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# Strabismus

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Strabismus refers to ocular misalignment and is one of the most common causes of pediatrician referrals to ophthalmologists. Affecting around 3% of the general population, strabismus is occasionally the presenting sign of major intracranial processes and both ocular and nonocular occult malignancies. Although strabismic patients may have normal visual acuity, strabismus is frequently associated with amblyopia or other sight-threatening diseases. Ocular misalignment interferes with the development and use of normal binocular vision. Furthermore, recent studies have confirmed the negative effects of strabismus on self-esteem and in arousing societal prejudices. For example, persons with strabismus were significantly less likely to be hired by prospective employers [1].

This article introduces basic concepts in the evaluation and treatment of strabismus, including diagnostic techniques applicable to the primary care examination.

# Strabismus terminology

- 1. Primary position –Resting eye position, with the head and both eyes pointed straight ahead
- 2. Duction –A monocular eye movement, designated by the direction of the movement (eg. elevation versus depression, abduction versus adduction)

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- 3. Abduction –A monocular eye movement in a temporal direction (eg, the left eye looking to the left, or the right eye looking to the right)
- 4. Adduction—A monocular eye movement in a nasal direction (eg, the left eye looking to the right, or the right eye looking to the left)
- 5. Elevation A monocular eye movement in an upward direction (sursumduction)
- 6. Depression—A monocular eye movement in a downward direction (infraduction)
- 7. Version—A binocular eye movement with both eyes moving in the same direction (eg, left gaze)
- 8. Vergence—A binocular eye movement with the eyes moving in opposite directions (eg, convergence and divergence)
- 9. Tropia -A manifest deviation or ocular misalignment, which may be constant or intermittent
- 10. Phoria –A latent deviation, which requires interruption of binocular fusion (eg, by covering one eye) to allow detection
- 11. Esodeviation –An inward turning of the eyes, which may be manifest (esotropia) or latent (esophoria)
- 12. Exodeviation –An outward turning of the eyes, which may be manifest (exotropia) or latent (exophoria)
- 13. Hyperdeviation –An upward turning of the eyes, which may be manifest (hypertropia) or latent (hyperphoria)
- 14. Hypodeviation –A downward turning of the eyes, which may be manifest (hypotropia) or latent (hypophoria)
- 15. Prism diopter -A unit of strabismus measurement (commonly abbreviated "prism diopters" or  $\Delta$ ). By definition, 1 prism diopter denotes the prism strength that will bend a light ray 1 centimeter at a 1-meter distance from the prism. Horizontal deviations greater than 10 prism diopters are usually clinically significant, whereas vertical strabismus may be problematic at much lower amplitudes.
- 16. Binocular vision The ability to process visual input from both eyes, which can vary in level of sophistication (from simultaneous perception to fusion to stereopsis). Just as visual acuity development may be permanently impeded during the amblyogenic pediatric age range, so too the ability to develop high levels of binocular vision is, largely, age-dependent.
- 17. Fusion Sensory fusion refers to the brain process wherein the images from the properly aligned right and left eyes are united into a single visual perception. Motor fusion refers to the efferent eye movements required to align the eyes to keep each eye fixated on the same target. Thus, motor fusion denotes the mechanical eye muscle adjustments required to allow the internal processing of sensory fusion [2].
- 18. Stereopsis-Depth perception, the highest level of binocular vision
- 19. Diplopia Double vision may be monocular or binocular in origin. Monocular diplopia typically results from the beam-splitting effect of an

- optical aberration in the affected eye. Causes of such aberrations may include corneal scars, cataracts, and retinal diseases.
- 20. Binocular (strabismic) diplopia occurs when the eyes are not properly aligned, but suppression is not available. Strabismic diplopia is rare in congenital or infantile deviations (because of suppression) but is common in acquired strabismus, especially beyond the amblyogenic age range.
- 21. Suppression—A facultative brain process by which binocular diplopia is eliminated by preventing the diplopic image (from the deviated or nonfixing eye) from reaching consciousness. Suppression of sensory input from the deviated eye is common when strabismus begins early in visual development. If visual input is consistently suppressed from the same eye during early visual development, strabismic amblyopia commonly results. Strabismic amblyopia may be avoided if the fixation freely alternates between the two eyes.
- 22. Comitant strabismus—Strabismic measurements remain similar in all gaze directions. The most common forms of childhood strabismus, including most congenital and early-onset esotropias and exotropias, fall into this category.
- 23. Incomitant strabismus—The degree (amplitude) of misalignment changes as the patient looks in different directions. This group includes paretic and restrictive strabismus, central nervous system gaze disorders, and syndromic strabismus.
- 24. Torticollis—A stereotypic anomalous head position (a turn or tilt) should be presumed to have an ocular cause until proven otherwise. Assumption of an eccentric head position is a common association with both paretic and restrictive strabismus. Head turning may serve to obstruct diplopic awareness from the deviated eye, to improve visual acuity by dampening nystagmus (null position), or even to achieve a pinhole effect for refractive errors. Secondary contracture (nonocular torticollis) and facial asymmetry may result from longstanding uncorrected ocular torticollis.
- 25. Asthenopia—Eyestrain caused by accommodative or convergence effort. Commonly referred to as eyestrain, asthenopia can be an expected consequence of prolonged near work, failure to wear appropriate hyperopic glasses, or untreated convergence insufficiency.

## **Pediatrician evaluation**

The pediatrician's responsibility regarding strabismus includes (1) establishing an office screening mechanism, (2) learning and using detection and diagnostic techniques, (3) referring appropriately, (4) supporting and reinforcing ophthalmologist-initiated treatment, (5) providing presurgical medical evaluation and clearance, and (6) facilitating ongoing strabismic monitoring and treatment.

An appropriately timed referral to the ophthalmologist for strabismus will promptly detect conditions that have significant neurologic causes or complications, facilitate detection and treatment of blinding or amblyogenic conditions, promote measures to preserve or develop stereopsis, and reduce parental anxiety. Factors that warrant prompt referral include constant tropic (versus intermittent or phoric) deviations, acutely acquired strabismus, fixation preference (implying strabismic amblyopia), cranial nerve palsies, diplopia, and any strabismus associated with suspected visual impairment, pupillary involvement, or eye pain.

Screening for strabismus should be part of all routine well-child check-ups. Parental information sheets should elicit family suspicions of possible strabismus and other ocular abnormalities. In evaluation of a strabismic complaint, the pediatrician should establish the period of onset, progression frequency and amplitude (amount), constancy versus intermittency, presence or absence of fixation preference (which eye is usually deviated?), family history of strabismus or amblyopia, and evidence of any associated neurologic or other disorders. Historical risk factors for strabismus include prematurity (including premature infants without retinopathy of prematurity), family history (first- or second-degree relative), cerebral palsy, most chromosomal and other major genetic anomalies, prenatal drug exposure (including fetal alcohol syndrome), major head trauma, and major congenital or acquired structural ocular defects.

Family photographs are frequently useful in the evaluation of strabismus. When evaluating these photographs, focus on the centration of the corneal light reflex, symmetry (or lack thereof) of the red reflex, and eyelid anatomy.

The corneal light reflex test, a measure of the decentration of the pupillary light reflex, is the least invasive method of detecting strabismus (Fig. 1A), although this test is less reliable than the cover test in detecting smaller deviations. The light reflex in an esotropic eye will be seen temporal to the pupillary center. Conversely, the light reflex in an exotropic eye is nasal to the pupillary center. The amount of deviation may be quantified by using a prism to center the light reflection (the Krimsky test) (Fig. 1B).





Fig. 1. Infantile esotropia. (A) The light reflection in the deviated right eye is temporal to the pupillary center. (B) Using a prism, the Krimsky test measures the amplitude of the esotropia by centering the light reflection in the right pupil.



Fig. 2. Cover test using readily available occlusive digit.

The cover test (Fig. 2) is the most useful test in detecting and characterizing strabismus. Using this simple method, the observant examiner can confirm the presence of a tropic deviation, establish which eye has been spontaneously fixing, and indicate the direction of the strabismus.

The alternate cover test is more sensitive than the cover test and detects phorias as well as tropias. Small phoric deviations uncovered by alternate cover testing may not be clinically important, however.

Brief periods of exotropia are commonly seen in newborns and through early infancy, particularly during the first 3 months of life (Fig. 3). Like esotropia, constant exotropia in infancy is occasionally found in association with neurologic abnormalities.

Broad epicanthal folds, a wide nasal bridge, or narrow-set eyes commonly result in pseudoesotropia (Fig. 4). Pseudoesotropic eyes may appear crossed because of the small amount of sclera that is visible nasally compared with temporally, especially when the patient looks to the side. Primary practitioners should be cautious in making this diagnosis, because true strabismus may coexist with this structural variation. The light reflex and cover tests are crucial in determining the presence or absence of a tropic deviation.

In recent years, various photoscreeners have been introduced to detect strabismus as well as refractive errors [3]. These devices have the ability to screen large numbers of children rapidly for strabismus, refractive errors, and



Fig. 3. Transient exotropia is relatively frequent during the neonatal period, but normally disappears by age 3 to 4 months.



Fig. 4. Pseudoesotropia produced by a somewhat wide nasal bridge viewed slightly eccentrically.

ocular media opacities. Interpretation of the results requires specialized training, however, which, along with expense issues, has limited widespread adaptation of this technology.

#### Causes of strabismus

The causes of strabismus may be roughly categorized as (1) comitant strabismus (sometimes termed benign or essential childhood strabismus), (2) paralytic strabismus, (3) restrictive strabismus, (4) sensory strabismus, and (5) syndromic strabismus. The most common particular types of strabismus encountered in a pediatric practice include infantile esotropia, accommodative esotropia, intermittent exotropia, sensory exotropia, cranial nerve palsies, and traumatic strabismus.

# Comitant strabismus

Comitant childhood strabismus occurs in the absence of identifiable neurologic, mechanical, sensory, or neurotransmitter deficits or causes. The comitant strabismus group includes infantile esotropia, acquired esotropia, infantile exotropia, and intermittent exotropia. Although the precise causative mechanisms for these conditions remain elusive, two main contributing themes emerge: (1) primary anomalies of motor innervation to the extraocular muscles, and (2) primary anomalies of binocular vision and fusion.

Common features of infantile (essential or congenital) esotropia (Fig. 5) include [4]

- 1. Onset usually by 6 months of age
- 2. Large-angle deviation (> 30 prism diopters), usually constant
- 3. Relatively less dense amblyopia, manifested by alternating fixation and cross-fixation (right eye held in an adducted position to view left visual space, and the left eye used to view toward right visual space)
- 4. Frequent concomitant vertical deviations (inferior oblique overaction, dissociated vertical deviation)

- 5. Frequent positive family history of strabismus
- 6. Relatively lower-grade hyperopia than later-onset accommodative esotropia (< 2 diopters)
- 7. No significant spectacle prescription asymmetry (anisometropia)
- 8. Most children with infantile esotropia are otherwise healthy, but an increased incidence is seen in patients with prematurity, chromosomal abnormalities, and other neurologic problems

Treatment is with eye muscle surgery, preferably within 12 months of onset. Acquired esotropia is usually subclassified as accommodative (related to focusing effort), nonaccommodative, or partly accommodative.

Common features of accommodative esotropia (Fig. 6) include

- 1. Age of onset usually 1.5 to 4 years
- 2. Intermittent at onset, often becoming more frequent; occasionally abrupt onset
- 3. Small- or large-angle deviation
- 4. Frequent and occasionally severe amblyopia, especially with associated anisometropia (asymmetry in glasses strength)
- 5. Frequent positive family history of strabismus or amblyopia
- 6. Higher degrees of hyperopia (2 to 6 diopters)

Treatment is with glasses.

Partly accommodative esotropia occurs when the eye crossing is incompletely improved with spectacle use. Surgery may be beneficial in some cases. Non-accommodative esotropia is not improved with glasses and is usually treated surgically. Nonaccommodative esotropia may be early in onset (as in infantile esotropia) or later (acquired).

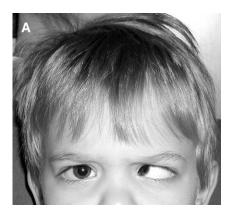




Fig. 5. Large-angle esotropia with alternating fixation and associated elevation in the adducted position (inferior oblique overaction). (A) Right eye fixing. (B) Left eye fixing.





Fig. 6. (A) Accommodative esotropia (B) treated with hyperopic spectacles.

Infantile exotropia is less common than infantile esotropia. Constant exotropia in infancy is found more frequently in children with neurologic abnormalities. Treatment is surgical.

Intermittent exotropia is usually later in onset than esotropia, often presenting with squinting (particularly outdoors, in bright lights, and with distance fixation), diplopia, and eyestrain. The exotropia often becomes more frequent and pronounced over time; spontaneous improvement occurs less commonly. There is some evidence that improvement in distance stereopsis is greater in exotropes corrected at earlier rather than later ages.

# Paralytic strabismus

The cranial nerves involved in ocular movement (oculomotor, trochlear, and abducens) may be affected by congenital, traumatic, infectious, ischemic, and compressive processes. Whereas acute cranial nerve palsies often present in dramatic fashion, more longstanding causes may have a subtle presentation, because of compensatory head positioning. Observers should be aware that patients occasionally fixate with the paretic eye—in other words, the (nonfixing) eye that appears deviated may not be paretic eye.

A sixth (abducens) cranial nerve palsy presents with an esotropia in the primary position and reduced or absent ability to abduct the affected eye. True isolated congenital sixth nerve palsies are rare; more common are the simulating conditions of infantile esotropia with apparent poor abduction caused by cross fixation, Duane syndrome (as discussed later), and Mobius syndrome. Intact abduction can be demonstrated in the infantile esotrope by rapidly spinning the patient or turning the head to the side (doll's head maneuver) (Fig. 7).

Acute unilateral isolated sixth nerve palsies in children most often have a postviral origin. Medical imaging can usually be safely deferred after ensuring that the child is otherwise neurologically intact. Slow improvement in abduction generally occurs within a few weeks; if no improvement follows, the palsy





Fig. 7. (A) Turning the head to the right to elicit leftward gaze. In this patient, decreased left abduction was observed. (B) Reasonably full left eye adduction was present in rightward gaze, with some narrowing of the palpebral fissure. This observation led to the diagnosis of Duane's syndrome, a form of nuclear abducens palsy with aberrant coinnervation of the lateral rectus with a branch from the oculomotor nerve.

worsens, or other neurologic signs or symptoms develop, brain and orbital MR imaging with contrast should be obtained.

Other causes of abducens palsy to consider include intracranial and orbital masses, arteriovenous malformations, trauma, and elevated intracranial pressure. Diabetic and hypertensive palsies, common in the adult population, occur infrequently in children.

Fourth (trochlear) nerve palsy usually presents with intermittent vertical or oblique diplopia and a compensatory head tilt. This palsy most commonly has a







Fig. 8. (A) Patient with right congenital superior oblique palsy manifests a compensatory left head tilt, in which position the eyes appear well aligned. (B) With the head tilted to the right, a right hypertropia becomes manifest. (C) In leftward gaze, the right inferior oblique muscle is overacting, with a large right hypertropia in this gaze position.

congenital or early idiopathic onset but may arise acutely following viral illness or head trauma. Congenital trochlear nerve palsy (Fig. 8) is frequently missed by family members, who may attribute the often subtle heat tilt to a longstanding habit. Vertical diplopia following closed head trauma is frequently caused by unilateral or bilateral superior oblique palsy; with bilateral palsy, patients may have an alternating hypertropia.

A third (oculomotor) nerve palsy presents with a primary position exotropia, with ptosis, enlarged pupil, decreased elevation, depression, and adduction. Oculomotor nerve palsies are less common than fourth or sixth nerve palsies but are often much more difficult to manage because of the involvement of more extraocular muscles. Because the pupillary fibers are located toward the periphery of the nerve, pupillary involvement (dilation) may indicate a compressive lesion, whereas pupil-sparing palsies are often ischemic in origin (unusual in children) [5]. The most common causes of pediatric oculomotor palsies are congenital, traumatic, and brain tumor. Oculomotor palsies are frequently incomplete, with wide variation in the amount of ptosis or elevation deficit, for example. Aberrant reinnervation over time (crossed wires) often results in unusual patterns of eye movement; for example, the eyelid may elevate (levator muscle) on attempted downgaze.

#### Restrictive strabismus

Whereas most strabismus results from neurogenic problems, mechanical forces play a primary role in restrictive strabismus. The rectus muscles may become stiff from inflammation or edema or tight from adherence in an orbital fracture or from postsurgical cicatrisation. Restrictive strabismus is usually incomitant, with increasing misalignment when trying to look away from the restricting muscle.

Blunt ocular trauma can cause sudden localized increased pressure within the orbital space, leading to a blowout fracture of the medial or inferior orbital walls (Fig. 9). Incomitant strabismus may result from localized edema and hematoma within the muscle as well as from actual muscle entrapment within the fracture. Eyelid swelling may preclude a complete ocular examination in some cases. Orbital plane radiographs may disclose the fracture, but orbital computed



Fig. 9. Patient with decreased left downgaze ability resulting from inferior blow-out fracture.

tomographic (CT) scanning, with both axial and coronal views, is indicated when visualization of the rectus muscles is desired.

The ocular findings of most blowout fractures resolve spontaneously. A short course of systemic steroids may be useful in reducing both orbital and eyelid edema. Indications for surgical repair of an inferior blowout fracture include evidence of inferior rectus muscle entrapment (difficulty looking up) and weakness (difficulty looking down), inferior globe displacement toward the maxillary sinus, diplopia progressively worsening in upgaze, and excessively large fractures. Surgical repair should not be undertaken lightly, because the manipulation and exogenous plate material employed can actually induce strabismus by excessive scar formation.

Restrictive strabismus may result from congenital disorders such as congenital orbital fibrosis and certain craniofacial disorders.

Strabismus caused by to dysthyroid (Graves') ophthalmopathy is rarely found in the pediatric population, whose clinical findings are usually limited to eyelid retraction and, occasionally, proptosis. In older Graves' patients, restrictive strabismus may result from inflammatory and restrictive changes within the inferior and medial rectus muscles

# Sensory strabismus

Sensory strabismus occurs when one or both eyes have severe visual loss, leading the brain, in effect, to stop trying to achieve ocular alignment. This process may occur early in life, for example with cortical visual loss, or following acquired vision damage. Sensory esotropia is fairly common in very young patients, but sensory exotropia is more common with longstanding visual loss in older children and adults. Sensory nystagmus (classically a pendular, to-and-fro movement) often accompanies strabismus when the visual loss is both profound and very early in onset.

In a child with strabismus and unilateral loss of vision, the ophthalmologist must distinguish a primary ocular misalignment with secondary strabismic amblyopia from primary visual loss with secondary sensory strabismus.

# Syndromic strabismus

Duane syndrome results from a congenital absence of the abducens nucleus, with aberrant innervation of the lateral rectus muscle by a branch of the oculomotor nerve. This syndrome generally appears as limited abduction and cocontraction of the medial and lateral rectus muscles during attempted adduction (Fig. 10). Clinically Duane syndrome resembles an abducens palsy, with some limitation of adduction and left right asymmetry in palpebral fissure size. Duane syndrome is most commonly unilateral, affecting the left eye about 60% of the time. The majority of affected patients are female, for unclear reasons. Duane





Fig. 10. Left Duane syndrome, with (A) marked eyelid fissure narrowing in right gaze and (B) decreased left eye abduction and fissure widening in left gaze.

syndrome may be isolated, or as a feature of Goldenhar syndrome (oculoauriculovertebral dysplasia), Wildervanck syndrome, or hemifacial microsomia.

Brown syndrome appears clinically to simulate weakness of the inferior oblique muscle but in fact represents a restriction created by a tight superior oblique tendon-sheath complex (Fig. 11). Brown's syndrome may be congenital or acquired and can be both transient and variable.

Myasthenia gravis is relatively uncommon in the pediatric population but should be considered whenever acquired strabismus is intermittent and especially in the presence of variable ptosis. Although often adequately controlled with appropriate pharmacologic therapy (Mestinon, pyridostigmine bromide, ICN Pharmaceuticals, Irvine, CA), prism glasses or strabismus surgery may be appropriate for larger deviations and refractory cases.

## Treatment options in strabismus

Patching is only rarely an appropriate treatment for strabismus per se and in fact may occasionally be contraindicated in intermittent strabismus because it may prevent binocular fusion. Occlusion therapy remains the mainstay of strabismic amblyopia treatment, however. Strabismic amblyopia is generally treated before strabismus surgery, in part because visual acuity takes precedence



Fig. 11. Failure of the right eye to elevate in left-and-up gaze (Brown syndrome).

over strabismus in importance, and because surgical results are improved if visual acuity asymmetry is minimized.

Glasses are useful in the treatment of strabismus when blurring from a significant uncorrected refractive error makes fusion difficult, as can happen with both esotropia and exotropia. Correction of hyperopia is particularly important in the treatment of accommodative esotropia. Recall that the near triad consists of accommodation (focusing), convergence, and miosis. Most young children are hyperopic (because of their shorter axial length) but do not require glasses for correction, because they have a high accommodative reserve, allowing them to overcome their hyperopia. (Presbyopic adults, conversely, have lost enough accommodative reserve to necessitate the use of reading glasses or bifocals.) Most children with accommodative esotropia see reasonably well without using glasses. The accommodative effort needed to overcome the hyperopia stimulates convergence, which contributes to their esotropia, however. Use of hyperopic glasses reduces accommodative drive and, thereby, excess accommodative convergence.

Bifocal glasses are generally indicated as a strabismic treatment only in accommodative esotropia when the distance fixation is well controlled but a significant accommodative near deviation remains.

In children, it is important that refraction be performed following use of cycloplegic eye drops. If cycloplegia is not performed, residual accommodation may result in inaccurate refractive measurements. In hyperopes, residual accommodation may make the far-sighted prescription seem weaker than the true amount of hyperopia; conversely, myopes may seem to require stronger prescriptions than would be measured if accommodation were adequately controlled. Some practitioners may use atropine drops or ointment (which, because of a longer onset of action, requires administration at home) to control accommodation more fully.

Glasses do not make the eyes stronger or weaker and do not alter the axial growth of the eyes, which is the main determinant of future refractive changes. Their use may be considered akin to the choice of shoes, which make walking and running more comfortable but do not change the wearer's future foot size.

Prism glasses may be used either optically to correct a strabismus or to stimulate sensory fusional effort (most commonly by triggering convergence). Ground-in prisms offer excellent clarity but are fixed in amount and are expensive and heavy. Press-on Fresnel prisms are thin and light, allowing for stronger prescriptions that would be too thick to allow ground-in correction. Fresnel prisms are also less costly and easier to change but may induce blurring and glare and are hard to keep clean.

Although most children do not grow out of strabismus, improvement in the crossed appearance does occur over time with pseudostrabismus (where nasal growth leads to normalization of wide epicanthal folds) and in accommodative esotropes whose hyperopia may naturally decline during the period of axial elongation (generally age 5 to 15 years). The mistaken concept that children will outgrow strabismus often delays presentation to an ophthalmologist, with

attendant detrimental effects on visual acuity, binocular vision, and surgical success rates.

Eye exercises (orthoptic therapy) are ineffective for esotropia, largely because the voluntary divergence ability (divergence amplitudes) is so limited. Convergence amplitudes, on the other hand, may usually be increased through exercises. Orthoptic therapy is frequently employed for intermittent exotropia and, in particular, for convergence insufficiency. Orthoptic treatment may also be a useful adjunct to surgical treatment. Conversely, eye exercises are not generally indicated for reading disorders (in the absence of convergence insufficiency), tracking problems, most vertical strabismus, or noncomitant strabismus.

Orthoptic exercises have traditionally been performed in an office setting, or at home using quite simple methods. With the advent of computer-based programs, sophisticated and effective treatment can be done outside the office, with attendant reduction of expense and increased convenience. Such programs usually include the ability to track the compliance of patient use, as well as the progression of strabismic control over time.

Botulinum toxin (Botox, Allergan Pharmaceuticals, Fort Worth, TX) may be injected into an extraocular muscle, leading to an iatrogenic paresis. This procedure is especially useful as a temporizing measure in paretic strabismus, when the antagonist of the paretic muscle in injected. For example, botulinum toxin may be injected in the medial rectus following an abducens palsy. Because the therapeutic effect dissipates over time, and because diffusion of the toxin toward the levator aponeurosis can cause a significant ptosis, botulinum injection is infrequently used in most pediatric strabismus.

In general, strabismus surgery is appropriately reserved for patients in whom nonsurgical methods are likely to be unsuccessful. On the other hand, untoward delay of surgical management can reduce the effectiveness of the operation by preventing the establishment of binocularity and prolonging the period of strabismic suppression.

In general, strabismus surgery consists of loosening (recession) and tightening (resection) procedures. A recession moves the muscle insertion closer to its origin, reducing the vector force, much as occurs when relaxing a tight rubber band. In a resection, by contrast, the muscle is stretched by removing a portion of muscle belly and tendon near its insertion. This tightening has a restrictive effect on globe movement in the gaze opposite the resected muscle.

The amount that a muscle is moved or resected and the number of muscles operated depends in part on the severity or amplitude of the ocular deviation. Usually the muscle is moved a fixed distance (in millimeters) which has been preselected by the surgeon. A suture refixates the muscle tendon to the scleral surface.

When an adjustable suture technique is employed, a temporary bow or slipknot is used intraoperatively to secure the muscle to the globe; the muscle position may be adjusted later, with the patient awake. In certain cases this technique may improve the predictability of the operation by giving the surgeon a second chance to adjust the final outcome.

There are different circumstances when the surgeon may operate on one rather than both eyes at once. Because the two eyes are yoked, most strabismus can be improved by either unilateral or bilateral surgery, even when there is a fixation preference and only one eye is observed by the parents to deviate.

The primary goals and benefits of strabismus surgery are (1) to maximize visual acuity, (2) to optimize conditions for binocular vision, (3) to improve versions, and (4) to correct disfiguring appearance (reconstructive, rather than cosmetic surgery).

From a functional standpoint, the surgeon aims to achieve an adequacy of alignment to allow gross binocularity (generally considered within 12 to 15 prism diopters of horizontal alignment). Recovery of binocularity may occur in adults following strabismus surgery, even if the duration of deviation is longstanding. Thus, even in adults, strabismus correction should not be considered purely cosmetic [6]. Finally, binocular peripheral visual fields are expanded following surgery for esotropia. This expansion is approximately commensurate with the strabismus correction achieved and occurs regardless of concurrent amblyopia or absence of binocular fusion [7,8].

Modern strabismus surgery is performed on an outpatient basis, usually requiring 30 to 90 minutes (depending on the number of muscles operated). General anesthesia is usually employed, although intravenous sedation and even topical anesthetic techniques may be appropriate in some teenagers and adults. Postoperative pain is controllable with acetaminophen or oral nonsteroidal anti-inflammatory agents, occasionally with the additional use of anti-emetics and codeine.

# **Surgical complications**

The most common complication of strabismus surgery remains over- and undercorrection. The rate of surgical success varies according to numerous factors, including age of onset, age of surgical correction, preoperative intermittency, preoperative binocularity, and presence of other neurologic deficits. Overall success rates in achieving satisfactory alignment are approximately 75% to 80% with each eye muscle procedure. Observation for at least 6 weeks postoperatively is usually recommended before considering reoperation for residual deviation following strabismus surgery.

Although popular opinion may suggest that the results of strabismus surgery are not lasting, and that multiple operations are the norm, patients who establish good binocular fusion postoperatively usually do not require reoperation [9]. When surgery for infantile esotropia is performed within 1 year of onset, success rates are significantly better than those obtained when surgery is delayed beyond this time [10].

Besides unsatisfactory ocular alignment, the other complications of strabismus surgery are infrequent and include infections, excessive bleeding, conjunctival scarring, scleral perforation, and loss of vision. Postsurgical diplopia is a

consideration when operating in older children and adults; the frequency of persistent diplopia seems to be significantly less than once suspected, however.

# **Summary**

The various forms of strabismus present clinicians with interesting diagnostic and treatment dilemmas. The importance in distinguishing the various structural, neurosensory, and developmental forces at work in the interplay between visual acuity, binocularity, and oculomotor function keeps even experienced strabismologists occupied. It is hoped that this article may help pediatricians approach their strabismic patients with a fuller understanding of the subject.

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