OPTOMETRIC CLINICAL PRACTICE GUIDELINE

Care of the Patient with

Hyperopia

American Optometric Association®

OPTOMETRY: THE PRIMARY EYE CARE PROFESSION

Doctors of optometry (ODs) are the primary health care professionals for the eye. Optometrists examine, diagnose, treat, and manage diseases, injuries, and disorders of the visual system, the eye, and associated structures as well as identify related systemic conditions affecting the eye.

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. Approximately 37,000 full-time equivalent doctors of optometry practice in more than 7,000 communities across the United States, serving as the sole primary eye care provider in more than 4,300 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.

Healthy People 2010 Vision®

American Optometric Association

OPTOMETRIC CLINICAL PRACTICE GUIDELINE
CARE OF THE PATIENT WITH HYPEROPIA

Reference Guide for Clinicians

Prepared by the American Optometric Association Consensus Panel on Care of the Patient with Hyperopia:

Bruce D. Moore, O.D., Principal Author
Arol R. Augsburger, O.D., M.S.
Elise B. Ciner, O.D.
David A. Cockrell, O.D.
Karen D. Fern, O.D.
Elise Harb O.D. M.S.

Reviewed by the AOA Clinical Guidelines Coordinating Committee:

David A. Heath, O.D., Chair
Diane T. Adamczyk, O.D.
John F. Amos, O.D., M.S.
Stephen C. Miller, O.D.

Approved by the AOA Board of Trustees August 9, 1997;
Reviewed February 2001, 2006; Revised 2008

243 N. Lindbergh Blvd., St. Louis, MO 63141-7881

Printed in U.S.A.

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.

TABLE OF CONTENTS

[redacted]

[redacted]

INTRODUCTION

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

The Optometric Clinical Practice Guideline on Care of the Patient with Hyperopia describes appropriate examination and treatment procedures to reduce the risk of visual disability from hyperopia. It contains recommendations for timely diagnosis, treatment, and, when necessary, referral for consultation with or treatment by another health care provider. This Guideline will assist optometrists in achieving the following goals:

- Accurately diagnose hyperopia
- Document the patient care treatment options for patients with hyperopia
- Identify patients at risk for the adverse effects of hyperopia
- Minimize the adverse effects of hyperopia
- Preserve the gains obtained through treatment
- Inform and educate parents, patients, and other health care practitioners about the visual complications of hyperopia and the availability of treatment.

I. STATEMENT OF THE PROBLEM

Hyperopia, also termed hypermetropia or farsightedness, is a common refractive error in children and adults. Its effect varies greatly, depending upon the magnitude of hyperopia, the age of the individual, the status of the accommodative and convergence system, and the demands placed on the visual system. Individuals with uncorrected hyperopia may experience:

- Blurred vision
- Asthenopia
- Accommodative dysfunction
- Binocular dysfunction
- Amblyopia
- Strabismus.

Early detection of hyperopia may help to prevent the complications of strabismus and amblyopia in young children. In older children, uncorrected hyperopia may affect learning ability.[1] In individuals of any age, it can contribute to ocular discomfort and visual inefficiency.

A. Description and Classification of Hyperopia

Refractive error is a manifestation of the relationship between the optical components of the eye (i.e., curvatures, refractive indices, and distances between the cornea, aqueous, crystalline lens, and vitreous) and the overall axial length of the eye. Hyperopia is a refractive error in which parallel rays of light entering the eye reach a focal point behind the plane of the retina, while accommodation is maintained in a state of relaxation.[2] The magnitude of hyperopia is described as the additional dioptric power of the converging lenses required to advance the focusing of light rays onto the retinal plane, while accommodation is relaxed. These correcting lenses may be spherical or spherocylindrical, depending upon the nature of the hyperopia and the amount of astigmatic refractive error co-existing with the hyperopia.

Refractive errors, including hyperopia, may be differentiated by the degree of variance from a model of the optical components of the eye

and their relationship to axial length.[3-6] The early refractive development of the eye reflects a pattern of growth and change in which the eye's overall axial length and various optical components are coordinated to minimize refractive error. As a result, long eyes tend to have flat corneas and short eyes tend to have steep corneas. This coordination is maintained as the child and the eyes grow, resulting in a trend toward emmetropization.

Hyperopia can be classified on the basis of structure and function. It most commonly occurs when one or more of the components of ocular refraction deviate moderately from normal, a condition known as correlational hyperopia. When one or more of these refractive components varies significantly from normal, the condition is known as component hyperopia. Relatively few individuals have the high refractive errors of component hyperopia. These classifications are based solely on the structure, not the function, of the eye and visual system. The classification of physiologic (functional) hyperopia includes persons with correlational hyperopia and those with component hyperopia who otherwise have normal ocular anatomy.

Clinically, hyperopia may be divided into three categories:[7]

- Simple hyperopia, due to normal biological variation, can be of axial or refractive etiology.
- Pathological hyperopia is caused by abnormal ocular anatomy due to maldevelopment, ocular disease, or trauma.
- Functional hyperopia results from paralysis of accommodation.

Hyperopia may also be categorized by degree of refractive error:[8]

- Low hyperopia consists of an error of +2.00 diopters (D) or less.
- Moderate hyperopia includes a range of error from +2.25 to +5.00 D.
- High hyperopia consists of an error over +5.00 D.

A hyperopia classification scheme that relates the role of accommodation to visual functioning adds an important dimension to structure-based classifications:

- Facultative hyperopia is that which can be overcome by accommodation.[8]
- Absolute hyperopia cannot be compensated with accommodation.[9]

The total magnitude of hyperopia is the sum of absolute and facultative hyperopia.

The classification of hyperopia can also be based upon the outcome of noncycloplegic and cycloplegic refractions:

- Manifest hyperopia, determined by noncycloplegic refraction, may be either facultative or absolute.
- Latent hyperopia, detected only by cycloplegia, can be overcome by accommodation.

The sum of latent and manifest hyperopia is equal to the magnitude of hyperopia.

The simplest functional classification system is based on the presence or absence of symptoms resulting from hyperopia. Significant hyperopia is defined as any degree of hyperopia sufficient to cause symptoms requiring remediation. These symptoms include vision that is blurred, inefficient, or causes discomfort. Patients with significant hyperopia may not even be aware of any problems experienced that are related to hyperopia.

The descriptions and classifications used in this Guideline are amalgamated from the above schema, because each is useful in illustrating different aspects of hyperopia and its effects on the patient. (See Appendix Figure 4 for the ICD-9-CM classification of hyperopia.) Table 1 provides an overview of physiologic and pathologic hyperopia.

1. Physiologic Hyperopia

The vast majority of cases of hyperopia are of a physiologic nature. From the perspective of physiologic optics, hyperopia occurs when the axial length of the eye is shorter than the refracting components the eye requires for light to focus precisely on the photoreceptor layer of the

Table 1. Clinical Classification of Hyperopia

	Description	Etiology	Symptoms, Signs, and Complications
Physiologic hyperopia	Hyperopia that occurs when the axial length of the eye is shorter than the refracting components the eye requires for light to focus precisely on the retina	• Hereditary factors, with some environmental influence • Relatively flat corneal curvature • Insufficient crystalline lens power • Increased lens thickness • Short axial length • Variance from normal separation of optical components of the eye	• Constant to intermittent blurred vision • Asthenopia • Red, teary eyes • Frequent blinking • Decreased binocularity • Difficulty reading • Amblyopia • Strabismus
Pathologic hyperopia	Hyperopia that results from other than normal biologic variation of the refracting components of the eye	• Maldevelopment of the eye during the prenatal or early postnatal period • Corneal or lenticular changes • Chorioretinal or orbital inflammation or neoplasms • Neurologic- or pharmacologic-based causes	• Related congenital or acquired ocular or systemic disorders

retina. Hyperopia can result from a relatively flat corneal curvature alone or in combination with insufficient crystalline lens power, increased lens thickness,[10] short axial length, or variance of the normal separation of the optical components of the eye relative to each other.

Astigmatism, the most common refractive error, is often present in conjunction with hyperopia.[4,11-18] High hyperopia is associated with high levels of astigmatism,[119] suggesting a breakdown in the process of emmetropization that results in a component-type refractive error. Hereditary factors are probably responsible for most cases of refractive error, including physiologic hyperopia, with environment playing some role in influencing the development and degree of the error.[20] However, environment probably plays a lesser role in influencing the course and magnitude of hyperopia than of myopia.

Physiologic hyperopia **is not** solely an anomaly of physiologic optics. Significant effects on visual system function are closely related to the underlying structural anomaly.[21] Active accommodation mitigates some or all of hyperopia's adverse effects on vision. The impact of accommodation is highly dependent upon age, the amount of hyperopia and astigmatism, the status of the accommodative and vergence systems, and the demands placed upon the visual system.

Active accommodation typically enables young patients to overcome facultative and latent hyperopia, but it may not be sustainable for long periods under conditions of visual stress. Signs and symptoms such as optical blur, asthenopia, accommodative and binocular dysfunction, and strabismus may develop. These signs and symptoms occur more readily and to a greater degree in manifest and absolute hyperopia. In general, younger individuals with lower degrees of hyperopia and moderate visual demands are less adversely affected than older individuals, who have higher degrees of hyperopia and more demanding visual needs.

2. Pathologic Hyperopia

Use of the term "pathologic" implies that the hyperopia has an etiology other than normal biologic variation of the refractive components of the eye. Pathologic hyperopia may be due to maldevelopment of the eye

during the prenatal or early postnatal period, a variety of corneal or lenticular changes, chorioretinal or orbital inflammation or neoplasms, or to neurologic- or pharmacologic-based etiologies. It is rare in comparison with physiologic hyperopia and may have a genetic inheritance pattern.[22, 23] Because of the relationship of pathologic hyperopia to potentially serious ocular and systemic disorders, proper diagnosis and treatment of the underlying cause may prove critical to the patient's overall health.

Microphthalmia (with or without congenital or early acquired cataracts and persistent hyperplastic primary vitreous) and this condition's often hereditary form, nanophthalmia, may produce hyperopia in excess of +20 D.[24-26] Anterior segment malformations such as corneal plana, sclerocornea, anterior chamber cleavage syndrome,[27] and limbal dermoids are associated with high hyperopia. Acquired disorders that can cause a hyperopic shift result from corneal distortion or trauma,[28] chalazion,[29] chemical or thermal burn, retinal vascular problems,[30] diabetes mellitus,[31-33] developing or transient cataract[34] or contact lens wear.[35] When extreme enough to lead to relative aphakia, ectopia lentis produces high hyperopia.[36] Conditions that cause the photoreceptor layer of the retina to project anteriorly (idiopathic central serous choroidopathy[37] and choroidal hemangioma from Sturge-Weber disease[38]) also induce hyperopia. Similarly, orbital tumors, idiopathic choroidal folds,[39-41] and edema can mechanically distort the globe and press the retina anteriorly, thereby causing hyperopia. Adie's pupil occasionally causes a mild hyperopic shift.[42] Cycloplegic agents may induce hyperopia by affecting accommodation,[43] and a variety of other drugs can produce transient hyperopia.[28]

A number of developmental disabilities and syndromes are associated with high hyperopia. Conditions having foveal hypoplasia (albinism, achromatopsia, and aniridia)[44] or early retinal degeneration (Leber's congenital amaurosis)[45] appear to disrupt emmetropization grossly and result in high hyperopia and astigmatism. Other disorders with a high prevalence of hyperopia are Aarskog-Scott, Kenny, Rubinstein-Taybi, fragile X,[46,47] and Down's syndromes.[48,49]

B. Epidemiology of Hyperopia

1. Prevalence and Incidence

Although it is difficult to specify the prevalence of hyperopia due to variations in its definition by researchers (e.g. with or without cycloplegia, spherical equivalent, least hyperopic meridian),[50] it is age related. Most full-term infants are mildly hyperopic (approximately +2.00 D),[51-53] while premature infants and those of low birthweight tend to be either less hyperopic or myopic (approximately +0.24 D).[54] The prevalence of refractive error among full-term infants has a normal bell-shaped distribution.[55] Approximately 4-9 percent of infants 6-9 months old have hyperopia greater than +3.25 D;[56-58] the prevalence of hyperopia (> +3.25 D) to 3.6 percent in the 1-year-old population.[59] Higher levels of astigmatism are associated with moderate to high hyperopia during infancy, but both tend to decrease by the age of 5 years.[60,61] Although at this age the prevalence of refractive error is reduced, its distribution still peaks toward mild hyperopia.[62] Over the next 10-15 years of life, there is a further decrease in the prevalence of hyperopia and an increase in the frequency of myopia.[63,64] With the development of presbyopia, latent hyperopia becomes manifest, contributing to an apparent increase in the prevalence of hyperopia.[65]

There is no known gender difference in the prevalence of hyperopia, but there is evidence of the influence of ethnicity on the prevalence of hyperopia. Native Americans, African Americans, and Pacific Islanders[66,67] are among the groups with the highest reported prevalence of hyperopia. A study of 1,880 Chinese schoolchildren in Malaysia showed that the prevalence of hyperopia greater than +1.25 D was only 1.2 percent.[68]

2. Risk Factors

The risk of developing clinically significant physiologic hyperopia is largely determined by a combination of hereditary factors and biologic variation.[69] Both the prevalence and magnitude of hyperopia are greatest during early childhood, decreasing in the first decade of life through the process of emmetropization. Physiologic hyperopia does not usually

develop after early childhood. There is, however, an apparent increase in the incidence of hyperopia in some presbyopic adults, likely the manifestation of latent hyperopia as a result of loss of ciliary muscle tonus and accommodation as well as modest configuration changes in the crystalline lens associated with presbyopia. In contrast, pathologic hyperopia may be associated with diabetes mellitus, contact lens wear, and a host of intraocular and orbital tumors and inflammations. Pathologic hyperopia can be acquired at any age.

C. Clinical Background of Hyperopia

1. Natural History

Most newborn infants have mild hyperopia (approximately +2.00D), with only a small number of cases falling within the moderate to high range (>3.5D).[70,71] Although emmetropization results in a gradual decrease in the level of hyperopia in most patients, the change occurs more rapidly in patients who have high degrees of hyperopia.[55,70] Infants with high hyperopia are more likely to remain significantly hyperopic throughout childhood. Infants tend to have almost twice the incidence of astigmatism as adults.[72] Young children with significant hyperopic astigmatism, especially against-the-rule astigmatism, exhibit a smaller reduction in hyperopia during emmetropization than children without significant astigmatism.[70,73]

Infants with moderate to high hyperopia (> +3.50 D) are up to 13 times more likely to develop strabismus by 4 years of age if left uncorrected, and they are 6 times more likely to have reduced visual acuity than infants with low hyperopia or emmetropia.[57,58] The association of high hyperopia with a greatly increased risk of amblyopia and strabismus is a major justification for universal vision evaluation of young children.[74,75] There is also a strong (almost 90%) association of at least modest degrees of hyperopia with infantile esotropia.[76] Anisometropic hyperopia persisting beyond 3 years of age is also a risk factor for the development of strabismus and amblyopia.[77,78]

During the school years, there is a slow but continued decreasing trend in the incidence and the magnitude of hyperopia,[53] except in patients with

high hyperopia, whose refractive error is more likely to remain relatively unchanged. During the years of presbyopia development, latent hyperopia may become manifest, requiring the use of both distance and near correction. Yet in older individuals (>75 years of age) a myopic refractive shift may ensue, likely due to crystalline lens changes.[79-81]

2. Common Signs, Symptoms, and Complications

The interrelationship between structure and function in the visual system is the basis for many of the signs and symptoms experienced by patients with hyperopia (see Table 1). Compound hyperopic astigmatism, especially when it is oblique or against the rule, causes correspondingly more visual problems than simple hyperopia of equal magnitude. As the levels of hyperopia and astigmatism increase, visual acuity decreases as a result of both optical blur and amblyopia.[8]

Young persons with hyperopia generally have sufficient accommodative reserve to maintain clear retinal images without producing asthenopia. However, both younger and older hyperopic patients, even those with mild hyperopia, may be symptomatic as a result of inadequate accommodative reserves for their levels of hyperopia. When the level of hyperopia is too great or the accommodative reserves are insufficient, due to age or fatigue, blurred vision and asthenopia develop. Presbyopia brings an increase in absolute hyperopia, causing blur, especially at near. The influence of accommodation on the vergence system also plays a role in the presence or absence of symptoms in patients with hyperopia. Individuals with esophoria and inadequate negative fusional vergence ability are frequently symptomatic because of the uncorrected hyperopia.

Among the signs and symptoms of hyperopia are red or tearing eyes, squinting and facial contortions while reading, ocular fatigue or asthenopia, frequent blinking, constant or intermittent blurred vision, focusing problems, decreased binocularity and eye-hand coordination, and difficulty with or aversion to reading. The presence and severity of these symptoms varies widely. Some young patients with hyperopia, including those with moderate and high hyperopia, may be relatively free of signs and symptoms.

The major complications of moderate and high physiologic hyperopia in children are amblyopia and strabismus. Children who had significant hyperopia during infancy are much more likely to develop amblyopia and strabismus by 4 years of age.[71,73,82-85] The majority of patients with early-onset esotropia are hyperopic.[76] The presence of anisometropic hyperopia further increases the risk of strabismus and amblyopia.[71] Early detection and treatment of hyperopia may reduce the incidence and severity of these complications.[86] Levels greater than 1.00 D of hyperopic anisometropia and 5.00 D of isometropic hyperopia are considered amblyogenic.*

Uncorrected hyperopia (>3.5 D in one meridian) may contribute to poor motor and cognitive development in younger children (9 months to 5.5 years)[87,88] and/or learning problems in some older children.[16, 89-91] The precise mechanism of this relationship is unclear, but optical blur, accommodative and binocular dysfunction, and fatigue all appear to play roles. Uncorrected infant hyperopia has been associated with mild delays in visuocognitive and visuomotor development.[58] However, it appears that this delay may be eliminated after weeks of full-time hyperopic spectacle wear by 3- to 5-year-olds.[92] The substantial number of school-age children and young adults who have uncorrected significant hyperopia is evidence of the potential impact of this learning-related vision problem and the need for early detection.

3. Early Detection and Prevention

The early detection of moderate and high hyperopia may be accomplished by effective vision screening of young children.[93-98] The available vision screening procedures have certain advantages and disadvantages:

*Refer to the Optometric Clinical Practice Guideline for Care of the Patient with Amblyopia.

- The +1.50 D test, usually as a component of the modified clinical procedure of vision screening, has been widely used for refractive screening for hyperopia.

- Photoscreening and autorefractor screening performed by professionals or lay persons can be a useful tool in early refractive screening.[98-101]

- Screening retinoscopy requires personnel with more skills than photoscreening or autorefraction screenings.

- Screening by visual acuity testing with age-appropriate techniques is likely to identify only hyperopia associated with high astigmatism and/or amblyopia. Persons with simple hyperopia are usually able to obtain good visual acuity through active accommodation.

- Stereopsis screening alone is of limited value in identifying significant hyperopia, unless it occurs in conjunction with amblyopia or strabismus.[98,102]

Inasmuch as physiologic hyperopia exists during infancy, prevention is impossible. Modification of the degree of hyperopia during early childhood has been the topic of recent speculation,[103] but clinical application of the possible procedures is not foreseeable. Full optical correction for hyperopia during infancy may interfere with the process of emmetropization.[104] However, partial spectacle correction in infants with significant hyperopia does not impair the normal emmetropization of refraction over the first 36 months of life.[105] and may reduce the incidence of subsequent strabismus.[57,73] Conversely, treatment reduces the risk for strabismus and amblyopia. One large study has shown that partial spectacle correction of hyperopia during infancy dramatically reduces the risk of amblyopia and strabismus.[57] Pathologic hyperopia, being rare and having multiple etiologies and associations, is usually detected only after significant visual problems have developed and the patient receives a comprehensive eye examination.

II. CARE PROCESS

A. Diagnosis of Hyperopia

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

1. Patient History

The major components of the patient history include a review of the nature of the presenting problem and chief complaint, ocular and general health history, developmental and family history, use of medications and medication allergies, and vocational and avocational vision requirements. Parents of young children may suspect an eye or vision problem if the child frequently has red, irritated or tearing eyes, difficulty with the clarity or comfort of vision, or actual or suspected crossing of the eyes. Older children may complain to parents or teachers about visual symptoms, or they may have failed vision screening performed at school or in the pediatrician's office. Adults with even mild hyperopia may develop visual problems after extensive use of the eyes and in poor illumination. Most presbyopic patients complain about increasing difficulty with near vision. Although blurred vision at near and unspecified visual discomfort are the most common complaints of patients with hyperopia, there are no complaints specifically pathognomonic of hyperopia.

A positive family history of hyperopia, amblyopia, or strabismus increases the likelihood that a young patient with suspected eye or vision problems actually has a similar problem.[106]

2. Ocular Examination

a. Visual Acuity

The effect of hyperopia on visual acuity depends upon the magnitude of the hyperopia and the patient's age, visual demands, and accommodative amplitude available to overcome the hyperopia. Young patients with low to moderate facultative hyperopia generally have normal visual acuity, but when visual demands are high, they may experience blurred vision and asthenopia. Visual acuity testing of patients with high hyperopia, even when the patients are young, is likely to reveal measurable deficits, especially under significant visual demand. Although visual acuity may be reduced at times, especially at near, the objective measure of visual acuity in patients with latent hyperopia is usually normal. However, when such patients become visually fatigued, they demonstrate inconsistent levels of near, and sometimes distance visual acuity. Patients with moderate and high hyperopia are at significantly increased risk for refractive and strabismic amblyopia. The patient who has never been optically corrected for a high degree of hyperopia, with or without astigmatism, is at risk for isoametropic amblyopia.[107-109]

Older patients with hyperopia invariably experience reduced vision, especially at near. Prepresbyopic and early presbyopic patients with hyperopia manifest deficits of near vision before distance visual acuity is adversely affected. In patients with absolute hyperopia, the reduction in visual acuity at both distance and near is proportionate to the degree of absolute hyperopia.

b. Refraction

Retinoscopy is the most widely used procedure for objective measurement of hyperopia.[110]

Procedures for measuring refractive error include static retinoscopy, subjective refraction, and autorefraction:

- **Static retinoscopy.** When the patient consistently views a distant object and accommodation is relaxed, this procedure provides an accurate and repeatable measure of manifest hyperopia.[111,112] Patients with significant hyperopia, latent hyperopia, or accommodative esotropia may mask much of their hyperopia during noncycloplegic retinoscopy. Additional latent hyperopia may be uncovered using a fogging procedure or cycloplegia.

  Although primarily a method of measuring refractive error, static retinoscopy provides other useful information. By directly viewing the color, brightness, and motion of the retinoscopic reflex, the clinician can assess the patient's accommodation, fixation, and other dynamic aspects of the visual system. These findings provide a better understanding of vision and refraction than is obtainable under cycloplegia.

  The patient's steady fixation and relaxation of accommodation are critical for accurate static retinoscopy. Off-axis retinoscopy and incomplete relaxation of accommodation induce substantial measurement error. The difficulty of obtaining adequate fixation in young children may necessitate the use of alternative fixation targets, such as videos or attention-grabbing toys instead of less interesting targets such as visual acuity charts. In addition, when performing retinoscopy on a strabismic patient, the examiner should consider monocular retinoscopy to reduce measurement error. The examiner can best ascertain the steadiness of fixation and accommodation by evaluating the appearance of the retinoscopic reflex. The use of bilateral fogging lenses (e.g., +2.00 D) and simultaneous retinoscopy of both eyes minimizes the risk of unbalanced refraction. Hand-held or frame-mounted trial lenses or retinoscopy racks are essential in reducing the distraction of the phoropter in young children and patients with special needs.[113]

- **Nearpoint retinoscopy.** This procedure may be a useful alternative for young children who are resistant to basic static retinoscopy because the child's attention is drawn to the only light source to be seen, the retinoscope. Less distracting than standard static retinoscopy, the procedure is performed in a completely

darkened room at a distance of 50 cm with either a retinoscopy rack or hand-held trial lenses for optical correction. The net refractive error is calculated by subtracting 1.25 D from the gross finding.[114,115] Although underestimation of hyperopia is more likely with near retinoscopy than with cycloplegic retinoscopy,[116,117] there may be occasions when cycloplegia is not feasible and near retinoscopy is the only refractive procedure available. For older children, MEM dynamic retinoscopy may be useful in identifying a lag of accommodation at near, which is a good indicator of latent hyperopia.

- **Cycloplegic retinoscopy.** This procedure measures the total amount of hyperopia, including the latent component. The use of cycloplegia is important in assessing hyperopic refractive error in children.[118] A variety of cycloplegic (anticholinergic) agents, including atropine, cyclopentalate, and tropicamide, have been recommended and used.[*] Atropine (0.5% and 1% concentrations in ointment and drop form, respectively) provides maximum cycloplegia; however, it usually requires administration of the drug up to 3 days before the refraction and its effects are excessively longlasting.[119] Cyclopentalate hydrochloride (0.5% and 1% drops) is a good compromise between efficacy and duration (maximum cycloplegic effect at 35-45 minutes), and is the most widely used cycloplegic agent available.[120] A 2% solution of cyclopentalate is available but generally should not be used because of increased potential for adverse effects. Tropicamide (0.5% and 1% drops) has been effective in detecting milder cases of hyperopia among school-age children,[121] but it may not provide as great a degree and consistency of cycloplegia as the other drugs, especially in patients with dark irides and significant hyperopia.[8] Although each of these drugs poses some risk for adverse reaction, tropicamide is the least likely to cause adverse effects. All of these agents dilate the pupil, making cycloplegic retinoscopy somewhat more difficult to interpret due to confusing peripheral light reflexes. Because

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

mydriasis can be greater than cycloplegia, paying careful attention to the appearance and quality of the retinoscopic reflex is a better method of confirming the completeness of cycloplegia than mydriasis alone. Although the amount of hyperopia identified by cycloplegic retinoscopy in cases of latent hyperopia and accommodative esotropia may be considerable, it is generally less than 1.00 D more than that measured by careful noncycloplegic retinoscopy conducted under fogging.[8]

- **Subjective refraction.** This procedure is preferred for determining the refractive correction to prescribe, especially for the older child or adult patient, because it is based on the patient's actual acceptance of the prescription. However, patients with hyperopia and accommodative esotropia or other binocular anomalies often require refractive corrections that differ from those derived from subjective refraction alone. Although the static retinoscopy finding may accurately represent the refractive error, the indicated correction in an unmodified form may not prove suitable for the patient's visual needs. Subjective refraction often employs the static retinoscopy finding as a starting point. Alternatively, subjective refraction can substitute for static retinoscopy by use of a series of procedures, including fogging, clock-dial and cross-cylinder techniques for cylindrical determination, duochrome and visual acuity assessment for the spherical component, and binocular balance. Regardless of the specific subjective refraction procedures used, the clinician should base the final prescription on careful consideration of the patient's individual visual needs. Studies have shown that intraexaminer and interexaminer reliability of subjective refraction is acceptably high.[122]

  A subjective refraction may follow cycloplegic retinoscopy. This refraction increases the precision of the retinoscopy finding to provide maximum visual acuity. Under cycloplegia, the patient's responses to changes in lens power are different in character and less precise than under noncycloplegic conditions; nevertheless, useful information may be obtained by this means.

- **Autorefraction.** As an alternative to static retinoscopy, autorefraction yields replicable results, but the procedure's reliability and validity are lower than for subjective refraction.[122] Few available instruments appear to control accommodation adequately in children. Noncycloplegic auto refractions are particularly inaccurate in measuring hyperopia,[123,124] but they are accurate compared with subjective refraction under cycloplegic conditions.[124] Autorefraction instruments have internal targets and lack the means of testing binocular balance; thus, they have limited use in young patients. Still, autorefractors are useful as a starting point for subjective refraction.

c. Ocular Motility, Binocular Vision, and Accommodation

Along with assessment of refractive error, patients with hyperopia should undergo evaluation of ocular motility, binocular vision, and accommodation. Anomalies of any of these visual functions may result in visual acuity and visual performance deficits. Among the procedures that may be used are versions, both monocular and alternating cover tests, and testing of near point of convergence, accommodative amplitude and facility, and stereopsis. The specific tests selected should be age-appropriate. To determine the best spectacle prescription for maintaining ocular alignment and comfortable accommodative demand, the optometrist should assess the effect of plus lens power on any dysfunctions prior to cycloplegia.

d. Ocular Health Assessment and Systemic Health Screening

The clinician should assess ocular health to rule out or diagnose any disease that may cause hyperopia. The assessment may include, but is not limited to, pupillary responses, confrontation visual fields, color vision, intraocular pressure when appropriate for age and history, and thorough assessment of the health of the anterior and posterior segments of the eye and adnexa. Examination through a dilated pupil by biomicroscopy and binocular indirect ophthalmoscopy is generally required for thorough evaluation of the ocular media and posterior segment.

B. Management of Hyperopia

1. Basis for Treatment

Significant hyperopia, if uncorrected, can produce visual discomfort, blurred vision, amblyopia, and binocular dysfunction, including strabismus, and contribute to learning problems. Treatment should be initiated both to remediate symptoms and to reduce the future risk of vision problems resulting from the hyperopia.

The clinician should tailor specific elements of treatment to individual patient needs. Among the factors to consider when planning treatment and management strategies are the magnitude of the hyperopia, the presence of astigmatism or anisometropia, the patient's age, the presence of an associated esotropia and/or amblyopia, the status of accommodation and convergence, the demands placed on the visual system, and the patient's symptoms. (See Appendix Figure 1 for an overview of patient management strategy.)

2. Available Treatment Options

Among several available treatments for hyperopia-related symptoms, optical correction of the refractive error with spectacles and contact lenses is the most commonly used modality. It is the optometrist's responsibility to advise and counsel the patient regarding the options and to guide the patient's selection of the appropriate spectacles or contact lenses. Vision therapy and modification of the patient's habits and environment can be important in achieving definitive long-term remediation of symptoms. Pharmaceutical agents or refractive surgery may also be useful in treating some patients.

a. Optical Correction

The primary modality for treating significant hyperopia is correction with spectacles. Plus-power spherical or spherocylindrical lenses are prescribed to shift the focus of light from behind the eye to a point on the retina. Accommodation plays an important role in determining the prescription. Some patients with hyperopia do not initially tolerate the

full correction indicated by the manifest refraction, and many patients with latent hyperopia do not tolerate the full correction of hyperopia indicated under cycloplegia. However, young children with accommodative esotropia and hyperopia generally require only a short period of adaptation to tolerate full optical correction. Patients with latent hyperopia who prove intolerant to the use of full or partial hyperopic correction may benefit from initially wearing the correction only for near viewing; alternatively, trial use of a short-acting cycloplegic agent (e.g., 1% cyclopentalate) may enhance acceptance of the optical correction.[125] Patients with absolute hyperopia are more likely to accept nearly the full correction, because they typically experience immediate improvement in visual acuity.

To determine the final spectacle lens prescription, the clinician should carefully consider the patient's vision needs. The lenses prescribed may be either single vision or multifocal. Newer high-index lens materials and aspheric lens designs have reduced the thickness and weight of high plus-power lenses, increasing their wearability and patient acceptance. Spectacles, especially those with lenses of polycarbonate material, provide protection against trauma to the eye and orbital area.

Soft or rigid contact lenses are an excellent alternative for some patients. In patients who resist wearing spectacles, compliance with wearing the optical correction may be enhanced due to improved cosmesis. Contact lenses reduce aniseikonia and anisophoria in persons with anisometropia, improving binocularity.[126] In persons with accommodative esotropia, contact lenses decrease the accommodative and convergence demands, reducing or eliminating esotropia at near to a greater extent than spectacles.[127] Multifocal or monovision contact lenses may be considered for patients who require additional near correction but resist the use of multifocal spectacles because of the appearance.

The initial cost of contact lenses may be higher than that of spectacles, and there are additional responsibilities and costs associated with the proper care of contact lenses. Patients who wear contact lenses are at increased risk for ocular complications due to corneal hypoxia, mechanical irritation, or infection; nevertheless, improved vision makes contact lens wear a valuable treatment option for compliant patients.

b. Vision Therapy

Vision therapy can be effective in the treatment of accommodative and binocular dysfunction resulting from the hyperopia.[128] Habitual accommodative response in persons with hyperopia often does not respond to lens correction alone, and vision therapy may be required to remediate accommodative dysfunction. Accommodative esotropia secondary to hyperopia that is moderate to high may reduce binocular skills, which can be improved by the wearing of a prescribed lens correction and vision therapy.[129]

c. Medical (Pharmaceutical)Therapy

Miotics may be indicated for selected patients who cannot tolerate wearing spectacles. The potentially serious adverse effects of anticholinesterase agents[130] limit their usefulness. Anticholinesterase agents such as diisopropylfluorophosphate (DFP) and echothiophate iodide (Phospholine Iodide, PI) have been used in some patients with accommodative esotropia and hyperopia to reduce a high accommodative convergence-to-accommodation (AC/A) ratio and improve alignment of the eyes at near.[131] These drugs mimic the accommodative effect of plus lenses without the use of spectacles or contact lenses.

d. Modification of the Patient's Habits and Environment

Reduction in visual demand does not reduce actual levels of hyperopia, but it can lessen the symptoms, even in patients with optical correction. Thus, modification of the patient's habits and visual environment is occasionally useful as an adjunct therapy. Such modifications include, but are not limited to, improving lighting or reducing glare, using better quality printed material, decreasing temporal demands, and ensuring optimal visual hygiene and ergonomic conditions at the computer terminal.

e. Refractive Surgery

Several refractive surgery techniques to correct hyperopia are under development. Among procedures studied as possible therapies for

hyperopia are Holmium:YAG laser thermal keratoplasty,[132] automated lamellar keratoplasty,[133] spiral hexagonal keratotomy,[134] excimer laser,[135] and clear lens extraction with intraocular lens implantation.[136] A review of 36 articles on studies of the efficacy and safety of refractive surgery for hyperopia found that surgery provides effective, safe correction for lower ranges of hyperopia (<3.00 D).[137] Although its long-term outcome still needs to be established, LASIK has received approval from the U.S, Food and Drug Administration for treating hyperopia up to +6.00 D.[138]

3. Management Strategies for Hyperopic Correction

There is no universal approach to the treatment of hyperopia. Each patient should be considered in terms of age, degree of symptoms, amount of hyperopia, state of accommodation, visual acuity, and efficiency during the performance of visual tasks.[139] The goals of treatment are to reduce accommodative demand and to provide clear, comfortable vision and normal binocularity. It is not simply determination of the lens power required to focus light onto the retina, but a complex approach encompassing the patient's vision needs and sensitivity. The following are specific management strategies appropriate for different age groups and conditions.

a. Young Children

Young children (birth-10 years of age) with low to moderate hyperopia, but without strabismus, amblyopia, or other significant vision problems, usually require no treatment. However, even occasional evidence of decreased visual acuity, binocular anomalies, or functional vision problems may signal the need for treatment. Whereas the effects of uncorrected hyperopia may manifest as visual perceptual dysfunction reading difficulties, or failure in school, any child with hyperopia who is experiencing learning or other school difficulties needs careful assessment and may require treatment.[91,140,141]

In most young hyperopic children, the process of emmetropization leads to a gradual reduction in the degree of hyperopia by 5-10 years of age. Some children do not go through this process however, they remain significantly hyperopic and at increased risk for developing strabismus

and amblyopia.[75] Although patients under age 5 who have over 3.25 D of hyperopia appear to benefit from early optical correction to reduce the risk for strabismus and amblyopia,[57,58] the results of animal studies suggest that early optical correction, especially in infants, can interfere with emmetropization. Thus, early treatment has the potential to result in maintenance of the refractive error throughout life.[103] Nevertheless, clinical pediatric studies suggest that partial hyperopic prescriptions do not impede emmetropization of infants up to the age of 3 years.[58]

Clinicians generally should prescribe optical correction of hyperopia for young children who have moderate to high hyperopia. A survey of prescribing patterns suggests that for 2-year-olds many practitioners use a threshold of +3.0 D of bilateral asymptomatic hyperopia, while some use a threshold of +5.0 D.[139] Hyperopic correction should also be prescribed along with other interventions (e.g., occlusion or active vision therapy) for all young patients with actual or suspected amblyopia or strabismus. Optical correction may be deferred for some patients with moderate hyperopia, but such patients should be considered "at risk" and re-examined periodically.

Optical correction should be based on both static and cycloplegic retinoscopy, accommodative and binocular assessment, AC/A ratio, and the correction should be modified as needed to facilitate binocularity and compliance.[140] Careful followup is essential, and frequent lens changes may be needed. A significant increase in hyperopia is not unusual after the patient has worn optical correction for even a short time, due to the manifestation of latent hyperopia. When compliance proves difficult, the clinician may encourage acceptance of the prescribed treatment by using cycloplegic agents to blur uncorrected vision.[125] Contact lenses may be a good alternative for patients who do not comply with prescriptions for spectacle wear, especially those with anisometropia, high hyperopia with or without nystagmus, and hyperopia with accommodative esotropia.

The clinician should give special consideration to several specific categories of problems in young children who have significant hyperopia. Prior to the onset of accommodative esotropia, which usually becomes evident at about 2-3 years of age, few children exhibit obvious signs of ocular problems, with the exception of intermittent esotropia in

children who are ill or very tired. Early screening for refractive error usually detects hyperopia, but due to the relative infrequency of refractive screening, many children with underlying moderate to high hyperopia go undetected until the appearance of frank strabismus. Appropriate treatment includes the use of either single-vision or multifocal spectacles depending upon binocular and accommodative status. Alternatives for treating concurrent amblyopia are patching and active vision therapy. In rare circumstances, optical correction converts hyperopia and accommodative esotropia to consecutive exotropia. With careful consideration of the status of accommodation and binocularity, the optometrist may adjust the optical correction to achieve resolution of this problem.[142]

Less commonly, young children with bilateral high hyperopia develop isoametropic amblyopia due to the resulting constant state of severely blurred vision.[108] Such patients may have an associated esotropia, or conversely, may not manifest esotropia because they make no attempt to accommodate. Optical correction of this condition is indicated, to prevent or treat the associated amblyopia and/or strabismus. This treatment warrants careful monitoring, because a previously nonexistent esotropia may present itself following correction. Partial correction may inadvertently stimulate accommodative esotropia, because the patient now has good reason to attempt to overcome the remaining uncorrected hyperopia. Treatment to improve vision in the child with amblyopia may take a few years, but improvement is usually possible with full-time spectacle wear and/or patching.[*]

Among mentally and multiply handicapped children, the prevalence of ocular problems, including significant hyperopia, is higher than in normal, healthy children.[123] The inability of many of these patients to describe their visual impairments verbally makes early and periodic eye and vision examinations imperative. These patients require the same manner of optical correction of their hyperopia as other children. Practical issues associated with providing and maintaining optical

[*]Refer to the Optometric Clinical Practice Guideline for Care of the Patient with Amblyopia.

correction may influence the actual use of and benefit from the correction.

b. Older Children and Pre-Presbyopic Adults (Ages 10-40 Years)

Many persons between the ages of 10 and 40 years who have low hyperopia require no correction, because they have no symptoms. Ample accommodative reserves shelter them from visual problems related to their hyperopia. Under increased visual stress, such persons may develop symptoms that require correction. Wearing prescribed lenses with low amounts of plus power usually alleviates the problem. Patients with moderate degrees of hyperopia are more likely to require at least part-time correction, especially those who have significant near demands or have accommodative or binocular anomalies.

Either optical correction or vision therapy may be useful in treating accommodative or binocular dysfunction associated with uncorrected low-to-moderate hyperopia. Vision therapy may be instituted initially or after optical correction for patients who have significant binocular vision problems. The effects of visual habits and environment play an increasing role in determining the need for and characteristics of treatment.

Relatively few persons with high degrees of hyperopia will have escaped detection and treatment by the age of 10-20 years. Visual stress and decreased visual acuity are likely in such patients, who must rely on optical correction. The wide spectrum of philosophies concerning appropriate treatment ranges from providing minimal plus lenses that may alleviate symptoms to prescribing full plus correction to relax accommodation. The middle position of prescribing one-half to two-thirds of plus lens power takes into account the relationship of latent hyperopia to manifest hyperopia and is a reasonable approach for many patients. Clinicians may base prescription decisions on the power required to provide optimal visual acuity and normal accommodative and binocular function. Patients often become quite dependent upon this correction.

By the age of 30-35 years, most previously asymptomatic, uncorrected patients begin to experience blur at near and visual discomfort under strenuous visual demand. Facultative hyperopia can no longer be sustained comfortably, due to decreasing accommodative amplitudes. Latent hyperopia should be suspected when symptoms occur in conjunction with a lower-than-expected amplitude of accommodation for the patient's age. Cycloplegic retinoscopy can help identify this latent component.

When persons reach their mid-thirties, accommodation takes noticeably longer, while facility decreases, causing associated vision problems in many hyperopic persons previously free of symptoms. A prescription for the distance manifest (noncycloplegic) refraction for the patient to wear as needed (i.e., part time) often suffices. With increasing age and visual demands at near, the patient may require additional correction. Before prescribing a permanent pair of spectacles, the optometrist may lend the patient a pair of spectacles (i.e., over-the-counter reading glasses) to demonstrate the potential benefits of optically correcting latent hyperopia. In addition, the optometrist should tell the patient that under certain circumstances, correcting near vision can adversely affect distance visual acuity. A good alternative for some patients is the prescription of contact lenses, which can relax accommodation more effectively than spectacles.

c. Presbyopia

With the onset of presbyopia, changing focus becomes progressively more difficult, especially in poor illumination. Increased blur at near necessitates correction for near and often for distance as well.[*] Prescribing an optical correction for most or all of the distance manifest refraction, along with a near addition, can greatly improve the patient's vision and comfort. Hyperopia equal to or greater than 1.00-1.50 D generally requires full-time distance correction, with a near addition for patients over about age 45. As facultative hyperopia becomes absolute, more plus power at distance is required. Progressive multifocal lenses enable clear focusing at a range of finite distances. A monovision,

[*]Refer to the Optometric Clinical Practice Guideline for Care of the Patient with Presbyopia.

bifocal, or multifocal contact lens prescription is an option for some patients.

d. Pathologic Hyperopia

The underlying cause, rather than hyperopia itself, is the chief concern in patients with pathologic hyperopia. Because the causes of pathologic hyperopia are both uncommon and diverse, general statements concerning treatment must be limited to the need to correct the hyperopia in the best manner possible, depending upon the underlying etiology. Conditions of a developmental or anatomic nature are rarely progressive. When useful vision is thought to be obtainable, the treatment of hyperopia resulting from non-progressive conditions is similar to that for physiologic hyperopia.

4. Patient Education

The optometrist should inform the patient, and parents of children with hyperopia, of the diagnosis, signs, symptoms, clinical course, and treatment options. Although hyperopia only occasionally threatens the maintenance of good vision, it may have an impact on the quality of vision and the patient's level of comfort. Education about hyperopia is especially important for parents and children for whom amblyopia, strabismus, and learning-related issues are critical.

Demonstrating hyperopia by placing minus lenses in front of the parents' eyes and having them attempt reading, provides parents a graphic experience. All patients, regardless of age or the characteristics of refractive error, should also receive education about how their visual environment and their personal habits affect their visual status. The optometrist can help patients and parents understand that slight modification of these factors may greatly benefit their visual comfort and efficiency.

5. Prognosis and Followup

Physiologic hyperopia is not progressive. Therefore, the prognosis, which the clinician can give at diagnosis, is generally excellent, except

for those patients with both hyperopia and amblyopia or strabismus, for whom the prognosis is less certain. Appropriate optical correction almost always leads to clear and comfortable single binocular vision. Younger children who have significant hyperopia associated with amblyopia, strabismus, or anisometropia require intensive followup and treatment for their more complex problems, starting as early as 3-6 months of age. The timing of periodic preventive optometric care for uncomplicated hyperopia depends upon the patient's age and circumstances.[*] Children with hyperopia may require followup as often as every 3-6 months, depending upon the concern for development of strabismus and/or amblyopia. For asymptomatic adults, followup every 1 or 2 years is generally sufficient.

Patients with pathologic hyperopia require treatment of their underlying conditions and, when indicated, referral to another eye care provider for special services. All patients receiving treatment for hyperopia with persistent symptoms require additional followup care to remediate their problems. (See Appendix Figure 3 for the frequency and composition of evaluation and management visits for hyperopia.)

[*]Refer to the Optometric Clinical Practice Guideline for Comprehensive Adult Eye and Vision Examination and the Optometric Clinical Practice Guideline for Pediatric Eye and Vision Examination.

CONCLUSION

Hyperopia is a common refractive disorder that has been overshadowed by myopia in the public perception, vision research, and the scientific literature.[144] Although uncorrected myopia has a greater adverse effect on visual acuity than uncorrected hyperopia, the close association between hyperopia, amblyopia, and strabismus, especially in children, makes hyperopia a greater risk factor than myopia for a greater degree of permanent vision loss. The early diagnosis and treatment of significant hyperopia and its consequences can prevent a significant amount of visual disability in the general population. Because hyperopia is usually not readily apparent, preventive examination of all young children is essential. Periodic eye and vision examinations are needed thereafter to help ensure the provision of treatment appropriate to the changing visual needs of the hyperopic patient.

III. REFERENCES

1. Williams, WR, Latif AHA, Hannington L, Watkins DR. Hyperopia and educational attainment in a primary school cohort. Arch Dis Child. 2005 Feb;90(2):150-3.

2. Donders FC. On the anomalies of accommodation and refraction of the eye. London: New Syndenham Society, 1864:80-6.

3. Sorsby A, Leary GA, Richards MJ. Correlation ametropia and component ametropia. Vision 1962; 2:309-13.

4. Sorsby A, Benjamin B, Davey JB, et al. Emmetropia and its aberrations. Medical Research Council Special Report Series no. 293. London: Her Majesty's Stationery Office, 1957.

5. Sorsby A, Benjamin B, Sheridan M, Leary GA. Refraction and its components during growth of the eye from the age of three. Medical Research Council Special Report Series no. 301. London: Her Majesty's Stationery Office, 1961.

6. Sorsby A. The functional anomalies. Section I. Refraction and accommodation. Modern ophthalmology. Philadelphia: JB Lippincott, 1972:9-29.

7. Benjamin WJ, Borish IM. Borish's clinical refraction, 2nd ed. St. Louis: Butterworth Heinemann, 2006: 9-11.

8. Augsburger AR. Hyperopia. In: Amos JF, ed. Diagnosis and management in vision care. Boston: Butterworths, 1987:101-19.

9. Morgan MW. The nature of ametropia. Am J Optom 1947; 24:253-61.

10. Mutti DO, Zadnik K, Fusaro RE, et al. Longitudinal changes in the equivalent refractive index of the crystalline lens in childhood. Invest Ophthalmol Vis Sci 1995; 36:S939.

11. Bannon RE, Walsh R. On astigmatism. I. Am J Optom 1945; 22:101-11.

12. Bannon RE, Walsh R. On astigmatism. II. Am J Optom 1945; 22:162-79.

13. Stenstrom S. Investigation of the variation and the covariation of the optical elements of human eyes. Part I. Am J Optom 1948; 25:218-32.

14. Stenstrom S. Investigation of the variation and the covariation of the optical elements of human eyes. Part II. Am J Optom 1948; 25:286-99.

15. Stenstrom S. Investigation of the variation and the covariation of the optical elements of human eyes. Part III. Am J Optom 1948; 25:340-50.

16. Stenstrom S. Investigation of the variation and the covariation of the optical elements of human eyes. Part IV. Am J Optom 1948; 25:388-97.

17. Stenstrom S. Investigation of the variation and the covariation of the optical elements of human eyes. Part V. Am J Optom 1948; 25:438-49.

18. Stenstrom S. Investigation of the variation and the covariation of the optical elements of human eyes. Part VI. Am J Optom 1948; 25:496-504.

19. Kronfield PC, Devney C. The frequency of astigmatism. Arch Ophthalmol 1930; 4:873-84.

20. Mutti DO, Zadnik K, Adams AJ. Myopia—the nature versus nurture debate goes on. Invest Ophthalmol Vis Sci 1996; 37:952-7.

21. Rosner J. Hyperopia. In: Grosvenor T, Flom M, eds. Refractive anomalies. Research and clinical applications. Boston: Butterworth-Heinemann, 1991:121-30.

22. Traboulsi EI. Ocular malformations and developmental genes. J APPOS 1998; 2:317-23.

23. Fuchs J, Holm K, Vilhelmsen K, et al. Hereditary high hypermetropia in the Faroe Islands. Ophthalmic Genet 2005; 26:9-15.

24. Moore BD. Changes in the aphakic refraction of children with unilateral congenital cataracts. J Pediatr Ophthalmol Strabismus 1989; 26:290-5.

25. Karr DJ, Scott WE. Visual acuity results following treatment of persistent hyperplastic primary vitreous. Arch Ophthalmol 1986; 104:662-7.

26. Zwann J. Interactions in lens development and eye growth: basic facts and clinical implications. In: Cottlier E, ed. Congenital cataracts. Austin, TX: RG Landes, 1994:261-7.

27. Waring GO, Rodrigues MM, Laibson PR. Anterior chamber cleavage syndrome. A stepladder classification. Surv Ophthalmol 1975; 20:3-27.

28. Casser-Locke L. Induced refractive and visual changes. In: Amos JF, ed. Diagnosis and management in vision care. Boston: Butterworths, 1987:313-67.

29. Halpern JI, Phillips SB. Acquired hyperopia due to a chalazion. Ann Ophthalmol 1995; 27:25-6.

30. Saxena RC, Saxena S, Nath R. Hyperopia in branch vein occlusion. Ann Ophthalmol 1995; 27:15-8.

31. Marmor MF. Transient accommodative paralysis and hyperopia in diabetes. Arch Ophthalmol 1973; 89:419-21.

32. Gwinup G, Villareal A. Relationship of serum glucose concentration to changes in refraction. Diabetes 1976; 25:29-31.

33. Eva PR, Pascoe PT, Vaughn DG. Refractive changes in hyperglycemia: hyperopia not myopia. Br J Ophthalmol 1982; 66:500-5.

34. Humphriss D. Transient monocular hypermetropia with transient lens opacities. Am J Optom 1969; 46:52-5.

35. Rengstorff RH. Corneal curvature and astigmatic changes subsequent to contact lens wear. J Am Optom Assoc 1965; 36:996-1000.

36. Nelson LB, Maumenee IH. Ectopia lentis. Surv Ophthalmol 1982; 27:143-60.

37. Keller JT, Polse KA. Central serous retinopathy with transitory monocular hypermetropia: a case report. Am J Optom 1972; 49:793-6.

38. Susac JO, Smith JL, Scelfo RJ. The "tomato catsup" fundus in Sturge-Weber syndrome. Arch Ophthalmol 1974; 92:69-70.

39. Duke-Elder S, MacFaul PA. The ocular adnexa. Lacrimal, orbital and paraorbital diseases. In: Duke-Elder S, ed. System of ophthalmology, vol 13, pt 2. St. Louis: CV Mosby, 1974:1023.

40. Dailey RA, Mills RP, Stimac GK, et al. The natural history and CT appearance of acquired hyperopia with choroidal folds. Ophthalmology 1986; 93:1336-42.

41. Jacobson DM. Intracranial hypertension and the syndrome of acquired hyperopia with choroidal folds. J Neuro-Ophthalmol 1995; 15:178-85.

42. Bell RA, Thompson HS. Ciliary muscle dysfunction in Adie's syndrome. Arch Ophthalmol 1978; 96:638-42.

43. Thompson HS, Newsome DA, Lowenfield IE. The fixed dilated pupil. Sudden iridoplegia or mydriatic drops? A simple diagnostic test. Arch Ophthalmol 1971; 86:21-7.

44. Nathan J, Kiely PM, Crewther SG, Crewther DP. Disease-associated visual image degradation and spherical refractive errors in children. Am J Optom Physiol Opt 1985; 62:680-8.

45. Foxman SG, Heckenlively JR, Bateman JB, Wirtschfter JD. Classification of congenital and early onset retinitis pigmentosa. Arch Ophthalmol 1985; 103:1502-6.

46. Maino D, Schlange D, Maino J, Caden B. Ocular anomalies in fragile X syndrome. J Am Optom Assoc 1990; 61:316-23.

47. Storm RL, Pebenito R, Ferretti C. Ophthalmologic findings in the fragile X syndrome. Arch Ophthalmol 1987; 105:1099-102.

48. Wesson M, Maino D. Oculo-visual findings in Down's syndrome, cerebral palsy, and mental retardation with non-specific etiology. In: Maino D, ed. Diagnosis and management of special populations. St. Louis: Mosby-Yearbook, 1995:17-54.

49. Catalano RA. Down syndrome. Surv Ophthalmol 1990; 34:385-98.

50. Tarczy-Hornoch K. The epidemiology of early childhood hyperopia. Optom Vis Sci 2007: 84:115-23.

51. Goldschmidt E. Refraction in the newborn. Acta Ophthalmol 1969; 47:570-8.

52. Cook RC, Glascock RE. Refractive and ocular findings in the newborn. Am J Ophthalmol 1951; 34:1407-13.

53. Hirsch MJ, Weymouth FW. Prevalence of refractive anomalies. In: Grosvenor T, Flom M, eds. Refractive anomalies. Research and clinical applications. Boston: Butterworth-Heinemann, 1991:15-38.

54. Banks MS. Infant refraction and accommodation. Int Ophthalmol Clin 1980; 20:205-32.

55. Saunders KJ, Woodhouse M, Westall CA. Emmetropisation in human infancy: rate of change is related to initial refractive error. Vision Res 1995; 35:1325-8.

56. Ingram R, Arnold P, Dally S, Lucas J. The results of a randomized trial of treating abnormal hypermetropia from the age of 6 months. Br J Ophthalmol 1990; 74:158-9.

57. Atkinson J, Braddick O, Bobier B, et al. Two infant vision screening programs: prediction and prevention of strabismus and amblyopia from photo and videorefractive screening. Eye 1996; 10:189-98.

58. Atkinson J, Braddick O, Nardini M, Anker S. Infant hyperopia: detection, distribution, changes and correlates-outcomes from the Cambridge infant screening programs. Optom Vis Sci 2007; 84:84-96.

59. Ingram RM, Walker C, Wilson JM, et al. Prediction of amblyopia and squint by means of refraction at age 1 year. Br J Ophthalmol 1986; 70:12-5.

60. Van Alphen GW HM. On emmetropia and ametropia. Ophthalmologica 1961; 142 (suppl):1-92.

61. Mohindra I, Held R. Refraction in humans from birth to five years. Doc Ophthalmol Proc Ser 1981; 28:19-27.

62. Kempf GA, Collins SD, Jarman EL. Refractive errors in the eyes of children as determined by retinoscopic examination with a cycloplegic. Public Health Bull no 182. Washington DC: U.S. Government Printing Office, 1928.

63. Hirsch MJ, Ditmas DL. Refraction of young myopes and their parents--a re-analysis. Am J Optom 1947; 24:601-8.

64. Jankiewicz H. Embryologic and genetic factors in the refractive state of the eye. In: Hirsch MJ, ed. Synopsis of the refractive state of the eye. Am Acad Optom Ser. Minneapolis: Burgess Publishing, 1967; 5:60-75.

65. Morgan M. Changes in refraction over a period of twenty years in a non-visually selected sample. Am J Optom 1958; 35:281-99.

66. Post RH. Population differences in visual acuity: review with speculative notes on selection relaxation. Eugenics Q 1962; 9:189-92.

67. Crawford HE, Haamar GE. Racial analysis of ocular deformities in schools of Hawaii. Hawaii Med J 1949; 9:90-3.

68. Chung KM, Mohidin N, Yeow PT, et al. Prevalence of visual disorders in Chinese schoolchildren. Optom Vis Sci 1996; 73:695-700.

69. Hammond CJ, Snieder H, Gilbert CE, Spector TD. Genes and environment in refractive error: the twin eye study. Invest Ophthalmol Vis Sci 2001; 42:1232-6.

70. Erlich D, Braddick OJ, Atkinson J, et al. Infant emmetropization: a longitudinal study of refraction components from 9 months of age. Optom Vis Sci 1997; 74:822-843.

71. Ingram RM, Barr A. Changes in refraction between the ages of 1 and 3 1/2. Br J Ophthalmol 1979; 63:339-42.

72. Fulton AB, Dobson V, Salen D, et al. Cycloplegic refractions in infants and young children. Am J Ophthalmol 1980; 90:239-47.

73. Atkinson J. Infant vision screening: prediction and prevention of strabismus and amblyopia from refractive screening in the Cambridge Photorefraction Program. In: Simons K, ed. Early visual development normal and abnormal. New York: Oxford University Press, 1993:335-48.

74. Ingram R, Arnold P, Dally S, Lucas J. Emmetropisation, squint, and reduced visual acuity after treatment. Br J Ophthalmol 1991; 75:414-6.

75. Aurell E, Norrsell K. A longitudinal study of children with family history of strabismus: factors determining the incidence of strabismus. Br J Ophthalmol 1990; 74:589-94.

76. Robb RM, Rodier DW. The broad clinical spectrum of early infantile esotropia. Trans Am Ophthalmol Soc 1986; 84:103-16.

77. Abrahamsson M, Fabian G, Sjostrand J. A longitudinal study of a population based sample of astigmatic children. II. The changeability of anisometropia. Acta Ophthalmol 1990; 68:435-40.

78. Almeder LM, Peck LB, Howland HC. Prevalence of anisometropia in volunteer laboratory and school screening populations. Invest Ophthalmol Vis Sci 1990; 31:2448-55.

79. Grosvenor T. Changes in spherical refraction during the adult years. In: Grosvenor T, Flom M, eds. Refractive anomalies. Research and clinical applications. Boston: Butterworth-Heinemann, 1991:131-45.

80. Lee KE, Klein BE, Klein R, Wong TY. Changes in refraction over 10 years in an adult population: the Beaver Dam Eye Study. Invest Ophthalmol Vis Sci 2002; 43:2566-71.

81. Panchapakesan J, Rochtchina E, Mitchell P. Myopic refractive shift caused by incident Cataract: the Blue Mountain Eye Study. Ophthalmic Epidemiol 2003; 10:241-7.

82. Ingram RM. Refraction of 1-year-old children after atropine cycloplegia. Br J Ophthalmol 1979; 63:343-7.

83. Ingram R, Traynar M, Walker C, Wilson J. Screening for refractive errors at age 1 year. A pilot study. Br J Ophthalmol 1979; 63:243-50.

84. Ingram R, Barr A. Refraction of 1-year-old children after cycloplegia with 1% cyclopentolate: comparison with finding after atropinization. Br J Ophthalmol 1979; 63:348-52.

85. Atkinson J, Braddick OJ, Durden K, et al. Screening for refractive errors in 6-9 month old infants by photorefraction. Br J Ophthalmol 1984; 68:105-12.

86. Amos JF. Refractive amblyopia: a preventable vision condition. J Am Optom Assoc 1979; 50:1153-9.

87. Atkinson J, Anker S, Nardini M, et al. Infant vision screening predicts failures on motor and cognitive tests up to school age. Strabismus 2002; 10:187-98.

88. Atkinson J, Nardini M, Anker S, et al. Refractive errors in infancy predict reduced performance on the movement assessment battery for children at 3 ½ and 5 ½ years. Dev Med Child Neurol 2005; 47:243-51.

89. Grosvenor T. Refractive state, intelligence test scores, and academic ability. Am J Optom Physiol Opt 1970; 47:355-61.

90. Rosner J, Rosner J. Comparison of visual characteristics in children with and without learning difficulties. Am J Optom Physiol Opt 1987; 64:531-3.

91. Grisham JD, Simons HD. Refractive error and the reading process: a literature analysis. J Am Optom Assoc 1986; 57:44-54.

92. Roch-Levecq AC, Brody BL, Thomas RG, Brown SI. Ametropia, preschoolers' cognitive abilities, and effects of spectacle correction. Arch Ophthalmol 2008; 126:252-8.

93. Schmidt PP. Vision screening. In: Rosenbloom AA, Morgan MW, eds. Principles and practice of pediatric optometry. Philadelphia: JB Lippincott, 1990:467-85.

94. Schmidt PP, Orel-Bixler D, Allen D, et al. Analytical comparisons of photorefraction in screenings. Optom Vis Sci 1995; 72:209.

95. Ingram RM. Refraction as a basis for screening children for squint and amblyopia. Br J Ophthalmol 1977; 61:8-15.

96. Gerali P, Flom MC, Raab EL. Report of Children's Vision Screening Task Force. Schaumburg, IL: National Society to Prevent Blindness, 1990.

97. Ingram RM, Walker C. Refraction as a means of predicting squint or amblyopia in preschool siblings of children known to have these defects. Br J Ophthalmol 1979; 63:238-42.

98. The Vision in Preschoolers Study Group: Preschool Vision Screening Tests Administered by Nurse Screeners Compared with Lay Screeners in the Vision in Preschoolers Study. Optom Vis Sci 2005; 46:2639-2648.

99. Duckman R. Using photorefraction to evaluate refractive error, ocular alignment and accommodation in infants, toddlers and multiply handicapped children. Probl Optom 1990; 2:333-53.

100. Hsu-Winges C, Hamer RD, Norcia AM et al. Polaroid photorefraction of infants: comparison to cycloplegic refraction. J Pediatr Ophthalmol Strabismus 1988; 26:254-60.

101. Ottar WL, Scott WE, Holgado SI. Photoscreening for amblyogenic factors. J Pediatr Ophthalmol Strabismus 1995; 32:289-95.

102. Simons K. Preschool vision screening: rationale, methodology, and outcome. Surv Ophthalmol 1996; 41:3-30.

103. Hung LF, Crawford ML, Smith EL. Spectacle lenses alter eye growth and the refractive status of young monkeys. Nature Med 1995; 1:761-5.

104. Saunders KJ. Early refractive development in humans. Surv Ophthalmol 1995; 40:207-16.

105. Atkinson J, Anker S, Bobier W, et al. Normal emmetropization in infants with spectacle correction for hyperopia. Invest Ophthalmol Vis Sci 2000; 84:181-8.

106. Sjostrand J, Abrahamsson M. Risk factors in amblyopia. Eye 1990; 4:787-93.

107. Fern KB. Visual acuity outcome in isometropic hyperopia. Optom Vis Sci 1989; 66:649-58.

108. Schoenleber DB, Crouch ER. Bilateral hypermetropic amblyopia. J Pediatr Ophthalmol Strabismus 1987; 24:75-7.

109. Abraham SV. Bilateral ametropic amblyopia. J Pediatr Ophthalmol Strabismus 1964; 1:57-61.

110. Rosenberg R. Static retinoscopy. In: Eskridge JB, Amos JF, Bartlett JD, eds. Clinical procedures in optometry. Philadelphia: JB Lippincott, 1991:155-67.

111. Rosenfield M, Chiu NN. Repeatability of subjective and objective refraction. Optom Vis Sci 1995; 72:577-9.

112. Zadnik K, Mutti DO, Adams AJ. The repeatability of measurements of the ocular components. Invest Ophthalmol Vis Sci 1992; 33:2325-33.

113. Ciner EB. Refractive error in young children. Evaluation and prescription. Practical Optom 1992; 3(4):182-90.

114. Mohindra I. A technique for infant vision examination. Am J Optom Physiol Opt 1975; 52:867-70.

115. Mohindra I. A non-cycloplegic refraction technique for infants and young children. J Am Optom Assoc 1977; 48:518-23.

116. Saunders KJ, Westall CA. Comparisons between near retinoscopy and cycloplegic retinoscopy in the refraction of infants and children. Optom Vis Sci 1992; 69:615-22.

117. Wesson D, Mann KR, Bray NW. A comparison of cycloplegic refraction to near retinoscopy technique for refractive error determination. J Am Optom Assoc 1990; 61:680-4.

118. Foteder R, Rochtchina E, Morgan I, et al. Necessity of cycloplegia for assessing refractive error in 12-year-old children: a population-based study. Am J Ophthalmol 2007; 144:307-9.

119. Moore BD. Pediatric ocular pharmacology. In: Press LJ, Moore BD, eds. Clinical pediatric optometry. Boston: Butterworth-Heinemann, 1993:347-54.

120. Amos JF, Amos DM. Cycloplegic refraction. In: Bartlett JD, Jaanus SD, eds. Clinical ocular pharmacology, 3rd ed. Boston: Butterworth-Heinemann, 1995:503-13.

121. Egashira SM, Kish LL, Twelker JD, et al. Comparison of cyclopentolate vs tropicamide cycloplegia in children. Optom Vis Sci 1993; 70:1019-26.

122. Goss DA, Grosvenor T. Reliability of refraction--a literature review. J Am Optom Assoc 1996; 67:619-30.

123. Zhao J, Mao J, Luo R, et al. Accuracy of noncycloplegic autorefraction in school-age children in China. Optom Vis Sci 2004; 81:49-55.

124. Choong YF, Chen AH, Goh PP. A comparison of qutorefraction and subjective refraction with and without cycloplegia in primary school children. Am J Ophthalmol 2006; 142:68-74.

125. Silbert JA, Alexander A. Cyclotherapy in the treatment of symptomatic latent hyperopia. J Am Optom Assoc 1987; 58:40-6.

126. Moore BD. Contact lens problems and management in infants, toddlers, and preschool children. Probl Optom 1990; 2:365-93.

127. Moore BD. Contact lens therapy for amblyopia. Probl Optom 1991; 3:355-68.

128. Calorosa EE, Rouse MW. Clinical management of strabismus. Boston: Butterworth-Heinemann, 1993:76-93.

129. Wick B. Vision therapy for preschool children. In: Rosenbloom AA, Morgan MW, eds. Principles and practice of pediatric optometry. Philadelphia: JB Lippincott, 1990:274-92.

130. Fraunfelder FT. Ocular toxicology. In: Physicians' desk reference for ophthalmology, 25th ed. Montvale, NJ: Medical Economics, 1996:19.

131. Goldstein JH. The role of miotics in strabismus. Surv Ophthalmol 1968; 3:31-46.

132. Koch DD, Abarca A, Villareal R, et al. Hyperopia correction by noncontact holmium: YAG laser thermal keratoplasty. Clinical study with two year follow-up. Ophthalmology 1996; 103:731-40.

133. Kezirian GM, Gremillion CM. Automated lamellar keratoplasty for the correction of hyperopia. J Cataract Refract Surg 1995; 21:386-92.

134. Grandon SC, Sanders DR, Anello RD, et al. Clinical evaluation of hexagonal keratotomy for the treatment of primary hyperopia. J Cataract Refract Surg 1995; 21:140-9.

135. Dausch D, Klein R, Schroder E. Excimer laser photorefractive keratectomy for hyperopia. J Refract Corneal Surg 1993; 9:20-8.

136. Siganos DS, Siganos CS, Pallikaris IG. Clear lens extraction with intraocular lens implantation in normally sighted hyperopic eyes. J Refract Corneal Surg 1994; 10:117-21.

137. Varley GA, Huang D, Rapuano CJ, et al. Ophthalmic Tecnology Assessment Committee Refractive Surgery Panel, American Academy of Ophthalmology. LASIK for hyperopia, hyperopic astigmatism, and mixed astigmatism: na report by the American Academy of Ophthalmology. Ophthalmology 2004; 111:1604-17.

138. U.S. Food and Drug Administration Ophthalmic Devices Panel. PMA Number: P970043/S7. September 22, 2000.

139. Ciner EB. Management of refractive error in infants, toddlers and preschool children. Probl Optom 1990; 2:(3)394-419.

140. Rosner J, Gruber J. Differences in the perceptual skills development of young myopes and hyperopes. Am J Optom Physiol Opt 1985; 62:501-4.

141. Rosner J, Rosner J. Some observations about visual perceptual skills development of young hyperopes and age of first correction. Clin Exp Optom 1986; 69:166-8.

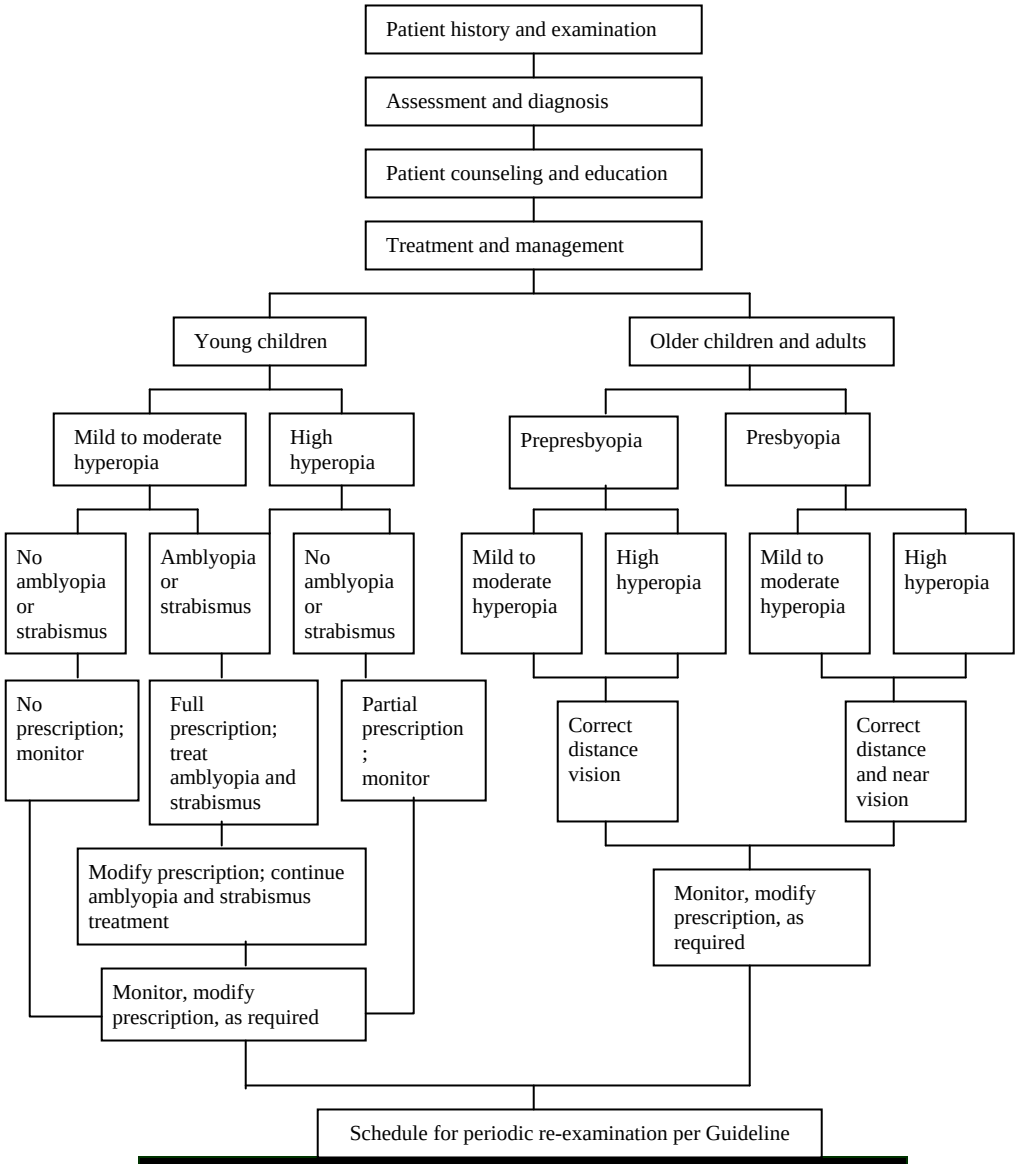
142. Ciner EB, Herzberg C. Optometric management of optically induced consecutive exotropia. J Am Optom Assoc 1992; 63:266-71.

143. Scheiman M. Assessment and management of the exceptional child. In: Rosenbloom AA, Morgan MW, eds. Principles and practice of pediatric optometry. Philadelphia: JB Lippincott, 1990:388-419.

144. Grosvenor T. The neglected hyperope. Am J Optom Physiol Opt 1971; 48:376-82.

IV. APPENDIX

Figure 1
Optometric Management of the Patient with Hyperopia: A Brief Flowchart



[redacted]

Figure 2
Potential Components of the Diagnostic Evaluation for Hyperopia

A. Patient history
   1. Nature of presenting problem, including chief complaint
   2. Ocular and general health history
   3. Developmental and family history
   4. Use of medications and allergies

B. Visual acuity
   1. Distance visual acuity testing
   2. Near visual acuity testing

C. Refraction
   1. Retinoscopy
      a. Static retinoscopy
      b. Nearpoint retinoscopy
      c. Cycloplegic retinoscopy
   2. Subjective refraction
   3. Autorefraction

D. Ocular motility, binocular vision, and accommodation
   1. Versions
   2. Monocular and alternating cover test
   3. Near point of convergence
   4. Accommodative amplitude and facility
   5. Stereopsis testing

E. Ocular health assessment and systemic health screening
   1. Assessment of pupillary responses
   2. Visual field screening
   3. Color vision testing
   4. Measurement of intraocular pressure
   5. Evaluation of anterior and posterior segments of eye and adnex

[redacted]

Figure 3
Frequency and Composition of Evaluation and Management Visits for Hyperopia*

Type of Patient	Number of Evaluation Visits	Treatment Options	Frequency of Followup Visits
Young child with mild to moderate hyperopia and no strabismus or amblyopia	1-2	Monitor Optical correction Modify habits and environment	3-12 mo
Young child with high hyperopia and no strabismus or amblyopia	1-2	Monitor if infant Optical correction Vision therapy Modify habits and environment	2-6 mo
Young child with mild to high hyperopia and strabismus or amblyopia	2-3	Optical correction Strabismus and amblyopia therapy Modify habits and environment Pharmaceuticals	2 wk-3 mo
Older child with mild to moderate hyperopia	1-2	Monitor Optical correction Modify habits and environment	6-18 mo
Older child with high hyperopia	1-2	Optical correction Vision therapy Modify habits and environment	6-12 mo
Pre-presbyopic adult	1	Optical correction Vision therapy Modify habits and environment	1-2 yr
Presbyopic adult	1	Optical correction Vision therapy Modify habits and environment	1-2 yr

*Figure 3 extends horizontally on page 46

Figure 3 (continued)

Composition of Followup Evaluations				
VA	**REF**	**A/V**	**OH**	**Management Plan**
Each visit	Each visit	Each Visit	p.r.n.	No treatment or provide refractive correction; monitor vision
Each visit	Each visit	Each Visit	p.r.n.	Provide refractive correction; treat any accommodative or binocular vision problem; monitor vision
Each visit	Each visit	Each Visit	p.r.n.	Provide refractive correction; treat any amblyopia or strabismus; monitor vision
Each visit	Each visit	Each visit	p.r.n.	No treatment or provide refractive correction; monitor vision
Each visit	Each visit	Each visit	p.r.n.	Provide refractive correction; treat any accommodative or binocular vision problem; monitor vision
Each visit	Each visit	Each visit	Each visit	No treatment or provide refractive correction; treat any accommodative or binocular vision problem; monitor vision
Each visit	Each visit	Each visit	Each visit	Provide refractive correction; treat any accommodative or binocular vision problem; monitor vision

VA = visual acuity testing
REF = refraction
A/V = accommodative/vergence testing
OH = ocular health assessment
p.r.n. = as needed

Figure 4
ICD-9-CM Classification of Hyperopia

Hypermetropia Far-sightedness Hyperopia	**367.0**
Astigmatism	**367.2**
Astigmatism, unspecified	**367.20**
Regular astigmatism	**367.21**
Irregular astigmatism	**367.22**
Anisometropia and aniseikonia	**367.3**
Anisometropia	**367.31**
Aniseikonia	**367.32**
Presbyopia	**367.4**
Disorders of accommodation	**367.5**
Paresis of accommodation Cycloplegia	**367.51**
Total or complete internal ophthalmoplegia	**367.52**
Spasm of accommodation	**367.53**

Abbreviations of Commonly Used Terms

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

D ..... Diopter

DFP ..... Diisopropylfluorophosphate

PI ..... Phospholine iodide

Glossary

Absolute hyperopia Hyperopia that cannot be overcome by accommodation.

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

Accommodative esotropia An esotropia that is associated with high hyperopia (refractive) and/or a high AC/A ratio (non-refractive).

Amblyopia A unilateral or bilateral reduction in corrected visual acuity in the absence of any obvious structural anomalies or ocular disease.

Anisometropic hyperopia Unequal and significant hyperopic refractive error.

Astigmatism Refractive anomaly due to refraction of light in different meridians of the eye, generally caused by a toroidal anterior surface of the cornea.

Component hyperopia Hyperopia resulting when one or more of the components of refractive error varies significantly from normal.

Correlational hyperopia Hyperopia resulting when one or more of the components of ocular refraction deviates modestly from normal.

Emmetropization The process by which significant neonatal refractive errors are reduced in the direction of emmetropia.

Facultative hyperopia Hyperopia that can be overcome by accommodation.

Hyperopia (farsightedness) Refractive condition in which the light entering the non-accommodated eye is focused behind the retina.

Isoametropic amblyopia Amblyopia caused by bilateral high refractive error.

Latent hyperopia Hyperopia that is habitually overcome by accommodation; determined by cycloplegic refraction.

Manifest hyperopia Hyperopia (either facultative or absolute) that is determined by noncycloplegic refraction.

Myopia (nearsightedness) Refractive condition in which the light entering the non-accommodative eye is focused in front of the retina.

Pathologic hyperopia Hyperopia due to abnormal anatomy, maldevelopment, ocular disease, or trauma, not to normal biological variation.

Physiologic hyperopia hyperopia due to correlational hyperopia or component hyperopiahaving otherwise normal ocular anatomy.

Presbyopia A reduction in accommodative ability that occurs normally with age and necessitates a plus lens addition for satisfactory seeing at near.

Significant hyperopia Any degree of hyperopia sufficient to cause symptoms requiring remediation.

Strabismus Condition in which binocular fixation is not present under normal seeing conditions, i.e., one eye is turned in relation to the other.

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

Visual acuity The clearness of vision that depends upon the sharpness of focus of the retinal image and the integrity of the retina and visual pathway.

Sources: Cline D, Hofstetter NW, 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.