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"America's Eyecare Provider"

Care of the Patient with
**Strabismus:
Esotropia
and
Exotropia**



American Optometric Association

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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.

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CARE OF THE PATIENT WITH STRABISMUS: ESOTROPIA AND EXOTROPIA

Reference Guide for Clinicians

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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 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 strabismus.

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

- Identify patients at risk of developing strabismus
- Accurately diagnose strabismus
- Improve the quality of care rendered to patients with strabismus
- Minimize the adverse effects of strabismus and enhance the patient's quality of life
- Preserve the gains obtained through treatment
- Inform and educate other health care practitioners including primary care physicians, teachers, parents, and patients about the visual complications of strabismus and the availability of treatment.

I. STATEMENT OF THE PROBLEM

Strabismus, often called “crossed-eyes” or “wall eyes,” is a condition in which the eyes are not properly aligned with each other. One eye is either constantly or intermittently turned in, out, up, or down. This ocular misalignment may be accompanied by abnormal motility of one or both eyes, double vision, decreased vision, ocular discomfort, or abnormal head posture. Although the exact cause cannot always be determined with reasonable certainty, strabismus is usually attributable to refractive, sensory or organic, anatomic or motor, or innervational causes. Any of these factors alone can result in strabismus; however, strabismus may be the result of multiple factors, which, occurring alone, might not cause the disorder.

For some individuals, strabismus can result in permanent vision loss. Young children with strabismus often develop amblyopia (lazy eye) and impaired stereopsis (binocular depth perception). Early identification and treatment of strabismic children may prevent amblyopia.¹ The strabismic child with amblyopia has a significantly higher risk of becoming blind by losing vision in the nonamblyopic eye due to trauma or disease.²

Normal binocular vision is required for many occupational and avocational tasks, as well as many other activities in daily life. Therefore, prompt diagnosis and treatment of strabismus are critical. For the individual with intermittent strabismus who has the potential for binocular vision and for the older individual who develops strabismus, symptoms such as diplopia, headaches, blurred vision, and ocular fatigue may cause the person to alter his or her activities of daily living. Studies comparing binocular with monocular performance on a variety of tasks for a group of normal individuals indicate that strabismus frequently leads to inefficient performance on various educational, occupational, and avocational tasks.^{3,4}

A student with intermittent strabismus may avoid reading, resulting in poor academic achievement. An employee with intermittent strabismus may suffer fatigue and headaches,

resulting in reduced productivity. Strabismus may also be cosmetically displeasing and produce significant psychological impact on patients, especially adolescents and those whose jobs involve substantial personal contact.⁵ In addition, delayed development (e.g., reaching milestones such as first walking and using single words) or difficulty with tasks involving visual perception has been found in young children with strabismus.⁶

Some types of strabismus, particularly those caused by high refractive errors, can be prevented. Esotropias that occur due to an abnormal amount of hyperopia are potentially preventable if the hyperopia is treated prior to the onset of the deviation. More than 30 percent of children with hyperopia that exceeds 4 diopters (D) develop esotropia by 3 years of age.⁷ Whereas adult-onset strabismus can result from a neurologic disorder (e.g., brain tumor) or systemic disease (e.g., diabetes mellitus), adequate control of the general health status of a person afflicted with such a condition may lessen the possibility of the individual developing strabismus.

Young children are not routinely screened for strabismus. Some school screenings require assessment of visual acuity with and without convex lenses, which can identify some children with significant hyperopia. Ideally, these screenings should be conducted early (i.e., before 2-3 years, the peak ages for the onset of accommodative esotropia). As a method of screening for refractive error, photorefractometry may gain popularity and may aid in earlier detection of some cases of accommodative esotropia.⁸ Nevertheless, because problems encountered in screening young children for refractive errors and strabismus may result in underdetection of vision problems, every child should undergo optometric examination by 6 months of age and again at the age of 3 years.*

Remediation of strabismus requires treatment by an eye care practitioner, and the results are usually best when treatment is instituted early. Preservation of vision and binocular function

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

result from proper diagnosis, treatment, and patient compliance. Periodic re-evaluation is important for appropriate patient management. Occasionally, reintervention is needed.

A. Description and Classification of Strabismus

Proper alignment of the visual axes of the eyes is necessary for normal binocular vision. The tendency for the eyes to deviate from each other can be classified as “latent” when the eyes are kept in alignment by the fusion mechanism and “manifest” when alignment is not maintained by fusion. Latent deviation of the eyes is called heterophoria; manifest deviation is heterotropia or strabismus.

Strabismus is defined as a manifest deviation of the primary lines of sight of 1 prism diopter (PD) or more. In strabismus, one eye is either constantly or intermittently not directed toward the same point as the other eye when the patient attempts to fixate an object. As a result, no image of the fixated object is formed on the fovea of the strabismic eye. The convergent (inward) misalignment of one eye is defined as esotropia; a divergent (outward) misalignment, exotropia; an upward misalignment, hypertropia; and a downward misalignment, hypotropia.

In strabismus the misalignment of the eyes can be classified in a number of ways⁹:

- Direction (convergent, divergent, or vertical)
- Comitant or incommitant (deviation equal or varying with the direction of gaze)
- Frequency (constant or intermittent)
- Involvement of accommodative system (accommodative or nonaccommodative)
- State of vergence system (convergence insufficiency or divergence excess; divergence insufficiency or convergence excess; basic esotropia or basic exotropia)
- Laterality (unilateral or alternating)
- Time of onset (congenital or acquired)
- Size (large, small, or intermediate).

The scope of this Guideline includes the diagnosis, treatment, and management of the most common clinical forms of nonparalytic esotropia and exotropia; it does not include vertical nor paretic strabismus. See Appendix Figure 3 for ICD-9-CM classification of esotropia and exotropia. The following sections describe various forms of esotropia and exotropia.

I. Esotropia

A convergent strabismus is termed an esotropia. Esotropia is usually constant. An intermittent angle of esotropia generally occurs in association with accommodative esotropia or decompensated esophoria (a tendency of one eye to deviate inward). The intermittency of accommodative esotropia is attributed to the fluctuating accommodative status of the patient at the onset of the deviation. Clinical forms of esotropia are discussed in the following paragraphs.

a. Infantile Esotropia

When esotropia begins in the neurologically normal child during the first 6 months of life, it is classified as “infantile.” When it occurs after 6 months of age, it is referred to as “early-acquired.” True “congenital” esotropia, which is present at birth, is considered extremely rare;¹⁰ however, the terms “infantile esotropia,” “essential infantile esotropia,” and “congenital esotropia” are often used interchangeably.

b. Acquired Esotropia

Acquired forms of esotropia usually occur at a later age than infantile esotropia. Generally, normal binocular vision has existed prior to the onset of the condition.

- **Accommodative esotropia.** This is an acquired strabismus associated with the activation of accommodation. The esotropia is attributed totally or partly to either uncorrected hyperopic refractive error and/or a high accommodative convergence/accommodation (AC/A) ratio. Accommodative strabismus has a better understood mechanism and a more straightforward treatment than any other form of strabismus.

- **Acute esotropia.** When a convergent strabismus develops without any apparent etiology in a patient with previously normal binocular vision, it is called acute esotropia. The sudden diplopia that usually occurs in acute esotropia may result from an underlying and potentially life-threatening disease process; thus it requires immediate evaluation. Its onset can often be traced to a precise hour of a particular day. Causes of acute esotropia are listed in Table 1.

Table 1
Causes of Acute Esotropia and Exotropia

-
- Neoplasm
 - Head trauma
 - Intracranial aneurysm
 - Hypertension
 - Diabetes mellitus
 - Atherosclerosis
 - Hydrocephalus
 - Multiple sclerosis
 - Meningitis/encephalitis
 - Myasthenia gravis
 - Ophthalmoplegic migraine
 - Chemotherapy
-

- **Mechanical esotropia.** A convergent strabismus caused by a mechanical restriction (e.g., fibrosis of muscle tissue) or a physical obstruction (e.g., blowout fracture) of the extraocular muscles is classified as a mechanical esotropia. A common form of mechanical esotropia is Duane's retraction syndrome type I, in which there is a limitation or absence of abduction. The palpebral fissure narrows when the eye rotates inward (adduction) and widens when abduction is attempted.

c. Secondary Esotropia

An esotropia that results from a primary sensory deficit or as a result of surgical intervention is classified as a secondary esotropia.

- **Sensory esotropia.** A convergent strabismus resulting from visual deprivation or trauma in one eye that limits sensory fusion is classified as a sensory esotropia. It may result from any number of conditions that limit visual acuity in one eye (e.g., uncorrected anisometropia, unilateral cataract, corneal opacity, macular disease). It occurs most frequently in persons under 5 years of age.¹¹
- **Consecutive esotropia.** A convergent strabismus that occurs iatrogenically after surgical overcorrection of an exodeviation, consecutive esotropia is frequently associated with other oculomotor anomalies (e.g., vertical or cyclo deviations) and may result in amblyopia in young children and diplopia in adults.

d. Microesotropia

When the angle of esotropia is less than 10 PD, it is classified as microesotropia. This condition often occurs with strabismus beginning in a child under 3 years of age, and, in some cases, may escape diagnosis by conventional methods. The esotropia is constant and usually unilateral. The terms "microtropia," "microsquint," "minitropia," and "small-angle deviation" may also be used to identify microesotropia.

2. Exotropia

Exotropia, or divergent strabismus, can be divided into a number of subclassifications. In basic exotropia, the angle of deviation is approximately the same at distance or near. In convergence insufficiency, the angle of deviation is greater at near. Divergence excess means the angle of deviation is greater at distance. Although exotropias may be constant or intermittent, most are intermittent. Other clinical classifications of exotropia are discussed in the following paragraphs.

a. Infantile Exotropia

A divergent strabismus that begins during the first 6 months of life is classified as infantile exotropia. It is less common than infantile esotropia. Some cases of exotropia in infants may be associated with neurological syndromes or defects.

b. Acquired Exotropia

An exotropia occurring after six months of age is generally considered to be acquired rather than congenital.

- **Intermittent exotropia.** In intermittent exotropia, the patient sometimes manifests diplopia, suppression, or anomalous correspondence, and at other times, normal binocular alignment with good stereopsis. The period of strabismus is variable for each patient. Basic intermittent exotropia accounts for approximately 50 percent of all cases of intermittent exotropia, with convergence insufficiency and divergence excess making up the balance of cases in approximately equal proportions.¹²
- **Acute exotropia.** When a divergent strabismus develops suddenly in a patient who previously had normal binocular vision, it is classified as acute exotropia. This condition can result from an underlying disease process (Table 1) or a decompensating exophoria.
- **Mechanical exotropia.** Mechanical exotropia is a divergent strabismus caused by a mechanical restriction (e.g., fibrosis of muscle tissue) or a physical obstruction of the extraocular muscles. One form of mechanical exotropia is Duane's retraction syndrome type II, in which there is a limitation or absence of adduction, accompanied by a narrowing of the palpebral fissure and retraction of the globe.

c. Secondary Exotropia

An exotropia that results from a primary sensory deficit or as a result of surgical intervention is referred to as a secondary exotropia.

- **Sensory exotropia.** A divergent strabismus resulting from a unilateral decrease in vision that disrupts fusion, sensory exotropia may be due to a sensory deficit such as uncorrected anisometropia, unilateral cataract, or other unilateral visual impairment. Sensory exotropia and sensory esotropia occur with equal frequency in children under age

5; however, sensory exotropia predominates in persons older than 5 years.¹¹

- **Consecutive exotropia.** Exotropia that occurs following surgical overcorrection of an esotropia is referred to as consecutive exotropia. This form of exotropia can also occur spontaneously in a formerly esotropic patient. A spontaneous change from esotropia to exotropia may be related to a sensory deficit of the deviating eye, to weak binocular function, or to excessive hyperopic refractive error.¹³

d. Microexotropia

A constant exotropia of less than 10 PD, microexotropia occurs much less frequently than microesotropia.

B. Epidemiology of Strabismus**1. Prevalence and Incidence**

The estimated prevalence of strabismus in the general population is from 2 to 6 percent.¹⁴ Between 5 and 15 million individuals in the United States may have this condition. Several studies of clinical populations have reported that esotropia appears to occur approximately 3 times as often as exotropia in children.¹⁵ However, the National Health Survey of individuals 4-74 years of age found a higher prevalence of exotropia (2.1%) than esotropia (1.2%) in the U.S. population. This difference is probably related to the fact that the overall prevalence of strabismus in persons 55-75 years of age (in whom exotropia is more common) is 6.1%, substantially greater than for very young children 1-3 years of age (1.9%) or children and adults 4-54 years of age (3.3%).¹⁴

Most cases of esotropia are accommodative or partially accommodative.¹⁶ Infantile esotropia accounts for 28-54 percent of all esotropia.¹⁷ Intermittent exotropia is the most common type of exotropia.¹⁸ Exotropia has been reported to be more prevalent among Asians than Caucasians.¹⁹ Women comprise 60-70 percent of patients with exotropia.²⁰

2. Risk Factors

Strabismus is more prevalent in persons with multiple handicaps, occurring in approximately 50 percent of patients with Down's syndrome,²¹⁻²³ 44 percent of patients with cerebral palsy,^{24,25} and 90 percent of patients with craniofacial dysostosis such as Apert-Crouzon syndrome.²⁶⁻²⁹ The prevalence of strabismus is also higher in families in which a parent or sibling has strabismus, ranging from 23 to 70 percent of family members.³⁰⁻³² Whether strabismus itself or the conditions underlying the strabismus are genetic is unclear. The common occurrence of sensorimotor anomalies in the pedigrees of strabismic probands suggests that all siblings of a strabismic child should be examined by 6 months of age to rule out the presence of strabismus.

C. Clinical Background of Strabismus

Although strabismus can develop at any age, it usually develops during childhood. Most cases begin prior to 6 years of age; the peak age of onset is around 3 years.³³ Strabismus acquired in adolescence or adulthood is frequently either motor or sensory in origin and can be a manifestation of systemic disease (e.g., diabetes mellitus) or neurologic disorder (e.g., brain tumor). Strabismus can also develop in adults following decompensation of a heterophoria. In patients of all ages, trauma to the head or orbit may result in strabismus.

1. Accommodative Esotropia

a. Natural History

Accommodative esotropia has an average age of onset of 2 1/2 years (a range of 1 to 8 years);³⁴ however, cases have been documented prior to age 1.³⁵ The esotropia usually occurs when the child first becomes interested in viewing near objects. It is frequently first seen by the parents when the child is tired or not feeling well; in fact, the onset may be precipitated by a febrile illness. As many as one-third of all children with esotropia obtain normal ocular alignment when wearing an optical correction for hyperopia, and another one-third obtain significant but not complete reduction of the esotropia.³⁶

b. Signs, Symptoms, and Complications

Patients with accommodative esotropia may exhibit any of the following:

- A small to moderate (generally 10-35 PD), often variable, angle of deviation that usually occurs more frequently at near. Depending on the AC/A ratio, the esotropia at near may be larger or similar to the esotropia at distance.
- Uncorrected hyperopia (generally 2-6 D) and/or a high AC/A ratio. For patients with normal AC/A ratios (similar distance and near deviations), the average amount of hyperopia is 4.75 D, whereas with high AC/A ratios (near deviation exceeds distance deviation by 10 PD or more), the average amount of hyperopia is only 2.25 D.³⁷ About 5 percent of patients with accommodative esotropia have minimal refractive error and manifest esotropia exclusively for near viewing distances. This type of esotropia is attributed solely to an elevated AC/A.
- Esotropia beginning as intermittent strabismus. When treatment is delayed or not provided, many deviations become constant, and sensory adaptations such as amblyopia, suppression, and anomalous correspondence may develop.
- Amblyopia (limited to cases that have become constant and unilateral and cases that are partially accommodative).
- Diplopia or closure of one eye when doing near work.
- Inferior oblique overaction, which develops in approximately 35 percent of cases.³⁸

c. Early Detection and Prevention

Children suspected of having accommodative esotropia should be examined immediately. The prognosis for achieving normal binocular vision is excellent provided treatment is not delayed. Better results usually occur when treatment is initiated during the intermittent phase.³⁹ If untreated, the patient may develop

a gradual nonaccommodative esotropia, accompanied by amblyopia, suppression, and anomalous correspondence. Subsequently, lens therapy is less successful in eliminating the total angle of deviation, and binocular vision can be lost.

2. Acute Esotropia and Exotropia

a. Natural History

Acute esotropia or exotropia generally develops in children older than 6 years, adolescents, and adults. Acute esotropia occurs less frequently than either infantile esotropia or accommodative esotropia. Acute esotropia can be associated with decompensated heterophoria,⁴⁰ late-onset accommodative esotropia,⁴¹ abducens nerve palsy,⁴² divergence paralysis/divergence insufficiency,⁴³⁻⁴⁵ or acute acquired comitant esotropia.⁴⁶⁻⁴⁸

Acute exotropia occurs less frequently than acute esotropia. Because isolated medial rectus palsy is rare, acute exotropia is usually associated with either decompensated exophoria or acquired oculomotor nerve palsy.

b. Signs, Symptoms, and Complications

Table 2 presents the signs, symptoms, and complications of the various types of acute esotropia.

c. Early Detection and Prevention

The patient with acute esotropia or exotropia should be examined immediately due to the possibility of an underlying disease process (See Table 1). Consultation with other health care professionals may be needed to determine the underlying cause of abducens nerve palsy, divergence paralysis, or acute acquired comitant esotropia, as well as acute exotropia in those cases in which the clinician is uncertain of the cause.

3. Consecutive Esotropia and Exotropia

a. Natural History

Consecutive esotropia is iatrogenic, developing only following surgical treatment of an exotropia; consecutive accommodative esotropia has been reported in some children following surgical

Table 2
Clinical Conditions Presenting as Acute Esotropia

Condition	Deviation in Primary Position	Motility	Refraction	Diplopia	Fusion	Systemic/Neurologic Disease	Comment
Decompensated heterophoria	10-20 PD at distance and near	Normal	Similar to non-strabismic population	Yes; at distance and near	Yes	No	History of pre-existing esophoria that has now become manifest; can be provoked by prolonged occlusion or febrile disease.
Late onset accommodative esotropia	10-35 PD; may be larger at near	Normal	Causative (2-6 D) hyperopia	Yes; usually more at near	Yes	No	Can be prevented by correcting high hyperopic refractive error during childhood.
Abducens nerve palsy	20-40 PD; larger at distance	Abnormal; restricted abduction in one or both eyes	Similar to non-strabismic population	Yes; more at distance	Yes, may have to use head turn	Frequent	Abducens nerve palsy should always be suspected with acute esotropia.
Divergence paralysis/insufficiency	8-30 PD at distance; 4-18 PD at near	Normal	Similar to non-strabismic population	Yes; only at distance	Yes	Usually	May precede abducens nerve palsy or be its sequelae.
Acute acquired comitant esotropia	15-75 PD at distance and near	Normal	Similar to non-strabismic population	Yes	Most of the time	Sometimes	Patients with related disease may also have poor fusion and other abnormal ophthalmic findings, such as nystagmus.

treatment of intermittent exotropia.⁴⁹ On the other hand, consecutive exotropia can develop either iatrogenically postsurgically or occur spontaneously. Predisposing factors for cases that occur spontaneously include hyperopia greater than 4.50 diopters, amblyopia, and poor fusion.¹³

b. Signs, Symptoms, and Complications

The patient with consecutive esotropia may exhibit these characteristics:

- Constant unilateral esotropia
- Refractive errors similar to those in the nonstrabismic population
- Amblyopia and loss of stereopsis which may occur if the esotropia is allowed to persist in young children
- Diplopia
- A history of a presurgical intermittent exotropia, which indicates that the potential for normal binocular vision is good when treatment is provided soon after onset.

The patient with consecutive exotropia may exhibit the following:

- Constant unilateral exotropia at distance and near
- Hyperopia over 4.50 D
- Amblyopia and medial rectus restriction, which are common in iatrogenic cases^{50,51}
- Reduced potential for normal binocular vision and high-level stereopsis
- Diplopia.

c. Early Detection and Prevention

In patients who receive surgical treatment for intermittent exotropia who then become esotropic, spontaneous reduction in the consecutive esotropia usually occurs within 2-3 weeks. Persistent esotropia may develop in 5-10 percent of these patients, who require immediate evaluation because amblyopia may develop in children less than 6 years old and loss of fusion may result at any age.⁵²⁻⁵⁴

On the other hand, patients with consecutive exotropia generally do not require immediate treatment unless diplopia develops. Diplopia is more likely to occur when the exotropia develops in adulthood and there is no suppression scotoma on the temporal hemiretina.⁵⁵

4. Infantile Esotropia

a. Natural History

Recent studies indicate that infantile esotropia is not congenital and most likely develops between 2 and 4 months of age, a period during which most infants are becoming orthotropic.^{10,56,57} A history of infantile esotropia in parents or siblings of affected patients is common. Characterized by a large deviation, infantile esotropia is commonly associated with cross-fixation (viewing targets in the right field of gaze with the left eye and visa versa) and severely interferes with the development of normal binocular vision. It must be distinguished from pseudoesotropia and other early onset esotropias, including Duane's retraction syndrome, congenital abducens nerve palsy, Moebius' syndrome, and sensory and accommodative esotropias.⁵⁸

b. Signs, Symptoms, and Complications

Patients with infantile esotropia may exhibit any of the following:

- A large-angle, constant esotropia (generally 40-60 PD) at distance and near that begins before 6 months of age
- Refractive errors skewed toward low hyperopia (approximately 50 percent have hyperopia exceeding 2 D)⁵⁹
- Amblyopia (about 40 percent of all cases)⁶⁰
- A high incidence of various ocular motility disorders, including limited abduction, inferior oblique overaction, and dissociated vertical deviation
- Limited potential for both normal binocular vision and good levels of stereopsis
- Latent or manifest nystagmus.

c. Early Detection and Prevention

Transient strabismic deviations occur frequently in neonates,⁵⁶

but in most cases they resolve by 3 months of age. Any infant with esotropia that persists at 3 months of age should be examined immediately. Because strabismus may be secondary to a more serious condition, early examination should be encouraged, especially in the presence of any additional ocular findings (e.g., leukocoria).

5. Infantile Exotropia

a. *Natural History*

Infantile exotropia, characterized by a large, constant deviation, severely interferes with the development of normal binocular vision. It must be differentiated from the rather common transient exotropias seen during the first few months of life,^{10,56,57} as well as from constant exotropia caused by poor vision in one eye. The development of constant exotropia in a neurologically normal child 6 months of age or younger is extremely rare, occurring in only 1 of 30,000 patients.⁶¹

b. *Signs, Symptoms, and Complications*

Patients with infantile exotropia can be characterized by the following:

- A large, constant deviation (generally 30-80 PD) at both distance and near, beginning before 6 months of age^{61,62}
- Refractive errors similar to those in the nonstrabismic population
- A high incidence of various ocular motility disorders, including limited adduction, inferior oblique overaction, and dissociated vertical deviation.

Infantile exotropia is rarely amblyopiogenic because most patients have an alternating fixation pattern. However, patients with infantile exotropia have limited potential for normal binocular vision and high-level stereopsis.

c. *Early Detection and Prevention*

Benign transient alternating exotropic deviations frequently occur in neurologically normal children during the first months of life.^{56,57} However, the infant with constant unilateral exotropia requires immediate evaluation to confirm or rule out

sensory exotropia. Any infant who has alternating exotropia that persists at 6 months of age and who has not previously been evaluated should be examined immediately.

6. Intermittent Exotropia

a. *Natural History*

Most exotropias are intermittent and may be of three types. In basic exotropia, the angle of deviation is approximately the same at distance and near. In convergence insufficiency, the angle of deviation is larger at near. In divergence excess, the angle of deviation is larger at distance. When it occurs in younger children, the exotropia is usually of the divergence excess type. The parents often note that the child is not looking at them properly or tends to close one eye when viewing at distance or in bright sunlight. Basic and convergence insufficiency exotropias are more likely in older individuals. Intermittent exotropia must be distinguished from constant exotropia (e.g., infantile, consecutive, and sensory exotropia, Duane's retraction syndrome type II, and oculomotor nerve palsy).

b. *Signs, Symptoms, and Complications*

Patients with intermittent exotropia may exhibit the following characteristics:

- A significant exodeviation at one or more fixation distances. The exotropia may not be apparent until the patient becomes fatigued or inattentive, or after prolonged dissociation.
- Minimal or no amblyopia.
- Reduced positive fusional vergence amplitudes and facility at one or more distances.
- Levels of stereopsis equal to or greater than 60 seconds of arc when fusing; no stereopsis when exotropic.
- Discomfort (e.g., headaches, difficulty reading, and eyestrain) during or following prolonged visual activity.
- Closing one eye in bright sunlight.
- Diplopia, which usually implies that the intermittent exotropia developed after early childhood.

- Suppression and/or anomalous correspondence in the patient who does not report diplopia when the eye is exotropic.
- Associated accommodative dysfunction, which is more likely with convergence insufficiency exotropia.⁶³

c. Early Detection and Prevention

Unlike other strabismic deviations, delay in treatment of intermittent exotropia is not likely to result in permanently worsened visual status for very young children. Nevertheless, professional confirmation is essential at an early age to differentiate intermittent exotropia from those exotropias that are amblyopiogenic and likely to cause loss of normal binocular vision. If treatment is postponed, the child should be monitored closely. Timely intervention is needed to prevent visual symptoms that may affect school performance for children and job performance for adults.

7. Mechanical Esotropia and Exotropia

a. Natural History

Mechanical strabismus can be either congenital or acquired. Duane's retraction syndrome has been documented in a 1-day-old child.⁵⁶ Fibrosis of most or all of the extraocular muscles can be present at birth. In thyroid myopathy, a condition that is usually acquired in adulthood, enlargement of the extraocular muscles, particularly the inferior rectus and medial rectus, causes esotropia and hypotropia.

b. Signs, Symptoms, and Complications

Mechanical esotropia or exotropia may be manifested in patients in the following ways:

- Minimal esotropia or exotropia in the primary position⁶⁴
- Significantly increased deviation in right or left gaze
- Restricted horizontal versions and ductions
- Frequent compensatory head turns, which usually indicate the absence of amblyopia and the potential for normal binocular vision.⁶⁵

c. Early Detection and Prevention

The patient with mechanical strabismus requires immediate evaluation to distinguish this condition from the more ominous paretic strabismus, which is more likely caused by a disease process.

8. Microtropia

a. Natural History

Microtropia frequently results from the treatment of a larger-angle esotropia or exotropia by optical correction, vision therapy, pharmacological agents, and/or extraocular muscle surgery. Microtropia can also occur idiopathically or secondary to anisometropia.^{66,67}

b. Signs, Symptoms, and Complications

The patient with microtropia usually exhibits the following:

- A constant, unilateral esotropia of less than 10 PD at distance and near (constant, unilateral exotropia of less than 10 PD is rare)
- Amblyopia
- Eccentric fixation
- Rudimentary binocular vision
- Anomalous correspondence
- Deficient stereopsis
- Anisometropia.

c. Early Detection and Prevention

Microtropia is usually diagnosed later than the more obvious types of strabismus; however, because of the high incidence of amblyopia, any child suspected of having microtropia should be evaluated immediately.

9. Sensory Esotropia

a. Natural History

Most cases of sensory esotropia develop within the first 5 years of life.¹¹ In this relatively uncommon type of esotropia, congenital or traumatic unilateral cataracts account for nearly 30 percent of all cases. The degree of monocular visual impairment leading to sensory esotropia ranges from 20/60 to light perception.¹¹

b. Signs, Symptoms, and Complications

Patients with sensory esotropia may exhibit the following:

- Constant unilateral esotropia at distance and near
- Anisometropia at degrees high enough to cause the esotropia
- Vertical deviations associated with overaction of the inferior and/or superior oblique muscles
- Functional amblyopia superimposed on the organically caused vision loss⁶⁸
- Limited potential for normal binocular vision and high-level stereopsis.

c. Early Detection and Prevention

Patients with suspected sensory esotropia should be evaluated immediately to determine the cause for the vision loss and strabismus. For example, there is greater urgency in infants, from whom congenital cataracts need to be removed within the first weeks of life in the hope of obtaining normal visual acuity and possible binocular vision.⁶⁹

10. Sensory Exotropia**a. Natural History**

Sensory exotropia occurs in both children and adults, but it is more common in adults. Although it occurs more frequently than sensory esotropia, it has the same causative factors. The relative decrease in tonic convergence with age is thought to result in the higher incidence of sensory exotropia.

b. Signs, Symptoms, and Complications

Patients with sensory exotropia may exhibit these characteristics:

- Constant unilateral exotropia at distance and near
- High degrees of anisometropia, which can be causative
- Frequently accompanying vertical deviations associated with overactivity of the inferior and/or superior oblique muscles
- Functional amblyopia which may be superimposed on the organically caused vision loss
- Reduced potential for normal binocular vision and high-level stereopsis for cases with early childhood onset.

c. Early Detection and Prevention

As with sensory esotropia, the patient with suspected sensory exotropia should be evaluated immediately to determine the cause of the vision loss and strabismus.

II. CARE PROCESS

A. Diagnosis of Strabismus

The examination of strabismic patients generally includes all areas of the evaluation of a comprehensive adult or pediatric eye and vision examination.* The evaluation of sensory, motor, refractive, and accommodative functions requires further, in-depth examination. Additional office visits may be required to complete the examination process, especially with younger children.

The evaluation of a patient with strabismus may include, but is not limited to, the following components. Professional judgment and individual patient symptoms and findings may have significant impact on the nature, extent, and course of the services provided. Some components of care may be delegated.

1. Patient History

In addition to gathering information about the patient's general and eye health history, the clinician should also determine:

- Probable time of onset of strabismus
- Nature of the onset (sudden or gradual)
- Frequency of deviation (constant or intermittent)
- Change in size or frequency of the deviation
- Which eye is strabismic
- Presence or absence of diplopia and other visual symptoms or signs
- History of neurologic, systemic, or developmental disorders
- Family history of strabismus
- Previous treatment, if any, and the type and results of such treatment.

* Refer to the Optometric Clinical Practice Guidelines on Comprehensive Adult Eye and Vision Examination and Pediatric Eye and Vision Examination.

2. Ocular Examination

a. Visual Acuity

Measurement of the visual acuity of each eye with best refractive correction helps to establish the presence or absence of amblyopia. In very young children (up to the age of 2 years), who do not respond reliably to visual acuity testing and in individuals who are nonverbal or otherwise not testable, a definite fixation preference with strabismus suggests the presence of amblyopia.⁷⁰ Any child with constant unilateral strabismus should be considered amblyopic until proven otherwise. An alternating fixation pattern, intermittent strabismus, or incomitant strabismus with a compensatory head posture usually precludes the development of amblyopia in the young child. When amblyopia exists, it should usually be addressed before any effort is made to establish normal binocular vision.*

Quantification of visual acuity for children 2 years old or younger can sometimes be accomplished by using preferential looking tests such as the Teller acuity cards.⁷¹ For children ages 3-5, visual acuity tests such as the Broken Wheel test, which uses Landolt C characters, can quantify visual acuity.⁷² With older individuals psychometric acuity cards can be used or standard Snellen visual acuity measurements can be taken.⁷³ Presenting isolated Snellen acuity targets may result in underestimation of the degree of amblyopia.⁷⁴

b. Ocular Motor Deviation

The diagnosis of strabismus, including the direction and frequency of the deviation, may be established by performing a unilateral cover test at distance and near while the patient fixates a target that controls for accommodation. The alternate cover test with prisms is useful in determining the magnitude of the ocular deviation. After initial measurement of the strabismus in the primary positions of gaze, measurements should be made in all other fields of gaze. When evaluating very young children and nonverbal or otherwise not testable

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

patients who cannot fixate on a target long enough for valid cover testing, the optometrist can estimate the degree of strabismus using the corneal reflex test with prisms (Krimsky test) or without prisms (Hirschberg test). The method of measurement and the presence or absence of refractive correction during the measurement should be documented.

c. Monocular Fixation

The method of choice for evaluating monocular fixation is visuoscopy using an ophthalmoscope with a calibrated fixation target.⁷⁵ The practitioner should determine whether eccentric fixation is present and, if so, assess its characteristics: location, magnitude, and steadiness. When there is no foveal reflex, entoptic testing, such as Haidinger's brushes or Maxwell's spot, can be useful in the assessment of monocular fixation.

d. Extraocular Muscle Function

To determine a reasonable prognosis and management approach, it is important to establish whether the ocular deviation is comitant or incomitant. Direct observation of any abnormal head position can aid the evaluation of comitancy. In addition, version and duction testing can provide objective determination of ocular muscle imbalances.

The evaluation of both versions and ductions should be performed without spectacle correction. Common extraocular muscle disorders include limited abduction, limited adduction, and overaction or underaction of the oblique muscles. The presence of an extraocular muscle palsy or mechanical restriction should be noted and may require additional clinical testing, such as the Maddox rod test, Parks three-step test, and the forced duction test for differential diagnosis.

e. Sensorimotor Fusion

The ability to determine the presence of fusion potential by sensory testing may be limited by the patient's age and cognitive ability. Tests such as the Worth dot test at distance and near and stereo tests such as the Random Dot E test, which uses a forced-choice paradigm, may be used. Among other commonly used measures of stereopsis are the Randot and

Titmus stereo tests. More detailed sensory testing (e.g., the Bagolini striated glass, Hering-Bielschowsky afterimage, and synoptophore tests) can be used to evaluate anomalous correspondence in older children and adults. In addition, performing sensory testing while the patient wears prisms to compensate for the amount of strabismus can help determine sensory fusion potential. Once normal sensory fusion has been established, motor fusion can be quantified using a prism bar or rotary or flipper prisms for the patient with intermittent strabismus or a stereoscope for the patient with constant strabismus.

f. Accommodation

When it is feasible, an evaluation of accommodative function should include tests of monocular accommodative amplitude (push-up or minus lens method), accommodative facility (plus/minus flipper method), and accommodative response (dynamic retinoscopy).

g. Refraction

An accurate objective measurement of refractive error is essential because it is often an important etiologic factor in the development of strabismus. The patient's refractive condition generally should be evaluated under both noncycloplegic and cycloplegic conditions.* The instillation of 1 drop of 1% cyclopentolate hydrochloride twice at 5-minute intervals followed by retinoscopy 30-40 minutes later is usually adequate.^{76,77} In cycloplegic retinoscopy, it is best to occlude the eye not being refracted and have the patient view the retinoscope to avoid being off axis. For examining young children, hand-held lenses or lens bars may be preferable. Repeated refractions may be required during the course of treating strabismus.

h. Ocular Health Assessment and Systemic Health Screening

Ocular health should be evaluated to rule out coexisting or

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

causal congenital anomalies or disease associated with strabismus. Pharmacologic dilation of the pupil is generally required for thorough evaluation of the ocular media and the posterior segment.

B. Management of Strabismus

The extent to which an optometrist can provide treatment for strabismus may vary depending on the state's scope of practice laws and regulations and the individual optometrist's certification. Management of the patient with strabismus may require consultation with or referral to an ophthalmologist for those services outside the optometrist's scope of practice.

The management of the strabismic patient is based on the interpretation and analysis of the examination results and overall evaluation. The goals of treatment may include (1) obtaining normal visual acuity in each eye, (2) obtaining and/or improving fusion, (3) eliminating any associated sensory adaptations, and (4) obtaining a favorable functional appearance of the alignment of the eyes. The significance of normal ocular alignment for the development of a positive self-image and interpersonal eye contact cannot be overemphasized.⁷⁸

1. Basis for Treatment

The indications for and specific types of treatment need to be individualized for each patient. In determining a course of therapy, the optometrist should consider the following:

- Age of the patient at the onset of strabismus
- Current age of the patient
- Overall health status of the patient
- Patient's developmental level and anticipated compliance with therapy
- Concerns of the patient and/or parents
- Symptoms and signs of visual discomfort
- Visual demands of the patient
- Comitancy of the deviation
- Size and frequency of the strabismus
- Presence or absence of fusion
- Presence or absence of amblyopia.

2. Available Treatment Options

The treatment of strabismus may include any or all of the following procedures.

a. Optical Correction

Regardless of the cause of the strabismus, the goal for strabismic patients, especially very young patients, is to allow binocularity to develop. The best optical correction that allows a clear retinal image to be formed in each eye is generally the starting point for all treatment. However, overcorrection or undercorrection of the refractive error may be prescribed to affect the angle of strabismus.

Hyperopia may be either partly or totally causative in as many as 50 percent of all cases of esotropia.¹⁶ Generally, when clinically significant amounts of hyperopia are present, the total amount of lens power needed to achieve ocular alignment is prescribed. Anisometropia and astigmatism should also be fully corrected. The full prescription of previously uncorrected refractive errors is usually well accepted by younger children; however, if sensory fusion is difficult or if the patient is unable to adapt to a full prescription, undercorrection of refractive errors may be prescribed initially.

Whereas a full correction of refractive error is often prescribed for esotropia and hyperopia, the presence of exotropia and hyperopia usually requires a more conservative approach. For preteens and teenagers, reduction of the full refractive correction can be attempted if the strabismus is still comfortably controlled. For adults, the refractive correction should be prescribed to the extent tolerated by the patient.

The clinician should continue to re-evaluate the prescribed lenses periodically to assess the effect on the angle of deviation and fusion potential. The patient should be advised that changes in the lenses may be needed during treatment.

b. Added Lens Power

Lenses can also be used to take advantage of the AC/A ratio to help obtain or maintain binocular vision. A bifocal lens pre-

scription may be used for the patient with fusion potential or when full plus acceptance at distance cannot be attained initially. Periodic followup is required to determine the efficacy of this treatment.

Bifocals are often prescribed for the patient with esotropia who has a high AC/A ratio in order to eliminate or decrease the angle of strabismus at near to an amount controllable by compensating divergence. Added convex lens power may also be indicated when the esotropic deviation is larger for near than for distance, or when the ocular alignment at distance with the hyperopic correction permits binocular vision but an esotropia remains at near. In that case, the optometrist may prescribe the minimum added convex lens power to allow fusion at near.

For young children, wide segment bifocals that bisect the pupils are generally prescribed. For older children and teenagers, the optometrist can prescribe standard bifocals or progressive-addition lenses,⁷⁹ or, as fusional vergence ranges increase, fit the patient with contact lenses.⁸⁰

Added minus lens power (e.g., an undercorrection of hyperopia or an overcorrection of myopia) can be prescribed for intermittent exotropia that measures the same for distance and near or is larger for distance.⁸¹ With this correction, the patient uses the added accommodative convergence response to stimulate the fusional vergence system. Fusional vergence sometimes increases to the extent that the added minus lenses are no longer needed. One study found that nearly 70 percent of patients with intermittent exotropia treated with added concave lenses developed improved fusion.⁸² Added minus lenses are contraindicated in patients' whose exotropia is associated with accommodative insufficiency or who are presbyopic.

Treatment with lenses should be discontinued when the frequency and duration of the exotropia remain unchanged despite the wearing of added minus lenses, or when fusion at near becomes disrupted. A large lag in accommodation, as measured by dynamic retinoscopy, may indicate that the

patient is having accommodative difficulties at near with the added minus lenses. In such cases, bifocal lenses can be prescribed. Concern that using added concave lenses might cause large increases in myopia has not been substantiated.⁸³

Patient compliance in wearing prescribed lenses is crucial to the success of any treatment plan. The lens prescription must be acceptable to the patient and worn as directed.

c. Prisms

Ophthalmic prisms can aid in the establishment or maintenance of sensory fusion by moving the image of the target of regard onto or closer to the fovea of each eye. Prisms are generally prescribed for patients with strabismic deviations of less than 20 PD who are capable of fusion.⁸⁴ The presence of amblyopia, deep suppression, and/or anomalous correspondence generally contraindicates the use of prisms. Press-on plastic (Fresnel) prisms to promote binocular vision in early childhood strabismus or to alleviate diplopia in late-onset strabismus are especially helpful in treating larger angles of strabismus.⁷⁶ Disruptive prisms (i.e., overcorrecting or inverse) may be prescribed to eliminate anomalous correspondence.⁸⁵ The effectiveness of such treatment, however, is unknown. In addition, inverse prisms may be used to improve the cosmetic appearance of the strabismic patient who has a poor prognosis for attaining normal binocularity and is not interested in surgery.⁸⁶

For patients who have some fusional vergence ranges, the prescription of prisms less than the strabismic angle may allow them to maintain some active motor fusion.⁷⁶ Relative to the magnitude of the deviation, less prism is generally needed for exotropia than for esotropia.

Prisms may also be used to reduce or eliminate mild compensatory head postures in patients with incomitant strabismus. Older patients who have diplopia in association with acquired extraocular muscle palsy, muscle restriction, or phoria decompensation also may benefit from prisms.

Patients treated with prisms need periodic evaluation to determine treatment efficacy. Some patients adapt, manifesting increased angles of strabismus while wearing the prisms. This change may represent an uncovering of the total deviation, some of which was kept latent by motor fusion.⁷⁶ Removal of these prisms usually allows a return to the original angle of strabismus within a few days.

d. Active Vision Therapy

Optometric vision therapy or orthoptics involves active training procedures to improve the patient's fixation ability and oculomotor control, to eliminate amblyopia, to improve sensory and motor fusion, and to increase facility and the range of accommodation and vergence responses.^{87,88} Used alone or in conjunction with refractive correction, added lens power, prisms, or surgery, these vision therapy procedures are adapted to the individual patient and modified as the patient achieves binocular vision.

Indications for treating strabismus with optometric vision therapy vary, depending on the type of strabismus and the number of visual adaptations. Vision therapy may be successful in the treatment of many forms of strabismus.⁸⁷⁻⁹⁰ The prognosis is most favorable for patients with intermittent strabismus who have sensory-motor fusion at some point in space and those with recently developed strabismus.⁸⁹ Nevertheless, constant or longstanding strabismus also can often be successfully treated with vision therapy.

The optometrist may prescribe active vision therapy or refer the patient to an optometrist who has advanced training or clinical experience with strabismus. The time required for therapy depends upon the type of strabismus, the presence or absence of associated visual adaptations and/or visual anomalies, and patient compliance. Office treatment usually requires 24-75 hours of therapy.⁹¹⁻⁹³ Patients are usually treated for 30-60 minutes once or twice a week in the office. In addition, home treatment may also be prescribed, often requiring 20-60 minutes per day. During office visits, the optometrist reviews home treatment and prescribes appropriate changes.

e. Pharmacological Agents

Pharmacological therapy* may provide a potentially useful treatment option for the management of some patients with strabismus. Anticholinesterase miotics, such as echothiophate iodide (Phospholine Iodide[®]) and diisopropylfluorophosphate (Floropryl[®], DFP), can serve as temporary alternatives to corrective glasses and bifocal lenses for children with accommodative esotropia.⁹⁴ These drugs act by reducing the patient's accommodative effort and decreasing the associated accommodative-convergence mechanism.⁷⁷ The initial dosage is usually 0.125% Phospholine Iodide[®] (1 drop q.d.), tapered downward to a level that maintains the desired result. If used, Floropryl[®] ointment in 0.025% concentration should be instilled at bedtime. A trial period of up to 8 weeks may be needed to determine if a reduction in esotropia is obtained.⁹⁴

Treatment with a pharmacological agent is less effective and less desirable than using corrective glasses and bifocal lenses because of the possibility of both local and systemic adverse effects. Such treatment should be considered for only those patients with accommodative esotropia who cannot wear glasses due to facial deformities, for children who continually remove, lose, or break their spectacles, or for other special cases.

f. Extraocular Muscle Surgery

The clinician should consider all aspects of the nonsurgical treatment of strabismus before recommending surgery. Surgical consultation is appropriate for patients whose strabismus is cosmetically objectionable, as well as for patients who may not display the intellectual, motivational, or physiological characteristics (including fusion potential) that warrant consideration of other treatment.⁸⁸

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

In general, surgery for esotropia may be considered when the manifest deviation exceeds 15-20 PD at both distance and near while the patient is wearing the full refractive correction. For patients with exotropia, deviations exceeding 20-25 PD generally are possible candidates for surgery. Patients with smaller deviations should not be considered for surgery except when adults have acquired symptomatic deviations that do not respond to nonsurgical therapy. Patients with totally accommodative esotropia should not be considered for extraocular muscle surgery because of the risk of inducing consecutive exotropia.⁹⁵

In patients without normal sensory-motor fusion whose best corrected strabismic angle remains too large for binocular comfort and acceptable cosmesis, strabismic surgery may be considered. Preoperative and/or postoperative optometric vision therapy should be considered, when appropriate, to enhance functional vision outcomes by reducing amblyopia and suppression.

The timing and urgency for surgical referral depend upon the type of strabismus, the age of the patient, and the likelihood of improving fusion. In general, children with infantile esotropia requiring surgical correction should undergo surgery prior to 2 years of age. Several studies have shown that peripheral binocular vision with limited stereopsis has a better chance of developing when surgery is performed at that early age.^{96,97} Peripheral binocular vision is best achieved when the postsurgical alignment is within 10 PD of orthophoria. Multiple surgeries are often needed to obtain this result.⁹⁸ Possible complications following surgery include diplopia, undercorrections, overcorrections, chronic inflammation of the conjunctiva, excessive scar tissue, lost muscle(s), perforation of the globe, endophthalmitis, anterior segment ischemia, and corneal dellen.⁹⁹ Surgery is rarely performed at such an early age for other childhood strabismic deviations such as intermittent exotropia.

g. Chemodenervation

The injection of Botulinum Toxin Type A (Oculinum, Botox®)

has been used as an alternative to conventional incisional surgery in selected strabismic patients.¹⁰⁰ The toxin selectively binds to nerve terminals and interferes with the release of acetylcholine, thereby functionally denervating muscles injected with small amounts of the drug. The dose-related but temporary paralysis of an extraocular muscle leads to a change in eye position, followed by some degree of contracture of the opposing muscle. This change has been reported to result in long-lasting and permanent alteration in ocular alignment. Although one injection is often sufficient to produce positive results, one-third to one-half of patients may require additional injections. Transient ptosis and vertical strabismus may develop after chemodenervation. This technique has been most successful when used in patients who have acute abducens nerve palsy and in adults with small-angle deviations. It is rarely used in children.¹⁰¹

3. Patient Education

The prognosis, advantages, and disadvantages of the various modes of treatment should be discussed with the patient and/or the patient's parents and a plan developed based on this dialogue. Patients who suddenly develop strabismus of undetermined etiology should be informed that such an event may be related to a systemic or neurologic disease that would necessitate referral for consultation with or treatment by another health care provider.

It is important for parents of strabismic children to learn about the condition and the child's risks of developing amblyopia and impaired binocular depth perception. Treatment plans formulated in consultation with the patient and parents should be responsive to their preferences. The optometrist should elicit the child's and/or the parents' expectations for outcomes, advise the persons involved, relate the findings, prepare treatment plans, discuss options, and recommend strategies for successful treatment. Parents and children must understand that timely examination and management are critical to reducing the risk for loss of vision and fusion and the development of other symptoms associated with strabismus.

4. Management Strategies for Strabismus

a. Accommodative Esotropia

After the diagnosis of accommodative esotropia has been confirmed, correction of the amount of hyperopia needed to obtain ocular alignment should be provided. If present, amblyopia should be treated. The clinician may prescribe active vision therapy procedures for the development and enhancement of normal sensory and motor fusion.

The treatment of accommodative esotropia may have the following possible sequelae:

- Alignment at distance with corrective lenses, but persistence of esotropia at near. The remaining esotropia at near is usually treated with bifocal lenses.⁷⁶
- Esotropia persisting both at distance and near while the patient wears corrective lenses. Cycloplegic refraction should be repeated to determine whether additional hyperopia should be corrected.
- Esotropia persisting both at distance and at near and cycloplegic refraction revealing no additional hyperopia, showing the presence of a residual nonaccommodative esotropia. Other treatment options (e.g., prisms, vision therapy, surgery) should be considered. Nonaccommodative esotropia exceeding 15-20 PD, that does not respond to prisms and/or vision therapy, may require extraocular muscle surgery. Surgery is performed with the intent of reducing or eliminating the nonaccommodative component, not the accommodative component, of the esotropia. The patient will continue to need to wear corrective lenses.¹⁰²

Some patients who at first achieve ocular alignment with corrective lenses may subsequently develop additional esotropia and risk losing binocular vision.^{103,104} This is more likely to occur in the following circumstances:

- Onset of accommodative esotropia during the first year of life
- Delay between the onset of the esotropia and the initiation of treatment
- Large increases in hyperopia

- Incomplete treatment (undercorrection of hyperopia or part-time wearing of corrective lenses)
- Elevated AC/A ratio.

Children with treated accommodative esotropia should be evaluated at intervals, according to the schedule in Appendix Figure 2. New or different findings may alter the frequency of followup care needed.

b. Acute Esotropia and Exotropia

Once the cause of the esotropia or exotropia has been determined, prisms may be used to correct deviations less than or equal to 20 PD (except in late-onset accommodative esotropia) to eliminate diplopia and re-establish binocular vision. For larger and transient deviations, the optometrist can prescribe Fresnel prisms.

Optometric vision therapy may be prescribed to expand fusional vergence amplitudes and facility. Surgical consultation may be considered for deviations that have become stable over time, when the angle of deviation exceeds 15-20 PD, and when the strabismus is cosmetically displeasing.

Acute esotropia associated mostly with abducens nerve palsy, to a lesser extent divergence paralysis/divergence insufficiency, and acute acquired comitant esotropia may change over time. Complete remission is more likely when the cause is associated with vascular disease (e.g., diabetes mellitus) and less likely when related to trauma.⁴² Patients with acute esotropia or exotropia should be followed as indicated in Appendix Figure 2 and therapy should be altered when necessary.

c. Consecutive Esotropia and Exotropia

Consecutive esotropia following surgery should be treated aggressively in young children with lenses, prisms, and optometric vision therapy to prevent possible amblyopia and loss of binocular vision. Older patients with consecutive esotropia following surgery frequently have diplopia and usually require similar treatment.

Consecutive exotropia that is spontaneous and optically induced can be treated by reducing the power of the hyperopic lenses. This is generally done in younger patients when the exotropia exceeds 20-25 PD. For older patients, reduction in the hyperopic correction may result in accommodative asthenopia, and alternative treatments may be needed.

d. Infantile Esotropia and Exotropia

Once the diagnosis of infantile esotropia has been confirmed, the clinician should make an effort to determine whether a superimposed accommodative component exists by evaluating the effect of correcting the hyperopic refractive error on the angle of deviation.¹⁰⁵ In most cases, a large esotropia persists, despite corrective lenses, and repeated cycloplegic refractions show little change in the amount of hyperopia.

Amblyopia, if present, should be treated.* In young patients who show fixation preference, occlusion therapy may be used until an alternating fixation pattern is established.⁸⁸ Children under 2 years of age who undergo occlusion therapy should be monitored biweekly; older children, at least monthly. The acquisition of alternating fixation should prevent amblyopia regression. Maintenance amblyopia therapy may be required for children who return to their original fixation pattern.

When a large, nonaccommodative esotropia exists, surgical ocular alignment should be considered. Most ophthalmic surgeons prefer to intervene before 24 months of age, in the hope of establishing binocular vision. However, the binocular vision achieved in these cases is usually not bifoveal, but peripheral, implying a fusion response detectable by the Worth dot test at near but not distance. Rudimentary stereopsis, when possible, is best accomplished if the postsurgical strabismic deviation is no greater than 10 PD, even in older patients.¹⁰⁶

Multiple surgical procedures are frequently needed in such cases.⁹⁸ The most accepted approach to timing the surgery for

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

infantile esotropia is to perform it only after the deviation can be accurately determined and after adequate attention has been directed to correcting any accommodative component and treating amblyopia. The development of binocular vision for infantile esotropia appears more related to the stability of the postsurgical alignment than to the age of the patient treated.¹⁰⁶

There are several possible sequelae to alignment for infantile esotropia:¹⁰⁷

- Recurring strabismus
- Accommodative esotropia
- Recurring amblyopia
- Increasing dissociated vertical deviation and inferior oblique overaction.

Due to the instability of visual acuity, ocular alignment, refractive error, and extraocular muscle function, young children who have been treated for infantile esotropia should be evaluated according to the schedule in Appendix Figure 2. New or different findings may alter the frequency of followup care.

The treatment of infantile exotropia is similar to those for infantile esotropia. Treatment often includes surgery because of the large angle of constant exotropia. Prisms are usually not helpful in cases with poor fusion potential. Surgery should be considered only after (1) the refractive error and fundus have been assessed; (2) amblyopia has been shown not to exist; (3) the angle of exotropia is stable on subsequent examinations; and (4) other treatment options are not appropriate or have failed. The likelihood of success in establishing peripheral fusion is less with infantile exotropia than with infantile esotropia.¹⁰⁸

e. Intermittent Exotropia

Individual cases of intermittent exotropia are treated in different ways and often by a combination of treatments.^{109,110} Therapy for intermittent exotropia should include correction of

significant refractive error. Usually, the full amount of myopia, anisometropia, and astigmatism should be corrected. Hyperopia may be undercorrected for younger patients.

Added minus lens power may be used temporarily to help facilitate fusion in children with divergence excess or basic intermittent exotropia. The minimum minus lens power (generally 1-3 D) that will allow the patient to maintain alignment easily, as determined with the unilateral cover test, may be prescribed. An overall success rate of 28 percent has been estimated for cases of intermittent exotropia with this treatment modality, which is often used in conjunction with active vision therapy.¹¹⁰

Compensatory base-in prisms can be used to facilitate fusion. The degree of fusion will vary, but generally enough prism should be prescribed to place the patient's deviation in balance with his or her fusional vergence amplitudes, so that the patient is able to maintain alignment without excessive effort. The overall success rate for prism therapy with intermittent exotropia has been estimated as 28 percent.¹¹⁰ Prism therapy is also often used in conjunction with active vision therapy.

Numerous vision therapy procedures, including but not limited to expanding fusional vergence amplitudes and vergence facility, diplopia awareness, biofeedback, and increasing accommodation, are frequently prescribed. Although success has been reported with home-oriented therapy,⁸⁸ more intensive office-based treatment may be required. The overall success rate of orthoptics and vision therapy with intermittent exotropia has been estimated as 59 percent.¹¹⁰

Surgical intervention should only be considered when, after a reasonable time, other treatment modalities have not been successful and the deviation persists or increases. Surgery is rarely performed in a child under 4 years of age (except in cases of a very large and frequent deviation) because of the increased risk for persistent consecutive esotropia with amblyopia and loss of stereopsis.^{53,54} The overall success rate of surgical therapy with intermittent exotropia has been estimated as 46 percent.¹¹⁰

The course of intermittent exotropia, when untreated or when treatment recommendations have not been followed, is unclear. Lack of compliance would likely result in the continuation of signs and symptoms. However, studies that have followed untreated patients over the years indicate that approximately 75 percent get worse, whereas 25 percent stay the same or get better.^{111,112} Getting worse implies that the frequency and magnitude of exotropia increase. Getting better is defined as the patient's becoming completely exophoric, with re-establishment of fusion.

For patients who have been treated (especially those treated surgically), the possibility of recurring intermittent exotropia exists. Patients with a history of intermittent exotropia should receive followup evaluations according to the schedule suggested in Appendix Figure 2.

f. Mechanical Esotropia and Exotropia

The patient with mechanical esotropia or exotropia may need no specific therapy if there is either minimal or no strabismus in the primary position of gaze and the patient does not experience diplopia. For example, treatment for Duane's retraction syndrome is generally restricted to cases in which there is an objectionable compensatory head turn, a large angle strabismus in the primary position, or extreme elevation or depression of the eye in adduction.^{64,65} Because head turning is prevalent, amblyopia is uncommon and high-level stereopsis usually exists. Prisms may be prescribed for slight head turns. For large head turns, surgery may be used but it does not improve the deficient abduction or adduction.

g. Microtropia

Microtropia is a fully adapted strabismus that rarely gives rise to symptoms unless other conditions become superimposed. Its treatment consists mostly of correcting significant refractive errors and any coexisting amblyopia. The use of vision therapy and prisms to establish bifoveal fusion has been successful in selected cases of microtropia.¹¹³

h. Sensory Esotropia and Exotropia

Infants diagnosed at birth with sensory esotropia or exotropia due to unilateral congenital cataracts should be treated within the first 2 months of life with cataract surgery, optical correction with contact lens, and occlusion therapy for amblyopia.¹¹⁴ Neutralizing prisms may be prescribed and, depending upon the size of the deviation, subsequent strabismus surgery may be performed. The attainment of normal binocular vision is generally not a realistic goal.

In older children and adults with acquired sensory exotropia due to dense traumatic unilateral cataracts, fusion may be lost if the cataract remains in situ for more than 2 years, despite subsequent cataract extraction, prisms, vision therapy, and strabismus surgery.¹¹⁵ Therefore, treatment should not be delayed.

5. Prognosis and Followup

The purpose of the followup evaluation is to assess the patient's response to therapy and to alter or adjust treatment as needed (See Appendix Figure 2). The schedule of followup visits depends upon the patient's condition and associated circumstances. Followup evaluation includes monitoring of several aspects of the patient's condition:

- Patient history
- Visual acuity
- Characteristics of strabismus at distance and near
- Fusion status
- Extraocular muscle function
- Refractive error
- Tolerance, efficacy, and side effects of therapy.

CONCLUSION

The optometrist should emphasize the diagnosis, timely and appropriate management, and careful followup of patients with strabismus. Proper care can result in reduction of personal suffering for those involved as well as a substantial cost savings for the involved individuals and their families.

III. REFERENCES

1. National Society to Prevent Blindness. Crossed eyes: a needless handicap. New York: NSPB. March 1981.
2. Tomilla V, Tarkkanen A. Incidence of loss of vision in the healthy eye in amblyopia. *Br J Ophthalmol* 1981; 65:575-7.
3. Jones RK, Lee DN. Why two eyes are better than one. *J Exp Psychol Hum Percept Perform* 1981; 7:30-40.
4. Sheedy JE, Bailey IL, Buri M, Bass E. Binocular vs monocular task performance. *Am J Optom Physiol Opt* 1986; 63:839-46.
5. Prosser PC. Infantile development: strabismic and normal children compared. *Ophthalm Opt* 1979; 19:681-3.
6. Tonge BJ, Lipton GL, Crawford G. Psychological and educational correlates of strabismus in school children. *Aust N Z J Psychiatry* 1984; 18:71-7.
7. Dobson V, Sebris SL. Longitudinal study of acuity and stereopsis in infants with or at risk for esotropia. *Invest Ophthalmol Vis Sci* 1989; 30:1146-58.
8. Duckman RH, Meyer B. Use of photoretinoscopy as a screening technique in the assessment of anisometropia and significant refractive error in infants/toddlers/children and special population. *Am J Optom Physiol Opt* 1987; 64:604-10.
9. von Noorden GK. Binocular vision and ocular motility, 4th ed. St. Louis: CV Mosby, 1990:129-33.
10. Sondhi N, Archer SM, Helveston EM. Development of normal ocular alignment. *J Pediatr Ophthalmol Strabismus* 1988; 25:210-1.
11. Sidikaro Y, von Noorden GK. Observations in sensory heterotropia. *J Pediatr Ophthalmol Strabismus* 1982; 19:12-9.
12. Wick B, Cotter SA, Scharre J, et al. Characteristics and prevalence of exotropia in clinic populations. *Optom Vis Sci* 1990; 67(10 suppl):81.
13. Ciner EB, Herzberg C. Optometric management of optically induced consecutive exotropia. *J Am Optom Assoc* 1992; 63:266-71.
14. Roberts J, Rowland M. Refractive status and motility defects of persons 4-74 years, United States 1971-1972. Vital and health statistics: series 11, DHEW publication no. (PHS) 78-1654. Hyattsville, MD: National Center for Health Statistics, 1978.
15. Nordlow W. Squint—the frequency of onset of different ages and the incidence of some defects in a Swedish population. *Acta Ophthalmologica* 1964; 42:1015-37.
16. Stidwell D. Orthoptic assessment and management. Oxford: Blackwell Scientific Publication, 1990:7.
17. Nordlow W. Age distribution of onset of esotropia. *Br J Ophthalmol* 1953; 37:593-600.
18. Cooper J. Intermittent exotropia of the divergence excess type. *J Am Optom Assoc* 1977; 48:1261-73.
19. Ing MR, Pang SWL. The racial distribution of strabismus. In: Reinecke RD, ed. *Strabismus*. New York: Grune & Stratton, 1978:107-9.
20. Krzystkowz K, Pajakowa J. The sensorial state in divergent strabismus. In: Mein J, Bierlaagh JJM, Brummel Kamp-Dons TEA, eds. *Proc II Int Orthoptic Congress*, Amsterdam: Excerpta Medica Foundation, 1972:72-6.

21. Harcourt B. Strabismus affecting children with multiple handicaps. *Br J Ophthalmol* 1974; 58:272-80.
22. Bankes JLK. Eye defects of mentally handicapped children. *Br Med J* 1974; 2:533-5.
23. Falls HF. Ocular changes in mongolism. *Ann N Y Acad Sci* 1970; 171:627-36.
24. Lossef S. Ocular findings in cerebral palsy. *Am J Ophthalmol* 1962; 54:1114-8.
25. Seaber JH, Chandler AC. A five-year study of patients with cerebral palsy and strabismus. In: Moore S, Mein J, and Stockbridge L, eds. *Orthoptics: past, present, and future*. New York: Stratton Intercontinental Medical Book Corp, 1976:271-7.
26. Carruthers JDA. Strabismus in craniofacial dysostosis. *Graefes Arch Clin Exp Ophthalmol* 1988; 226:230-4.
27. Caputo AR, Lingua RW. Aberrant muscle insertions in Crouzon's disease. *J Pediatr Ophthalmol Strabismus* 1980; 17:239-41.
28. Miller M, Folk E. Strabismus associated with craniofacial anomalies. *Am Orthopt J* 1975; 25:27-36.
29. Spir M, Gilad E, Ben-Sira I. An unusual extraocular muscle anomaly in a patient with Crouzon's disease. *Br J Ophthalmol* 1982; 66:253-7.
30. Maumenee IH, Alston A, Mets MB, et al. Inheritance of congenital esotropia. *Trans Am Ophthalmol Soc* 1986; 84:85-93.
31. Griffin JR, Asano GW, Somers RJ, Anderson CE. Heredity in congenital esotropia. *J Am Optom Assoc* 1979; 50:1237-65.
32. Schlossman A, Priestly BJ. Role of heredity in etiology and treatment of strabismus. *Arch Ophthalmol* 1952; 47:1-20.
33. Adelstein AM, Scully J. Epidemiological aspects of squint. *Br Med J* 1967; 3:334-38.
34. Parks MM. Management of acquired esotropia. *Br J Ophthalmol* 1974; 58:240-7.
35. Baker JD, Parks MM. Early onset accommodative esotropia. *Am J Ophthalmol* 1980; 90:11-8.
36. Adler FH. Pathologic physiology of convergent strabismus. Motor aspects of the non-accommodational type. *Arch Ophthalmol* 1945; 33:362-77.
37. Parks MM. Abnormal accommodative convergence squint. *Arch Ophthalmol* 1958; 59:364-80.
38. Wilson ME, Parks MM. Primary inferior oblique overaction in congenital esotropia, accommodative esotropia, and intermittent exotropia. *Ophthalmology* 1989; 96:950-5.
39. Wick B. Accommodative esotropia: efficacy of therapy. *J Am Optom Assoc* 1987; 58:562-6.
40. Swan KC. Esotropia following occlusion. *Arch Ophthalmol* 1947; 37:444-51.
41. Rutstein RP. Acute comitant esotropia simulating late onset accommodative esotropia. *J Am Optom Assoc* 1988; 59:446-9.
42. Richards BW, Jones FR, Younge BR. Causes and prognosis in 4,278 cases of paralysis of the oculomotor, trochlear, and abducens cranial nerves. *Am J Ophthalmol* 1992; 113:489-96.

43. Scheiman M, Gallaway M, Ciner E. Divergence insufficiency: characteristics, diagnosis and treatment. *Am J Optom Physiol Opt* 1986; 63:425-31.
44. Curran RE. True and simulated divergence paresis as a precursor of benign sixth nerve palsy. *Binoc Vis Eye Muscle Surg* 1989; 4:125-30.
45. Kirkham TH, Bird AC, Sanders MD. Divergence paralysis with raised intracranial pressure. An electro-oculographic study. *Br J Ophthalmol* 1972; 56:776-82.
46. Williams AS, Hoyt CS. Acute comitant esotropia in children with brain tumors. *Arch Ophthalmol* 1989; 107:376-8.
47. Goldman HD, Nelson LB. Acute acquired comitant esotropia. *Ann Ophthalmol* 1985; 17:777-8.
48. Clark AC, Nelson LB, Simon JW, et al. Acute acquired comitant esotropia. *Br J Ophthalmol* 1989; 73:376-8.
49. Raab EL. Consecutive accommodative esotropia. *J Pediatr Ophthalmol Strabismus* 1985; 22:58-67.
50. Folk RF, Miller MR, Chapman L. Consecutive exotropia following surgery. *Br J Ophthalmol* 1983; 67:546-8.
51. Bradbury JA, Doran RML. Secondary exotropia. A retrospective analysis of matched cases. *J Pediatr Ophthalmol Strabismus* 1993; 30:163-6.
52. Keech RV, Stewart SA. The surgical overcorrection of intermittent exotropia. *J Pediatr Ophthalmol Strabismus* 1990; 27:218-20.
53. Pratt-Johnson JH, Barlow JM, Tillson G. Early surgery in intermittent exotropia. *Arch Ophthalmol* 1977; 84:684-94.
54. Edelman PM, Broan MH, Murphree LH, Wright KW. Consecutive esodeviation: the what? *Am Orthopt J* 1988; 38:111-6.
55. Eskridge JB. Persistent diplopia associated with strabismus surgery. *Optom Vis Sci* 1993; 70:849-53.
56. Archer SM, Sondhi N, Helveston EM. Strabismus in infancy. *Ophthalmology* 1989; 96:133-7.
57. Nixon RB, Helveston EM, Miller K, et al. Incidence of strabismus in neonates. *Am J Ophthalmol* 1985; 100:798-801.
58. Christenson GN, Rouse MW, Adkins DA. Management of infantile-onset esotropia. *J Am Optom Assoc* 1990; 61:559-72.
59. Costenbader FD. Infantile esotropia. *Trans Am Ophthalmol Soc* 1961; 59:397-429.
60. von Noorden GK. A reassessment of infantile esotropia. *Am J Ophthalmol* 1988; 105:1-10.
61. Rubin SE, Nelson LB, Wagner RS, et al. Infantile exotropia in healthy children. *Ophthalmic Surg* 1988; 19:792-4.
62. Moore S, Cohen RL. Congenital exotropia. *Am Orthopt J* 1985; 35:68-70.
63. Rutstein RP, Daum KM. Exotropia associated with defective accommodation. *J Am Optom Assoc* 1987; 58:548-54.
64. Rutstein RP. Duane's retraction syndrome. *J Am Optom Assoc* 1992; 63:419-29.

65. Mende A. Duane's retraction syndrome and relief of secondary torticollis and near point asthenopia with prism. *J Am Optom Assoc* 1990; 61:556-8.
66. Rutstein RP, Eskridge JB. Stereopsis in small-angle strabismus. *Am J Optom Physiol Opt* 1984; 61:491-8.
67. Helveston EM, von Noorden GK. Microtropia. A newly defined entity. *Arch Ophthalmol* 1968; 78:272-81.
68. Kushner BJ. Functional amblyopia associated with organic ocular disease. *Am J Ophthalmol* 1981; 91:39-45.
69. Drummond GT, Scott WE, Keech RV. Management of monocular congenital cataracts. *Arch Ophthalmol* 1989; 107:45-51.
70. Zipf RF. Binocular fixation pattern. *Arch Ophthalmol* 1976; 94:401-4.
71. Stager DR, Birch EE. Preferential looking acuity and stereopsis in infantile esotropia. *J Pediatr Ophthalmol Strabismus* 1986; 23:160-5.
72. Richman JE, Petito GT, Cron MT. Broken wheel acuity test. A new and valid test for preschool and exceptional children. *J Am Optom Assoc* 1984; 55:561-71.
73. Davidson DW, Eskridge JB. Reliability of visual acuity measures of amblyopic eyes. *Am J Optom Physiol Opt* 1977; 54:756-66.
74. Burian HM, Cortimiglia RM. Visual acuity and fixation pattern in patients with strabismic amblyopia. *Am Orthopt J* 1962; 12:169-74.
75. Griffin JR. Binocular anomalies: procedures for vision therapy, 2nd ed. Chicago: Professional Press, 1982:86-8.
76. London R. Passive treatments for early onset strabismus. *Probl Optom* 1990; 2(3):480-95.
77. Amos DM. Pharmacologic management of strabismus. In: Bartlett JD, Jaanus SD, eds. *Clinical ocular pharmacology*, 2nd ed. Boston: Butterworths, 1989:725-32.
78. Groffman S. Psychological aspects of strabismus and amblyopia/a review of the literature. *J Am Optom Assoc* 1978; 49:995-9.
79. Smith JB. Progressive-addition lenses in the treatment of accommodative esotropia. *Am J Ophthalmol* 1985; 99:56-62.
80. Calcutt C. Contact lenses in accommodative esotropia therapy. *Br Orthopt J* 1989; 46:59-65.
81. Kennedy JR. The correction of divergent strabismus with concave lenses. *Am J Optom* 1954; 31:605-14.
82. Caltrider N, Jampolsky A. Overcorrecting minus lens therapy for treatment of intermittent exotropia. *Ophthalmology* 1983; 90:1160-5.
83. Rutstein RP, Marsh-Tootle W, London R. Changes in refractive error for exotropes treated with overminus lenses. *Optom Vis Sci* 1989; 66:487-91.
84. von Noorden GK, Helveston EM. *Strabismus. A decision making approach*. St. Louis: CV Mosby, 1994:178-9.
85. Rouse MW. Overcorrecting prism therapy for anomalous correspondence. In: Cotter S, London R, eds. *Clinical uses of prism: a spectrum of applications*. St. Louis: Mosby-Year Book, 1995:237-56.
86. Caloroso EE, Cotter S. Prescribing prisms for strabismus. In: Cotter S, London R, eds. *Clinical uses of prism: a spectrum of applications*. St. Louis: Mosby-Year Book, 1995:220-1.

87. Griffin JR. Binocular anomalies. Procedures for vision therapy, 2nd ed. Chicago: Professional Press, 1982:189-93.
88. Rosner J, Rosner J. Vision therapy in a primary care practice. New York: Professional Press Books Fairchild Publications, 1988:31.
89. Hellerstein LF, Dowis RT, Maples WC. Optometric management of strabismus patients. J Am Optom Assoc 1994; 65:621-5.
90. Cohen AH, Lowe SE, Steele GT, et al. The efficacy of optometric vision therapy. J Am Optom Assoc 1988; 59:95-105.
91. Ludlam WH. Orthoptic treatment of strabismus. Am J Optom 1961; 38:369-88.
92. Goldrich SG. Optometric therapy of divergence strabismus. Am J Optom 1980; 57:7-14.
93. Etting GL. Strabismus therapy in private practice. Cure rates after three months of therapy. J Am Optom Assoc 1978; 49:1367-73.
94. Hiatt RL, Ringer C, Cope-Troupe C. Miotics vs glasses in esodeviation. J Pediatr Ophthalmol Strabismus 1979; 16:213-7.
95. Jampolsky A, von Noorden GK, Spiritus M. Unnecessary surgery in fully refractive accommodative esotropia. Int Ophthalmol 1992; 16(2):129-31.
96. Ing MR. Early surgical alignment for congenital esotropia. J Pediatr Ophthalmol Strabismus 1983; 20:11-8.
97. Zak TA, Morin D. Early surgery for infantile esotropia: results and influence of age upon results. Can J Ophthalmol 1982:213-8.
98. Scheiman M, Ciner E, Gallaway M. Surgical success rates in infantile esotropia. J Am Optom Assoc 1989; 60:22-31.
99. Caloroso EE, Rouse MW. Clinical management of strabismus. Boston: Butterworth-Heinemann, 1993:148-56.
100. Scott AB. Botulinum toxin injection of eye muscles to correct strabismus. Trans Am Ophthalmol Soc 1981; 79:734-70.
101. Ing MR. Botulinum alignment for congenital esotropia. Ophthalmology 1993; 100:318-22.
102. Raab EL. The accommodative portion of mixed esotropia. J Pediatr Ophthalmol Strabismus 1991; 28:73-6.
103. Ludwig IH, Parks MM, Getson PR, Kammerman LA. Rate of deterioration in accommodative esotropia correlated to the AC/A relationship. J Pediatr Ophthalmol Strabismus 1983; 25:8-12.
104. Dickey CF, Scott WE. The deterioration of accommodative esotropia: frequency characteristics, and predictive factors. J Pediatr Ophthalmol Strabismus 1988; 25:172-5.
105. Nelson LB, Wagner RS, Simon JW, Harley RD. Congenital esotropia. Surv Ophthalmol 1987; 31:363-83.
106. Morris RJ, Scott WE, Dickey CF. Fusion after surgical alignment for long-standing strabismus in adults. Ophthalmology 1993; 100:135-8.
107. Scheiman MM, Wick B. Optometric management of infantile esotropia. Probl Optom 1990; 2(3):459-79.
108. Williams F, Beneish R, Polomeno RC, Little JM. Congenital exotropia. Am Orthopt J 1984; 34:92-4.

109. Cooper J, Medlow N. Intermittent exotropia. Basic and divergence excess type. *Binoc Vis* 1993; 8:185-216.
110. Coffey B, Wick B, Cotter S, et al. Treatment options in intermittent exotropia: a critical appraisal. *Optom Vis Sci* 1992; 69:386-404.
111. von Noorden GK. Some aspects of exotropia. Presented before meeting of the Wilmer Resident Assn., Johns Hopkins Hospital 1966, as cited in *Binocular vision and ocular motility: theory and management of strabismus*, 4th ed. St. Louis: CV Mosby, 1990:326.
112. Hiles DA, Davies GT, Costenbader FD. Long-term observations on unoperated intermittent exotropia. *Arch Ophthalmol* 1968; 80:436-42.
113. Wick B. Visual therapy for small angle esotropia. *Am J Optom Physiol Opt* 1974; 51:490-6.
114. Robb RM, Mayer DL, Moore BD. Results of early treatment of unilateral congenital cataracts. *J Pediatr Ophthalmol Strabismus* 1987; 24:178-81.
115. Pratt-Johnson JH, Tillson G. Intractable diplopia after vision restoration in unilateral cataract. *Am J Ophthalmol* 1989; 107:23-6.

IV. APPENDIX

Figure 1
Optometric Management of the Patient With Strabismus: A Brief Flowchart

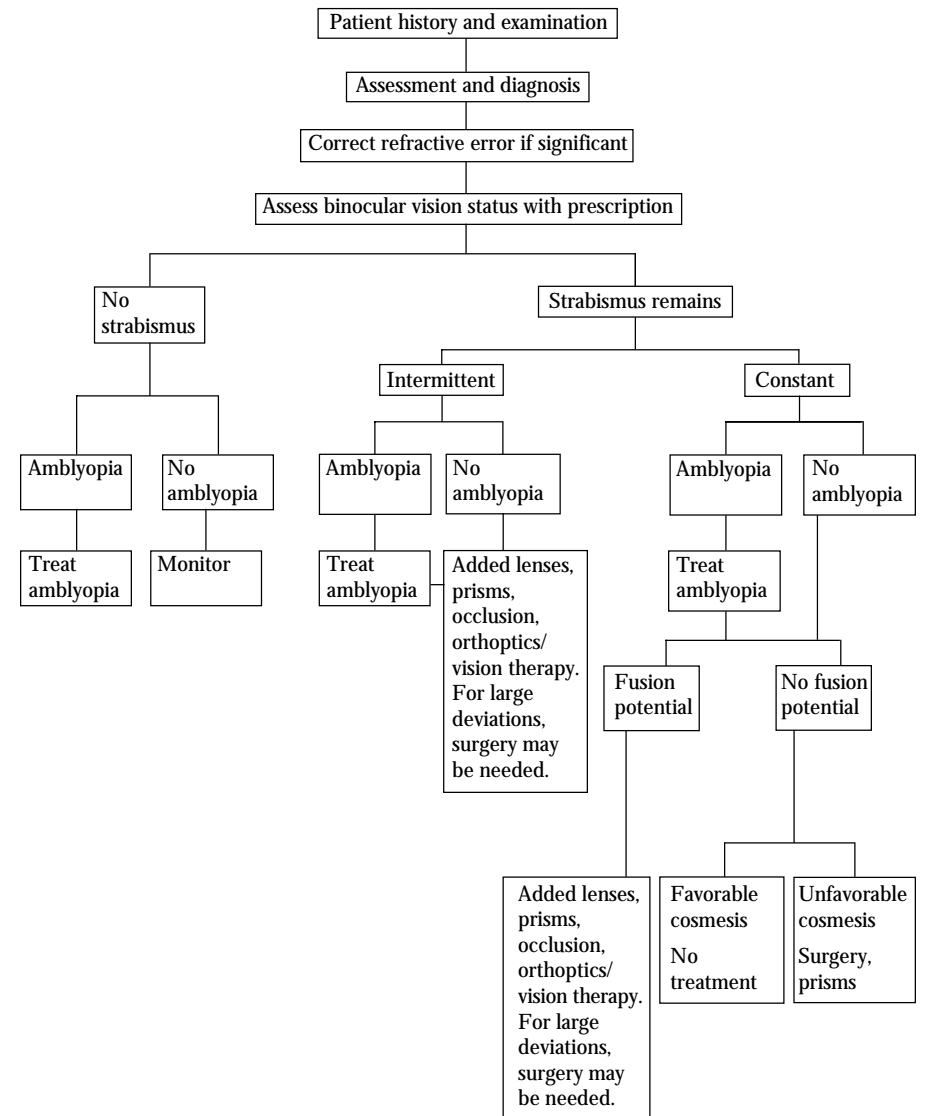


Figure 2
Frequency and Composition of Evaluation and Management Visits for Esotropia and Exotropia*

Type of Patient	Number of Evaluation Visits	Treatment Options
Accommodative esotropia	1-3	Optical correction Vision therapy
Acute esotropia and exotropia	1-3	Prisms Vision therapy Surgery
Consecutive esotropia and exotropia	1-3	Optical correction Prisms Vision therapy Surgery
Infantile or early-acquired esotropia and exotropia	1-3	Optical correction Prisms Vision therapy Surgery
Intermittent exotropia	1-3	Optical correction Prisms Vision therapy Surgery
Mechanical esotropia and exotropia	1-3	Prisms Surgery
Microtropia	1-3	Optical correction Prisms Vision therapy
Sensory esotropia and exotropia	1-3	Optical correction Prisms Vision therapy Surgery

* Figure 2 extends horizontally on page 55.

* Vision therapy would require additional visits.

Figure 2 Continued . . .

Frequency of Followup Visits*	Management Plan
<6 years: every 4-6 mo 6-10 years: every 6-12 mo ≥11 years: every 12 mo	Provide refractive correction; treat any amblyopia; use added plus at near if needed to facilitate fusion; prescribe vision therapy to develop/enhance normal sensory and motor fusion.
Every 3-12 mo	Use prisms to eliminate diplopia and re-establish binocular vision; prescribe vision therapy; in stable deviations over 20-25 PD, consult with ophthalmologist regarding extraocular muscle surgery.
Variable, depending on etiology	Provide refractive correction; prescribe prism and/or vision therapy to prevent amblyopia, eliminate diplopia, and establish normal sensory fusion, if applicable.
<2 years: every 3 mo 2-5 years: every 4-6 mo 6-10 years: every 12 mo ≥11 years: every 12-24 mo	Provide refractive correction; treat any amblyopia; use prism to establish normal sensory fusion, if applicable; consult with ophthalmologist regarding extraocular muscle surgery.
<5 years: every 4-6 mo 5-10 years: every 6-12 mo ≥11 years: every 12-24 mo	Provide refractive correction; use added minus lens power or base-in prism if needed to facilitate fusion; prescribe vision therapy; if deviation persists or increases, consult with ophthalmologist regarding extraocular muscle surgery.
Variable, depending on etiology	No therapy if strabismus is not present in the primary position of gaze and no diplopia. Consider prisms and/or surgery to treat head turn.
Every 3-12 mo	Provide refractive correction; treat any amblyopia; prescribe vision therapy and/or prism to establish bifoveal fusion, if applicable.
Every 3-12 mo	Consult with ophthalmologist regarding treatment of any underlying ophthalmic disease; provide refractive correction; treat any amblyopia; prescribe vision therapy and/or prism, if applicable; if deviation persists or increases, consult with ophthalmologist regarding extraocular muscle surgery.



Figure 3
ICD-9-CM Classification of Esotropia and Exotropia

Strabismus and other disorders of binocular eye movements	378
<i>Excludes: nystagmus and other irregular eye movements (379.50-379.59)</i>	
Esotropia	378.0
Convergent concomitant strabisms	
<i>Excludes: intermittent esotropia (378.20-378.22)</i>	
Exotropia, unspecified	378.00
Monocular esotropia	378.01
Monocular esotropia with A pattern	378.02
Monocular esotropia with V pattern	378.03
Monocular esotropia with other noncomitancies	378.04
Monocular esotropia with X or Y pattern	
Alternating esotropia	378.05
Alternating esotropia with A pattern	378.06
Alternating esotropia with V pattern	378.07
Alternating esotropia with other noncomitancies	378.08
Alternating esotropia with X or Y pattern	
Exotropia	378.1
Divergent concomitant strabismus	
<i>Excludes: intermittent exotropia (378.20, 378.23-378.24)</i>	
Exotropia, unspecified	378.10
Monocular exotropia	378.11
Monocular exotropia with A pattern	378.12
Monocular exotropia with V pattern	378.13
Monocular exotropia with other noncomitancies	378.14
Monocular exotropia with X or Y pattern	

Figure 3
ICD-9-CM Classification of Esotropia and Exotropia
(continued)

Alternating exotropia	378.15
Alternating exotropia with A pattern	378.16
Alternating exotropia with V pattern	378.17
Alternating exotropia with other noncomitancies	378.18
Alternating exotropia with X or Y pattern	
Intermittent heterotropia	378.2
<i>Excludes: vertical heterotropia (intermittent) (378.31)</i>	
Intermittent heterotropia, unspecified	378.20
Intermittent:	
esotropia NOS	
exotropia NOS	
Intermittent esotropia, monocular	378.21
Intermittent esotropia, alternating	378.22
Intermittent exotropia, monocular	378.23
Intermittent exotropia, alternating	378.24
Other and unspecified heterotropia	378.3
Heterotropia, unspecified	378.30
Monofixation syndrome	378.34
Microtropia	
Accommodative component in esotropia	378.35
Mechanical strabismus	378.6
Mechanical strabismus, unspecified	378.60

Abbreviations of Commonly Used Terms

AC/A	Accommodative convergence/accommodation
D	Diopter
PD	Prism diopter
q.d.	Daily

Glossary

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

Alternating strabismus Each eye not only takes up fixation but holds fixation while both eyes are open. In very young children with strabismus, an alternating fixation pattern generally prevents amblyopia.

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

Anomalous correspondence A type of retinal correspondence, occurring frequently in strabismus, in which the foveae of the two eyes do not give rise to a common visual direction; the fovea of one eye has the same functional direction with an extrafoveal area of the other eye.

Comitant A condition in which the magnitude of deviation remains essentially the same in all positions of gaze and with either eye fixating.

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

Ductions Ability of the eyes to show a full range of motion under monocular (one eye) viewing conditions.

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

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

Heterophoria A latent condition of the eyes from the orthophoric position which requires vergence for bifixation to be maintained.

Incomitant A condition in which the magnitude of deviation is not the same in the different positions of gaze or with either eye fixating. Generally, the magnitude must change by at least 5 PD to be incomitant.

Inferior oblique overaction Elevation or upturning of the eye when it adducts.

Motor fusion The ability to align the eyes in such a manner that sensory fusion can be maintained.

Nystagmus Rhythmic oscillations or tremors of the eyes which occur independent of the normal eye movements.

Primary position The position of the eyes when looking straight ahead, with the head erect and still.

Prism diopter (PD) The unit of measurement for strabismus. One PD is the angle defined by a deviation of 1 centimeter at a distance of 1 meter.

Pseudoesotropia The false appearance of having esotropia when actually no convergent misalignment of the visual axes exists. Common with infants and very young children who have flat and broad nasal bridges with prominent epicanthal folds.

Ptosis Drooping of the upper eyelid below its normal position.

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

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

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

Unilateral strabismus A condition where only the nonstrabismic eye can maintain fixation while both eyes are open. In young children, a constant unilateral strabismus causes amblyopia.

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

Source:

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.