The efficacy of optometric vision therapy

The 1986/87 Future of Visual Development/Performance Task Force[a]

The purpose of this paper is to offer supporting documentation for the efficacy and validity of vision therapy for modifying and improving vision functioning.

Optometry is an independent primary health care profession. Its scope of practice includes the prevention and remediation of disorders of the vision system through the examination, diagnosis, treatment, and/or management of visual efficiency and eye health as well as the recognition and diagnosis of related systemic manifestations, all of which are designed to preserve and enhance the quality of our lives and environment.

Optometrists examine the eyes and related structures to determine the presence of vision problems, eye disease, and other abnormalities. They gather information on the vision system during the optometric examination, diagnose any conditions discovered, and prescribe individual or combinations of interventions such as corrective lenses, prescription drugs, contact lenses, and vision therapy.

The American Optometric Association considers vision therapy an essential and integral part of the practice of optometry.[1] Forty-three states specifically describe vision training, orthoptics, or some synonym in their definitions of the profession of optometry. The Institute of Medicine of the National Academy of Sciences,[2] the Dictionary of Occupational Titles of the Employment and Training Administration,[3] the U.S. Public Health Service,[4] the U.S. Dept. of Labor, Employment and Training Administration,[5] the National Center for Health Statistics,[6] the Bureau of Labor Statistics,[7] the Dept. of Health and Human Services,[8] and the Association of Academic Health Centers[9] all include vision therapy in their definitions of the profession of optometry.

The theory and procedures underlying the diagnosis and management of vision disorders are taught in all the schools and colleges of optometry.[9] In addition, the National Board of Examiners in Optometry[10] and the majority of the various state licensing agencies examine applicants for their theoretical and clinical knowledge in vision therapy.

What is vision therapy/ visual training?

Vision therapy (also called vision training, orthoptics, eye training, and eye exercises) is a clinical approach for correcting and ameliorating the effects of eye movement disorders, non-strabismic binocular dysfunctions, focusing disorders, strabismus, amblyopia, nystagmus, and certain visual perceptual (information processing) disorders. The practice of vision therapy entails a variety of non-surgical therapeutic procedures designed to modify different aspects of visual function.[11] Its purpose is to cure or ameliorate a diagnosed neuromuscular, neurophysiological, or neurosensory visual dysfunction.

Vision therapy typically involves a series of treatments during which carefully planned activities are carried out by the patient under professional supervision in order to relieve the visual problem. The specific procedures and instrumentation utilized are determined by the nature and severity of the diagnosed condition. Vision therapy is not instituted to simply strengthen eye muscles, but rather is generally done to treat functional deficiencies in order for the patient to achieve optimal efficiency and comfort.

The treatment may appear to be relatively uncomplicated, such as patching an eye as part of amblyopia therapy. Or, it may require complex infrared sensing devices and computers which monitor eye position and provide feedback to the patient to reduce the uncontrolled jumping of an eye with nystagmus. Treatment of strabismus, or turned eye, can involve complex optical and electronic instruments or such simple devices as a penlight or a mirror. The particular procedures and instruments are dependent on the nature of the visual dysfunction and the doctor's clinical judgment.

Who can benefit?

Vision therapy is utilized for conditions which include oculomotor dysfunctions, non-strabismus binocular coordination problems, accommodative disorders, strabismus, amblyopia, and nystagmus.

These disorders and dysfunctions have a prevalence rate second only to refractive conditions, such as myopia and hyperopia, and are far greater than most ocular diseases.[12-16] Graham[17] reports overt strabismus in almost 4% of over

4,000 school children. Among clinical cases, Fletcher and Silverman[18] found 8% of 1,100 to be strabismic. Other studies have generally found rates between these two levels.[19]

The reported prevalence of amblyopia varies somewhat depending upon the specific criteria used, with low estimates at approximately 2%,[20] and ranging up to 8.3% in the Rand HIE report,[21] and also in the study by Ross, Murray and Steed.[22] The National Society to Prevent Blindness estimates 127,000 new cases of amblyopia per year in the United States.[23]

Non-strabismic binocular coordination anomalies have an even higher incidence. Convergence insufficiency is reported in 15% of adults by Duke-Elder.[24] Graham[15] reports high heterophorias in over 13%, while Hokoda[25] found fusion or accommodative problems in 21% of a non-presbyopic clinical population. The recently developed New York State Vision Screening Battery probes oculomotor, binocular, accommodative, and visual perceptual function. Testing of 1,634 children with this battery revealed a failure rate of 53%.[27]

When "special" populations are considered, the incidence of ocular coordination and visual processing problems becomes very high. Among children who are reading disabled, as many as 80% show deficiency in one or more basic visual skills.[26] Grisham[28] has recently reported that children with reading problems showed greater than a 50% prevalence of visual deficiencies in accommodation, fusional vergence or gross convergence, compared to their normally achieving peers. Cerebral palsied patients show an incidence of strabismus as high as 50%.[29,30]

The hearing impaired,[31,32] emotionally impaired,[33] and developmentally disabled[34,35] also demonstrate unusually high prevalence rates of visual problems. This is of particular importance because almost 11% of the school population has been identified as having one of the above handicapping conditions.[36]

Our culture continues to foster higher educational standards and produces work related tasks which are increasingly visually demanding. This is evident in the difficulties encountered by video display terminal (VDT) operators. A majority of surveys have shown that more than 50% of VDT workers report they experience some type of ocular discomfort or blurring.[37,38] The National Academy of Sciences[39] concluded that the oculomotor and binocular vision changes noted at video display terminals are similar to those that occur during standard nearpoint tasks.

What are oculomotor skills and oculomotor dysfunctions?

Clear vision occurs when a precisely focused image of the object of regard is centered on the fovea and when accurate eye movements maintain this relationship. The components of the oculomotor or eye movement system include fixations, vestibular and optokinetic movements, saccades, and pursuit movements.[40]

Each one of the components has its own distinct and different neuroanatomical substrate and functional neurophysiology.[41] There are times when several components interact. An example of this occurs when the pursuit system interacts with other systems to create the ocular stabilization or position maintenance system[42] to hold the eyes steady.

Nystagmus, a to-and-fro involuntary movement of the eyes, is caused by disturbances in the mechanisms that hold images steady (position maintenance) and may be exhibited in over a dozen different clinical patterns of movement.[43] This loss of ability to maintain central fixation and eye position with the foveal area is one of the characteristics of pathological nystagmus.[40]

Patients with amblyopia represent another class of individuals with impaired central fixational ability. Lack of ability to steadily fixate with the fovea is accompanied by reduced visual acuity and is commonly observed in anisometropic and especially strabismic amblyopes. Their characteristics have been described extensively.[44–46] Abnormal saccadic and pursuit eye movements are exhibited in strabismic amblyopes and appear to be related to dysfunctions in the monocular motor control center for position maintenance.[47–49]

When nystagmus or nystagmoid movements are present, the clinical identification of fixation pauses, regressions, and progressions during reading become difficult. The erratic eye movements interfere with efficient visual information processing.[50,51]

During reading, the function or behavior of the eye movement system involves more than the physical movement of the eyes alone. This functional component involves the integration of the eye movements with higher cognitive processes including attention, memory, and the utilization of the perceived visual information.[52]

Clinical and research evidence strongly suggest that many children and adults who have difficulty with both reading and non-reading visual information processing tasks exhibit abnormal eye movements.[53–66]

Numerous studies[67–69] indicate that there is a distinct difference in the oculomotor (eye movement) patterns between children with reflective strategies or styles of processing visual information and those with impulsive styles. There is evidence that children and adults with attentional difficulties and hyperactivity exhibit inefficient eye movement patterns that interfere with visual information processing.[70–74]

In summary, there are a variety of dysfunctions in the oculomotor system. Their clinical manifestations are quite often related to problems with functional visual performance and the efficient processing of information.

Can eye movement skills be modified?

Improvement in eye movement control and efficiency has been reported in individual case studies following vision therapy.[75–77]

Wold et al[78] reported on 100 consecutive optometric vision therapy patients whose eye movement skills were rated on the Heinsen-Schrock Performance Scale.[79] This is a 10-point observational scale for scoring saccadic and pursuit eye movement performance. Only 6% of the children passed the eye movement portion prior to therapy. Post-therapy re-evaluation revealed that 96% of the children were able to pass.

Heath[80] discussed the influence of ocular-motor proficiency on reading. Sixty third and fourth graders who scored below the 40th percentile on the Metropolitan Reading Test and failed the ocular pursuit subtest of the Purdue Perceptual Motor Survey were divided into control and experimental groups. Results of the study showed significant improvement in ocular pursuit ability for the experimental compared to the control group. In addition, those children receiving therapy were found to score significantly better on a post-test of the Metropolitan Reading Test.

Fujimoto et al[81] compared the use of various techniques for saccadic fixation training. In this controlled clinical trial, both of the treated groups showed a statistically significant improvement in speed and accuracy of eye movements compared to an untreated control group.

A controlled study of pursuit eye movements was conducted by Busby[82] in an enhancement program for special education students. The subjects were rated on their ability to maintain fixation on a moving target. The rating procedure was shown to have a high interrater reliability. The results showed statistically significant improvement by the experimental group in pursuit eye movement and persistence of the therapeutic effect on re-testing at a 3 month interval after conclusion of the therapy.

Punnett and Steinhauer[83] conducted a controlled study investigating the effects of eye movement training with and without feedback and reinforcement. There were clear post-training differences between the eye movement skills of the control and experimental group of reading disabled students. This demonstrated that the use of reinforcement in training oculomotor facility can improve those skills. There was an improvement in reading performance following the oculomotor training as well. Similar results demonstrating the trainability of eye movements have been obtained in studies employing behavior modification and reinforcement.[84,85]

Modifying and improving the oculomotor ability to maintain central fixation and eye position in nystagmus patients has been reported over the years in various studies.

The use of after-images[86,87] and Emergent Textual Contour training to provide visual biofeedback regarding eye position and stability has had some success in improving fixational ability. Orthoptics, as well as verbal feedback techniques, have helped some patients in reducing their nystagmus.[88–90]

More recently, the application of eye movement auditory biofeedback in the control of nystagmus has shown positive results. Ciufredda et al[91] demonstrated a significant reduction in the amplitude and velocity of eye movements in congenital nystagmus patients. Vision was improved, and positive cosmetic and psychological changes were reported as well. Abadi et al[92] reported reduction in nystagmus and improvement of contrast sensitivity after auditory biofeedback training. In addition to nystagmus, the use of auditory biofeedback has been successfully used in expanding the range of eye movement in gaze limitations.[93]

There is evidence[94] that large and unsteady eye movements occur in the eyes of amblyopic patients during attempted monocular fixation. A number of studies report the successful treatment of amblyopia resulting in improved vision and oculomotor control.[95–98] Occlusion therapy, a passive procedure, has been a standard and relatively successful approach for many years.[99–111] However, there are individuals that either do not or cannot respond to occlusion therapy. There is evidence that occlusion with active vision therapy is more effective than occlusion alone.[112] Pleoptics[113,114] is an active vision therapy procedure in which patients receive visual feedback about their position of fixation and direction of gaze. These procedures are designed to correct the positional fixation problem and thereby improve the vision of the patient. Pleoptics has been used successfully in treating eccentric fixation in individuals not responding to regular occlusion therapy.[115–118]

Vision therapy for amblyopia incorporates a broad spectrum of procedures, including occlusion techniques, pleoptic techniques, and visual-motor spatial localization feedback techniques using after-images and entoptic phenomena[45,79] with a high success rate.[119–124]

The question of age and its influence on the efficacy of amblyopia therapy has been addressed in a number of studies and reviews. These indicate that a significant improvement in oculomotor and vision function can be achieved even in adulthood.[125] It is clear from the evidence that amblyopia and its oculomotor components can be successfully treated with occlusion and active vision therapy for a wide range of patients of all ages.

Studies have demonstrated that it is possible to change and improve inefficient and inadequate visual information processing strategies and visual attention patterns. Many of these changes have been accompanied by enhanced eye movements.[126–138]

A number of techniques used to improve these poor visual scanning and attention problems in children and adults, e.g., tachistoscopic procedures, pursuit and fixation activities, and eye-hand coordination techniques have been described and utilized professionally for many years.[79,139–143]

What are accommodative dysfunctions and their remediation?

Accommodative (focusing) dysfunctions have been described in detail[144–146] in numerous sources and are clinically classified as accommodative spasm, accommodative infacility, accommodative insufficiency, and ill-sustained accommodation. There are also clearly defined syndromes associated with accommodative dysfunctions.[147–155]

The literature discusses many symptoms common to accommodative dysfunctions as a group. These have been described as reduced nearpoint acuity, a general inability to sustain nearpoint activity, asthenopia, excessive rubbing of the eyes, headaches, periodic blurring of distance vision after prolonged near activities, periodic double vision at near, and excessive fatigue at the end of the day.[152,154,156–160]

The efficacy of applying vision therapy procedures in improving accommodative functioning has considerable basic science and clinical research support. Studies have shown that accommodative findings, although under autonomic nervous system control, can respond to voluntary command[161–163] and can be conditioned.[164] These studies demonstrate that voluntary control of accommodation can be controlled, trained, and transferred.

Once pathological or iatrogenic causes have been eliminated, the treatment of accommodative deficiencies includes plus lenses for near work and vision therapy aimed at improving the functioning of the accommodative mechanism.[165–168] Levine et al[156] established baseline statistics for diagnostic accommodation findings which differentiate symptomatic from asymptomatic patients. Their findings were in close agreement with a similar study by Zellers and Rouse.[152] The significant element of these studies is the relationship between symptoms and inadequate accommodative facility.

Wold[78] reported on 100 children who had undergone accommodative vision therapy procedures. These clinically selected cases showed an 80% rate of improvement in accommodative amplitude and 76% in accommodative facility using a pre- and post-treatment ordinal criterion referenced scaling method. These results are similar to those reported by Hoffman and Cohen[168a] in which 70 patients were successfully treated for accommodative insufficiency and infacility based on clinical findings.

Liu et al[169] investigated accommodative facility disorders by objective laboratory methods using a dynamic optometer with an infrared photomultiplier. They objectively identified the dynamic aspects of the accommodative response that were improved by vision therapy. Young adults with symptoms related to focusing difficulties were treated by procedures commonly used in orthoptic or vision therapy practice. Significant improvement in their focus flexibility occurred and these changes correlated with marked reduction or elimination of symptoms. Standard clinical measures of accommodative facility were found to correlate well with the more objective measures.

Bobier and Sivak[170] replicated the work of Liu et al[169] using a greater degree of recording precision with a dynamic photorefractor (television camera and monitor with light-emitting diodes). They found no evidence of regression in improved focusing flexibility during an 18-week interval after cessation of training. The subjects' symptoms also abated as accommodative function normalized. Hung et al[171] demonstrated the efficacy of accommodation, vergence, and accommodative vergence orthoptic therapy using a dynamic binocular simulator. This experiment objectively validated optometric vision therapy procedures through use of photoelectric eye movement recording systems and an optometer.

There is a higher prevalence of accommodative insufficiencies and infacilities in persons with cerebral palsy.[172] Duckman demonstrated that accommodative abilities can be modified and improved in a cerebral palsy population using vision therapy techniques.[173,174]

Since accommodative changes take place when looking from near to far and back to near, Haynes and McWilliams[175] investigated the effects of training this near-far response on school age and college students. Their results indicate that this near-far response ability is trainable and can be improved with vision therapy.

Weisz[176] has shown that improvement in accommodative ability transfers to improvement in near point task performance. In a double blind clinical study following vision therapy, her experimental group was found to improve significantly in accuracy of performance on a Landolt-C resolution task as compared with the controls.

Hoffman[160] investigated the impact of accommodative deficiencies on visual information processing tasks. He compared the results of vision therapy for the accommodative problems in an experimental and control group of school age children. This study indicated that by improving accommodative skills, there was a concomitant improvement in his subject's visual perceptual skills.

Recently, in a detailed series of analyses involving retrospective studies, Daum[177–180] investigated the full range of accommodative disorders. He used a stepwise discriminant analysis of regression variables in patient care records, to establish a model to determine the length of treatment necessary, and to predict the success of treatment for accommodative disorders.

In conclusion, these studies demonstrate that accommodative disorders can cause significant discomfort, inefficiency or avoidance of nearpoint tasks. They further demonstrate that when diagnosed and treated appropriately, these dysfunctions may be ameliorated or eliminated through vision therapy.

What are binocular vision disorders and their remediation?

Normal and efficient binocular vision is based on the presence of motor alignment and coordination of the two eyes and sensory fusion. The range of binocular disorders extends from constant strabismus with no binocular vision present to non-strabismic binocular dysfunctions, e.g., convergence insufficiency.[146]

The first category is non-strabismic binocular disorders. Standard techniques and diagnostic criteria in the assessment of the vergence system and binocular sensory fusion ability have been described in detail elsewhere.[181–185]

Patients exhibiting non-strabismic anomalies of binocular vision quite often report feeling ocular discomfort and asthenopia.[186] Some of the patient complaints include eyestrain, soreness of the eyes, frontal and occipital headaches, and ocular fatigue which result in an aversion to reading and studying.[187,187a]

Vision therapy has long been advocated as a primary intervention technique for the amelioration of non-strabismic anomalies of binocular vision.[188–194] Suchoff and Petito[146] have concluded that vision therapy for these conditions is directed toward several therapeutic goals: First, to increase the efficiency of the accommodative system so as to facilitate a more effective interaction between this system and the vergence system. Second, to maximize the functioning of the fusional vergence system (i.e., divergence and convergence) and the binocular sensory system. Since the training of accommodation has been covered in the previous section, the remainder of this section will be devoted to the evidence of the modifiability of the vergence system.

Clinical vision therapy procedures are intended to improve the patient's ability to compensate for fusional stress which may result in asthenopia, headache, and/or diplopia. A number of studies will be reviewed showing that improvements can be made in fusional vergence skills by vision therapy procedures.

The clinical assumption that fusional vergences can be trained is not a new one. Over 50 years ago, Berens et al advocated the use of this aspect of orthoptics for all non-strabismic anomalies of binocular vision.[195] Within the past several years a number of investigators have sought to determine experimentally whether the clinical assumption of the trainability of the vergence system was a valid one.

Daum[196] prospectively studied a group of 35 young adults. The results of daily vision therapy showed statistically significant improvement in convergence ranges. The gains persisted on post-testing 24 weeks after completion of the therapy program. The conclusion was that relatively short periods of training can provide long-lasting increases in vergence ability.

Daum[197] conducted a retrospective study of 110 patients who received treatment for convergence insufficiency. The patients were classified according to the effectiveness of the treatment program into total success, partial success or no success categories. Post training diagnostic findings and changes in patient symptomatology were used to define the classification categories. A comparison of pre- and post-training findings revealed statistically significant improvement. In a companion report,[198] a portion of the above data[197] was used to investigate and identify which of 14 common diagnostic measures best predicted the success of the vision training program. These measures were 75% accurate in predicting efficacy of the vision therapy program.

Another study[199] utilized tonic and phasic vergence training and demonstrated impressive changes in convergence and divergence abilities. The 34 subjects were randomly assigned in a double crossover design, wherein subjects served as their own controls, and learning effects were controlled.

In another study, Veagan used a motor-driven prism stereoscope (ophthalmic ergograph) to train divergence and convergence.[200] Forty-seven adults were divided into convergence and divergence experimental and control groups. The findings led Veagan to conclude that sustained divergence and convergence training showed large and significant immediate and stable improvement in the trained vergence ranges of the experimental groups.

Vaegan and McMonnies[201] utilized a recording device that measured eye movements during vergence activity. They were able to objectively demonstrate that convergence training with prism-induced changes resulted in sustained improvement of convergence ability. In a companion study, Vaegan[202] demonstrated substantial long-lasting gains in convergence and divergence ability from both tonic and phasic vergence training.

Pantano[203] studied over 200 subjects with convergence insufficiency who underwent vision therapy and evaluated them 2 years later. The majority remained asymptomatic with normal clinical findings. Those subjects who had learned to control convergence and accommodation together had the best success.

Grisham[204,205] used vergence latencies, velocity, and step vergence tracking rate by measuring them objectively with infra-red eye monitor recordings. He reported improved step vergence tracking after vision therapy of 4 to 8 weeks.

Cooper and Duckman, in their extensive review of convergence insufficiency, stated that 95% of the patients reported in these studies responded favorably to vision therapy for this binocular disorder.[206]

Cooper and Feldman[207] investigated the role and clinical use of operant conditioning in vision therapy based on random dot stereograms (RDS). They demonstrated that response-contingent positive reinforcement, immediate feedback, and preprogrammed systematic changes during discrimination learning improves convergence ability. Control and experimental groups were formed with subjects matched in baseline convergence ability and randomly assigned to each group. The convergence ranges of the experimental group improved significantly while there were little or no increases for the control group.

Cooper et al[208] conducted a controlled study of vision therapy and its relationship to symptomatology for a group of patients with convergence insufficiency. A vision therapy program of fusional vergence activities was administered in a matched-subjects control group crossover design to reduce placebo effects. They used a written assessment scale for rating asthenopia in terms of discomfort and/or fatigue, and conclusively demonstrated that the symptoms were eliminated or relieved. Clinical findings also improved, corroborating the subjective assessments.

Dalziel[209] reported on 100 convergence insufficiency patients who did not meet Sheard's criterion, and were given a program of vision therapy. After vision therapy, clinical findings were again assessed and 84% of the patients successfully met Sheard's criterion. Eighty-three percent of the patients reported they had symptoms of discomfort or loss of efficiency prior to treatment. Only 7% reported these symptoms after therapy. The post-training group who failed to meet Sheard's criterion correlated well with those still reporting subjective symptoms.

Wold[78] reported on the results of 100 patients who underwent vision therapy. Based on standard clinical tests, only 25% of the children had adequate binocular sensory fusion prior to vision therapy and 9% had adequate binocular fusional vergence. Post-training evaluation showed 96% had achieved appropriate sensory fusion findings and 75% demonstrated adequate fusional vergence ranges.

Wittenberg et al,[210] along with Saladin and Rick,[211] used slightly different techniques and demonstrated that stereopsis thresholds could be improved in normal subjects. In Dalziel's[212] study there was a statistically significant improvement in stereopsis after vision therapy.

Another category of binocular vision disorders is strabismus. Strabismus may be described as a misalignment of the eyes (referred to as crossed-eyes, eye turn, weak eye muscle, etc.). Many forms and variations of strabismus exist, depending upon direction and amount of the eye turn, the number of affected nerves or muscles, and the degree to which it is associated with reduced vision. The clinical characteristics and diagnostic criteria have been described in detail.[212-215]

Numerous comprehensive reviews and studies relating to the success of vision therapy for strabismus exist. Flom[216] reviewed studies and used detailed multifactorial analysis. This revealed an overall functional cure rate for strabismics receiving vision therapy of 50%, with esotropia less responsive than exotropia. Ludlam[217] evaluated a sample of 149 unselected strabismics who received vision therapy and determined a 73% overall success rate utilizing the rigorous criteria established by Flom.

In a longitudinal follow-up study of this population, Ludlam and Kleinman[218] found 89% of these patients had retained their functional cure (binocular vision present). The long-term overall success rate of vision therapy was calculated at 65%. If one adopts a less stringent definition of "success," such as the cosmetic criterion of "straight-looking eyes" employed in some less precise studies, the success rate increases to 96% of the re-analyzed population, or a 71% long-term success rate.

Flax and Duckman,[219] in their literature review of treatment for strabismus, found strong support for the efficacy of vision therapy for strabismus. They gathered data from numerous studies, each of which met rigorous criteria for success, and reported an overall success rate of 86%.

In a controlled study of 100 cases[220] Gillan reported that 76% of strabismic patients attained a cosmetic cure with orthoptics. None of those in the control group, treated with glasses alone, showed a spontaneous cure.

In a series of controlled studies conducted by Guibor,[221-223] 50% of the experimental group achieved alignment of the eyes with glasses and vision therapy (orthoptics) as compared with only 12.5% of the control group who received glasses without vision therapy.

More recently, Ziegler et al[224] conducted a literature review of the efficacy of vision therapy for strabismus. An important contribution is their comparative analysis of published papers using the functional cure criteria defined by Flom. They noted the study conducted by Etting[225] in which he reported a 65% overall success rate in patients with constant strabismus (57% of esotropes and 82% of exotropes), 89% success rate with intermittent strabismus (100% of esotropes and 85% of exotropes), and a 91% success rate when retinal correspondence was normal.

In a study designed to investigate the effectiveness of vision therapy utilizing computer generated stereo graphics for subjects with strabismus, Kertesz and Kertesz[226] reported a 74% success rate in 57 strabismics. They combined traditional vision therapy techniques with computer generated stimuli as successfully applied by Cooper[207] to the remediation of non-strabismic binocular vision anomalies. The functional cures obtained persisted on long-term follow-up visits for a period of up to 5 years.

Sanfilippo and Clahane[227] designed a prospective study of the results of orthoptic therapy for divergent strabismus (exotropia). Of the patients who completed the study, 64.5% attained a functional cure upon completion, and 51.7% retained this status on an average follow-up interval of 5 years and 4 months.

In two studies on the effectiveness of orthoptics (vision therapy) for intermittent and constant exotropes, Altizer[228] and Chryssanthou[229] found the majority of their patients had significant improvement in clinical findings as well as relief of symptoms.

Goldrich[230] reviewed records of patients completing a vision therapy program for exotropia of the divergence excess type. Of the patients reviewed, 71.4% attained a functional cure following approximately 5 months of standardized sequential therapy procedures used in-office as well as at home.

Several studies have applied biofeedback in vision therapy to assist in training patients to align their eyes.[231-236] The use of biofeedback to enhance traditional vision therapy, provide reinforcement, and increase motivation was supported in these studies.

Strabismic patients exhibiting esotropia with anomalous correspondence tend to be the most difficult to successfully treat. The use of more aggressive and sophisticated techniques for vision therapy has been reported with a better success rate for anomalous correspondence and esotropia than earlier studies.[237,238] In general, the treatment period tends to be longer for anomalous correspondence and esotropia than other types of strabismus.

Summary and conclusion

Vision is not simply the ability to read a certain size letter at a distance of 20 feet. Vision is a complex and adaptable information gathering and processing system which collects, groups, analyzes, accumulates, equates, and remembers information.

In this review, some of the essential components of the visual system and their disorders which can be physiologically and clinically identified. i.e., the oculomotor, the accommodative, and the fusional vergence systems have been discussed. Any dysfunctions in these systems, can lessen the quality and quantity of the initial input of information into the visual system.

Deficiencies in one or more of these visual subsystems have been shown to result in symptoms, such as blurred or uncomfortable vision or headaches, or behavioral signs such as rubbing of the eyes, eyes turning inward or outward, reduced job efficiency or reading performance, or simply the avoidance of near point tasks. In addition, these signs/symptoms may contribute to reducing a person's attention and interest in near tasks. The goal of vision therapy is to eliminate visual problems, thereby reducing the frequency and severity of the patient's signs and symptoms. Vision therapy should only be expected to be of clinical benefit to patients who have detectable visual deficiencies.

In response to the question, "How effective is vision therapy in remediating visual deficiencies?," it is evident from the research presented that there is sufficient scientific support for the efficacy of vision therapy in modifying and improving oculomotor, accommodative, and binocular system disorders, as measured by standardized clinical and laboratory testing methods, in the majority of patients of all ages for whom it is properly undertaken and employed.

The American Optometric Association reaffirms its long-standing position that vision therapy is an effective therapeutic modality in the treatment of many physiological and information processing dysfunctions of the vision system. It continues to support quality optometric care, education, and research and will cooperate with all professions dedicated to providing the highest quality of life in which vision plays such an important role.[1]

■■

Corresponding author:
Allen H. Cohen, O.D.
SUNY State College of Optometry
100 E. 24th St.
New York, NY 10010

Acknowledgment

The Task Force would like to acknowledge Jack E. Richman, O.D., Nathan Flax, O.D. and Leonard Press, O.D. for their major contributions to the research and preparation of this document. A number of editorial revisions were based on the recommendations of the following individuals and organizations: Arol Augsburger, O.D., Louis G. Hoffman, O.D., Mike Rouse, O.D., Ralph T. Garzia, O.D., the College of Optometrists in Vision Development, and the Optometric Extension Program Foundation. The members of the 1985-86 Task Force also contributed to the initial development of this document: Donald J. Getz, O.D., chairman; Paul A. Harris, O.D.; Paul J. Lederer, O.D.; Ronald L. Bateman, O.D.; and D. Gary Thomas, O.D.

Footnote

a. Allen H. Cohen, O.D., chairman; Sue E. Lowe, O.D.; Glen T. Steele, O.D.; Irwin B. Suchoff, O.D.; Daniel D. Gottlieb, O.D., consultant; Torrance L. Trevorrow, O.D., staff.

References

1. Special report: Position statement on vision therapy. J Am Optom Assoc 1985; 56:782-83.
2. Costs of education in the health professions — 1974. Institute of Medicine of the National Academy of Sciences.
3. Dictionary of occupational titles, 1977, 4th ed. GPO No. 029-013-00079-9, Employment and Training Administration.
4. Facts about medical and dental practitioners 1975-76. US Department of

Health, Education and Welfare Public Health Service Health Resources Administration, Bureau of Health Resources Development.
5. Health careers guidebook 1979, 4th ed. GPO No. 029-000-00343-2, Department of Labor, Employment and Training Administration and US Department of Health and Human Services, Health Resources Administration.
6. National Center for Health Statistics 1976-77. US Department of Health, Education, and Welfare, Health Manpower and Health Facilities, Health Resources Statistics.
7. Occupational outlook handbook, April 1982. US Department of Labor Bureau of Labor Statistics, Bulletin 2200.
8. Third report to the president and congress on the status of health professions personnel in the United States. Publication No. (HRA) 82-2. January 1982, Department of Health and Human Services.
9. National Board of Examiners in Optometry. New content outline. Implementation plans for the new entry-level examinations. Washington, DC: National Board of Examiners in Optometry, 1986.
10. Special Committee Report, Association of Schools and Colleges of Optometry. Curriculum model for oculomotor, binocular, and visual perception dysfunctions. Washington, DC 1987.
11. Flax N, ed. Vision therapy and insurance: A position statement. New York: State University of New York, State College of Optometry, 1986.
12. National Center for Health Statistics. Eye examination findings among children, United States. DHEW Publication No. 72-1057, Series 11, No 115. Rockville, MD: Department of Health, Education, and Welfare, 1972.
13. National Center for Health Statistics. Refraction status and motility defects of persons 4-74 years, United States. DHEW Publication No. 78-1654, Series 11, No 206. Hyattsville, MD: Department of Health, Education and Welfare, 1978.
14. National Center for Health Statistics. Refraction status of youths 12-17 years, United States. DHEW Publications No. 75-1630, Series 11, No 148. Rockville, MD: Department of Health, Education and Welfare, 1974.
15. Bennett GR, Blondin M, Ruskiewicz J. Incidence and prevalence of selected visual conditions. J Optom Assoc 1982; 53:647-56.
16. Blum HL, Peters HB, Bettman JW. Vision screening for elementary schools: The Orinda study. Berkeley: University of California Press, 1959.
17. Graham PA. Epidemiology of strabismus. Br J Ophthalmol 1974; 58:224-31.
18. Fletcher CF, Silverman SJ. Strabismus. Part I. A summary of 1110 consecutive cases. Am J Ophthalmol 1966; 61:86-94.
19. Frendsen AD. The occurrence of squint. Acta Ophthalmol (Suppl) 1960; 62.
20. Flom MC, Neumaier RW. Prevalence of amblyopia. Am J Optom Arch Am Acad Optom 1966; 73:732-51.
21. Rubenstein RS, Lohr KN, Brook RH, et al. Measurement of the physiological health of children. Vol 4, Vision Impairments. Santa Monica: Rand Corp, 1985.
22. Ross E, Murray AL, Stead S. Prevalence of amblyopia in grade 1 school children in Saskatoon. Can J Public Health 1977; 68:491-3.
23. Operational Research Department of the National Society to Prevent Blindness. Vision problems in the United States: A statistical analysis. New York: National Society to Prevent Blindness, 1980.
24. Duke-Elder S. The physiology of the eye and of vision. In: Duke-Elder S, ed. System of ophthalmology, vol IV. St. Louis: CV Mosby, 1968.
25. Hokoda SC. General binocular dysfunctions in an urban optometry clinic. J Am Optom Assoc 1985; 56:560-3.
26. Hoffman LH. Incidence of vision difficulties in children with learning disabilities. J Am Optom Assoc 1980; 51:447-51.
27. Lieberman S, Cohen A, Stolzberg M, et al. Validation study of the New York State Optometric Association (NYSOA) vision screening battery. Am J Optom Physiol Opt 1985; 62:165-8.
28. Grisham JD. Computerized visual therapy — year 1 report. Palo Alto: American Institutes for Research, 1986.
29. LoCascio GP. A study of vision in cerebral palsy. Am J Optom Physiol Opt 1977; 54:332-7.
30. Scheiman MN. Optometric findings in children with cerebral palsy. Am J Optom Physiol Opt 1984; 61:321-1.
31. Gottlieb DD, Allen W. Incidence of visual disorders in a selected population of hearing impaired students. J Am Optom Assoc 1985; 56:292-6.
32. Mohindra I. Vision profile of deaf children. Am J Optom Physiol Opt 1976; 53:412-9.
33. Lieberman S. The prevalence of visual disorders in a school for emotionally disturbed children. J Am Optom Assoc 1985; 56:800-5.
34. Levy B. Incidence of oculo-visual anomalies in an adult population of mentally retarded persons. Am J Optom Physiol Opt 1984; 61:324-6.
35. Woodruff ME, Cleary E, Bader D. The prevalence of refractive and ocular anomalies among 1242 institutionalized mentally retarded persons. Am J Optom Physiol Opt 1980; 57:70-6.
36. Plisko VW, Stern JD, eds. The condition of education, 1985. Washington: United States Department of Education, 1985.
37. Smith MJ, Cohen BGF, Stammerjohn LW, Jr, et al. An investigation of health complaints and job stress in video display operation. Hum Factors 1981; 23:387-400.
38. National Institute of Occupational Safety and Health. Potential health hazards of video display terminals. DHHS (NIOSH) Publication No. 81-129. Cincinnati: National Institute for Occupational Safety and Health, 1981.
39. Panel on Impact of Video Viewing on Vision of Workers. Video displays, work, and vision. Washington: National Academy Press, 1983.
40. Leigh JR, Zee DS. The diagnostic value of abnormal eye movements: A pathophysiological approach. Johns Hopkins Med J 1982; 151:122-35.
41. Leigh JR, Zee DS. The neurology of eye movements. Philadelphia: FA Davis, 1984: 11-89.
42. Leigh JR, Zee DS. The neurology of eye movements. Philadelphia: FA Davis, 1984: 6.
43. Leigh JR, Zee DS. The neurology of eye movements. Philadelphia: FA Davis, 1984: 192-94.
44. Schor C. A directional impairment of eye movement control in strabismus amblyopia. Invest Ophthalmol Vis Sci 1975; 15:692-7.
45. Schapero M. Amblyopia. Philadelphia: Chilton, 1971.
46. Von Noorden GK. Burian-Von Noorden's binocular vision and ocular motility, 2nd ed. St. Louis: CV Mosby, 1980: 219-49.
47. Von Noorden GK, Mackensen G. Pursuit movements of normal and amblyopic eyes — an electromyographic study. II. Pursuit movements of amblyopic patients. Am J Ophthalmol 1962; 53:477-87.
48. Ciufredda KJ, Kenyon RV, Stark L. Abnormal saccadic substitution during small amplitude pursuit tracking in amblyopic eyes. Invest Ophthalmol Vis Sci 1979; 18:506-16.
49. Ciufredda KJ, Kenyon RV, Stark L. Saccadic intrusions in strabismus. Arch Ophthalmol 1979; 97:1673-9.
50. Metz HS, Jampolsky A, O'Meara DM. Congenital ocular nystagmus and nystagmoid head movements. Am J Ophthalmol 1974; 6:1131-3.
51. Ciufredda KJ, Bahill AT, Kenyon RV, et al. Eye movements during reading: case reports. Am J Optom Physiol Opt 1976; 53:389-95.
52. Senders JW, Fisher DF, Monty RA, eds. Eye movements and higher psychological functions. Hillsdale, NJ: Lawrence Erlbaum Assoc, 1978.
53. Lefton LA. Eye movements in reading disabled children. In: Senders JW, Fisher DF, Monty RA, eds. Eye movements and higher psychological functions. Hillsdale NJ: Lawrence Erlbaum Assoc, 1978: 225-37.
54. Senders JW, Fisher DF, Monty RA, eds. Eye movements: cognition and visual perception. Hillsdale, NJ: Lawrence Erlbaum Assoc, 1981.
55. Leigh JR, Zee DS. The neurology of eye movements. Philadephia: FA Davis, 1984: 44-6.
56. Fisk JD, Goodale MA, Burkart G, et al. Progressive superanuclear palsy: The relationship between ocular motor dysfunction and psychological test performance. Neurology 1982; 32:698-705.
57. Pavlidis GT. Eye movements in dyslexia: Their diagnostic significance. J

Learn Disabil 1985; 18:42–50.
58. Pirozzolo FJ. Eye movements and reading disability. In: Rayner K, ed. Eye movements in reading: perceptual and language processes. New York: Academic Press, 1983: 499–509.
59. Rayner K. Eye movements in reading and information processing. Psychol Bull 1978; 85:618–60.
60. Poynter HL, Schor C, Haynes HM, et al. Oculomotor functions in reading disability. Am J Optom Physiol Opt 1982; 59:116–27.
61. Pollatsek A. What can eye movements tell us about dyslexia? In: Rayner K, ed. Eye movements in reading: perceptual and language processes. New York: Academic Press, 1983: 511–21.
62. Senders JW, Monty RA, eds. Eye movements and psychological processes. Hillsdale, NJ: Lawrence Erlbaum Assoc, 1976.
63. Kundel HL, Nodine CF. Studies of eye movements and visual search in radiology. In: Senders JW, Fisher DF, Monty RA, eds. Eye movements and higher psychological functions. Hillsdale, NJ: Lawrence Erlbaum Assoc, 1978: 317–28.
64. Locher PJ, Worms PP. Visual scanning strategies of neurologically impaired, perceptually impaired, and normal children viewing the Bender-Gestalt drawings. Psychol In The Schools 1977; 14:147–57.
65. Locher PJ, Worms PF. Visual scanning strategies of perceptually impaired and normal children viewing the motor-free visual perception test. J Learn Disabil 1981; 14:416–9.
66. Tinker MA. Bases for effective reading. Minneapolis, MN: University of Minneapolis Press, 1966: 81–94.
67. Ault RL, Crawford DE, Jeffrey WE. Visual scanning strategies of reflective, impulsive, fast-accurate, and slow-inaccurate children on the Matching Familiar Figures Test. Child Dev 1972; 43:1412–7.
68. Sato K. An investigation of visual scanning strategies of reflective and impulsive children and adults. Jap Educ Psych 1976; 24:224–34.
69. Drake DM. Perceptual correlates of impulsive and reflective behavior. Dev Psychol 1970; 2:202–14.
70. Cohen B, Bala S, Morris AG. Do hyperactive children have manifestations of hyperactivity in their eye movements? ERIC Document ED 112 601, 1975.
71. Bala SP, Cohen B, Morris AG, et al. Saccades of hyperactive and normal boys during ocular pursuits. Dev Med Child Neurol 1981; 23:323–36.
72. Richman JE. Use of a sustained visual attention task to determine children at risk for learning problems. J Am Optom Assoc 1986; 57:20–6.
73. Simon MJ. Use of a vigilance task to determine school readiness of preschool children. Percept Mot Skills 1982; 54:1020–2.
74. Berch DB, Kanter DR. Individual differences. In: Warm JS, ed. Sustained attention in human performance. New York: John Wiley and Sons, 1984: 143–70.
75. Ludlam WM. Visual training, the alpha activation cycle and reading. J Am Optom Assoc 1979; 50:111–5.
76. Ludlam WM. Optometric visual training for reading disability — a case report. Am J Optom Physiol Opt 1973; 50:58–66.
77. Camuccio D, Griffin JR. Visual skills therapy — a case report. Optom Mon 1982; 73:94–6.
78. Wold RM, Pierce JR, Keddington J. Effectiveness of optometric vision therapy. J Am Optom Assoc 1978; 49:1047–59.
79. Griffin JR. Binocular anomalies — procedures for vision therapy. Chicago: Professional Press, 1982: 349–65.
80. Heath EJ, et al. Eye exercises and reading efficiency. Academic Therapy 1976; 11:435–45.
81. Fujimoto DH, Christensen EA, Griffin JR. An investigation in use of videocassette techniques for enhancement of saccadic eye movements. J Am Optom Assoc 1985; 56:304–8.
82. Busby RA. Vision development in the classroom. J Learn Disabil 1985; 18:266–72.
83. Punnett AF, Steinhauer GD. Relationship between reinforcement and eye movements during ocular motor training with learning disabled children. J Learn Disabil 1984; 17:16–9.
84. Punnett AF. Relationship between reinforcement and eye movements during vision therapy with dyslexic children. PhD thesis. Ann Arbor: University of Microfilms, 1981: 24.
85. Feldman J. Behavior modification in vision training: facilitating prerequisite behaviors and visual skills. J Am Optom Assoc 1981; 52:329–40.
86. Stohler T. Afterimage treatment of nystagmus. Am Orthopt J 1973; 23:65–7.
87. Goldrich SG. Emergent textual contours: A new technique for visual monitoring in nystagmus, oculomotor dysfunction, and accommodative disorders. Am J Optom Physiol Opt 1981; 58:451–9.
88. Healy E. Nystagmus treated by orthoptics. Am Orthopt J 1952; 2:53–5.
89. Stegall FW. Orthoptic aspects of nystagmus. Am Orthopt J 1973; 23:30–4.
90. Ciufredda KJ, Kenyon RV, Stark L. Supression of fixational saccades in strabismic and anisometropic amblyopia. Ophthalmic Res 1979; 11:31–9.
91. Ciufredda KJ, Goldrich SG, Neary C. Use of eye movement auditory feedback in the control of nystagmus. Am J Optom Physiol Opt 1982; 59: 396–409.
92. Abadi RV, Carden D, Simpson J. A new treatment for congenital nystagmus. Br J Ophthalmol 1980; 64:2–4.
93. Letourneau JE. Biofeedback reinforcement in the training of limitation of gaze: A case report. Am J Optom Physiol Opt 1976; 53:672–6.
94. Schor CM, Flom MC. Eye position control and visual acuity in strabismus amblyopia. In: Lennerstrand G, Bach-y-Rita P, Collins CC, et al, eds. Basic mechanisms of ocular motility and their clinical manifestations. New York: Pergamon Press, 1975.
95. Ciufredda KJ. Visual system plasticity in human amblyopia. In: Hilfer RS, Sheffield JB, eds. Development of order in the visual system. New York: Springer-Verlag, 1986: 212–44.
96. Burian HM, Von Noorden GK. Binocular vision and ocular motility, 3rd ed. St. Louis: CV Mosby, 1985.
97. Schapero M. Amblyopia. New York: Chilton Book Co, 1971.
98. Duke-Elder S, ed. System of ophthalmology, vol VI. Ocular motility and strabismus. St. Louis: CV Mosby, 1973: 424–56.
99. Gortz H. The corrective treatment of amblyopia with eccentric fixation. Am J Ophthalmol 1960; 49:1315–21.
100. Gregersen E. Occlusion treatment of squint amblyopia in young adults. Acta Ophthalmol 1966; 44:166–8.
101. Brown MH, Edleman PM. Conventional occlusion in the older amblyope. Am Orthopt J 1976; 26:34–6.
102. Eibschitz N, Friedman Z, Neuman E. Comparative results of amblyopia treatment. Metab Ophthalmol 1978; 2:111–2.
103. Garzia, RP. The efficacy of visual training in amblyopia: A literature review. Am J Optom Physiol Opt 1987; 64: 393–404.
104. Nawratzki I. Treatment of amblyopia. Ir J Med Sci 1972; 8:1475–9.
105. Massie H. Fixing eye occlusion: Survey of approximately 1000 case histories of patients who received occlusion of the fixing eye. Trans Ophthalmol Soc Aust 1965; 24:39–46.
106. Kupfer C. Treatment of amblyopia ex anopsia in adults. Am J Ophthalmol 1957; 43:918–22.
107. Gregersen E, Rindziunski E. Conventional occlusion in the treatment of squint amblyopia. Acta Ophthalmol 1965; 43:462–74.
108. Scott WE, Stratton VB, Fabre J. Full-time occlusion therapy for amblyopia. Am Orthopt J 1980; 30:125–30.
109. Haldi B, Mitchelson JE. Amblyopia therapy: Expected results from standard techniques. Am Orthopt J 1981; 31:19–28.
110. Ingram RM, Rogers S, Walker C. Occlusion and amblyopia. Br Orthopt J 1977; 34:11–22.
111. Goodier HM. Some results of conventional occlusion. Br Orthopt J 1974; 31:55–8.
112. Francois J, James M. Comparative study of amblyopic treatment. Am Orthopt J 1955; 5:61–4.
113. Bangerter A. Amblyopienhandling. Aufl 2. Basel: Karger, 1955.
114. Bangerter A. Die occlusion in der pleoptik and orthoptic. Klin Monatsbl Augenheilkd 1960; 136:305–31.
115. Girard LJ, Fletcher MC, Tomlinson E, et al. Results of pleoptic treatment of supperession amblyopia. Am Orthopt J 1962; 12:12–31.
116. Jablonski M, Tomlinson E. A new look at pleoptics. Ophthalmology 1979; 86:2112–4.

117. Mayweg S, Massie HH. A preliminary report of the more recent methods of treatment of amblyopia, especially when associated with eccentric fixation in cases of strabismus. Br J Ophthalmol 1958; 42:257–69.
118. Deller M, Streiff EB. Apropos de l'amblyopie a fixation excentrique. Ophthalmologica 1965; 150:76–82.
119. Selenow A, Ciufredda KJ. Vision function recovery during orthoptic therapy in an adult esotropic amblyope. J Am Optom Assoc 1986; 57:132–40.
120. Ciuffreda KJ, Kenyon RV, Stark L. Different rates of functional recovery of eye movements during orthoptics treatment in an adult amblyope. Invest Ophthalmol Vis Sci 1979; 18:213–9.
121. Hoffman L, Cohen AH, Feuer G, et al. Effectiveness of optometric therapy for strabismus in a private practice. Am J Optom Arch Am Acad Optom 1970; 47:990–5.
122. Shippman S. Video games and amblyopia treatment. Am Orthopt J 1985; 35:2–5.
123. Porter EE. Treatment of amblyopia. Am Orthopt J 1962; 12:157–61.
124. Cohen AH. Monocular fixation in a binocular field. J Am Optom Assoc 1981; 52:801–6.
125. Birnbaum MH, Koslowe K, Sanet R. Success in amblyopia therapy as a function of age: A literature survey. Am J Optom Physiol Opt 1977; 54:269–75.
126. Blackman S, Goldstein KM. Cognitive styles and learning disabilities. J Learn Disabil 1982; 15:106–15.
127. Messer SB. Reflection-impulsivity: a review. Psychol Bull 1976; 83:1026–52.
128. Abikoff H. Cognitive training interventions in children: Review of a new approach. J Learn Disabil 1979; 12:123–35.
129. Meichenbaum DH, Goodman J. Training impulsive children to talk to themselves: A means of developing self-control. J Abnorm Psychol 1971; 77:115–26.
130. Egeland B. Training impulsive children in the use of more efficient scanning techniques. Child Dev 1974; 45:165–71.
131. Zelniker, Oppenheimer L. Modification of information processing of impulsive children. Child Dev 1973; 44:445–50.
132. Orbach I. Impulsive cognitive style: Three modification techniques. Psychol in the Schools 1977; 14:353–9.
133. McKinney JD, Haskins R. Cognitive training and the development of problem-solving strategies. Except Educ Q 1980; 1:41–51.
134. Douglas VI, Parry P, Marton P, et al. Assessment of a cognitive training program for hyperactive children. J Abnorm Child Psychol 1976; 4:389–410.
135. Brown RT, Wynne ME. Correlates of teacher ratings, sustained attention, and impulsivity in hyperactive and normal boys. J Clin Child Psychol 1982; 11:262–7.
136. Brown RT, Conrad KJ. Impulse control or selective attention: Remedial programs for hyperactivity. Psychol in the Schools 1982; 19:92–7.
137. Brown RT, Alford N. Amelioration attentional deficits and concomitant academic deficiencies in learning disabled children through cognitive training. J Learn Disabil 1984; 17:20–6.
138. Sherman CF, Anderson RP. Modification of attending behavior in hyperactive children. Psychol in the Schools 1980; 17:372–9.
139. Schrock RE. Introduction to vision training, Series 1–2, 1965–67, OEP Foundation, Santa Ana, CA.
140. MacDonald LW. Visual training, Series 1–2. 1978–79, OEP Foundation, Santa Ana, CA.
141. Forrest EB. Visual imagery: An optometric approach, OEP Foundation, Santa Ana, CA, 1981.
142. Richman JE, Cron MT, Cohen E. Basic vision therapy: A clinical handbook. Big Rapids, MI: Ferris State College Press, 1983.
143. Smith W. Clinical orthoptic procedure: A reference book on clinical methods of orthoptics. St. Louis: CV Mosby, 1950.
144. Borish IM. Clinical refraction, 3rd ed. Chicago: Professional Press, 1970: 184–5.
145. Griffin JR. Binocular anomalies: Procedures for vision therapy, 2nd ed. Chicago: Professional Press, 1982: 377–93.
146. Suchoff IB, Petito TG. The efficacy of visual therapy: accommodative disorders and non-strabismic anomalies of binocular vision. J Am Optom Assoc 1986; 57:119–25.
147. Duane A. Anomalies of the accommodation, clinically considered. Trans Am Ophthalmol Soc 1915; 1:386–402.
148. Duane A. Anomalies of the accommodation, clinically considered. Arch Ophthalmol 1916; 45:124–36.
149. Pierce J, Greenspan S. Accommodative rock procedures in vision training, a clinical guide. Optom Wkly 1972; 62(33):754–7.
150. Pierce J, Greenspan S. Accommodative rock procedures in vision training, a clinical guide. Optom Wkly 1971; 62(34):776–80.
151. Weisz CL. How to find and treat accommodative disorders. Rev Optom 1983; 120:48–54.
152. Zellers JA, Alpert TL, Rouse MW. A review of the literature and a normative study of accommodative facility. J Am Optom Assoc 1984; 55:31–7.
153. Garzia R, Richman J. Accommodative facility: A study of young adults. J Am Optom Assoc 1982; 53:821–4.
154. Bieber J. Why nearpoint retinoscopy with children? Optom Wkly 1974; 65(3):54–7.
155. Apell R. Clinical application of bell retinoscopy. J Am Optom Assoc 1975; 46:1023–7.
156. Levine S, Ciuffreda KJ, Selenow A, et al. Clinical assessment of accommodative facility in symptomatic and asymptomatic individuals. J Am Optom Assoc 1985; 56:286–90.
157. Hoffman LG, Rouse MW. Referral recommendations for binocular function and/or developmental perceptual deficiencies. J Am Optom Assoc 1980; 51:119–26.
158. Hennessey D, Iosue R, Rouse M. Relation of symptoms to accommodative infacility of school-aged children. Am J Optom Physiol Opt 1984; 61:177.
159. Daum K. Accommodative dysfunction. Doc Ophthalmol 1983; 55:177–98.
160. Hoffman L. The effect of accommodative deficiencies on the development level of perceptual skills. Am J Optom Physiol Opt 1982; 59:254–62.
161. Marg E. An investigation of voluntary as distinguished from reflex accommodation. Am J Optom Arch Am Acad of Optom 1951; 28:347–56.
162. Randall R. Volitional control of visual accommodation. In: Conf Proceedings, Advisory Group for Aerospace Research and Development, 1970; 82:15–7.
163. Provine R, Enoch J. On voluntary ocular accommodation. Percept Psychophys 1975; 17:209–12.
164. Cornsweet TN, Crane H. Training the visual accommodative system. Vision Res 1973; 13:713–5.
165. Prangen A. Subnormal accommodation. Arch Ophthalmol 1931; 6:906–18.
166. Duane A. Anomalies of accommodation clinically considered. Arch Ophthalmol (Old Series) 1916; 45:124–36.
167. Prakash P, Agarwal L, Nag S. Accommodational weakness and convergence insufficiency. Orient Arch Ophthalmol 1972; 10:261–4.
168. Von Noorden G, Brown D, Parks M. Associated convergence and accommodative insufficiency. Doc Ophthalmol 1973; 34:393–403.
168a. Hoffman L, Cohen AH. Effectiveness of non strabismic optometric vision training in a private practice. Am J Optom Arch Am Acad Optom 1973; 50:813–6.
169. Liu JS, Lee M, Jang J, et al. Objective assessment of accommodation orthoptics: 1. dynamic insufficiency. Am J Optom Physiol Opt 1979; 56:285–91.
170. Bobier WR, Sivak JG. Orthoptic treatment of subjects showing slow accommodative responses. Am J Optom Physiol Opt 1983; 60:678–87.
171. Hung GK, Ciuffreda KJ, Semmlow JL. Static vergence and accommodation: population norms and orthoptic effects. Doc Ophthalmol 1986; 62:165–79.
172. Duckman RH. The incidence of visual anomalies in a population of cerebral palsy children. J Am Optom Assoc 1979; 50:1013–6.
173. Duckman RH. Effectiveness of visual training on a population of cerebral palsied children. J Am Optom Assoc 1980; 51:607–14.
174. Duckman RH. Accommodation in cerebral palsy: Function and remediation. J Am Optom Assoc 1984; 55:281–3.
175. Haynes HM, McWilliams LG. Effects of training on near-far response time as measured by the distance rock test. J Am Optom Assoc 1979; 50:715–8.
176. Weisz CL. Clinical therapy for accommodative responses: Transfer effects

upon performance. J Am Optom Assoc 1979; 50:209–21.
177. Daum K. Accommodative dysfunction. Doc Ophthalmol 1983; 55:177–98.
178. Daum K. Accommodative insufficiency. Am J Optom Physiol Opt 1983; 60:352–9.
179. Daum K. Orthoptic treatment in patients with inertia of accommodation. Aust J Optom 1983; 66:68–72.
180. Daum K. Predicting results in the orthoptic treatment of accommodative dysfunction. Am J Optom Physiol Opt 1984; 61:184–9.
181. Borish IM. Clinical refraction, 3rd ed. Chicago: Professional Press, 1970: 859–937.
182. Duke-Elder S. System of ophthalmology, vol VI. Ocular motility and strabismus. St. Louis: CV Mosby, 1973: 513–76.
183. Griffin JR. Binocular anomalies: Procedures for vision therapy, 2nd ed. Chicago: Professional Press, 1982: 126–30.
184. Schor C, Ciuffreda KJ, eds. Vergence eye movements: Basic and clinical aspects. Boston: Butterworths, 1983: 1–538.
185. Burian H, von Noorden GK. Binocular vision and ocular mobility, 2nd ed. St. Louis: CV Mosby, 1974: 181–211.
186. Borish IM. Clinical refraction, 3rd ed. Chicago: Professional Press, 1970: 327.
187. Burian H, von Noorden K. Binocular vision and ocular motility, 2nd ed. St. Louis: CV Mosby, 1974: 167.
187a. Sheedy JE, Saladin JJ. Association of symptoms with measures of oculomotor deficiencies. Am J Optom Physiol Opt 1978; 55:670–6.
188. Scobee R. The oculorotary muscles, 2nd ed. St. Louis: CV Mosby, 1952: 160–8.
189. Duke-Elder S. System of ophthalmology, vol V: Ophthalmic optics and refraction. St. Louis: CV Mosby, 1970: 469–87.
190. Lancaster JE. A manual of orthoptics. Springfield, IL: CC Thomas, 1951.
191. Dendy HM, Shaterian ET. Practical ocular motility. Springfield, IL: CC Thomas, 1967.
192. Hugonnier R, Hugonnier Sc. Strabismus, heterophoria, ocular motor paralysis: clinical ocular muscle imbalance. St. Louis: CV Mosby, 1969.
193. Gibson HW. Textbook of orthoptics. London: Hatton Press Ltd, 1955.
194. Hurtt J, Rasicovici A, Windsor CE. Comprehensive review of orthoptics and ocular motility. St. Louis: CV Mosby, 1972.
195. Berens C, Connolly P, Kern D. Certain motor anomalies of the eye in relation to prescribing lenses. Am J Ophthalmol 1933; 5:199–213.
196. Daum KM. The course and effect of visual training on the vergence system. Am J Optom Physiol Opt 1982; 59: 223–7.
197. Daum KM. Convergence insufficiency. Am J Optom Physiol Opt 1984; 61:16–22.
198. Daum KM. Classification criterion for success in the treatment of convergence insufficiency. Am J Optom Physiol Opt 1984; 61:10–5.
199. Daum KM. A comparison of the results of tonic and phasic training on the vergence system. Am J Optom Physiol Opt 1983; 60:769–75.
200. Vaegan JL. Convergence and divergence show longer and sustained improvement after short isometric exercise. Am J Optom Physiol Opt 1979; 56:23–33.
201. Vaegan JL, McMonnies C. Clinical vergence training. Aust J Optom 1979; 62:28–36.
202. Vaegan JL. Convergence and divergence show large and sustained improvement after short isometric exercise. Am J Optom Physiol Opt 1979; 56:23–33.
203. Pantano R. Orthoptic treatment of convergence insufficiency: A two year follow-up report. Am Orthopt J 1982; 32:73–80.
204. Grisham J. The dynamics of fusional vergence eye movements in binocular dysfunction. Am J Optom Physiol Opt 1980; 57:645–55.
205. Grisham J. Treatment of binocular dysfunction. In: Schor C, Ciuffreda KJ, eds. Vergence eye movements. Boston: Butterworths, 1983: 605–46.
206. Cooper J, Duckman R. Convergence insufficiency: Incidence, diagnosis and treatment. J Am Optom Assoc 1978: 49:673–80.
207. Cooper J, Feldman J. Operant conditioning of fusional convergence ranges using random dot stereograms. Am J Optom Physiol Opt 1980; 57:205–13.
208. Cooper J, Selenow A, Ciuffreda KJ, et al. Reduction of asthenopia in patients with convergence insufficiency after fusional vergence training. Am J Optom Physiol Opt 1983; 60:982–9.
209. Dalziel CC. Effect of vision training on patients who fail Sheard's criterion. Am J Optom Physiol Opt 1981; 58:21–3.
210. Wittenberg S, Brock FW, Folsom WC. Effect of training on stereoscopic acuity. Am J Optom Arch Am Acad Optom 1969; 46:645–53.
211. Saladin JJ, Rick JO. Effect of orthoptic procedures on stereoscopic acuities. Am J Optom Physiol Opt 1982; 59: 718–25.
212. Duke-Elder S. System of ophthalmology, Vol VI. Ocular motility and strabismus. St. Louis: CV Mosby, 1973: 245–77.
213. Burian H, von Noorden GK. Binocular vision and ocular mobility, 2nd ed. St. Louis: CV Mosby, 1974: 175–274.
214. Parks M. Oculomotility and strabismus. In: Duane TD, ed. Clinical ophthalmology. Hagerstown, MD: Harper & Row, 1979: 1.
215. Flax N. Strabismus diagnosis and prognosis. In: Schor C, Ciuffreda KJ, eds. Vergence eye movements: basic and clinical aspects. Boston: Butterworths, 1983: 579–95.
216. Flom MC. Treatment of binocular anomalies of vision. In: Hirsch MJ, Wick RE, eds. Vision of children. Philadelphia: Clinton, 1963: 197–228.
217. Ludlam WM. Orthoptic treatment of strabismus. Am J Optom Arch Am Acad Optom 1961; 38:369–88.
218. Ludlam WM, Kleinman BI. The long range results of orthoptic treatment of strabismus. Am J Optom Arch Am Acad Optom 1965; 42:647–84.
219. Flax N, Duckman RH. Orthoptic treatment of strabismus. J Am Optom Assoc 1978; 49:1353–61.
220. Gillan RU. An analysis of one hundred cases of strabismus treated orthoptically. Br J Ophthalmol 1945; 29:420–8.
221. Guibor GP. Practical details in the orthoptic treatment of strabismus. Arch Ophthalmol 1934; 12:887–901.
222. Guibor GP. Some possibilities of orthoptic training. Arch Ophthalmol 1934; 11:433–61.
223. Guibor GP. The possibilities of orthoptic training — a further report. Am J Ophthalmol 1934; 17:834–9.
224. Ziegler D, Huff D, Rouse MW. Success in strabismus therapy: a literature review. J Am Optom Assoc 1982; 53:979–83.
225. Etting G. Strabismus therapy in private practice: Cure rates after three months of therapy. J Am Optom Assoc 1978; 49:1367–73.
226. Kertesz AE, Kertesz J. Wide-field stimulation in strabismus. Am J Optom Physiol Opt 1986; 63:217–22.
227. 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.
228. Altizer LB. The non-surgical treatment of exotropia. Am Orthopt J 1972; 22:71–6.
229. Chryssanthou G. Orthoptic management of intermittent exotropia. Am Orthopt J 1974; 24:69–72.
230. Goldrich SG. Optometric therapy of divergence excess strabismus. Am J Optom Physiol Opt 1980; 57:7–14.
231. Van Brocklin MD, Vasche TR, Hirons RR, et al. Biofeedback enhanced strabismus therapy. J Am Optom Assoc 1981; 52:731–6.
232. Hirons RR, Yolton RL. Biofeedback treatment of strabismus: Case studies. J Am Optom Assoc 1978; 49:875–82.
233. Afanador AJ. Auditory biofeedback and intermittent exotropia. J Am Optom Assoc 1982; 53:481–3.
234. Goldrich SG. Oculomotor biofeedback therapy for exotropia. Am J Optom Physiol Opt 1982; 59:306–17.
235. Flom MC, Kirschen DG, Bedell HE. Control of unsteady, eccentric fixation in amblyopic eyes by auditory feedback of eye position. Invest Ophthalmol Vis Sci 1980; 19:1371–91.
236. Scheiman MM, Peli E, Libassi D. Auditory biofeedback used to enhance convergence insufficiency therapy. J Am Optom Assoc 1983; 54:1001–3.
237. Pigassou-Albouy R. Use of prisms in pre-operative and post-operative treatment. In: Fells P, ed. The First Congress of the International Strabismus Assoc. St. Louis: CV Mosby, 1971: 242–75.
238. Berard PV. Constant wearing of prisms in treatment of concomitant strabismus. In: Ferrer OM, ed. Int Ophthalmol Clin, Ocular Motility. Boston: Little, Brown & Co, 1971: 283–91.