than to BI prism.^{170,171} Prolonged wearing of prisms not only alters the heterophoria position, but it also results in a readjustment of horizontal fusional amplitudes.¹⁷¹ 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.¹⁷²⁻¹⁷⁴ 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 parietic deviations; and large-angle, decompensated, older deviations. Studies have shown that patients with these deviations differ in their adaptation responses to vertical prism.^{170,175} 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 after removal of the prism. 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

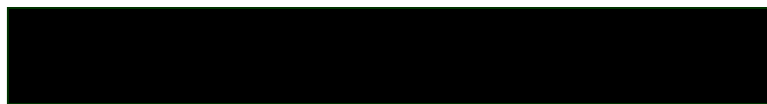


predicted to occur whenever a heterophoria increases dramatically after repeated, prolonged cover testing.

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 equal (i.e., PRA/NRA).
- **Minus lenses.** Minus lenses may be used to change the motor demand of the vergence system by reducing 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; however, the 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.¹⁷⁶

Surgery may be considered for cases of noncomitant vertical deviations that have a significant torsional component. Newly acquired large-angle vertical deviations that cannot be resolved within 6 months may require surgery.²⁵ 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 and management of accommodative and vergence anomalies are 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 and management 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 the patient diagnosed with an accommodative anomaly or with an abnormally high AC/A ratio. 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 disorder 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 linear 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 relation 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. This guideline will use the term "vision therapy" rather than similar terminology such as "vision training" or "orthoptics."

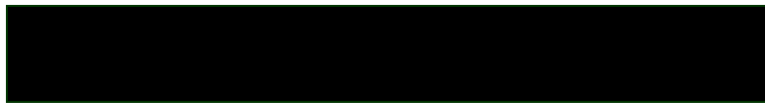


The first phase of therapy is to normalize accommodative and vergence amplitudes. Most clinicians use large targets to slowly change convergence and divergence demand. The patient is encouraged to exert maximum effort to increase his or her vergence amplitudes. Accommodative facility exercises are performed 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.¹⁵

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, subjects who underwent 8 weeks of vision therapy that consisted of push-ups and fusional amplitude therapy had improved vergence adaptation and fusional amplitudes.¹⁷⁵ Subsequent studies have demonstrated that vision therapy alters the fixation disparity curve (FDC), specifically, flattening the FDC and concurrently reducing the symptoms.¹²



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.¹⁷⁷ 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. If in-office therapy is not available, home computerized vision therapy with push-ups should be offered as an alternative.²⁰⁻²³

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 or CE. With myasthenia gravis, trial use of MESTINON® 60 mg (1–4 times daily) may be appropriate.* CE patients may benefit occasionally from the judicious use of Phospholine Iodide® 0.06% in 2.5% Neo-Synephrine® at bedtime.¹⁷⁸

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.

* 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.



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.¹³⁷ Therapy should focus on increasing accommodative amplitudes. Alternatively, plus lenses may be prescribed at near, if the patient is not interested in or is unable to meet the time requirements for vision therapy.¹⁷⁹

b. Ill-Sustained Accommodation

Plus lenses and vision therapy are effective in treating ill-sustained accommodation.¹⁷⁹ Vision therapy is used to improve the speed of the accommodative response, and it is generally the treatment of choice.

c. Accommodative Infacility

Plus lenses may be prescribed initially, but vision therapy is highly effective in correcting accommodative infacility.¹³⁵ 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.¹⁸⁰ 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. 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.^{16,181}

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). 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.^{27,30,68,69,145,182-192}

b. Divergence Excess

Among the variety of treatments for DE are occlusion, over-minus lenses, base-in prism, 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.¹⁹ When 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.

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.¹⁹



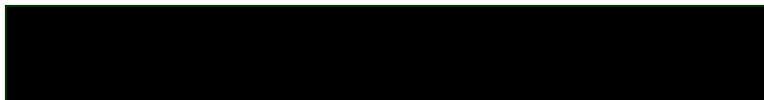
Table 6
Vision Therapy Success Rate for
Convergence Insufficiency Patients in Large Studies *

Author	Number	% Cured	% Improved	% Failed
Mayou ¹⁸⁷	87	92	6	2
Lyle and Jackson ¹⁸⁴	300	83	10	7
Mann ¹⁸²	142	68	30	3
Cushman and Burri ¹⁸³	80	66	30	4
Duthie ¹⁸⁸	123	88	6	6
Mayou ¹⁸⁶	420 [†]	72	7	5
Mayou ¹⁸⁶	100	93	5	2
Mellick ¹⁸⁵	88	77	10	12
Hirsch ⁶⁹	48	77	12	10
Passmore and MacLean ⁶⁸	100	82	18	0
Norn ³¹	65	10	60	30
Hoffman et al. ²⁷	17	94	6	0
Wick ¹⁴⁵	134	93	4	3
Dalziel ¹⁹⁰	100	84	9	7
Pantano ¹⁹¹	207	79	6	5
Daum ¹⁸⁹	110	41	56	3
Cohen and Soden ¹⁹²	28	96	4	0
Total	2,149	78[*]	15	5

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

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

^{*}Mean weighted cure rate; 2% did not complete orthoptics.



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.¹⁷⁸ 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, with caution, because wearing the prism at near might cause asthenopia.¹⁹³

Vision therapy is usually successful in patients with DI. If it 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

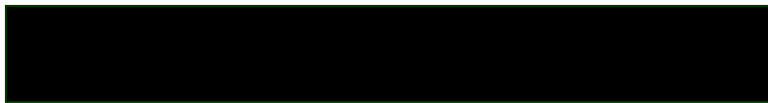
Patients with basic esophoria often have uncorrected hyperopia, and correcting the hyperopia may eliminate the deviation. If correcting the hyperopia does not eliminate the deviation, prismatic correction may be prescribed. Generally, the patient should be given the least amount of prism needed to eliminate 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 Heterophorias

Management of vertical heterophorias 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.¹⁶⁰ 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.¹⁹⁴



5. Patient Education

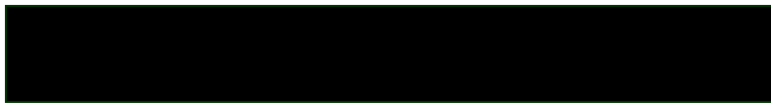
Patients should be advised that many accommodative and vergence anomalies are neuromuscular problems and not refractive problems. Thus, the most effective treatment relies on not only spectacles, but active vision therapy to eliminate neuromuscular dysfunction. Patients should also be told that treatment improves accommodative and vergence reflexes. Proper management usually results in improvement, due to changes in the slow vergence system.^{195,196}

6. Prognosis and Follow-up

When the patient is cooperative, the prognosis for the elimination of accommodative and vergence dysfunction is excellent (see Appendix Figure 7). 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 dysfunctions. The difficulty with lenses is that they do not affect either the fast vergence or slow vergence systems. Furthermore, the effectiveness of prisms and lenses may be reduced by adaptation.¹⁷¹ 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 as necessary. Many practitioners schedule a follow-up visit after the patient has worn his/her prescribed spectacles for 1 month and again 3–6 months later.

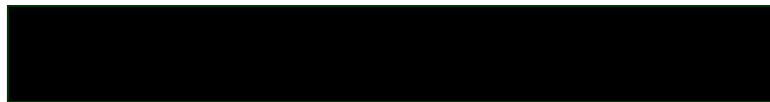




CONCLUSION

Accommodative and vergence dysfunction comprises a group 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 dysfunctions respond to the appropriate use of lenses, prisms, or vision therapy. It is medically necessary for the optometrist to diagnose the condition accurately, to discuss the diagnosis and the risks and potential benefits of existing treatment options with the patient, and to initiate treatment when appropriate. Management, 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.



III. REFERENCES

1. Flax N. General issues. In: Scheiman M, Rouse M. Optometric management of learning-related vision problems. St. Louis: CV Mosby, 1994:138-43.
2. Eames TH. Low fusional convergence as a factor in reading disability. *Am J Ophthalmol* 1934; 17:709-10.
3. Stein JF, Riddell PM, Fowler S. Disordered vergence control in dyslexic children. *Br J Ophthalmol* 1988; 72:162-6.
4. Buzzelli AR. Stereopsis, accommodative and vergence facility: do they relate to dyslexia? *Optom Vis Sci* 1991; 68:842-6.
5. Graybiel A, Jokl E, Trapp C. Russian studies in vision-related activity and sports. *Res Q Am Assoc Health Phys Educ* 1955; 26:480-5.
6. Olson CA. Relationship between psychological capacities and success in college athletes. *Res Q Am Assoc Health Phys Educ* 1956; 27:79-89.
7. Solomon H, Zinn WJ, Vacroux A. Dynamic stereoacuity: a test for hitting a baseball? *J Am Optom Assoc* 1988; 59:522-6.
8. Laby DM, Rosenbaum AL, Kirschen DG, et al. The visual function of professional baseball players. *Am J Ophthalmol* 1996; 122:476-85.
9. Sheedy JE, Parsons SD. The Video Display Terminal Eye Clinic: clinical report. *Optom Vis Sci* 1990; 67:622-6.
10. Bergqvist UO, Knave BG. Eye discomfort and work with visual display terminals. *Scand J Work Environ Health* 1994; 20:27-33.

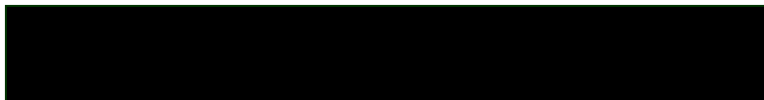


62 Accommodative and Vergence Dysfunction

11. Neugebauer A, Fricke J, Russmann W. Asthenopia: frequency and objective findings. *Ger J Ophthalmol* 1992; 1:122-4.
12. Gur S, Ron S. Does work with visual display units impair visual activities after work? *Doc Ophthalmol* 1992; 79:253-9.
13. Schor CM. Fixation disparity and vergence adaptation. In: Schor CM, Ciuffreda KJ, eds. *Vergence eye movements: basic and clinical aspects*. Boston: Butterworths, 1983:465-516.
14. Duke-Elder S. *The practice of refraction*, 5th ed. St. Louis: CV Mosby, 1949:141-51.
15. Cooper J. Accommodative dysfunction. In: Amos JF, ed. *Diagnosis and management in vision care*. Boston: Butterworths, 1987:431-59.
16. Rutstein RP, Daum KM, Amos JF. Accommodative spasm: a review of 17 cases. *J Am Optom Assoc* 1988; 59:527-38.
17. Duane A. A new classification of the motor anomalies of the eye based upon physiologic principles. Part 2. Pathology. *Ann Ophthalmol* 1897; 6:247-60.
18. Cooper J, Duckman R. Convergence insufficiency: incidence, diagnosis, and treatment. *J Am Optom Assoc* 1978; 49:673-80.
19. Cooper J, Medow N. Major review: intermittent exotropia; basic and divergence excess type. *Binocul Vis Eye Muscle Surg Q* 1993; 8(3 suppl):187-216.
20. Scheiman M, Wick B. *Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders*. Philadelphia: JB Lippincott, 1994:331.
21. Altizer LB. The nonsurgical treatment of exotropia. *Am Orthopt J* 1972; 22:71-6.



22. Scheiman M, Wick B. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. Philadelphia: JB Lippincott, 1994:270.
23. von Noorden G. Binocular vision and ocular motility, 5th ed. St. Louis: CV Mosby, 1996:129.
24. Scheiman M, Wick B. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. Philadelphia: JB Lippincott, 1994:310.
25. Amos JF, Rutstein RP. Vertical Deviations. In: Amos JF, ed. Diagnosis and Management in Vision Care. Boston: Butterworths, 1987:515-83.
26. Hokoda SC. General binocular dysfunctions in an urban optometry clinic. J Am Optom Assoc 1985; 56:560-2.
27. Hoffman L, Cohen AH, Feuer G. Effectiveness of non-strabismus optometric vision training in a private practice. Am J Optom Arch Am Acad Optom 1973; 50:813-6.
28. Ong E, Ciuffreda KJ. Accommodation, nearwork in myopia. Santa Ana, CA: Optometric Extension Program Foundation, 1997:97-142.
29. Prokopich CL, Bartlett JD, Jaanus SD. Ocular adverse effects of systemic drugs. In: Bartlett JD, Jaanus SD, eds. Clinical ocular pharmacology, 5th ed. Boston: Butterworths, 2008:701-59.
30. Kent PR, Steeve JH. Convergence insufficiency: incidence among military personnel and relief by orthoptic methods. Mil Surg 1953; 112:202-5.
31. Norn MS. Convergence insufficiency: incidence in ophthalmic practice results of orthoptic treatment. Acta Ophthalmol 1966; 44:132-8.

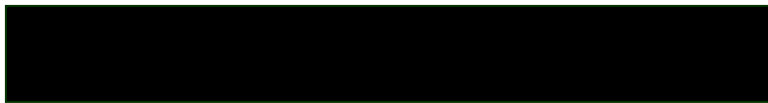


64 Accommodative and Vergence Dysfunction

32. Mahto RS. Eye strain from convergence insufficiency. *Br Med J* 1972; 2:564-5.
33. Rouse MW, Borsting E, Hyman L, et al. Frequency of convergence insufficiency among fifth and sixth graders. The Convergence Insufficiency and Reading Study (CIRS) group. *Optom Vis Sci* 1999;76:643-9.
34. Borsting E, Rouse MW, Deland PN, et al. Association of symptoms and convergence and accommodative insufficiency in school-age children. *Optometry* 2003;74:25-34.
35. Scheiman M, Gallaway M, Coulter R, et al. Prevalence of vision and ocular disease conditions in a clinical pediatric population. *J Am Optom Assoc* 1996; 67:193-302.
36. Daum KM. Characteristics of exodeviations: I. A comparison of three classes. *Am J Optom Physiol Opt* 1986; 63:237-43.
37. Scheiman M, Wick B. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. Philadelphia: JB Lippincott, 1994:309.
38. Bannister JM. A contribution to the study of the dynamics of the ocular muscles. *Ann Ophthalmol* 1898; 7:17-32.
39. Field PC. Phorometry of normal eyes in young male adults. *Arch Ophthalmol* 1911; 40:526-31.
40. Scheiman M, Wick B. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. Philadelphia: JB Lippincott, 1994:406.
41. Krohel GB, Kristan RW, Simon JW, Barrows NA. Posttraumatic convergence insufficiency. *Ann Ophthalmol* 1986; 18:101-4.

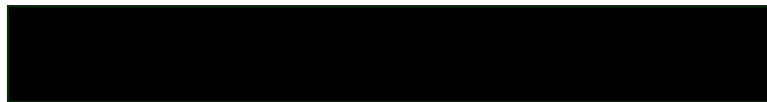


42. Carroll RP, Seaber JH. Acute loss of fusional convergence following head trauma. *Am Orthopt J* 1974; 24:57-9.
43. Anderson M. Orthoptic treatment of loss of convergence accommodation caused by road accidents ("whiplash" injury). *Br Orthopt J* 1961; 18:117-20.
44. Raskind R. Problems at the reading distance. *Am Orthopt J* 1976; 26:53-9.
45. Brown B. The convergence insufficiency masquerade. *Am Orthopt J* 1990; 40:94-7.
46. Oleszewski SC. Parkinson disease. In: Marks ES, Adamczyk DT, Thomann KH, eds. *Primary eyecare in systemic disease*. Norwalk, CT: Appleton & Lange, 1995:106-9.
47. Campbell FW, Westheimer G. Dynamics of accommodation responses of the human eye. *J Physiol* 1960; 151:285-95.
48. Fisher RF. Presbyopia and the changes with age in the human crystalline lens. *J Physiol* 1973; 228:765-79.
49. Takahashi E. Visual acuity. *Annu Rev Psychol* 1962; 16:359-78.
50. Campbell FW. Twilight myopia. *J Opt Soc Am* 1953; 43:925-6.
51. Knoll HA. A brief history of "nocturnal myopia" and related phenomena. *Am J Optom Arch Am Acad Optom* 1952; 29:69-81.
52. Lancaster W, Williams E. New light on the theory of accommodation with practical applications. *Trans Am Acad Ophthalmol Otolaryngol* 1914; 19:170-95.

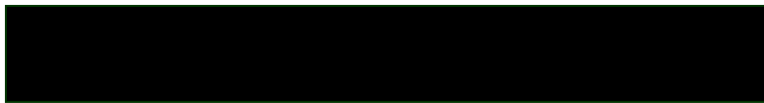


66 Accommodative and Vergence Dysfunction

53. Berens C, Stark E. Studies in ocular fatigue. IV. Fatigue of accommodation, experimental and clinical observations. *Am J Ophthalmol* 1932; 15:527-42.
54. Hofstetter HW. An ergographic analysis of fatigue of accommodation. *Am J Optom* 1943; 20:115-35.
55. Prakash G, Sharma N, Sharma P, et al. Accommodative spasm after laser-assisted in situ keratomileusis (LASIK). *Am J Ophthalmol* 2007;143:540; author reply 40-1.
56. Airiani S, Braunstein RE. Accommodative spasm after laser-assisted in situ keratomileusis (LASIK). *Am J Ophthalmol* 2006; 141:1163-4.
57. Sitole S, Jay WM. Spasm of the near reflex in a patient with multiple sclerosis. *Semin Ophthalmol* 2007; 22:29-31.
58. al-Qurainy IA. Convergence insufficiency and failure of accommodation following midfacial trauma. *Br J Oral Maxillofac Surg* 1995; 33:71-5.
59. Kowal L. Ophthalmic manifestations of head injury. *Aust N Z J Ophthalmol* 1992; 20:35-40.
60. Monteiro ML, Curi AL, Pereira A, et al.. Persistent accommodative spasm after severe head trauma. *Br J Ophthalmol* 2003; 87:243-4.
61. Sloane AE, Kraut JA. Spasm of accommodation. *Doc Ophthalmol* 1973; 34:365-9.
62. Porter JD, Baker RS, Ragusa RJ, Brueckner JK. Extraocular muscles: basic and clinical aspects of structure and function. *Surv Ophthalmol* 1995; 39:451-84.
63. Maddox EE. The clinical use of prism and the decentering of lenses. Bristol, England: John Wright & Sons, 1893:83-106.

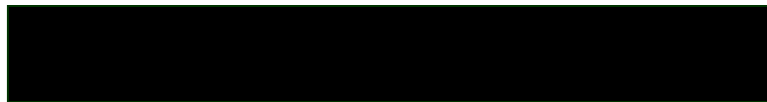


64. Guyton DL. The 10th Bielschowsky Lecture. Changes in strabismus over time: the roles of vergence tonus and muscle length adaptation. *Binocul Vis Strabismus Q* 2006; 21:81-92.
65. Burian HM, Spivey BE. The surgical management of exodeviations. *Am J Ophthalmol* 1965; 59:603-20.
66. Grisham JD. Visual therapy results for convergence insufficiency: a literature review. *Am J Optom Physiol Opt* 1988; 65:448-54.
67. Davies CE. Etiology and management of convergence insufficiency. *Am Orthopt J* 1956; 6:124-7.
68. Passmore JW, MacLean F. Convergence insufficiency and its management: an evaluation of 100 patients receiving a course of orthoptics. *Am J Ophthalmol* 1957; 43:448-56.
69. Hirsch MJ. A study of forty-eight cases of convergence insufficiency at the near point. *Am J Optom* 1953; 20:52-8.
70. Daum KM. Characteristics of convergence insufficiency. *Am J Optom Physiol Opt* 1988; 65:426-38.
71. Sheard C. Zones of ocular comfort. *Am J Optom* 1930; 7:9-25.
72. Bugola J. Hypoaccommodation and convergence insufficiency. *Am Orthopt J* 1977; 27:85-90.
73. Marran LF, De Land PN, Nguyen AL. Accommodative insufficiency is the primary source of symptoms in children diagnosed with convergence insufficiency. *Optom Vis Sci* 2006;83:281-9
74. Hiles DA, Davies GT, Costenbader FD. Long-term observations on unoperated intermittent exotropia. *Arch Ophthalmol* 1968; 80:436-42.



68 Accommodative and Vergence Dysfunction

75. Richards BW, Jones FR Jr, Younge BR. Causes and prognosis in 4,278 cases of paralysis of the oculomotor trochlear, and abducens cranial nerves. *Am J Ophthalmol* 1992; 113:489-96; comment 1992; 114:777-8.
76. Granet DB, Gomi CF, Ventura R, Miller-Scholte A. The relationship between convergence insufficiency and ADHD. *Strabismus* 2005;13:163-8.
77. Borsting E, Rouse M, Chu R. Measuring ADHD behaviors in children with symptomatic accommodative dysfunction or convergence insufficiency: a preliminary study. *Optometry* 2005; 76:588-92.
78. Schor C. Influence of accommodative and vergence adaptation on binocular motor disorders. *Am J Optom Physiol Opt* 1988; 65:464-75
79. Brautaset RL, Jennings JA. Horizontal and vertical prism adaptation are different mechanisms. *Ophthalmic Physiol Opt* 2005; 25:215-8.
80. Capobianco NM. The subjective measurement of the near point of convergence and its significance in the diagnosis of convergence insufficiency. *Am Orthopt J* 1952; 2:40-2.
81. Costenbader FD. The physiology and management of divergent strabismus. In: Allen JH, ed. *Strabismus Ophthalmic Symposium I*. St. Louis: CV Mosby, 1950:349-76.
82. Cooper J, Feldman J. Panoramic viewing, visual acuity of the deviating eye, and anomalous retinal correspondence in the intermittent exotropia of the divergence excess type. *Am J Optom Physiol Opt* 1979; 56:422-9.
83. Scheiman M, Wick B. *Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders*. Philadelphia: JB Lippincott, 1944:268.

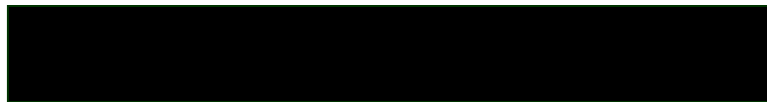


84. Tait EF. Some factors in the diagnosis and treatment of accommodative convergence excess. *Am Orthopt J* 1956; 6:90-7.
85. Taylor DM. Is congenital esotropia functionally curable? *Trans Am Ophthalmol Soc* 1972; 70:529-76.
86. von Noorden G. *Binocular vision and ocular motility*, 5th ed. St. Louis: CV Mosby, 1996:6.
87. Bennett GR, Blondin M, Ruskiewicz J. Incidence and prevalence of selected visual conditions. *J Am Optom Assoc* 1982; 53:647-56.
88. Nicholls JV. Ophthalmic disturbances. In: Keeney AH, Kennedy VT, eds. *Dyslexia: diagnosis and treatment of reading disorders*. St. Louis: CV Mosby, 1968:49-52.
89. Atzmon D. Positive effect of improving relative fusional vergence on reading and learning disabilities. *Binocul Vis Eye Muscle Surg Q* 1985; 1:39-43.
90. Rouse MW, Borsting EJ, Mitchell GL, et al. Validity and reliability of the revised convergence insufficiency symptom survey in adults. *Ophthalmic Physiol Opt* 2004; 24:384-90.
91. Feldman JM, Cooper J, Eichler R. The effect of stimulus parameters (size, complexity, depth, and line thickness) on horizontal fusional amplitudes in normal humans. *Binocul Vis Eye Muscle Surg Q* 1993; 8:23-30.
92. von Noorden GK. *Binocular vision and ocular motility*, 5th ed. St. Louis: CV Mosby, 1996:168.
93. Griffin J, Grisham JD. *Binocular anomalies. Diagnosis and vision therapy*. Boston: Butterworth-Heinemann, 1995:17-61.

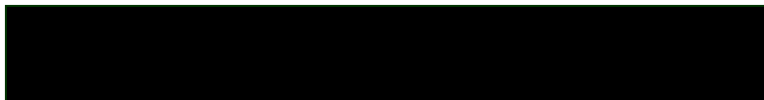


70 Accommodative and Vergence Dysfunction

94. Scheiman M, Gallaway M, Frantz KA, et al. Nearpoint of convergence: test procedure, target selection, and normative data. *Optom Vis Sci* 2003; 80:214-25.
95. Scheiman M, Wick B. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. Philadelphia: JB Lippincott, 1994:46-7.
96. Scheiman M, Mitchell GL, Cotter S, et al. A randomized clinical trial of vision therapy/orthoptics versus pencil pushups for the treatment of convergence insufficiency in young adults. *Optom Vis Sci* 2005; 82:583-95.
97. Alpern M. The zone of clear single vision at the upper levels of accommodation and convergence. *Am J Optom Arch Am Acad Optom* 1950; 27:491-513.
98. Ellerbrock VJ. Tonicity induced by fusional movements. *Am J Optom Arch Am Acad Optom* 1950; 27:8-20.
99. Daum KM. Vergence amplitude. In: Eskridge JB, Amos JF, Bartlett J, eds. *Clinical procedures in optometry*. Philadelphia: JB Lippincott, 1991:91-8.
100. Feldman J, Cooper J, Eichler R. Effect of various stimulus parameters on fusional horizontal amplitudes in normal humans. *Binocul Vis Eye Muscle Surg Q* 1993; 8:23-32.
101. Scheiman M, Wick B. Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders. Philadelphia: JB Lippincott, 1994:22.
102. Garcia R, Richmond J. Accommodative facility; a study of young adults. *J Am Optom Assoc* 1986; 53:821-4.
103. Levine S, Ciuffreda KJ, Selenow A, Flax N. Clinical assessment of accommodative facility in symptomatic and asymptomatic individuals. *J Am Optom Assoc* 1985; 56:286-90.

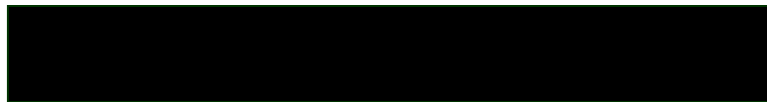


104. Hennessey D, Iosue RA, Rouse MW. Relation of symptoms to accommodative infacility of school-aged children. *Am J Optom Physiol Opt* 1984; 61:177-83.
105. Cooper J, Feldman J. Operant conditioning of fusional convergence ranges using random dot stereograms. *Am J Optom Physiol Opt* 1980; 57:205-13.
106. Cooper J, Feldman J. Random-dot-stereogram performance by strabismic, amblyopic, and ocular-pathology patients in an operant-discrimination task. *Am J Optom Physiol Opt* 1978; 55:599-609.
107. Cooper J. Clinical stereopsis testing: contour and random dot stereograms. *J Am Optom Assoc* 1979; 50:41-6.
108. Rutstein RP, Daum KM. *Anomalies of binocular vision: diagnosis and management*. St. Louis: CV Mosby, 1998:275-324.
109. Goss, DA. *Ocular accommodation, convergence, and fixation disparity: a manual of clinical analysis*, 2nd ed. Newton, MA: Butterworth-Heinemann, 1995:14.
110. Fry GA. The effect of age on the AC/A ratio. *Am J Optom Arch Am Acad Optom* 1959; 36:299-303.
111. Alpern M, Kincaid WM, Lubeck MJ. Vergence and accommodation. III. Proposed definitions of the AC/A ratios. *Am J Ophthalmol* 1959; 48:141-8.
112. Buzzelli AR. Vergence facility: developmental trends in a school age population. *Am J Optom Physiol Opt* 1986; 63:351-5.
113. Pellizzer S, Siderov J. Assessment of vergence facility in a sample of older adults with presbyopia. *Optom Vis Sci*. 1998; 75:817-21.

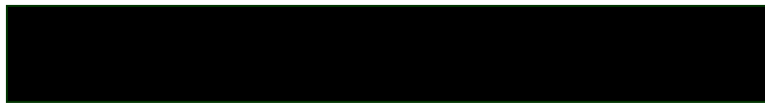


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114. Gall R, Wick B, Bedell H. Vergence facility: establishing clinical utility. *Optom Vis Sci* 1998; 75:731-42.
115. Gall R, Wick B. The symptomatic patient with normal phorias at distance and near: what tests detect a binocular vision problem? *Optometry* 2003; 74:309-22.
116. Rouse MW, London R, Allen DC. An evaluation of the monocular estimate method of dynamic retinoscopy. *Am J Optom Physiol Opt.* 1982 ; 59:234-9.
117. Jackson TW, Goss DA. Variation and correlation of clinical tests of accommodative function in a sample of school-age children. *J Am Optom Assoc.* 1991; 62:857-66.
118. Hofstetter HW. The zone of clear single binocular vision. Part I. *Am J Optom* 1945; 22:301-33.
119. Hofstetter HW. The zone of clear single binocular vision. Part II. *Am J Optom* 1945; 22:361-84.
120. Percival A. *The prescribing of spectacles*, 3rd ed. Bristol, England: John Wright & Sons, 1928.
121. Morgan MW. The clinical aspects of accommodation and convergence. *Am J Optom* 1944; 21:301-13.
122. Ogle KN, Mussey F, Prangen AD. Fixation disparity and the fusional processes in binocular single vision. *Am J Ophthalmol* 1949; 32:1069-87.
123. Ogle KN, Prangen AD. Observations on vertical divergences and hyperphorias. *Arch Ophthalmol* 1953; 49:313-34.
124. Mallet RF. Fixation disparity—its genesis in relation to asthenopia. *Ophthalmic Optician* 1974; 14:1159-68.

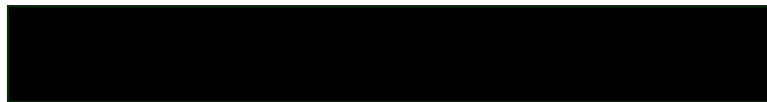


125. Carter DB. Fixation disparity and heterophoria following prolonged wearing of prisms. *Am J Optom Arch Am Acad Optom* 1965; 42:141-52.
126. Sheedy JE, Saladin JJ. Phoria, vergence and fixation disparity in oculomotor problems. *Am J Optom Physiol Opt* 1977; 54:474-8.
127. Sheedy JE, Saladin JJ. Association of symptoms with measures of oculomotor deficiencies. *Am J Optom Physiol Opt* 1978; 55:670-6.
128. Carr H, Allen JB. A study of certain relations of accommodation and convergence to the judgment of the third dimension. *Psychol Rev* 1906; 13:258-75.
129. Sisson ED. Voluntary control of accommodation. *J Gen Psychol* 1938; 18:195-8.
130. Marg E. An investigation of voluntary as distinguished from reflex accommodation. *Am J Optom Arch Am Acad Optom* 1951; 28:347-56.
131. Cornsweet TN, Crane HD. Training the visual accommodation system. *Vision Res* 1973; 13:713-5.
132. Morris CW. A theory concerning adaptation to accommodative impairment. *Optom Weekly* 1959; 59:255-62.
133. Russell GE, Wick B. A prospective study of treatment of accommodative insufficiency. *Optom Vis Sci* 1993; 70:131-5.
134. Daum KM. Predicting results in the orthoptic treatment of accommodative dysfunction. *Am J Optom Physiol Opt* 1984; 61:184-9.
135. Cooper J, Feldman JM, Selenow A, et al. Reduction of asthenopia after accommodative facility training. *Am J Optom Physiol Opt* 1987; 64:430-6.



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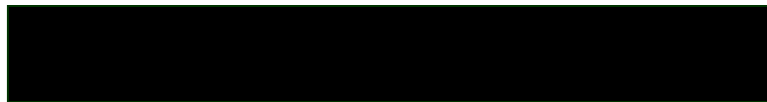
136. Randle RJ, Murphy MR. The dynamic response of visual accommodation over a seven-day period. *Am J Optom Physiol Opt* 1974; 51:530-44.
137. Liu JS, Lee M, Jang J, et al. Objective assessment of accommodation orthoptics. I. Dynamic insufficiency. *Am J Optom Physiol Opt* 1979; 56:285-94.
138. Bobier WR, Sivak JG. Orthoptic treatment of subjects showing slow accommodative response. *Am J Optom Physiol Opt* 1983; 60:678-87.
139. Ciuffreda KJ, Kenyon RV. Accommodative vergence and accommodation in normals, amblyopes, and strabismics. In: Schor CM, Ciuffreda KJ, eds. *Vergence eye movements: basic and clinical aspects*. Boston: Butterworths, 1983:101-73.
140. Grisham JD, Bowman MC, Owyang LA, Chan CL. Vergence orthoptics: validity and persistence of training effect. *Optom Vis Sci* 1991; 68:441-51.
141. Daum K. Double-blind placebo-controlled examination of timing effects in the training of positive vergences. *Am J Optom Physiol Opt* 1986; 63:807-12.
142. Daum KM, Rutstein RP, Eskridge JB. Efficacy of computerized vergence therapy. *Am J Optom Physiol Opt* 1987; 64:83-9.
143. Vaegan. Convergence and divergence show large and sustained improvement after short isometric exercise. *Am J Optom Physiol Opt* 1979; 56:23-33.
144. Goodson RA, Rahe AJ. Visual training effects on normal vision. *Am J Optom Physiol Opt* 1981; 58:787-91.
145. Wick B. Vision training for presbyopic nonstrabismic patients. *Am J Optom Physiol Opt* 1977; 54:244-7.



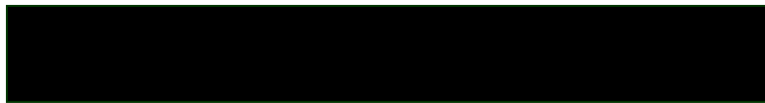
146. Birnbaum MH, Soden R, Cohen AH. Efficacy of vision therapy for convergence insufficiency in an adult male population. *J Am Optom Assoc* 1999;70:225-32.
147. Cooper J, Selenow A, Ciuffreda KJ, et al. Reduction in asthenopia in patients with convergence insufficiency after fusional vergence training. *Am J Optom Physiol Opt* 1983; 60:982-9.
148. Group CITTTS. Randomized Clinical Trial of Treatments for Symptomatic Convergence Insufficiency in Children. *Archives of Ophthalmology*. 2008;126(10):1336-1349.
149. Scheiman M, Mitchell GL, Cotter S, et al. Convergence Insufficiency Treatment Trial Study Group. A randomized clinical trial of treatments for convergence insufficiency in children. *Arch Ophthalmol* 2005; 123:14-24.
150. Convergence Insufficiency Treatment Trial Study Group. Long-term effectiveness of treatments for symptomatic convergence insufficiency in children. *Optom Vis Sci* 2009; 86:1096-103.
151. Cooper J, Feldman J. Reduction of asthenopia in non-strabismic binocular anomalies using computerized home therapy—HTS. *Optometry* 2009; 80:481-6.
152. Coffey B, Wick B, Cotter S, et al. Treatment options in intermittent exotropia: a critical appraisal. *Optom Vis Sci* 1992; 69:386-404.
153. Sanfilippo S, Clahane AC. The immediate and long-term effects of orthoptics in exodeviations. *Trans 1st Int Congr Orthoptists*. St. Louis: CV Mosby, 1968:300-12.
154. Sanfilippo S, Clahane AC. The effectiveness of orthoptics alone in selected cases of exodeviation: the immediate results and several years later. *Am Orthopt J* 1970; 20:104-17.



155. Mann D. The role of orthoptic treatment. *Br Orthopt J* 1947; 4:30-4.
156. Durran J. Orthoptic treatment of intermittent divergence strabismus of the divergence excess type. *Br Orthopt J* 1961; 18:110-3.
157. Cooper EL, Leyman IA. The management of intermittent exotropia. A comparison of the results of surgical and nonsurgical treatment. In: Moore S, Mein J, Stockbridge L, eds. *Orthoptics, past, present, and future*. New York: Stratton Intercontinental Medical Book Corp, 1976:563-8.
158. Chryssanthau G. Orthoptic management of exotropia. *Am Orthopt J* 1974; 24:69-72.
159. Daum KM. Divergence excess: characteristics and results of treatment with orthoptics. *Ophthalmic Physiol Opt* 1984; 4:15-24.
160. Goldrich SG. Optometric therapy of divergence excess strabismus. *Am J Optom Physiol Opt* 1980; 57:7-14.
161. Gallaway M, Scheiman M. The efficacy of vision therapy for convergence excess. *J Am Optom Assoc* 1997; 68:81-6.
162. Cooper J. Orthoptic treatment of vertical deviations. *J Am Optom Assoc* 1988; 59:463-8.
163. Robertson KW, Kuhn L. Effect of visual training on the vertical vergence amplitude. *Am J Optom Physiol Opt* 1985; 62:659-68.
164. Cohen AH, Rein L. The effect of head trauma on the visual system: the doctor of optometry as a member of the rehabilitative team. *J Am Optom Assoc* 1992; 63:530-6.
165. Cage I. Rehabilitative optometric management of a traumatic brain injury patient. *J Behav Optom* 1994; 5:143-8.

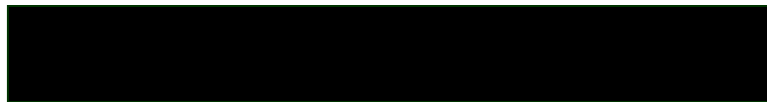


166. Worrell BE, Hirsch MJ, Morgan MW. An evaluation of prism prescribed by Sheard's criterion. *Am J Optom Arch Am Acad Optom* 1971; 48:373-6.
167. Grisham D, Buu T, Lum R, et al. Efficacy of prism prescription by the associated phoria criterion. *Optom Vis Sci* 1996; 73(12 suppl):158.
168. Scheiman M, Cotter S, Rouse M, et al.; Convergence Insufficiency Treatment Trial Study Group. Randomised clinical trial of the effectiveness of base-in prism reading glasses versus placebo reading glasses for symptomatic convergence insufficiency in children. *Br J Ophthalmol* 2005; 89:1318-23.
169. Teitelbaum B, Pang Y, Krall J. Effectiveness of base in prism for presbyopes with convergence insufficiency. *Optom Vis Sci* 2009; 86:153-6.
170. Ogle KN, Martens T, Dyer J. Oculomotor imbalance in binocular vision and fixation disparity. Philadelphia: Lea & Febiger, 1967:272-94.
171. Carter DB. Effects of prolonged wearing of prisms. *Am J Optom Arch Am Acad Optom* 1963; 40:265-73.
172. Cusick PL, Hawn HW. Prism compensation in cases of anisometropia. *Arch Ophthalmol* 1963; 25:651-8.
173. Pascal JI. Compensatory imbalance in correcting anisometropia. *World* 1949; 37:23-4.
174. Ellerbrock VJ, Fry GA. Effects induced by anisometric corrections. *Am J Optom* 1942; 19:444-59.
175. North RV, Henson DB. The effect of orthoptic treatment upon the vergence adaptation mechanism. *Optom Vis Sci* 1992; 69:294-9.

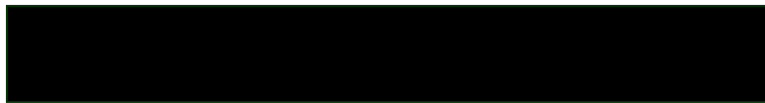


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176. Hawkeswood H. A case of surgery for convergence insufficiency. *Aust Orthopt J* 1970-71; 11:47-8.
177. Grisham JD. Treatment of binocular dysfunctions. In: Schor CM, Ciuffreda KJ, eds. *Vergence eye movements: basic and clinical aspects*. Boston: Butterworths, 1983:605-46.
178. Scheiman M, Wick B. *Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders*. Philadelphia: JB Lippincott, 1994:269-70.
179. Scheiman M, Wick B. *Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders*. Philadelphia: JB Lippincott, 1994:342.
180. Scheiman M, Wick B. *Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders*. Philadelphia: JB Lippincott, 1994:347.
181. Scheiman M, Wick B. *Clinical management of binocular vision: heterophoric, accommodative, and eye movement disorders*. Philadelphia: JB Lippincott, 1994:360.
182. Mann I. Convergence deficiency. *Br J Ophthalmol* 1940; 24:373-90.
183. Cushman B, Burri C. Convergence insufficiency. *Am J Ophthalmol* 1941; 24:1044-52.
184. Lyle K, Jackson S. *Practical orthoptics in the treatment of squint*. London: Lewis Co, 1937:203-7.
185. Mellick A. Convergence deficiency: an investigation into the results of treatment. *Br J Ophthalmol* 1950; 34:41-6.
186. Mayou S. The treatment of convergence deficiency. *Br Orthopt J* 1945; 3:72-82.

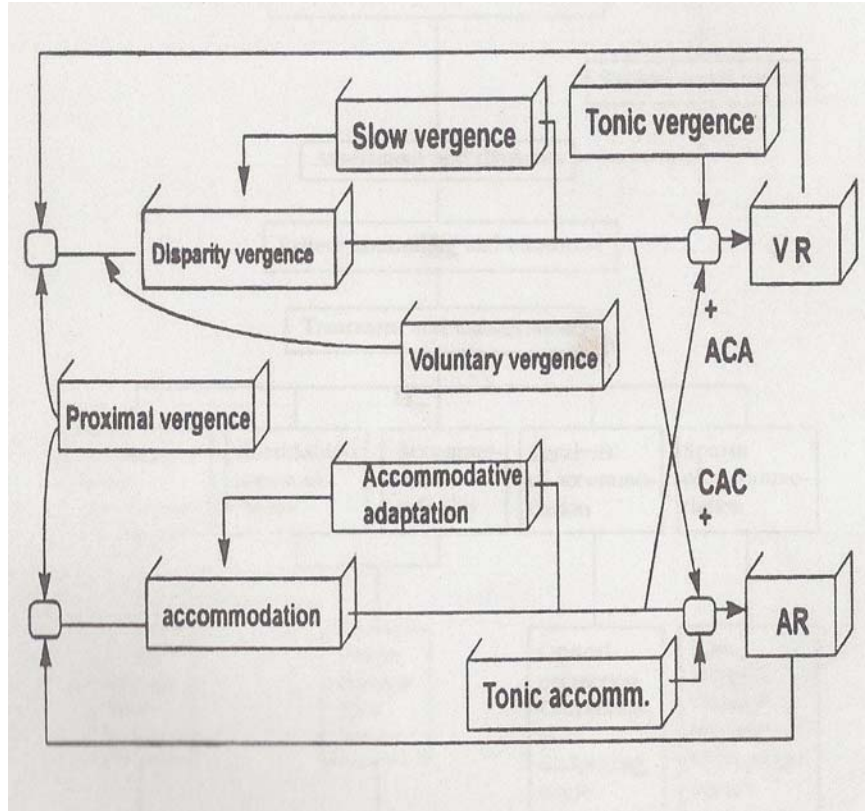


187. Mayou S. The treatment of convergence deficiency, 1933-1944. *Br J Ophthalmol* 1946; 30:354-70.
188. Duthie OM. Convergence deficiency. *Br Orthopt J* 1944; 2:38-41.
189. Daum KM. Convergence insufficiency. *Am J Optom Physiol Opt* 1984; 61:16-22.
190. Dalziel CC. Effect of vision therapy on patients who fail Sheard's criterion. *Am J Optom Physiol Opt* 1981; 58:21-3.
191. Pantano F. Orthoptic treatment of convergence insufficiency: a two year follow-up report. *Am Orthopt J* 1982; 32:73-80.
192. Cohen AH, Soden R. Effectiveness of visual therapy for convergence insufficiencies for an adult population. *J Am Optom Assoc* 1984; 55:491-4.
193. Saladin JJ. Horizontal prism correction. In: Cotter S, ed. *Clinical uses of prism—a spectrum of applications*. St. Louis: Mosby, 1995:109-48.
194. Rutstein R, Daum K, Cho M, Eskridge JB. Horizontal and vertical vergence training and its effect on vergences, fixation disparity, and prism adaptation: II. Vertical data. *Am J Optom Physiol Opt* 1988; 65:8-13.
195. Schor C. Introduction to the Symposium on Basic and Clinical Aspects of Vergence Eye Movements. *Am J Optom Physiol Opt* 1980; 57:535-6.
196. North RV, Henson DB. Effect of orthoptics upon the ability of patients to adapt to prism-induced heterophoria. *Am J Optom Physiol Opt* 1982 59:983-6.



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.

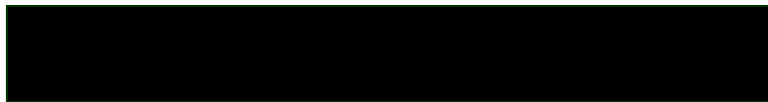


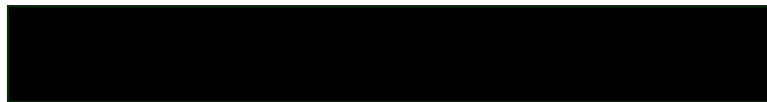
Figure 2
ICD-10-CM Classification
of Accommodative and Vergence Dysfunction

Presbyopia	367.4
Disorders of accommodation	367.5
Paresis of accommodation	367.51
Cycloplegia	
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	367.89
Drug-induced disorders of refraction and accommodation	
Toxic disorders of refraction and accommodation	
Unspecified disorder of refraction and accommodation	367.9
Visual disturbances	368
<i>Excludes: electrophysiological disturbances (794.11-794.14)</i>	
Subjective visual disturbances	368.1
Subjective visual disturbance, unspecified	368.10
Visual discomfort	368.13
Asthenopia	Photophobia
Eye strain	
Other visual distortions and entoptic phenomena	368.15
Photopsia	Visual halos
Refractive:	
diplopia	
polyopia	



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Diplopia	368.2
Double vision	
Other disorders of binocular vision	368.3
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	368.8
Blurred vision NOS	
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	378.8
<i>Excludes: nystagmus (379.50-379.56)</i>	
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	378.87
Skew deviation	

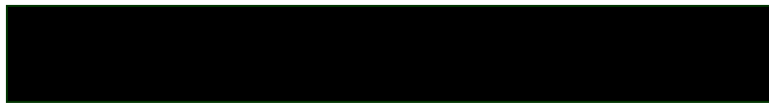


Figure 3
Potential Components of the Diagnostic Evaluation
for Accommodative and Vergence Dysfunction

- 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/associated phoria
 - 3. Distance fusional vergence amplitudes
 - 4. Vergence facility
 - 5. Accommodative lag

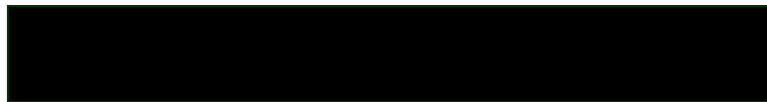


Figure 4
Standardized Convergence Insufficiency Symptom Survey

Clinician instructions: Read the following subject instructions and then each item exactly as written. If subject responds with “yes,” please qualify with frequency choices. **Do not give examples.**

Subject instructions: Please answer the following questions about how your eyes feel when reading or doing close work.

		Never	Infre- quently (not very often)	Some- times	Fairly often	Always
1.	Do your eyes feel tired when reading or doing close work?					
2.	Do your eyes feel uncomfortable when reading or doing close work?					
3.	Do you have headaches when reading or doing close work?					
4.	Do you feel sleepy when reading or doing close work?					
5.	Do you lose concentration when reading or doing close work?					
6.	Do you have trouble remembering what you have read?					
7.	Do you have double vision when reading or doing close work?					



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		Never	Infrequently (not very often)	Sometimes	Fairly often	Always
8.	Do you see the words move, jump, swim or appear to float on the page when reading or doing close work?					
9.	Do you feel like you read slowly?					
10.	Do your eyes ever hurt when reading or doing close work?					
11.	Do your eyes ever feel sore when reading or doing close work?					
12.	Do you feel a "pulling" feeling around your eyes when reading or doing close work?					
13.	Do you notice the words blurring or coming in and out of focus when reading or doing close work?					
14.	Do you lose your place while reading or doing close work?					
15.	Do you have to re-read the same line of words when reading?					
		__ × 0	__ × 1	__ × 2	__ × 3	__ × 4
	Total Score					

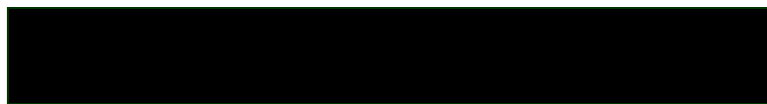


Figure 5
Optometric Management of the Patient
with Accommodative Dysfunction:
A Brief Flowchart

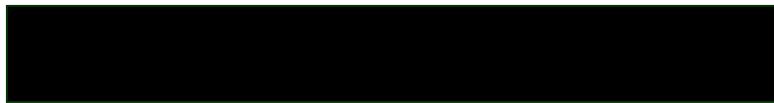
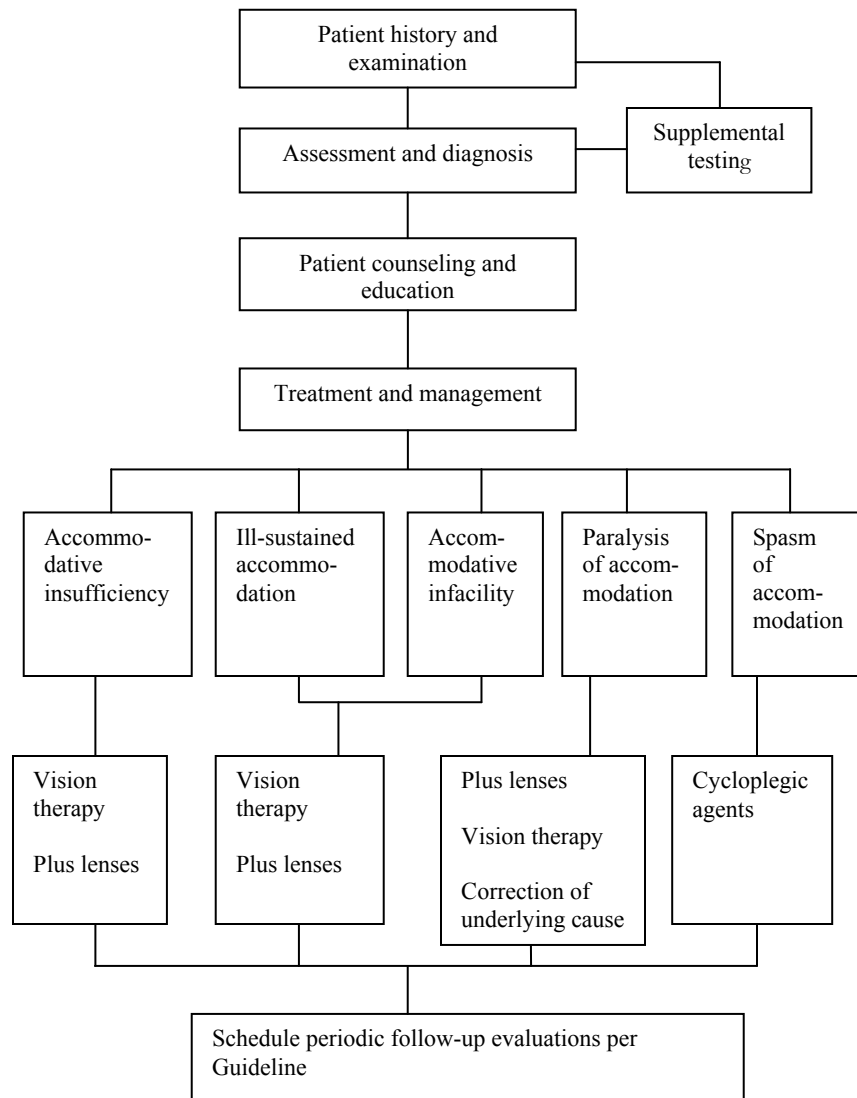


Figure 6
Optometric Management of the Patient
with Vergence Dysfunction:
A Brief Flowchart

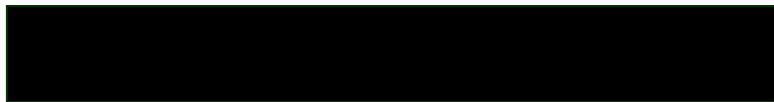
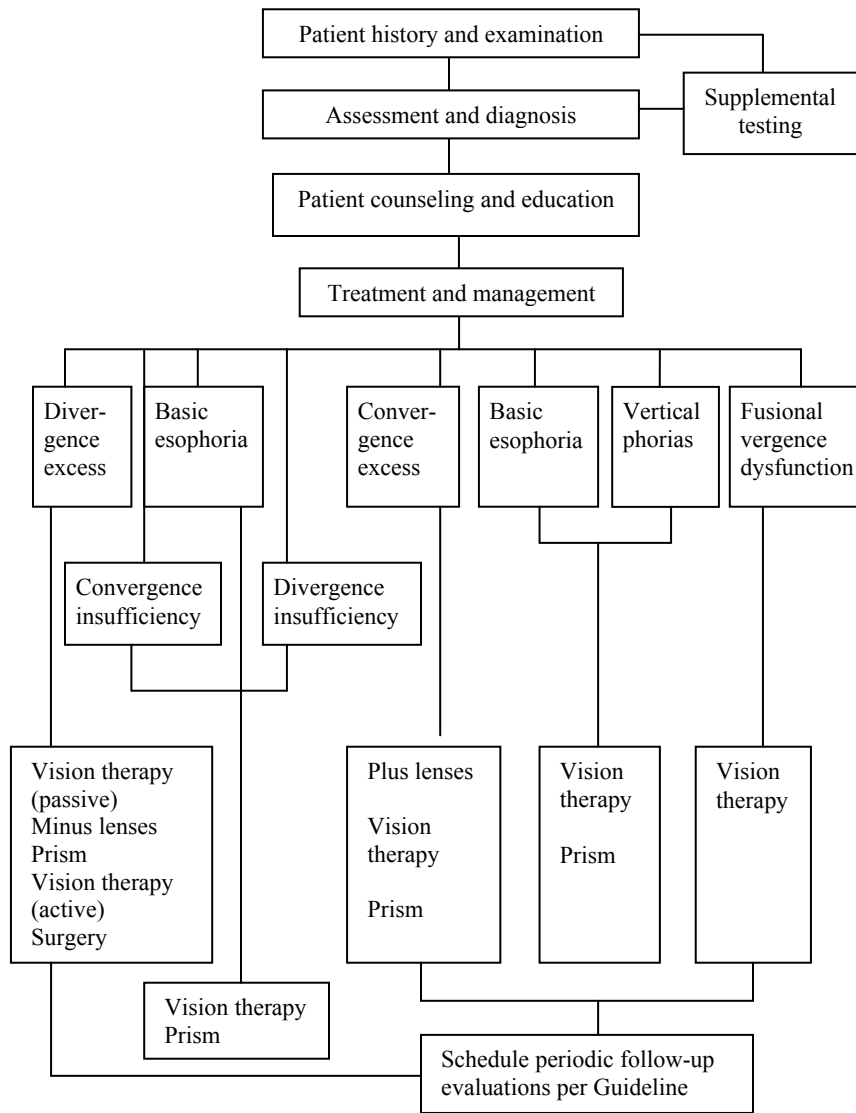
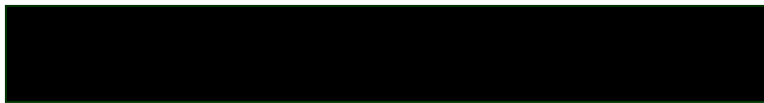


Figure
Frequency and Composition of Evaluation and Management

Dysfunction	No. of Evaluation Visits	Management Options
Convergence insufficiency	1	Vision therapy; prisms
Divergence excess	2	Vision therapy; prism; minus lenses
Basic exophoria	1	Prism; vision therapy
Convergence excess	1	Plus lenses; vision therapy
Divergence insufficiency	1–2	Vision therapy; prism
Basic esophoria	1	Prism; vision therapy
Fusional vergence dysfunction	1	Vision therapy
Vertical phorias	1–2	Prism; vision therapy

VT = vision therapy. MRI = magnetic resonance imaging.



7

Visits for Accommodative or Vergence Dysfunction

Prognosis	No. of Follow-up Visits	Management Plan*
Excellent	12-20	Provide in-office VT with supplement home VT; use prisms if patient is not able to participate in VT; educate patient.
Good	30	Provide active VT; use passive VT including occlusion, base-in prisms, lenses; surgery and minus lenses for noncommunicative patient; surgery if VT is not successful or the deviation is too large; educate patient.
Good	30	Treat near problems like CI; treat distance problems like DE; educate patient.
Excellent	5-25	Prescribe plus lens addition at near; provide VT for residual symptoms; prism; increase plus acceptance; use prism for the nonresponsive patient; educate patient.
Fair	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.
Good	20	Eliminate deviation by correcting hyperopia; prescribe prismatic correction; provide VT for residual asthenopia and to eliminate prism; educate patient.
Excellent	15-20	Provide VT balanced between convergence and divergence; treat abnormal accommodative system; educate patient.
Good	20	Correct vertical deviation with prism; if vergence dysfunction, proceed with horizontal vergence VT; educate patient.

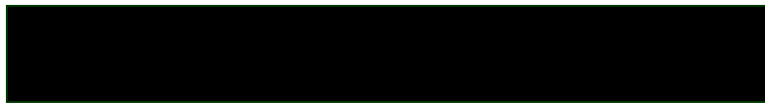
*See Guideline for other management strategies.



Figure

Dysfunction	No. of Evaluation Visits	Management Options
Accommodative insufficiency	1	Vision therapy; plus lenses
Ill-sustained accommodation	1	Vision therapy; plus lenses
Accommodative infacility	1	Plus lenses; vision therapy
Paralysis of accommodation	1	Optical correction
Spasm of accommodation	1–2	Plus lenses; vision therapy; cycloplegic drug

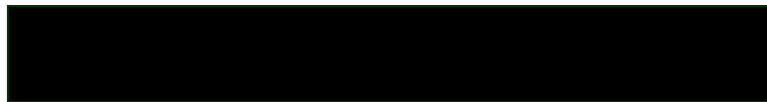
VT = vision therapy. MRI = magnetic resonance imaging.



7 (cont.)

Prognosis	No. of Follow-up Visits	Management Plan*
Excellent	10	Provide VT to build accommodative amplitudes and accommodative facility,
Excellent	10	Treat with VT or plus lenses; educate patient.
Excellent	10	Improve speed of accommodation with plus lenses initially; proceed with vision therapy; educate patient.
Poor	--	Determine underlying cause; correct with progressive lens when necessary; educate patient.
Fair	10	Begin with plus lenses and VT; if VT fails, use cycloplegic drug agent temporarily; educate patient.

*See Guideline for other management strategies.



Abbreviations of Commonly Used Terms

AA	Amplitude of accommodation
AC/A	Accommodative convergence/accommodation ratio
AI	Accommodative insufficiency
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
E	Esophoria at distance
E'	Esophoria at near
ET	Constant alternating esotropia at DV
E(T)	Intermittent esotropia at distance
FD	Fixation disparity
FDC	Fixation disparity curve
IPD	Interpupillary distance
LET	Unilateral esotropia (left)
LH	Hyperphoria (left)
LHT	Constant hypertropia (left)
LH(T)	Intermittent hypertropia (left)
LXT	Unilateral exotropia (left)



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MEM	Monocular estimate (estimation) 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
RDS	Random dot stereogram
RET	Unilateral esotropia (right)
RH	Hyperphoria (right)
RHT	Constant hypertropia (right)
RH(T)	Intermittent hypertropia (right)
RXT	Unilateral exotropia (right)
SNR	Spasm of the near reflex
X	Exophoria at distance
X'	Exophoria at near
XT	Constant alternating exotropia at distance
X(T)'	Intermittent exotropia at near



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.

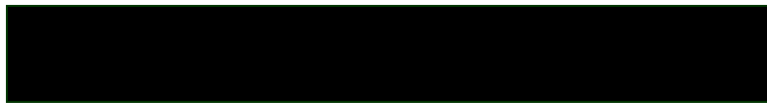
Amplitude of accommodation (AA) The difference between the farthest point and the nearest point of maximum accommodation denoted by first sustained blur with respect to the spectacle plane, the entrance pupil, or some other reference point of the eye, expressed in diopters.

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.

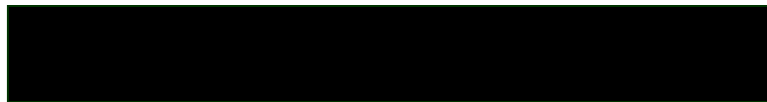
Esophoria, basic Vergence position of the eyes in which the two eyes' lines of sight cross closer to the patient than the object of regard when binocular fusion is disrupted, the magnitude of the deviation being the same at both far and near fixation distances.

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.

Sources:

Cline D, Hofstetter HW, Griffin JR. Dictionary of visual science, 4th ed. Radnor, PA: Chilton, 1989.

Grosvenor TP. Primary care optometry. Anomalies of refraction and binocular vision, 3rd ed. Boston: Butterworth-Heinemann, 1996:575-91.

