OPTOMETRIC CLINICAL PRACTICE GUIDELINE

Care of the Patient with

Amblyopia

American Optometric Association

CPG 4

OPTOMETRY:
THE PRIMARY EYE CARE PROFESSION

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

Optometrists provide more than two-thirds of the primary eye care services in the United States. They are more widely distributed geographically than other eye care providers and are readily accessible for the delivery of eye and vision care services. There are approximately 30,000 full-time equivalent doctors of optometry currently in practice in the United States. Optometrists 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.

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American Optometric Association

CARE OF THE PATIENT WITH AMBLYOPIA

Reference Guide for Clinicians

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

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

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INTRODUCTION

Optometrists, through their clinical education, training, experience, and broad geographic distribution, 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 amblyopia.

This Optometric Clinical Practice Guideline for the Care of the Patient with Amblyopia describes appropriate examination and treatment procedures to reduce the risk of visual disability from amblyopia. 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:

- Identify patients at risk of developing amblyopia
- Accurately diagnose amblyopia
- Improve the quality of care rendered to patients with amblyopia
- Minimize the adverse effects of amblyopia
- Preserve the gains obtained through treatment
- Inform and educate parents, patients, and other health care practitioners about the visual complications of amblyopia and the availability of treatment.

I. STATEMENT OF THE PROBLEM

Amblyopia, also referred to by the public as "lazy eye", is a unilateral or infrequently bilateral condition in which the best corrected visual acuity is poorer than 20/20 in the absence of any obvious structural anomalies or ocular disease.[1] Amblyopia represents a syndrome of compromising deficits, rather than simply reduced visual acuity, including:

- Increased sensitivity to contour interaction effects[2,3]
- Abnormal spatial distortions and uncertainty[4]
- Unsteady and inaccurate monocular fixation[5]
- Poor eye tracking ability[6]
- Reduced contrast sensitivity[7]
- Inaccurate accommodative response.[8,9]

The loss of visual acuity in amblyopia ranges from slightly worse than normal (6/6, 20/20) to functionally blind (6/60, 20/200, or worse). Functional amblyopia only develops in children up to the age of 6-8 years,[10] although it may persist for life once established. In the under-20 age group, amblyopia causes more vision loss than trauma and all other ocular diseases.[11] The Visual Acuity Impairment Study, sponsored by the National Eye Institute, found functional amblyopia to be the leading cause of monocular vision loss in the 20-70+ age group, surpassing diabetic retinopathy, glaucoma, macular degeneration, and cataract.[12]

A. Description and Classification of Amblyopia

The classification of amblyopia is based on the clinical conditions responsible for its development (Table 1).[1,13-17] This classification serves as a practical method for identifying its etiology and applying appropriate management strategies. Functional amblyopia occurs before 6-8 years of age and is attributable to form deprivation, strabismus, or anisometropia.[1]

Table 1
Differential Diagnosis of Cases of Reduced Visual Acuity

Functional amblyopia causes:

1. Form deprivation
   Congenital or traumatic cataract
   Early complete blepharoptosis
   Corneal opacity
   Hyphema
   Vitreous hemorrhage
   Uncontrolled occlusion therapy
   Uncontrolled penalization therapy
2. Constant unilateral strabismus
3. Amblyopiogenic uncorrected refractive error
   Anisometropia (spherical or astigmatic)
   Isoametropia
4. Combined aniso-strabismus

Psychogenic causes:

1. Conversion hysteria
2. Malingering

Structural/pathological causes:

1. Achromatopsia
2. Coloboma
3. Myelinated retinal nerve fibers
4. Retinopathy of prematurity
5. Degenerative myopia
6. Hypoplastic optic nerve
7. Keratoconus
8. Opacities of the media
9. Macular, perimacular chorioretinal scar
10. Macular pathology (e.g., Stargardt's disease)
11. Optic atrophy
12. Retrobulbar neuritis
13. Nystagmus (congenital, latent, manifest latent)
14. Craniopharyngioma

Other forms of vision loss are sometimes incorrectly classified as amblyopia:

- Psychogenic or hysterical vision loss is characterized by the substitution of physical signs or symptoms (e.g. reduced visual acuity) for anxiety or emotional repression.
- Organic vision loss is attributable to ocular disease or anatomical anomalies in the visual pathways.

The scope of this Guideline covers the diagnosis, treatment, and management of functional amblyopia (See Appendix Figure 4 for ICD-9-CM classification of functional amblyopia).

1. Form Deprivation Amblyopia

When a physical obstruction along the line of sight prevents the formation of a well-focused, high-contrast image on the retina, the result is form deprivation amblyopia.[1] This obstruction can occur in one or both eyes and must take place before the age of 6-8 years for amblyopia to develop. The degree to which amblyopia develops depends on the time of onset and the extent of the form deprivation. Congenital cataract is the most frequent cause of form deprivation amblyopia. Other conditions that can lead to the development of form deprivation amblyopia include traumatic cataract, corneal opacities, congenital ptosis,[18,19] hyphema, vitreous opacification or clouding, prolonged uncontrolled patching (occlusion therapy),[20] prolonged unilateral blepharospasm, and prolonged unilateral atropinization for occlusion therapy.[21]

2. Refractive Amblyopia

Refractive amblyopia results from either high but equal (isoametropic) or clinically significant unequal (anisometropic) uncorrected refractive errors (Table 2).[15,16,22]

a. Isoametropic Amblyopia

Isoametropic amblyopia is an uncommon form of amblyopia caused by a high but approximately equal uncorrected bilateral refractive error that creates a blurred image on each retina. Over time, this subtle type of visual form deprivation delays normal neurophysiological development of the visual pathway

and visual cortex. Hyperopia greater than 5.00 diopters (D), myopia greater than 8.00 D, and astigmatism greater than 2.50 D are common causes of isoametropic amblyopia. Patients with isoametropic amblyopia have a wide range of visual acuity loss, from slightly worse than 20/20 to 20/200, although the majority of patients have initial best corrected visual acuity of 20/50 or better.[23]

Table 2
Potentially Amblyopiogenic Refractive Errors

Isoametropia	**Diopters**
Astigmatism	>2.50 D
Hyperopia	>5.00 D
Myopia	>8.00 D
Anisometropia	
Astigmatism	>1.50 D
Hyperopia	>1.00 D
Myopia	>3.00 D

b. Anisometropic Amblyopia

Anisometropic amblyopia is caused by an uncorrected refractive error in which the difference between the corresponding major meridians of the two eyes is at least l D.[13] This refractive difference causes a blurred image in the eye with the greater refractive error, disrupting the normal neurophysiological development of the visual pathway and visual cortex.[1]

Generally, the greater the anisometropia, the more severe the amblyopia.[24-26] Patients with hyperopic anisometropia with as little as 1 D difference between the eyes may develop amblyopia,[27] but those with myopic anisometropia usually do not have amblyopia until the amount of anisometropia reaches 3-4 D.[26] The patient with myopic anisometropia uses the more myopic eye at near and the less myopic eye at far until the myopia exceeds 3 D, thereby maintaining foveal fixation and good corrected acuity in each eye.

Because the person with hyperopic anisometropia uses the less hyperopic eye for fixation at all distances, the more hyperopic eye never receives a clear image, resulting in amblyopia. Patients with anisometropic amblyopia have a wide range of visual acuities, from slightly worse than 20/20 to poorer than 20/200. The average best corrected visual acuity is approximately 20/60.[28] When the etiology is a combination of anisometropia and strabismus, the average visual acuity is approximately 20/94.[28]

3. Strabismic Amblyopia

Strabismic amblyopia is most commonly associated with an early onset (<6-8 years of age) of constant unilateral strabismus. Due to the absence of bifoveal fixation, the two eyes receive different visual images, causing confusion and diplopia. To eliminate these problems, the visual system actively inhibits or suppresses the image from the turned eye. This active inhibition over time causes cortical spatial changes that result in a loss of visual acuity.[4] In addition, these patients often use an off-foveal point for monocular fixation.[5]

Eccentric fixation, in which the patient has difficulty directing the fovea at the target, is the most significant motor anomaly associated with strabismic amblyopia. This behavior contributes to the loss of visual acuity in strabismic amblyopia.[29,30] The size and steadiness of the eccentric fixation are important factors[31] in determining both the passive and active treatment options needed to restore foveal fixation and normal visual acuity.

Patients with strabismic amblyopia have a wide range of visual acuity loss, from slightly worse than 20/20 to poorer than 20/200. The average best corrected visual acuity is approximately 20/74. When anisometropia and strabismus are the combined etiologies, the average visual acuity is approximately 20/94.[28]

Amblyopia may also occur in intermittent or alternating strabismus, but it is most frequently shallow (20/60 visual acuity or better). Although patients with an alternating strabismus typically exhibit normal acuity in each eye, some have shallow amblyopia in the more frequently deviating eye.

B. Epidemiology of Amblyopia

1. Prevalence and Incidence

Controversy over which visual acuity criteria should be adopted for the clinical definition of amblyopia has caused confusion as to the prevalence of amblyopia.[32,33] Estimates of the prevalence can vary substantially depending on which criteria[28] and population are selected (e.g., 3.5% for patients whose visual acuity is 20/30; 1.4% for 20/40). The best estimate of the prevalence in the general population is 2 percent.[1,34]

Refractive and strabismic amblyopia account for the vast majority of amblyopia. Anisometropic amblyopia and/or strabismic amblyopia account for over 90 percent of all amblyopia. Isoametropic amblyopia is rare, accounting for only 1-2 percent of all refractive amblyopia.[16] The exact prevalence of form deprivation amblyopia is unclear, but it is also considered rare.

The incidence of amblyopia in the preschool years is approximately 0.4 percent per year.[32,33,35] If the prevalence after this period is approximately 2 percent, the annual incidence in the general population can be broadly estimated by assuming that 2-3 percent of healthy infants born each year will suffer visual loss from amblyopia.[35,36]

2. Risk Factors

The risk of developing amblyopia is associated with strabismus, significant refractive error, and conditions that may cause form vision deprivation by physically blocking or occluding the visual axis of one or both eyes during the sensitive period from birth to 6-8 years of age. A number of additional risk factors have been identified:

- Prematurity[37,38]
- Low birth weight[39,40]

- Retinopathy of prematurity (retrolental fibroplasia)[40]
- Cerebral palsy[41-43]
- Mental retardation[44,45]
- Family history of anisometropia, isoametropia, strabismus, amblyopia, or congenital cataract.[46-50]

Maternal smoking and the use of drugs or alcohol are associated with increased risk for amblyopia and strabismus.[51,52] The risk for amblyopia also increases fourfold following extraocular muscle surgery for early-onset esotropia.[53,54]

C. Clinical Background of Amblyopia

1. Natural History

The visual pathways develop from birth to approximately 6-8 years of age, with the most rapid development occuring in infancy. During this time, the visual system is susceptible to known amblyopiogenic factors: form deprivation, optical defocus, and misalignment of the eyes. If left untreated, the two amblyopiogenic mechanisms, form deprivation and abnormal binocular inhibition, cause a progressive reduction of visual acuity until approximately 6-8 years of age, at which time visual acuity stabilizes. The child's age when exposed to an amblyopia-inducing condition appears to be the most important determinant for the development of amblyopia.[55]

Untreated unilateral form deprivation extending past the first 3 months of age profoundly affects visual acuity development. Untreated bilateral visual form deprivation has a similar effect if it extends past 6 months of age. If treatment for these conditions is not initiated during this critical developmental period, the prognosis for normal vision development is poor. When the onset of the cause of deprivation occurs after the first 6-12 months, the prognosis for vision recovery is improved with early treatment.

Untreated unequal or high equal refractive errors occuring during the developmental period can also have a significant effect on vision development. Because the onset and stability

of refractive anomalies vary significantly, especially between 1 and 4 years of age, the exact onset of refractive amblyopia is often difficult to establish. In isoametropic amblyopia the uncorrected refractive anomaly creates a blurred image on each retina. Over time, this subtle type of visual form deprivation disrupts the normal neurophysiological development of the visual pathway and visual cortex. In anisometropia, the uncorrected refractive error creates a blurred image on one retina. Thus the mechanisms of both visual form deprivation and abnormal binocular inhibition contribute to the loss of acuity. The chances for improving visual acuity to 20/40 or better in both types of refractive amblyopia are favorable.

Strabismic amblyopia is generally associated with a constant, unilateral strabismus that develops prior to 6-8 years of age. To eliminate the problem when the two eyes receive different images, the visual system actively inhibits or suppresses the image from the strabismic eye. This abnormal binocular inhibition is the primary mechanism causing vision loss. The untreated strabismic eye may develop a number of sensorimotor anomalies, most notably eccentric fixation, that can worsen the prognosis and significantly increase the length of treatment.

2. Common Signs, Symptoms, and Complications

Amblyopia of one eye (as in anisometropic and strabismic amblyopia) usually produces little handicap and few symptoms because the patient typically has good visual acuity in the normal eye. The most significant problems usually result from a decrease in stereopsis, which may result in occupational exclusions and less efficient vision performance in certain tasks, such as driving and near eye-hand coordination activities. In addition, amblyopia may contribute to later onset of strabismus.

An additional problem with amblyopia is the potential loss of vision in the better eye.[56] The patient with amblyopia is at a greater risk (3 times that of a normal adult; 17 times that of a normal child) of losing the vision of the better eye.[57] There are many reports of improved vision in the amblyopic eye after loss

of normal vision in the better eye. However, the chance of spontaneous improvement of visual acuity to a usable level (20/70 or better) is relatively low (~17%) unless there is complete loss of vision in the better eye.[58] Intense therapeutic effort is needed to recover usable vision in adults who have strabismic amblyopia with eccentric fixation.[59]

Twenty-seven percent[23] of patients with hyperopic isoametropic amblyopia may have an accompanying visual perceptual skills deficit associated with early learning problems. The prevalence of perceptual deficits is approximately three times greater for children whose refractive errors are corrected after 4 years of age than for those corrected earlier.[23,60]

3. Early Detection and Prevention

Amblyopia is a preventable and a treatable condition especially if detected early. Screening for causes of form deprivation amblyopia should be conducted by the infant's primary care physician within the first 4-6 weeks after birth, and children at risk should be monitored yearly throughout the sensitive developmental period (birth to 6-8 years of age).

Screening for amblyopiogenic refractive error and strabismus should also begin during the first year of life. At-risk children will need to be monitored yearly because from birth to 4 years of age there is considerable variability of refractive anomalies, especially astigmatism[61-64] and anisometropia.[61,62,65] Screening children with a positive family history of strabismus or amblyopia may be a cost-effective strategy.[66] A screening program to detect and treat amblyopia at age 4 has been extremely successful in other countries.[67] Unfortunately, the percentage of the preschool population that is properly screened for amblyopia in the United States is low.[68] Therefore, optometrists should alert parents to the prevalence of and risk for the development of amblyopia and encourage them to seek regular professional vision care for their children.

II. CARE PROCESS

A. Diagnosis of Amblyopia

The evaluation of a patient with amblyopia 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; visual, ocular and general health history; developmental and family history; and use of medications.

There are typically few symptoms associated with amblyopia. The patient or the patient's parent may report poor vision in one or possibly both eyes and difficulty doing tasks requiring binocular depth perception. If amblyopia is associated with strabismus, the patient or parent may report a cosmetically noticeable eye turn or signs of strabismus that might include closing or winking one eye or reporting diplopia. The patient with isoametropic amblyopia may present with signs and symptoms indicating an associated visual perceptual skills deficit.

2. Ocular Examination

a. Visual Acuity

Reliable assessment of visual acuity in infants and young children can be accomplished by selecting procedures appropriate for the child's cognitive or chronological age.*[69-71]

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

Relying on single object tests may seriously overestimate the patient's visual acuity level and result in a failure to diagnose amblyopia (a false negative). For school-age children, psychometric visual acuity charts, such as the S-chart Visual Acuity slides,[72,73] the Psychometric Visual Acuity cards,[74] or the Bailey-Lovie chart[75] are useful. A psychometric evaluation of visual acuity results (plotted as visual acuity versus responses) provides the most reliable measure of the amblyopic patient's visual acuity.[72,74] If a psychometric chart is not available, the practitioner can conduct a psychometric evaluation of the full-chart Snellen results,[74] which correlate reasonably well with the more formal psychometric charts.[76]

For strabismic patients presenting with associated latent nystagmus,[77] the practitioner can evaluate the amblyopic eye's visual acuity under binocular conditions using vectographic or anaglyphic (red-green) methods if possible, or holding a fogging (plus) lens in front of the nonamblyopic eye.[78,79]

b. Refraction

The patient's refractive condition should be evaluated under both noncycloplegic and cycloplegic* conditions to determine whether the amblyopia has a refractive (anisometropic or isoametropic) etiology.[24,80] Reassessment of visual acuity with best refractive correction is needed to avoid misdiagnosis of amblyopia. Subjective refraction is typically unreliable in patients with amblyopia and should only be used in conjunction with objective techniques.

c. Monocular Fixation

The method of choice for evaluating monocular fixation is visuoscopy using an ophthalmoscope with a calibrated fixation target.[17,81] The practitioner should identify whether eccentric fixation is present and assess the characteristics of eccentric fixation: location, magnitude, and steadiness. When there is

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

no foveal reflex, entoptic testing, such as Haidinger's brushes, Maxwell's spot, or Emergent Textural Contour,[82] can be useful in assessing monocular fixation.

d. Ocular Motor Deviation

The breadth and depth of the investigation of the ocular motor deviation should parallel the patient's age and cognitive development.[70] Objective tests (e.g., Hirschberg, Brückner, and unilateral/alternate cover testing) are preferred for use with amblyopic patients. The practitioner should determine whether strabismus is present, and when it is, determine the frequency (constant or intermittent), the laterality (unilateral or alternating), and magnitude of the deviation.*

e. Sensorimotor Fusion

An evaluation of sensorimotor fusion may be conducted either "out-of-instrument" (in open or free space) or "in-instrument." The out-of-instrument evaluation is most appropriate for patients with nonstrabismic amblyopia. The red lens, Worth's four dot, and random dot stereopsis tests are good examples of out-of-instrument tests to evaluate sensorimotor fusion, specifically to determine the presence of suppression and the level of stereopsis.

An in-instrument evaluation is most appropriate for the patient with strabismic amblyopia. Testing conducted at the patient's angle of deviation, which eliminates the vergence demand, gives a clearer picture of the patient's sensory fusion potential. However, an out-of-instrument evaluation provides useful information about the patient's sensory fusion status under normal or near-normal seeing conditions. The major amblyoscope is the instrument of choice for evaluating sensorimotor fusion at the patient's angle of deviation. If a major amblyoscope is not available, it is possible to conduct an investigation using the mirror stereoscope trainer or, if angle of deviation is less than 25 prism diopters (PD), the Brewsters stereoscope.

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

f. Accommodation

An evaluation of accommodative function involves testing monocular accommodative amplitude (push-up or minus-lens method) and facility (plus-minus flipper method).[83,84] If the patient is nonstrabismic, the evaluation of accommodative accuracy (lag) may be assessed using the monocular estimation method (MEM).[85]

g. Ocular Motility

Examination of ocular motility should be done to evaluate the quality of fixation maintenance and saccadic and pursuit eye movements. Ocular motility can be assessed by observation, rating scale-assisted observation,[83,86,87] simple quantifying tests,[88] or, if available, sophisticated eye-monitoring instrumentation.[89]

h. Ocular Health Assessment and Systemic Health Screening

Ocular health should be evaluated to rule out coincidental or causal congenital anomalies or disease, associated with the amblyopia. This evaluation may include assessment of pupillary function, monocular color vision, and the anterior segment with the biomicroscope. Pharmacologic dilation of the pupil is generally required for thorough evaluation of the ocular media and posterior segment.

To rule out psychogenic or organic causes of decreased vision, the practitioner may evaluate visual fields in patients with bilaterally reduced visual acuity but no significant isoametropia, history of bilateral form deprivation, or retinal disease.

i. Supplemental Testing

Additional procedures may be indicated for the identification of associated conditions. A differential diagnosis may require an additional office visit. Electrodiagnostic testing by flash visual evoked potential or electroretinogram may be indicated to rule out conversion hysteria or malingering. Electrodiagnostic testing may also be indicated for patients in whom acquired diseases or congenital anomalies of the optic nerve or retina may account for the vision loss. Such testing assists in

establishing the diagnosis and prognosis prior to treatment. Functional amblyopia and acquired disease or congenital anomalies often coexist, and together are called relative amblyopia. In many cases treatment of the functional amblyopia component is successful.[90-92]

B. Management of Amblyopia

1. Basis for Treatment

Untreated amblyopia is an important health hazard. The risk for blindness is considerably higher for the amblyopic patient than for the general population.[57] Vision loss in the healthy eye is often due to trauma, but in cases of functional amblyopia, diseases that usually affect both eyes first attack the eye with less functional impairment.[93]

Normal vision in both eyes substantially decreases the lifetime risk for ocular injury and incapacitating vision loss, thus reducing the socioeconomic cost of legal blindness. The treatment of amblyopia is justified not only because it improves vision in the amblyopic eye and decreases the risk of blindness in the fellow eye, but also because it facilitates fusion in a high percentage of cases, which, in turn, helps maintain eye alignment.[94] Normal binocular vision and visual acuity are required in a variety of visually demanding careers.

Treatment should be directed toward the two primary etiologies of amblyopia: form deprivation and binocular inhibition. Amblyopia therapy effectively restores normal or near-normal visual function by eliminating eccentric fixation and/or developing more extensive synaptic input to the visual cortex.[95-99] It improves monocular deficits of visual acuity, monocular fixation, accommodation, and ocular motility. The final step in amblyopia therapy, if possible, is to develop normal binocular vision. The establishment of binocular vision eliminates or significantly reduces the underlying binocular inhibition in unilateral amblyopia, which increases the probability of maintaining visual acuity improvements (See

Appendix Figure 1 for an overview of patient management strategy).

2. Available Treatment Options

a. Optical Correction

The rationale for correcting the refractive anomaly with spectacles or contact lenses is to ensure that the retina of each eye receives a clear optical image. Full correction of the ametropia is effective in some patients, especially isoametropic and anisometropic (< 2 D) patients who are binocular.[25,100]

The use of spectacles versus contact lenses for optical correction has been the subject of debate. Selection of the optical correction involves consideration of the relative advantages of each.

Contact lenses appear to have certain advantages, including:

- Reduction of aniseikonia in cases of refractive and axial anisometropia[101-107]
- Improved cosmesis, which encourages better compliance with wearing the optical correction
- Elimination or reduction of prismatic imbalance, weight problems, tilt, peripheral distortions, and visual field restrictions experienced by users of spectacle lenses.

Spectacles have the advantages of:

- Being more economical in most cases
- Providing a level of safety against injury to the better seeing eye
- Serving as a modality for other optical modifications (bifocal or prism) in the management of residual binocular anomalies.

b. Occlusion

Occlusion has been the cornerstone of treatment of amblyopia for over 200 years. The rationale for using occlusion is that occluding the better eye stimulates the amblyopic eye, decreasing inhibition by the better eye. Occlusion enables the amblyopic eye to enhance neural input to the visual cortex. It is also important in eliminating eccentric fixation. Both convenient and economical, occlusion requires minimal in-office participation. Occlusion can be classified in several ways:[108-112]

- Type (direct, inverse, alternating)
- Time (full-time, part-time, minimal)
- Occluder (bandage, tie-on, spectacles, contact lenses, pharmacologically induced).

Numerous reports in the literature document the successful use of direct occlusion in the treatment of deprivation amblyopia,[113] strabismic amblyopia with eccentric fixation,[114-119] and anisometropic amblyopia.[25,120-126] However, noncompliance with occlusion represents a significant factor in occlusion failures,[127-129] especially in patients over 8 years of age in whom up to 50 percent noncompliance is common.[129]

Potential side effects of occlusion include:

- Occlusion amblyopia (amblyopia of the better eye) resulting from indiscriminate or poorly supervised occlusion[20]
- Precipitation of strabismus[25,130,131] or an increase in the magnitude of strabismus[132,133]
- Precipitation of diplopia
- Poor compliance due to reduced vision during school and work-related visual tasks
- Cosmetic concerns
- Skin allergies and irritations with bandage-type occluders.

c. Active Vision Therapy

Optometric vision therapy or orthoptics is used to correct or improve specific dysfunctions of the vision system.[134] Vision therapy refers to the total treatment program, which may include passive therapy options (e.g., spectacles, occlusion, pharmacologic agents) and active therapy.[17] With such passive treatment options as optical correction and occlusion, the patient experiences a change in visual stimulation without any conscious effort. Active therapy is designed to improve visual

performance by the patient's conscious involvement[17,135] in a sequence of specific, controlled visual tasks or procedures that provide feedback about the patient's performance.[17,136,137] When a reflexive response is achieved, it is anticipated that improved performance will transfer to other noncontrolled visual tasks,[138] ultimately changing the underlying visual processing mechanism.[139,140]

Active vision therapy for amblyopia is designed to remediate deficiencies in four specific areas: eye movements and fixation, spatial perception, accommodative efficiency, and binocular function.[141] The goal of vision therapy is remediation of these deficiencies, with subsequent equalization of monocular skills and, finally, integration of the amblyopic eye into binocular functioning.[142]

Active monocular and binocular amblyopia therapies, as opposed to passive management (e.g., occlusion), reduce the total treatment time needed to achieve the best visual acuity.[143-145] Monocular therapy involves stimulation techniques that enhance amblyopic resolution[6] and foster more normal eye movements,[146] central fixation,[147,148] and accommodation of the amblyopic eye.[149] Because active binocular inhibition is one of the underlying etiologies of unilateral types of amblyopia,[150,151] antisuppression procedures are performed under binocular conditions[152] or in-instrument conditions simulating binocular conditions.[17]

d. Management of Deprivation Amblyopia

When a significant physical obstruction (e.g., congenital cataract) is diagnosed early, the initial management should involve consultation with an ophthalmologist regarding removal of the obstruction within the first 2 months of life.[153-155] In the case of bilateral physical obstruction, surgery on the second eye typically follows the operation on the first eye by 1-2 weeks to minimize the period of binocular inhibition. Any significant refractive anomaly should be corrected, preferably with contact lenses, within 1 week after surgery. Part-time occlusion (2 hours per day) combined with visual stimulation techniques may also be prescribed.[35] It is recommended the patient be

followed at 2-4 week intervals for 1 year to monitor visual acuity and binocular development. If after 1 year the practitioner is satisfied with the optical correction, corneal physiology is normal, and visual acuity has improved and stabilized, the patient can then be monitored at 6 month intervals.

In the patient over 12 months old who has a physical obstruction, there may be a question about whether the condition is congenital or was acquired within the first 4-6 months of life. In these cases the prognosis for any significant improvement in visual acuity is poor. Electrodiagnostic testing is recommended to establish the prognosis before initiating any treatment or surgical consultation.

e. Management of Isoametropic Amblyopia

Initial treatment of isoametropic amblyopia involves full correction of the refractive error with spectacles or contact lenses. Within 4-6 weeks the practitioner should re-evaluate the visual acuity and refractive status and, if necessary, modify the optical correction to maintain full correction of the ametropia. Thereafter, followup may be conducted every 4-6 months to monitor visual acuity improvement. The patient may not reach his or her best visual acuity for 1-2 years after the initial correction of the refractive anomaly.[23] Whereas these patients often have a severe accompanying accommodative insufficiency,[156,157] the optometrist may prescribe active monocular vision therapy or consult with an optometrist who has advanced training or clinical experience in vision therapy. Ten to fifteen office visits plus home vision therapy 15-20 minutes daily may be sufficient to improve monocular vision function and establish stable binocular vision. More office visits may be necessary if home therapy is not possible.

f. Management of Anisometropic Amblyopia

The initial step in managing anisometropic amblyopia is full correction of the refractive error with spectacles or contact lenses. Contact lenses have been advocated as the optical treatment of choice in myopic anisometropic amblyopia.[158] A few patients, especially adults, may need to begin with less than the full anisometropic prescription to ensure acceptance of the prescription and avoid diplopia. Simply correcting the refrac-

tive error improves visual acuity in some cases. This response is expected more often in younger patients or patients in whom the degree of anisometropia is reasonably small (< 2 D).[25] For children under the age of 6, the recommended initial treatment consists of having the patient wear the refractive correction for 4-6 weeks, then re-evaluating visual acuity before prescribing additional therapy.[17] For older children, adults, and those younger children who do not respond to refractive correction alone, the practitioner may prescribe part-time direct occlusion and active vision therapy or consult with an optometrist who has advanced training or clinical experience in vision therapy.[17,80] Part-time occlusion with an opaque or translucent occluder may be used 2-5 hours per day.

Active vision therapy is recommended because several studies have shown a significant reduction in treatment time when procedures to improve monocular visual function are added.[143-145] Once visual acuity has improved to a shallow amblyopia level (20/40-20/60), residual binocular anomalies, especially suppression, should be re-evaluated and treated.

Therapy involving optical correction and occlusion only may last from 6 to 11.5 months,[127] with the maximum effect of occlusion reached in the first 3-4 months.[128,159] The addition of active vision therapy may reduce occlusion therapy time by up to 50 percent.[145] When all treatment options are combined, the estimated treatment time to attain best visual acuity and establish normal binocular function in nonstrabismic anisometropic amblyopia is 15-25 office visits. For amblyopic patients who have a combined strabismus and anisometropia, the estimated treatment time is greater. Additional steps would be needed to treat the strabismus and, if possible, establish binocular vision.

g. Management of Strabismic Amblyopia

If the patient with strabismic amblyopia has a poor prognosis for binocular vision, the practitioner should set a treatment goal of shallow amblyopia to avoid the possibility of producing diplopia. When the prognosis is questionable because amblyopia complicates the evaluation of important prognostic factors,

the practitioner should also set a goal of shallow amblyopia. Once this level has been reached, the prognosis for binocular vision can be made. For the patient with a good prognosis for establishing binocular vision, the practitioner should set a goal of best possible visual acuity.

The first step in managing strabismic amblyopia is full correction of the refractive error with either spectacles or contact lenses. In certain cases the full correction may need modification to address binocular vision considerations or patient acceptance. The refractive correction alone seldom results in improvement of visual acuity. Occlusion and active vision therapy or consultation with an optometrist who has advanced training or clinical experience in vision therapy may also be part of the initial management plan.

Full-time occlusion is recommended for constant strabismus; part-time occlusion, for intermittent strabismus. When full-time occlusion is prescribed, performance issues need consideration. To allow the patient to function at school, at work, or when good visual acuity is critical, inverse occlusion may be considered with direct occlusion prescribed during less critical seeing tasks.

In children under age 5 who have either central or eccentric fixation, direct full-time occlusion, with adjustments for performance, is the treatment of choice.[116] For patients age 5 and over, eccentric fixation is a significant complication that requires more aggressive treatment.[147] For older patients with eccentric fixation that does not respond to a trial period of 4-6 weeks direct occlusion, active vision therapy should be considered. Active vision therapy may include procedures to improve monocular visual function, especially procedures that promote central fixation.[17] Once visual acuity has improved to 20/40-20/60, the optometrist should re-evaluate and treat residual binocular anomalies, especially suppression and strabismus, if appropriate.

The duration of therapy using only optical correction and occlusion ranges from 6 to 11.5 months[127] with the maximum

effect of occlusion reached in the first 3-4 months.[128,159] The addition of active vision therapy can reduce therapy time by up to 50 percent. The estimated treatment using a combination of treatment options is 25-35 office visits to attain best visual acuity and central fixation. In some cases improved visual acuity will result in improved fusion and eye alignment. For patients who have a residual strabismus, the estimated treatment time may be greater; additional steps would be needed to treat the strabismus and establish normal binocular vision.

3. Patient Education

The patient and/or parent should be informed of the diagnosis, the positive and negative aspects of the prognosis, the treatment options and sequence, and the estimated treatment time. (See Appendix Figure 3). The final management plan formulated in consultation with the patient and/or parents should be responsive to their expectations and preferences. The optometrist should discuss the risks of no treatment (including the impact on future occupational opportunities and the probability of harm to the good eye) and the importance of protective eye wear and regular monitoring of the patient's condition.

4. Prognosis

The prognosis for recovery of visual acuity and improvement of monocular deficits depends on the interplay of several factors:

- Patient compliance
- Specific type of amblyopia
- Monocular fixation status
- Age at onset
- Initial visual acuity
- Age of the patient when treatment is initiated
- Type of treatment prescribed.

There is a sensitive or critical period early in life when an impediment to the development of normal binocular vision (constant strabismus or anisometropia) will cause abnormal visual input (suppression and/or monocular form deprivation),

frequently resulting in amblyopia. In contrast, there is a period which extends much farther in years where the physiological effects of abnormal visual experience can be reversed.[1,80,160]

Considerable plasticity of the visual system is possible in humans with functional amblyopia up to 60 years of age. Numerous clinical studies have reported marked improvement in visual acuity in the amblyopic eyes of older patients.[25,80,127,145,146,161-166] However, the rate, degree, and extent of recovery may be somewhat diminished in the older patient.[167] Motivation, interest, dedication, and commitment to a rigorous treatment program are critical.[17,167]

Amblyopia is a syndrome of visual processing abnormalities. Although improvement of visual acuity has been the emphasis of most clinical studies, it should not be the single measure of success in the treatment of amblyopia.[168] There is interest in developing broader measures of visual function more appropriate for evaluating the condition of amblyopia and its treatment.[169] Unfortunately, review of current literature shows the focus limited almost entirely to the prognosis for visual acuity improvement.

a. Deprivation Amblyopia

The prognosis for improvement to 20/50 or better is good for a patient with unilateral congenital cataract if it is treated within the first 2 months of life. Unfortunately, the prognosis for binocular function remains poor, emphasizing the need for frequent followup through the critical period to maintain visual acuity improvements.[113]

b. Isoametropic Amblyopia

For patients with hyperopic isoametropic amblyopia the likelihood of improving visual acuity to between 20/20 and 20/30 is excellent.[170] This prognosis appears independent of the magnitude of the hyperopia, the initial aided visual acuity, or the age at initial correction. The prevalence of strabismus in patients with uncorrected hyperopic isoametropia is 67 percent. Approximately 30 percent of the patients remain

strabismic even with the optical correction.[23] Therefore, it is important to identify all associated conditions. Although there are no published studies, the general clinical impression is that the prognosis for patients with isoametropic myopic amblyopia due to form deprivation is also good. The practitioner, however, needs to be careful to rule out structural or pathological causes of reduced vision in these cases.

The patient with nonstrabismic isoametropic amblyopia should be monitored on a 4-6 month schedule. Maximal visual acuity improvement is expected within the first 2 years following correction. To speed improvement of visual acuity, active vision therapy may be prescribed following the initial correction of the refractive anomaly to improve accommodative[156] and binocular function.

c. Anisometropic Amblyopia

In cases of hyperopic and astigmatic anisometropic amblyopia, the chances of improving visual acuity to 20/40 or better are considered good; reported success rates are 80-90 percent.[25,67,121,171,172] The prognosis for myopic anisometropic amblyopia, once considered hopeless, is now considered fair; reported success rates are 55-80 percent.[158,173-175]

Post-treatment followup is critical to maintain treatment success. Estimates that 25-87 percent of patients experience some decrease in their visual acuity after treatment[127,169,171,176] emphasize the importance of closely monitoring patients with amblyopia once they have been treated. Followups at 2, 4, 6, and 12 months are recommended during the first year after treatment of amblyopia even when treatment is successful.[17] Most recurrent amblyopia can be attributed to failure to establish normal binocular vision once normal visual acuity has been achieved and the patient's failure to continue wearing the optical correction.

d. Strabismic Amblyopia

The prognosis for the patient with strabismic amblyopia was, until recently, considered poorer than that of one with anisometropia. Recent studies have shown that treating compliant young patients has a success rate similar to that reported for treatment of anisometropic amblyopia.[67,128] The key to successful treatment of strabismic amblyopia is good patient compliance with the recommended treatment. The length of treatment needed to achieve success is age-related; on average, less time is needed for treatment of younger children. The treatment is longer for older children because they often develop eccentric fixation, which lengthens the treatment time and reduces the prognosis for successful treatment of the amblyopia.[31,92,176] A more favorable prognosis is typically associated with early diagnosis, early treatment, and central fixation.

For the patient who remains strabismic, there is a strong chance that the amblyopia will recur. When the patient is over 5 years old, the possibility of creating an alternating strabismus to help maintain approximately equal visual acuity is significantly less.[177] The best method of maintaining improved visual acuity is to establish normal binocular vision.[94] However, the fact there is no guarantee that amblyopia will not recur[17] emphasizes the importance of monitoring amblyopic patients closely once they have been treated. Even following successful treatment of amblyopia and strabismus, followups at 2, 4, 6, and 12 months are recommended.[17]

CONCLUSION

During the period of rapid development of the visual pathways, from birth to approximately 6-8 years of age, the visual system is susceptible to the development of amblyopia caused by form deprivation, optical defocus, or misalignment of the eyes. The optometrist's major diagnostic task is to identify the underlying etiology and associated conditions. The prognosis for success is generally good, especially if the amblyopia is diagnosed and treated early. Optical correction, occlusion, and vision therapy are the major treatment options.

Untreated amblyopic patients are at a greater risk for loss of vision in the better eye. The optometrist should carefully outline the possible outcome without treatment, including the risk for impact on future occupational opportunities, the possibility of harm to the good eye, and the importance of protective eye wear and regular monitoring of the patient's condition.

The prevalence, potential risks, and possible costs of untreated amblyopia contrasted with the good prognosis for patients treated at any age necessitate the involvement of optometrists in the diagnosis and treatment, or referral for consultation of patients with amblyopia.

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IV. APPENDIX

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

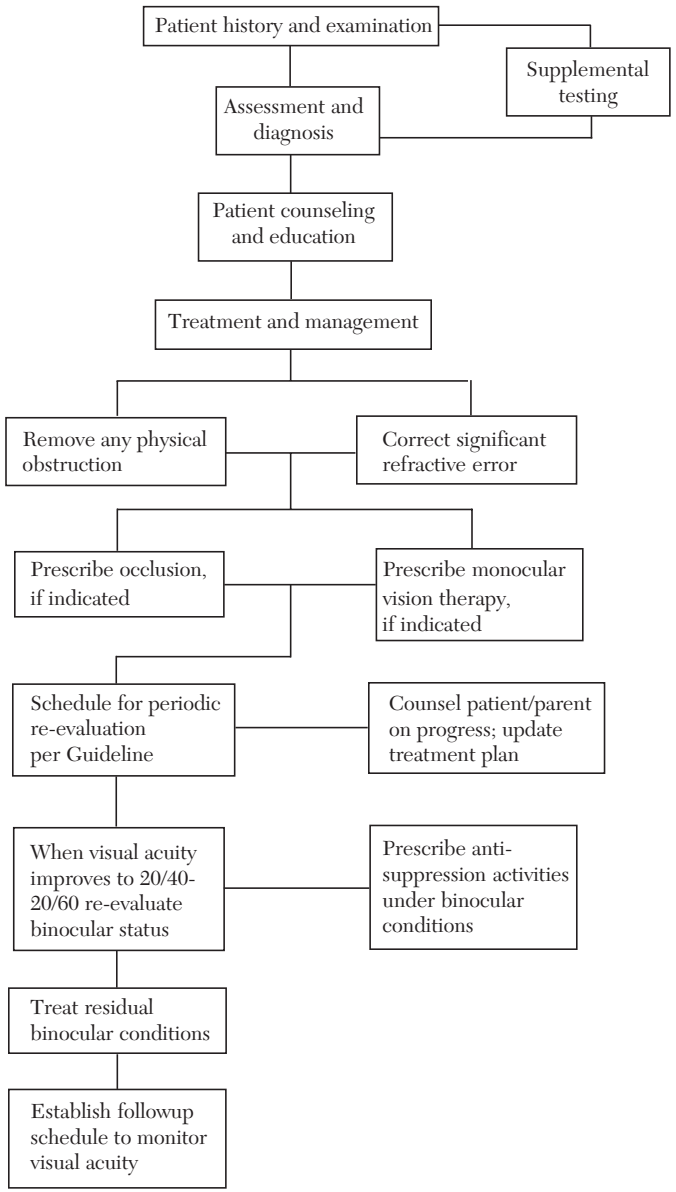

Figure 2
Potential Components of the Diagnostic Evaluation for Amblyopia

A. Patient history

B. Visual acuity

C. Refraction (noncycloplegic and cycloplegic)

D. Monocular fixation

E. Ocular motor deviation

F. Sensorimotor fusion

G. Accommodation

H. Ocular motility

I. Ocular health assessment and systemic health screening

J. Supplemental testing
   1. Electrodiagnostic testing
   2. Additional differential diagnostic testing

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

Type of Patient	Evaluation Visits	Prognosis*	Treatment Options+
Monocular Form Deprivation Amblyopia	1-2	Fair, (if diagnosed and treated during critical period)	1. Surgery optical correction 2. Surgery optical correction visual stimulation
Binocular Form Deprivation Amblyopia	1-2	Fair, (if diagnosed and treated during critical period)	1. Surgery optical correction 2. Surgery optical correction visual stimulation
Isometropic Refractive Amblyopia	1-2	Good	1. Optical correction 2. Optical correction vision therapy
Anisometropic Refractive Amblyopia	1-2	Good	1. Optical correction 2. Optical correction occlusion (part-time) 3. Optical correction occlusion (part-time) vision therapy
Strabismic Amblyopia (Central Fixation)	1-2	Good	1. Optical correction occulsion 2. Optical correction occlusion vision therapy
Strabismic Amblyopia (Eccentric Fixation)	1-2	Fair	1. Optical correction occlusion 2. Optical correction occlusion vision therapy

* Figure 3 extends horizontally on page 47.

* General prognosis; prognosis is improved during critical sensitive period of development, but compliance and motivation afford improvements into adulthood.

+ Surgery is indicated in cases of congenital cataract and ptosis.

Figure 3 Continued . . .

Frequency of FU visits	Estimated Total VT visits‡	Composition of FU Evaluations			
1. Every 2-4 wks for 1 yr; every 6 mos thereafter 2. Every 2-4 wks for 1 yr; every 6 mos thereafter		Each visit	Each visit	____	Each visit
1. Every 2-4 wks for 1 yr; every 6 mos thereafter 2. Every 2-4 wks for 1 yr; every 6 mos thereafter		Each visit	Each visit	____	Each visit
1. Re-evaluate in 4-6 wks; every 4-6 mos FU 2. Re-evaluate in 4-6 wks; 2-6 mos FU after VT	10-15	Each visit	PRN	PRN	Each visit
1. Re-evaluate in 4-6 wks; every 2-6 mos FU 2. Re-evaluate in 4-6 wks; every 2-4 wks FU 3. Re-evaluate in 4-6 wks; 2-6 mos FU after VT	15-25	Each visit	PRN	PRN	Each visit
1. Re-evaluate in 4-6 wks; every 2-4 wks FU 2. Re-evaluate in 4-6 wks; 2-6 mos FU after VT	15-25	Each visit	PRN	PRN	Each visit
1. Re-evaluate in 4-6 wks; every 2-4 wks FU 2. Re-evaluate in 4-6 wks; 2-6 mos FU after VT	25-35	Each visit	PRN	Each visit	Each visit

‡ Estimated visits may vary based on co-existing conditions, patient compliance, etc.

VA=visual acuity, REF=refractive status, MF=monocular fixation, BS=binocular status, FU=followup visit, VT=vision therapy, PRN=as necessary

Figure 4
ICD-9-CM Classification of Functional Amblyopia

Amblyopia ex anopsia	**368.0**
Amblyopia, unspecified	**368.00**
Strabismic amblyopia Suppression amblyopia	**368.01**
Deprivation amblyopia	**368.02**
Refractive amblyopia	**368.03**

Glossary

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

Amblyopiogenic factors Underlying clinical conditions that are responsible for amblyopia, e.g., uncorrected anisometropia.

Amblyopiogenic mechanisms Physiological mechanisms of form vision deprivation and binocular inhibition that actually cause amblyopia.

Aniseikonia Relative difference in the size or shape of the ocular images.

Anisometropia Condition of unequal refractive state for the two eyes, in which one eye requires a different lens correction than the other.

Anisometropic amblyopia Refractive amblyopia attributed to previously uncorrected anisometropia, typically in the eye with the greater refractive error.

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

Binocular inhibition Cortical process that inhibits or suppresses information coming from one eye, i.e., the eye with the higher refractive error in anisometropia or the turned eye in strabismus.

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

Eccentric fixation Monocular fixation that does not use the central foveal area.

Electroretinogram (ERG) The electrical effect originated by a pulse of light recorded from the surface of the eyeball. Measures general retinal function.

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

Latent nystagmus Nystagmus induced by covering either of the two eyes but otherwise absent, commonly found in early-onset strabismus patients.

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

Occlusion The act of obscuring or blocking the vision of an eye with an occluder.

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

Penalization therapy Procedure in which the dominant eye is occluded or blurred with an optical lens or pharmacological agent so as to favor the nondominant eye.

Psychogenic Of mental origin or causation.

Psychometric Process of analyzing visual acuity data, specifically visual acuity data from amblyopic patients, to arrive at a more accurate and reliable estimate of true visual acuity.

Sensorimotor fusion The process in which stimuli seen separately by the two eyes are combined, synthesized, or integrated into a single image; and the relative movements of the two eyes necessary to maintain this single image.

Stereopsis Perception of three-dimensional depth or solidity due to retinal disparity.

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

Suppression The inability to perceive all or part of objects in the field of vision of one eye.

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

Visual evoked potential Electrical discharge that occurs in the visual cortex in response to visual stimuli, recorded through electrodes on the scalp at the back of the head, generally reflecting foveal function and visual pathway integrity from the eye to the visual cortex.

Visuoscopy Examination of the eye using an ophthalmoscope with a calibrated fixation target to determine whether the patient has central or eccentric fixation.

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