

Exodeviations

Classification and Etiology

Even though the classification and etiology of strabismus are discussed in Chapters 8 and 9, a few specific remarks regarding exodeviations are in order in this chapter. Although fair agreement has been reached with respect to the classification and etiology of esodeviations, the same cannot be said for exodeviations.

Most current classifications of exodeviations are derived from Duane,⁴⁹ who championed the view that exodeviations are caused by an innervational imbalance that upsets the reciprocal relationship between active convergence and divergence mechanisms. According to Duane, an exodeviation greater at distance than at near fixation is caused by hypertonicity of divergence (*excess*), a deviation greater at near than at distance is caused by *convergence insufficiency*, and a deviation at distance equal to that at near fixation (*basic exotropia*) is caused by a divergence excess combined with a convergence insufficiency.

Some have argued against the etiologic concept Duane implied by this terminology; however, his classification has survived and is in current usage by many strabismologists.

Duane's classification is based on the assumption that divergence is an active process rather than relaxation of convergence with a return of the eyes to parallelism or a divergent position by mechanical or elastic forces. Most modern investigators share this view, which has been confirmed by electromyographic studies^{18–20, 122, 170} (see Chapter 22). Bielschowsky,¹⁴ on the other hand, al-

though not denying the existence of an active divergence mechanism, questioned Duane's claim that the majority of exodeviations are based on hyperactive tonic divergence. He argued that Duane's theory did not take into account the abnormal position of rest associated with exodeviations. This abnormal position is determined by anatomical and mechanical factors such as topographic and physical properties of the extrabulbar tissues, the shape and axis of the orbits, the interpupillary distance, and the size of the globe. As early as 1896, Weiss¹⁷⁸ had shown how the growth and depth of the orbit, as well as the length and insertion of the horizontal rectus muscles, may influence the functional equilibrium between medial and lateral rectus muscle actions. That orbital factors indeed may have etiologic significance in causing exodeviations is a view also supported by the high prevalence of exodeviations in patients with craniofacial dysostosis (Crouzon's disease) in whom shallow and laterally directed orbits are prominent clinical findings.

Bielschowsky,^{14, 15} in support of his theory that an anomalous position of rest contributes to the occurrence of exodeviations, cited the high incidence of sensory exotropia after disruption of fusion by unilateral blindness (see also Chavasse³⁹ and p. 345). He also pointed out that development of divergence excess secondary to convergence insufficiency is an untenable concept in view of the fact that patients with defective convergence are frequently orthophoric or even esophoric at distance fixation.

Most current theories on the etiology of exode-

viations combine the ideas of Duane and Bielschowsky and revolve around the concept that exodeviations are caused by a *combination of mechanical and innervational factors*,^{28: 44, p. 349; 98; 146} the innervational factors consisting of variation of convergence innervation or disturbed equilibrium between convergence and divergence.

Burian²⁸ summarized this thinking by stating that patients with exodeviations have a basic misalignment of the eyes caused by mechanical and anatomical (*static*) factors, the nature of which must remain speculative at the present stage of our knowledge. This basic exotropia may be defined as the relative position of the visual axes when there is no stimulus to fusion, when the refractive errors of the eyes are corrected, and when the dominant eye is fixating on a distant object with the eyes in primary position.¹⁶⁹ To this basic deviation are added innervational (*dynamic*) factors that tend to maintain ocular alignment by convergence or to impair it by divergence. Normal interplay between these innervational influences provides for gross alignment of the eyes, and any abnormality in this interplay is the primary factor in the pathogenesis of exodeviations. Burian further pointed out that during childhood, “exuberant” (Chavasse³⁹) functioning of the convergence mechanism may obscure a basic exodeviation at near fixation (*simulated divergence excess*) and that special tests are needed to elicit the true deviation.

In addition to interplay between the convergence and divergence mechanisms, refractive errors may further modify the innervational pattern that influences the position of the eyes. For instance, in a patient with uncorrected myopia, less than normal accommodative effort is required during near vision, thus causing decreased accommodative convergence. According to Donders,⁴⁸ this constant understimulation of convergence may cause an exodeviation to develop. It must be emphasized, however, that the role of myopia in the etiology of exodeviations is far less prominent than that of hypermetropia in esodeviations.

A similar mechanism prevails in patients with a hypermetropia. If a high degree of hypermetropia is uncorrected, such patients make no effort to overcome the refractive error by an accommodative effort, and clear vision is unattainable.¹⁴¹ As in the previous case, an exodeviation may develop on the basis of an understimulated and thus underactive convergence mechanism that causes the accommodative convergence–accommodation (AC/

A) ratio to remain low or even flat. In moderate degrees of hypermetropia, spectacles correction will decrease the accommodative demand and an underlying exodeviation, previously controlled by accommodative convergence, will increase and may require treatment.

Jampolsky and coworkers⁹³ emphasized that although an equal degree of myopia in both eyes cannot be correlated with exodeviations, anisomyopia and anisoastigmatism bear distinct relationships to exodeviations. Unequal clarity of retinal images may present an obstacle to fusion, facilitate suppression, and therefore contribute to the pathogenesis of exotropia.

The view that tonic convergence is a factor in masking exodeviations at near fixation and that excessive divergence may cause an exodeviation at distance fixation has been challenged by Jampolsky,⁹² who refutes the existence of convergence and divergence innervation other than that caused by fusional or accommodative stimuli (see Chapters 5 and 22). Although we cannot accept this reasoning unequivocally, we are aware that at this time no clinical or laboratory evidence exists for excessive tonic divergence innervation in exodeviations. Thus the term *divergence excess* introduced by Duane may well be a misnomer. Likewise, Duane’s term *convergence insufficiency* for describing exodeviations that are greater at near than at distance fixation is not identical with a synonymous condition (see Chapter 22) which may or may not be accompanied by an exodeviation. In fact, a patient with convergence insufficiency may have orthophoria or even esophoria at near fixation. On the other hand, a “convergence insufficiency *type or pattern*” exodeviation may be associated with a normal near point of convergence and normal or even excessive fusional convergence amplitudes. Thus we use Duane’s classification merely in a descriptive sense without accepting all its etiologic implications. With this reservation in mind, we classify exodeviations into the following patterns:

1. *Divergence excess pattern.* The exodeviation is at least 15^Δ larger at distance than at near fixation.
2. *Basic exodeviation.* The distance deviation is approximately equal to the near deviation.
3. *Convergence insufficiency pattern.* The near deviation is at least 15^Δ greater than the distance deviation.
4. *Simulated divergence excess pattern.* The prism and cover test will show an exodevia-

tion that is significantly larger at distance than at near fixation. However, a larger, static deviation at near fixation is obscured by dynamic factors such as persistent convergence innervation (vergence aftereffect; see p. 202), and special tests are required to reveal the deviation at near fixation, which will then often equal or even exceed that at distance fixation. It is necessary to distinguish between the contributions. As will be pointed out later in this chapter, it is necessary to distinguish between a reduction of the near deviation caused by a fusional convergence aftereffect (Burian's pseudodivergence excess type) or by the addition of plus lenses.^{76, 106, 137}

In terms of the state of fusion, exodeviations can be classified further as exophoria (X), intermittent exotropia (X[T]), and exotropia (XT) (see Chapter 8).

In addition to classic theories regarding the etiology of exodeviations discussed in the preceding paragraphs, an interesting and unconventional possibility was introduced by Mitsui.¹²³ He noted that in exotropia a slight adductive force applied to the fixating eye with a fixation forceps causes the deviated eye to adduct (magician's forceps phenomenon). Mitsui concluded from this and other observations¹²⁴ that abnormal proprioceptive impulses originating from the dominant eye are the cause of the exodeviation. However, this phenomenon can be explained on the basis of a visually elicited refixation reflex; that is, by adducting the fixating eye with a forceps, the retinal image is displaced nasally. The patient now attempts to refixate with an abduction saccade, but this movement cannot be executed because the eye is mechanically stabilized. The impulse to abduct will be transmitted to the deviated eye as an adduction impulse (Hering's law).^{102, 138} As one may expect, this phenomenon cannot be elicited when the fixating eye is prevented, by means of a translucent occluder, from registering the image displacement.¹³⁸ Thus, it is unlikely that a mechanism other than a visually elicited fixation reflex accounts for the phenomenon described by Mitsui.

Exodeviations also may be associated with vertical anomalies, and the angle of deviation may change in upward or downward gaze (A and V patterns). In this respect, they do not differ from esodeviations; this type of strabismus is discussed in Chapter 19.

Primary Exodeviations

Clinical Characteristics

Exotropia differs from esotropia not only in direction and size of the deviation but also with respect to prevalence, sex predilection, age of the patient at onset, progression of the disease, prognosis, nature of the underlying sensorial adaptation, and the etiologic significance of associated refractive errors. Also, exodeviations are much more common in a latent or intermittent form than are esodeviations. A patient may exhibit a manifest exotropia during one examination, and at another time an exophoria or intermittent exotropia. Indeed, it is common to observe rapid switching from one phase to the other during the same examination. Mechanisms responsible for these variations include the degree of fusional control with varying levels of alertness, the convergence-accommodation relationship, and the change of the angle of deviation at different fixation distances. For this reason, it is often impossible to distinguish clearly between exophoria and exotropia or, from a clinical point of view, to consider them as different entities. Therefore exophoria, intermittent exotropia, and exotropia will be discussed together, but efforts will be made to point out distinguishing features among these conditions. We should point out, however, that these three entities present with differential clinical features. A decompensation of exophoria is noticed quite soon as it is always accompanied by diplopia. Children will often close one eye and complain about visual disturbances. On the other hand, monocular eye closure in intermittent exotropia has a different explanation, as will be discussed later in this chapter. Subjective symptoms are usually absent and this condition may not be readily recognized by the parents. In constant exotropia, binocular vision is absent and no symptoms are present.

PREVALENCE. Exodeviations occur less frequently than esodeviations. During ophthalmic screening of 38,000 children aged 1 to 2½ years and observed at child welfare clinics in Israel, Friedmann and coworkers⁶² detected strabismus in 498 infants, of whom 72.2% had esotropia and 23% exotropia. This ratio of approximately 1:3 in the prevalence of exotropia and esotropia has also been established in surveys from Scandinavia,^{61, 134} Great Britain,⁶⁶ western Canada,¹⁰³ and the United States.^{43, 161} We have the distinct impression, based

on having been involved in the screening of populations in other parts of the world, that exodeviations occur more commonly in the Middle East, subequatorial Africa, and the Orient than in the United States. They appear to occur least commonly in central Europe. On the other hand, Chew and coworkers⁴³ reported no difference in the frequency of exodeviations in white and African-American children in the United States.

In comparing the prevalences reported from different countries, Jenkins⁹⁴ made the interesting observation that the nearer a country is to the equator, the higher the prevalence of exodeviations. A comprehensive epidemiologic study in which ethnic population differences⁸⁷ and even climatic and heliotropic⁵⁴ factors are considered is needed to explore the significance of these observations. The implication of a recent report that the prevalence of systemic and ocular disease is higher during the first year of life in children with exotropia than in those with esotropia⁸² is not at once obvious.

AGE OF ONSET AND NATURAL HISTORY. Contrary to common belief, onset of the majority of exodeviations is shortly after birth. In Costenbader's^{44, p. 353} series of 472 patients with intermittent exotropia of the divergence excess type, the deviation was present at birth in 204 and appeared in 16 at 6 months of age and in 72 between 6 and 12 months of age. In only 24 of his patients did exotropia develop after 5 years of age. Krzyzkowa and Pajakowa¹⁰⁵ reported the age of onset to be before 2 years of age in 34.5% of their patients; Hall,⁷⁰ in 37%; and Holland,⁸⁰ in 70%. In a more recent study, the mean age at diagnosis was 7.8 months.¹⁶

It is not always possible to ascertain by history alone whether a constant exotropia was present at birth or occurred shortly thereafter or was preceded by a period of intermittency. Yet such information would be important in assessing the prognosis. In the latter instance the chances for restoring normal binocular functions are better than in the former. Moore and Cohen¹²⁸ reported that fusion was unattainable after surgical alignment in patients with a genuine "congenital" exotropia.

Jampolsky⁹¹ made the point that, with rare exceptions, exodeviations begin as an exophoria that may deteriorate into intermittent and constant exotropia as suppression develops. He considered suppression to be the key that unlocks the fusion

mechanism. Such deviations usually occur first at distance and later at near fixation. Obviously, the prognosis for recovery of normal binocular function is infinitely better in patients who experience a long phase of intermittency than in those with a manifest deviation since early childhood.

Factors that may influence progression are the decrease in tonic convergence with advancing age, development of suppression, gradual lessening of accommodative power, and increased divergence of the orbits with advancing age. Progression may take several forms. The deviation may increase at near or at distance fixation,^{97, 130} exophoria may become intermittent or change to manifest exotropia, or suppression may develop.

Burian²⁷ observed that the divergence excess type of deviation tends to remain more or less stable, whereas with simulated divergence excess the near deviation tends to increase. In patients with the convergence insufficiency type of deviation, binocular function degenerates rapidly and progressively, and in those with a basic exotropia there is a tendency for the deviation to increase or for secondary convergence insufficiency to develop.

The generally progressive nature of the disease has important therapeutic implications in regard to indications for and timing of surgery. At this point, therefore, it is necessary to emphasize that not all exodeviations are progressive and that some remain unchanged over many years of observation; in fact, some improve without therapy.^{57, 78} Von Noorden¹³⁵ followed for an average of 3.5 years 51 patients ranging from 5 to 10 years of age with intermittent exotropia who, for one reason or another, were not operated on. One or more signs of progression, as defined above, were present in 75%, no change occurred in 9%, and 16% improved without therapy. From these studies, it follows that patients with intermittent exodeviations need to be evaluated over a period of time to ascertain whether progression is taking place and surgery is warranted, particularly those in whom a constant deviation is present less than 50% of the time.

Constant, infantile exotropia with an onset shortly after birth and with a large angle deviation that does not change at near and distance fixation may also occur and its clinical characteristics are said to be similar to those in infantile esotropia.¹⁵⁸ An association between early-onset exotropia with a large angle and delayed visual maturation has been reported.¹⁸⁴

SEX DISTRIBUTION. Several authors have commented on the preponderance of women in a population of patients with exodeviations. Cass³⁷ reported a prevalence of 62 (70%) women in 88 patients; Gregersen,⁶⁷ 61% women in 231 patients; and Krzystkova and Pajakowa,¹⁰⁵ 67% women in 620 patients with exodeviations.

REFRACTIVE ERRORS. The prevalence of refractive errors associated with exodeviations varies according to different investigators. Donders⁴⁸ found 70% of “comparatively high” myopes in a group of 100 patients with exotropia and concluded that reduction of accommodation in such patients is pivotal in the etiology of exodeviations. From more recent studies,^{28, 67, 105, 161} it appears, however, that distribution of refractive errors in exotropes resembles that in the nonstrabismic population and that the etiology usually is unrelated to the underlying refractive error.

SIGNS AND SYMPTOMS. Generally, signs and symptoms of exodeviations are no different from those observed in patients with other forms of strabismus, as discussed in Chapter 10. Patients with exophoria commonly complain of eyestrain, blurring of vision, difficulties with prolonged periods of reading, headaches, and diplopia. Children with intermittent or constant exodeviations are less frequently symptomatic because, unless the deviation is of recent onset, a well-developed suppression mechanism eliminates diplopia.⁹⁵ On the other hand, adults with intermittent exotropia are commonly symptomatic, and their complaints are not different from those with inadequately compensated exophorias.

PHOTOPHOBIA. One symptom that deserves special comment is photophobia, for it occurs commonly in association with intermittent exodeviations. In spite of the frequency of its occurrence,^{81, p. 219; 100, p. 234; 113; 146} little if any attempt has been made in the literature to satisfactorily explain photophobia in connection with exodeviations. It has been assumed that when a child is outdoors and looking at infinity, there are no near clues to stimulate convergence and that bright sunlight dazzles the retinas so that fusion is somehow disrupted, causing the deviation to become manifest.¹¹⁸ These explanations imply that one eye is closed to avoid diplopia and visual confusion. This view is held, for instance, by Wang and Chryssanthou,⁷⁵ who found that patients with anomalous retinal correspondence are less apt to complain about photophobia than those with nor-

mal correspondence. Jampolsky⁸⁹ assumes that the intermittent exotropes shuts one eye in bright light to avoid the many perceptual visual field changes that take place in bright light diffusion, which in turn may trigger the “hemiretinal suppression mechanism” (see pp. 218, 365).

Wirtschafter and von Noorden¹⁸³ demonstrated that bright light adversely affects the amplitude of fusional convergence in patients who maintain a delicate balance between exophoria and intermittent exotropia (see also Campos and Cipolli³⁶). Orthophoric patients, those with exophoria and adequate fusional amplitudes, and those with well-established intermittent exotropia were not affected in this manner. Eustace and coworkers⁵⁴ also noted that bright light causes exophoria to become manifest and suggested the use of photochromatic lenses to relieve photophobia in such patients. Graefe⁶⁵ reported an increase in the deviation at near fixation under the influence of bright light in patients with exophoria.

It is a common misconception that habitual monocular eye closure in bright sunlight is limited to intermittent exotropes and that eye closure is triggered by diplopia. Wiggins and von Noorden¹⁸⁰ observed photophobia predominantly in intermittent exotropes but also in constant exotropia, esotropia, and normal subjects. None of the subjects was aware of diplopia before closing one eye or under any other circumstances. The common factor in these patients was a significantly decreased binocular photophobia threshold, which was measured by exposure to an intense artificial illumination. This finding was unrelated to the presence or absence of anomalous correspondence as proposed by Wang and Chryssanthou.¹⁷⁵ The significance of these results and the reason for the highest incidence of photophobia in the intermittent esotropia group remains elusive at this time.

MICROPSIA. Another less well-known symptom of intermittent exodeviations is micropsia. We have seen several patients with this anomaly since we first became aware of it through the patient in Case 17-1.

CASE 17-1

This 21-year-old woman has had intermittent exotropia since childhood. Her right eye had been operated on by another ophthalmologist. She described her current problem as follows: “When I look in the distance, things go in and out of focus and then get

much smaller, like looking through the wrong end of binoculars." Examination revealed an intermittent exotropia of 22^Δ at distance and 18^Δ at near fixation. The deviation was fused at distance but changed from exophoria to exotropia, and the angle increased as soon as the patient was asked to read the 6/6 line on the acuity chart.

Clearly, the patient in Case 17–1 used accommodative convergence to control her exodeviation at distance. Since convergence and accommodation are associated with objects appearing smaller and closer, micropsia was experienced whenever these mechanisms were involved.

EXAMINATION AND SPECIAL TESTS. In addition to the testing procedures outlined in Chapters 12 and 13, several comments need to be made regarding tests specifically applicable to patients with exodeviations.

OCCCLUSION TEST OF SCOBEE-BURIAN. The occlusion test for differentiation between true and simulated divergence excess patterns is important because, as discussed under surgical management on page 368, the outcome of this test determines our choice of surgical procedure. Scobee¹⁶³, p. 172 pointed out that in patients with intermittent exodeviations the angle of exotropia elicited by the alternate cover test is greater at distance more often than at near fixation. By unilaterally occluding the eyes of such patients for 24 hours or by teaching them voluntary relaxation of convergence, he found that exotropia would increase at near fixation and become greater than at distance fixation. He explained control of the near deviation on the basis of the greater fusional stimuli provided by an object at near, such as the larger size of the retinal images, the increased brightness and proximity of the object, and the effect of accommodative convergence. Burian²⁶ reported independently that in many patients with an apparent divergence excess type of deviation, only brief periods of unilateral occlusion (30 to 45 minutes) are sufficient to cause an increase in the near deviation so that it equals or even exceeds that at distance fixation. He described such patients as having *simulated divergence excess* and distinguished this group from those in whom the near deviation is not influenced by brief periods of occlusion (*true divergence excess type*).

In addition to the factors enumerated by Scobee to explain this phenomenon, we believe that extremely active convergence tonus during childhood may be a factor in obscuring the exodevia-

tion at near fixation. This mechanism enables patients with a basic deviation to keep their eyes aligned for near vision but not for distance vision, where convergence is less active. Momentary disruption of fusion by alternately covering each eye in a rapid fashion, as during the prism and cover test, is obviously insufficient to disrupt this powerful compensatory mechanism that may have been exerted during all waking hours for years (vergence aftereffect, p. 202). Kushner introduced the term "tenacious proximal fusion" for the persistent convergence innervation that hides the exodeviation at near fixation.^{109, 110} This term seems awkward and does not add to the clarification of the issue. The term "convergence aftereffect" is a more appropriate description of this phenomenon.

One of the reasons why most patients with intermittent exotropia do not use fusional convergence also to overcome the exodeviation at distance fixation may be that the basic exodeviation is larger at distance than at near fixation and exceeds the limits of the fusional convergence amplitude. Other factors to be considered are the lack of proximal stimuli at distance fixation and the unaccustomed and difficult task of converging upon visual objects at infinity.

The patch test is illustrated in Figure 17–1. Momentary binocular stimulation may reinstate the mechanism by which the patient controls the deviation at near fixation. Thus before the measurement after the patching period, the fellow eye must be occluded before the patch is removed; one may then proceed in the usual manner with the prism cover test. Whether the dominant or nondominant eye is occluded does not seem to influence the results of this test.¹³² Publications in the European literature frequently and unjustifiably refer to the patch test as "Marlow occlusion." Marlow¹¹⁹ used unilateral occlusion of the dominant eye for as long as 1 to 2 weeks in nonstrabismic patients to "relax the muscles" and unmask horizontal or vertical heterophorias of a magnitude not exceeding a few prism diopters. If an eponym is to be attached to the patch test, which is different from the Marlow test in purpose and execution, it would be more appropriate to name the test after Scobee and Burian.

In a group of 46 patients in whom the exodeviation was greater at distance than at near fixation, von Noorden¹³⁶ found a true divergence excess pattern in only 14; in the remainder the occlusion test revealed simulated divergence excess. In 237 consecutive patients with exodeviations, Burian

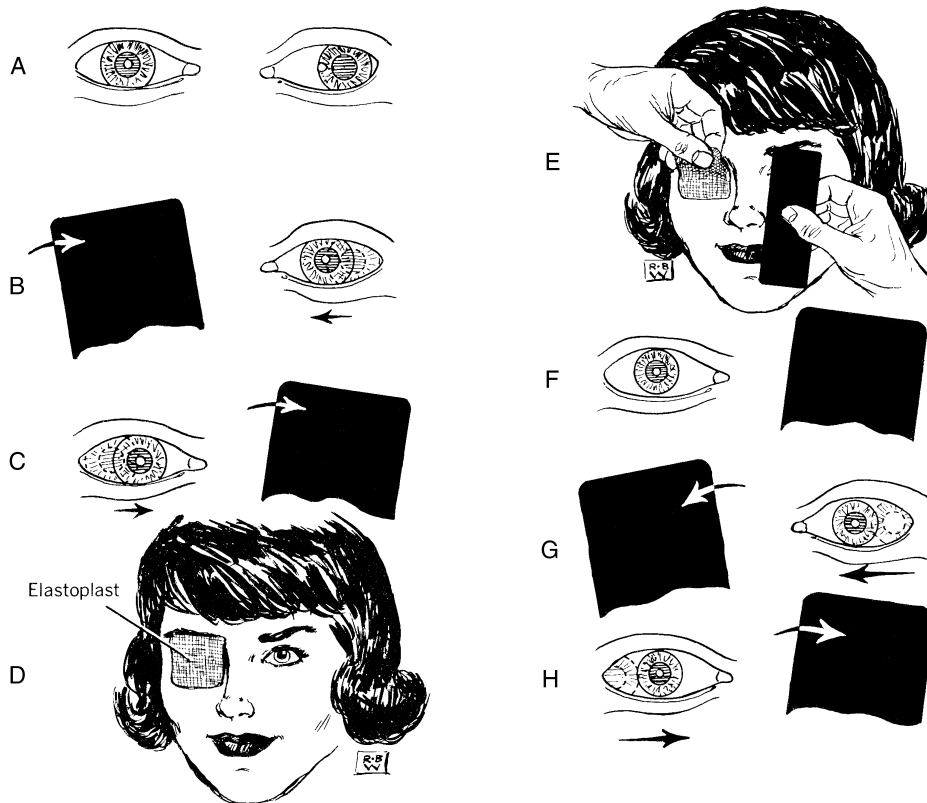


FIGURE 17-1. The patch test. *A–C*, Alternate cover test has revealed an exotropia that is significantly (15^{Δ}) smaller at near than at distance fixation. *D*, Patch is placed over one eye for 1 hour to thoroughly dissociate the eyes. *E*, Before removal of the patch the fellow eye is covered with an occluder. After the patch has been removed, it is important to prevent the patient from using the eyes together, even momentarily, since only a brief binocular exposure may be sufficient to again obscure the near deviation by fusional convergence. *F*, Patch has been removed. *G* and *H*, If simulated divergence excess (basic exotropia) is present, rapid alternate covering will reveal a markedly increased near deviation that may match or even exceed the distance deviation, whereas with true divergence excess the near deviation will remain unchanged. (From Noorden GK von: Atlas of Strabismus, ed 4. St Louis, Mosby-Year Book, 1983.)

and Franceschetti²⁹ found only 10 with a true divergence excess pattern. Thus it seems that the majority of patients with exodeviations in whom the deviation at distance fixation exceeds that at near belong in the simulated divergence excess category.

+3.00 SPHERICAL LENS TEST. The effect of brief periods of unilateral occlusion on an exodeviation at near fixation must not be confused with the effect of +3.00D spherical lenses. Occlusion removes the vergence aftereffect, whereas +3.00 lenses suspend accommodation and thus accommodative convergence. Elimination of the accommodative requirement at near fixation will have little influence on the positions of the eyes in a patient with an exodeviation and a low AC/A ratio; the angle of strabismus will increase only slightly when measured through +3.00 lenses. On

the other hand, with a high AC/A ratio, if the deviation is measured through +3.00 lenses, it will increase substantially at near fixation and under certain circumstances may equal that at distance.

This information may be clinically important for several reasons. Brown²² suggested that preoperative determination of the AC/A ratio may be helpful in predicting the extent to which a patient may respond to plus lenses when surgical overcorrection is obtained. Also, a patient with a high AC/A ratio and a basic exodeviation will manage to keep the eyes aligned when exerting normal accommodative effort at near fixation. Such a patient will respond well to minus lenses prescribed to reduce the distance deviation. Brown²³ and Jampolsky⁹² have commented on the high incidence of high AC/A ratios in patients with exodevi-

ations. On the other hand, von Noorden,¹³⁶ who determined the AC/A ratio over a range of 6D with +3.00 and -3.00 lenses in 46 patients with an apparent divergence excess, found that the AC/A ratio ranged from 3.3 to 9.

The purpose and interpretation of the Scobee-Burian and the +3.00 spherical lens tests are frequently confused in the literature. It must be understood that plus lenses do not uncover a deviation that is held in check by accommodative convergence, similar to uncovering by occlusion a deviation that is kept latent by the vergence aftereffect. The increase of the exodeviation under the influence of plus lenses is triggered by optically reducing the accommodative demand, a situation that is *artificially* induced by the examiner and different from ordinary conditions of seeing. Since different mechanisms are involved in the occlusion and +3.00 lens tests, it can be expected that patients with an apparent divergence excess type of deviation may respond differently to each test. That this is indeed so was shown by Burian and Franceschetti.²⁹ With respect to the choice of surgical procedure, we rely primarily on the occlusion test.

MEASUREMENT OF THE DEVIATION. Methods for determining the angle of strabismus are described in Chapter 12, and only a few special comments need be made in this chapter in connection with exodeviations. We have mentioned that young patients with an exodeviation may use voluntary convergence to overcome the deviation at near fixation, and in certain cases this compensatory mechanism may extend so as to control the deviation at distance fixation as well. Voluntary convergence then is enlisted to maintain single binocular vision at distance fixation. These patients obviously put up with the induced myopia and prefer blurred and single vision over sharp and double vision. Unless the target used for distance fixation forces patients to relax accommodation, and with it convergence, true deviation of the eyes at distance fixation may remain concealed. Therefore, we prefer to measure the angle of strabismus at distance while a patient reads the 6/9 line on the visual acuity chart. To recognize these letters, the patient must relax accommodation. When a patient with an exodeviation complains about intermittent blurred vision at distance, an accommodative spasm based on this mechanism must be considered. This situation may be confusing to the inexperienced ophthalmologist, who

finds visual acuity at distance to be decreased but who on refraction is unable to detect myopia in spite of the patient's complaints. Determination of binocular visual acuity is a simple method of detecting *accommodative spasm* in patients with exodeviations.^{15, 24, 165} When accommodative spasm is present, binocular visual acuity will be significantly reduced in comparison with unocularly obtained visual acuity (see Chapter 22).

Another important aspect to be considered when measuring the angle of deviation in patients with exodeviations is the *testing distance*. White¹⁷⁹ was the first to point out that to elicit the maximal deviation, measurements should be performed at fixation distances greater than 6 m. Many investigators agree with this modification of testing procedure^{38; 92; 98, p. 364; 107} since a larger angle of deviation may be detected and, more important, the fusional state of the patient may be revealed under a more natural visual condition than within the confines of an examination lane. Burian and Smith³⁰ noted the exodeviation to increase significantly in 31 of 105 patients when measured at 30 m.

Mention should be made also about the *variability of fusional control in patients* with intermittent exotropia. The extent to which an exodeviation is controlled by fusion depends not only on the size of the angle but also to a large extent on the general health, alertness, attention span, and level of anxiety of the patient at the time of examination. Considerable variation in the degree of fusional control from one examination to another is not a surprising finding. Repeated examinations, preferably at different times during the day, are required to assess the clinical situation thoroughly. For instance, a child who, when seen in the morning, may fuse steadily at near and distance fixation in spite of a large exophoria may exhibit manifest exotropia without fusional recovery in the late afternoon. The opposite also may be observed. For instance, a patient with unstable fusion in whom the deviation is mostly intermittent or manifest on repeated examinations may exhibit transient improvement of fusional control when admitted for surgery. Obviously, anxiety associated with the impending operation releases extra energy, permitting the patient to keep the eyes aligned. This should not deter the experienced surgeon from proceeding as planned with the operation. We have observed on several occasions that a less experienced physician may become intimidated by this apparent improvement

and send a patient home without surgery who will then have to be readmitted at a later date.

Finally, attention must be paid to the *angle of deviation in lateral gaze*. Parks¹⁴⁶ recommended decreasing the amount of recession of the lateral rectus muscles ordinarily done for an exodeviation in primary position when the measurements in right and left gaze show less exodeviation than in the primary position. This impression was confirmed by Moore,¹²⁷ who reported that surgical overcorrection is likely to occur in patients with intermittent exotropia whose deviation decreases in lateroversion as compared with the primary position (lateral gaze incomitance). This was true regardless of the type of intermittent exodeviation or type of surgery used. A 20% or greater decrease in the lateral gaze deviation is considered to be significant.¹⁰⁰ Moore did not mention whether, as might be expected, her patients with lateral gaze incomitance had limitation of abduction. However, her findings are in accordance with our philosophy that it is not advisable to perform a conventional amount of recession on an already underacting muscle. We confirmed the occurrence of lateral gaze incomitance in 55 of 92 exotropic patients and found that the decrease in the deviation in lateral gaze was asymmetrical in most instances.¹²¹ Caldeira³² reported similar findings. Reduction of the surgical dosage is advisable when the decrease in the deviation in lateral gaze is significant.^{121, 127} To avoid false measurements in lateral gaze, care must be taken not to rotate a loose plastic prism but to keep its back surface perpendicular to the optical axis of the eye.¹⁵⁵

SENSORIAL ADAPTATIONS. The sensory behavior associated with exodeviations differs in several respects from that in patients with esodeviations, which may be caused partly by differences in evolution of the disease. Anatomical and possibly physiologic variances between nasal and temporal retina are other factors held responsible for differences in the characteristics of sensorial adaptations in these two conditions.^{55, 131} In patients with the divergence excess type of exodeviation, a latent strabismus at near fixation often coexists with a manifest strabismus at distance fixation. Thus, normal binocular vision is constantly being reinforced, and sensorial adaptations are infrequent or, when present, are only superficially established. Deep amblyopia with eccentric fixation is a rare finding with an exodeviation and limited to unilateral deviations, usually caused by partial or com-

plete oculomotor paralysis. Likewise, deep-seated anomalous retinal correspondence occurs primarily with unilateral constant exotropia.⁷⁹ The majority of patients have an alternating type of strabismus with normal visual acuity in each eye and suppression of the nonfixating eye. In patients with intermittent exotropia, normal and anomalous retinal correspondence may coexist (see Chapter 13), and the afterimage test may indicate abnormal correspondence with one eye deviated and normal retinal correspondence with the eyes aligned.^{25; 81, p.198}

In certain patients with a large angle constant exotropia the results of sensory testing with a red glass may reveal a most puzzling finding. The patient will report paradoxical, that is, uncrossed, diplopia.^{1, 17, 37, 45, 47, 60} Homonymous localization of all binocularly perceived images is present. This confusing situation, which has been referred to as *panoramic vision*, seems to indicate a lack of any retinal correspondence, normal or abnormal, as though each eye functioned independently of the other. Indeed, it has been suggested that such patients may have regressed to a latently present, lower phylogenetic level at which the visual messages from each eye are independently received in the visual cortex.⁴⁷ Tests for retinal correspondence yield confusing results since some patients seem to be unable to relate one afterimage to the other.⁶⁰ Abraham¹ suggested that such patients suffer from a congenital absence of binocular function. However, we have occasionally observed recovery of normal binocular vision after surgical alignment (see also Forrer⁶⁰). We have been unable thus far to find a satisfactory explanation for this phenomenon, but have been impressed by the functional benefit some exotropic patients derive from panoramic vision, as shown in the following case.

CASE 17-2

A 46-year-old mailman servicing a rural mail route came for surgical correction of an exotropia that had been present since childhood. He was concerned about his appearance, but had no visual complaints. His uncorrected visual acuity was 6/6 OU, and he had a constant exotropia of 50^a at near and distance fixation. He strongly preferred OS for fixation. The afterimage test showed suppression of OD. With a dark-red glass the patient indicated homonymous diplopia. After surgical alignment the patient regained peripheral fusion without stereopsis. However, he was most displeased with the result. Be-

fore surgery he had been able to keep his left eye on the road when driving his truck while scanning the mailboxes with his right eye. After surgery he found his field of vision substantially reduced, and it took several months of adjustment before he was able to resume his occupation.

While enlargement of the field of vision may be experienced as a functional advantage by some patients with a large exodeviation, the opposite is true for esotropia where the field of vision is restricted and enlarges after surgery (see p. 333).

Jampolsky⁸⁸ and Pratt-Johnson and Wee¹⁵⁰ showed that suppression in patients with exodeviations may be regional; that is, the scotoma extends from the fovea into the temporal retinal periphery (Jampolsky⁸⁸) or, in the case of alternating constant exotropia, may include the entire temporal and nasal retina of the deviated eye.¹⁵⁰ Campos³⁵ pointed out that the hemianopic suppression scotoma reported by Jampolsky can be found only when putting a dense red filter before the fixating eye. When using a less dissociating method (e.g., Bagolini striated lenses), the scotoma extended well beyond the midline into the nasal retina (Fig. 17–2).

In view of these findings (see also Chapter 13) the concept of “hemiretinal suppression” becomes untenable. The great variations in location and size of suppression scotomas in patients with intermittent or manifest exodeviations was emphasized also in the studies of Herzau⁷⁷ and of Awaya and coworkers.⁴

Stereoacuity deteriorates concomitantly with loss of fusional control in patients who have inter-

mittent exodeviations.¹⁶⁸ Awaya and coworkers⁵ and Ikeyama and Awaya⁸⁵ made the astonishing observation that some patients with exotropia may preserve normal stereoacuity by rapid alternation.

Therapy

As stated on page 358, fundamental differences exist between exodeviations and esodeviations in terms of many clinical characteristics. Since fusion can be restored in a substantial number of patients and since normal binocular function may be present even in the preoperative stage during the exophoria period of intermittent exotropia, the less experienced ophthalmologist is inclined to approach treatment of this condition with brazen optimism—a functional cure seems to be just around the corner. However, as experience grows, it will become apparent to most that treatment of intermittent exotropia may be quite difficult and frustrating. Because functional restoration of binocular vision is apparently within easy reach, a partial or perhaps temporary improvement by surgery followed by subsequent deterioration of binocular function is doubly disappointing to the surgeon and patient.

NONSURGICAL TREATMENT. Therapy is not required for patients who have exophoria without muscular asthenopia. The treatment of symptomatic exophoria and intermittent and constant exodeviations is generally surgical. However, certain nonsurgical measures may be indicated to create optimal sensory conditions before surgery or, when surgery must be postponed, to reinforce fusion during the waiting interval. The functional

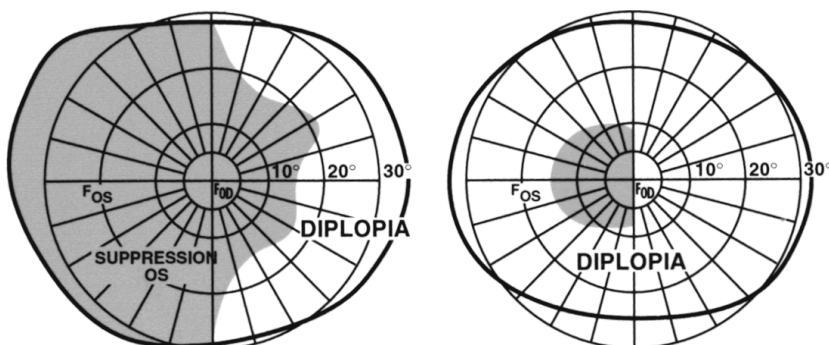


FIGURE 17-2. *Left*, Binocular visual field of a patient with right exotropia of 50 prism diopters. The suppression scotoma in the left eye overrides the midline and extends well into the nasal field when the binocular visual field is tested with a nondissociating technique consisting of a fixation light and striated glasses before both eyes of the patient. *Right*, When plotting the field with a dissociating dark-red filter over the left eye and a striated glass before both eyes, a hemianopic scotoma is detected which must be considered an artifact.

prognosis is poor when constant exotropia occurs in early infancy and when there is no history of intermittency. Preoperative treatment is not required.

CORRECTION OF THE REFRACTIVE ERROR AND THE USE OF MINUS LENSES. Significant refractive errors, especially astigmatism and anisometropic differences, should be corrected in patients with intermittent exodeviations to create sharp retinal images, which in turn increase the stimulus to fuse. Full correction is advisable in myopic patients to maintain active accommodative convergence. Whether hypermetropia should be fully corrected, partially corrected, or corrected at all depends entirely on its degree, the age of the patient, and the AC/A ratio. Since correction of any hypermetropic refractive error will decrease the demand on accommodative convergence and thus increase the exodeviation, each patient should be evaluated on an individual basis. As a rule, we do not correct a hypermetropia of less than +2.00D sph in children with exodeviations. In the older patient, correction of the hypermetropia is usually necessary to avoid refractive asthenopia, even though an underlying exophoria that was previously controlled by accommodative convergence then may become manifest and require therapy.

An exophoric patient with beginning presbyopia presents a special problem. As the accommodative range decreases, the exodeviation will increase and cause symptoms. Before one assesses the increased exodeviation in such patients, it is important to correct any underlying hypermetropia as well as prescribe the weakest bifocal lens that will permit comfortable near vision. If this fails to alleviate the patient's visual discomfort, we prescribe prisms base-in for near vision. Only about half of the exodeviation should be corrected prismatically to stimulate rather than relax accommodative convergence.

If the AC/A ratio is sufficiently high, minus lenses may be used to decrease an exodeviation by stimulating accommodative convergence.¹¹² In younger children with the convergence insufficiency type of exodeviation, minus lenses prescribed as lower segment bifocals may be of functional benefit as a temporizing measure, and in those with the divergence excess type of exodeviation, minus lenses prescribed as upper segment bifocals may be beneficial. Thus normal binocular stimulation can be reinforced while the child is awaiting surgery. Jampolsky^{90, p.150} makes the point

that 3D to 5D of accommodation stimulation with minus lenses is well tolerated by many children. He also observed that patients with orthophoria at near fixation and intermittent exotropia at distance fixation may become exophoric at near fixation under the influence of minus lenses.⁹¹ It is of interest that this initial esophoria is replaced by orthophoria within a matter of weeks. When the minus lenses are removed, an exodeviation may then be present at near fixation, indicating a transient change in the AC/A ratio. Jampolsky believes that surgical alignment of the eyes is facilitated by this change and advocates operating during this period. Caltrider and Jampolsky³⁴ reported that a significant number of patients from a group with intermittent exotropia treated by means of overcorrecting minus lenses manifested improved fusion as well as a decrease in their original deviation. This response persisted for as long as 1 year after therapy was discontinued in 70% of those who showed improvement. We use this form of therapy sparingly and, at most, only as a temporizing measure in patients with a high AC/A ratio. Stimulation of accommodation with minus lenses is tolerated well by younger children, does not cause myopia,¹¹¹ but may cause accommodative asthenopia as the child grows older and the amount of near work increases.⁶⁴

PRISMS. Although most ophthalmologists advocate the use of prisms in the surgically overcorrected exotrope (see consecutive esotropia, p. 372), some use prisms preoperatively to improve fusional control.^{148, 174, 177} Bérard¹¹ corrects one half to one third of the deviation in a preoperative trial to enforce bifoveal stimulation. Ravault and coworkers¹⁵³ claimed that surgery may be avoided in certain patients in whom a satisfactory functional result is obtained by means of full prismatic correction of the deviation, followed by gradual reduction of the prismatic power. Following surgery, Jampolsky⁸⁸ recommended overcorrection of a residual exodeviation with prisms to elicit diplopia and to stimulate fusion (see also Hardesty^{71, 72}). We do not use prisms preoperatively.

ORTHOPTICS. Knapp^{98, p.368} summarized the opinion of most strabismologists by stating that orthoptics should not be used as a substitute for surgery but rather as a supplement. With the exception of energetic preoperative treatment for amblyopia, we rarely use orthoptics before surgery. Even though some authors advocate such therapy, especially for suppression in patients with

intermittent deviations^{42, 83, 117, 160} and anomalous retinal correspondence,⁴⁰ it has not been proved that surgical results are functionally superior to those in patients who have not received orthoptics^{2, 126, 173} or that surgery can be avoided altogether by using this form of treatment. Flynn and coworkers⁵⁹ observed an improved sensory state and better motor control in a group of patients with intermittent exotropia who were treated with alternating occlusion. The dominant eye received occlusion more frequently than the non-dominant eye (see also Knapp⁹⁹ and Iacobucci and Henderson⁸⁴). We have occasionally found it beneficial to use alternating occlusion in lieu of surgery in patients with small angle intermittent exodeviations, as shown in Case 17-3.

CASE 17-3

A 5-year-old girl presented with a history of intermittent exotropia at distance fixation. Measurements showed orthotropia at near and an exophoria of 10^Δ distance fixation. The distance deviation was easily dissociated and the patient did not spontaneously re-fuse. The patch test was negative. An insignificant hypermetropic refractive error was present. Stereopsis at near was 60 seconds of arc on the Titmus test and no stereopsis could be elicited at distance with the Mentor B-VAT. In view of the small deviation at distance we decided against surgery for fear of causing an overcorrection. We ordered alternating occlusion instead and after 3 months the patient fused at near and distance. She had an esophoria of 3^Δ at near and distance. Stereopsis had improved to 15 seconds of arc at near and 60 seconds of arc at distance fixation. After 3 months without treatment the findings were the same.

Intensive orthoptic treatment may be indicated postoperatively when suppression persists or if a convergence insufficiency type of exodeviation is present. Inagaki and coworkers⁸⁶ reported abolishment of suppression and development of sensory and motor fusion by treatment consisting of preoperative simultaneous bifoveal stimulation with a checkerboard pattern in patients with constant and intermittent exotropia.

Surgical Treatment

INDICATIONS FOR SURGERY. The need for surgery is determined by the state of fusional control, the angle size of deviation, and the age of the patient. In patients with *manifest exotropia* present

at or shortly after birth with no history of intermittency, surgery should be performed as soon as reliable and constant measurements can be obtained, the patient can alternate freely, and the angle of deviation measures at least 15^Δ. We usually operate on such children when they are between 1 and 2 years of age. In adults with a large angle constant exodeviation, surgery is performed as soon as the diagnosis has been established. The prognosis for return of normal binocular function in such cases is poor when the deviation has been present since early childhood. Such patients usually retain a residual small angle exotropia with alternating fixation. However, exceptions to this rule do occur, and we have seen several patients who had a history of manifest exotropia for many years in whom unexpected and fortuitous return of normal binocular vision with stereopsis occurred after surgical alignment of the eyes (see also Ball and coworkers⁹).

Surgical treatment of *intermittent exotropia* or of constant exotropia preceded by a long period of intermittency is directed at normalization of binocular function. There is good evidence for an improvement of distance stereoacuity after surgery.^{144, 186} Unless there is definite evidence of existing defective binocular vision, surgery should be preceded by several months of observation since the disease does not progress in all patients^{68, 128, 135} (see p. 359). Signs of progression include gradual loss of fusional control as evidenced by increasing frequency of the manifest phase of the strabismus. A patient whose eyes turn outward only occasionally and who is asymptomatic does not need surgery. However, when the exotropia occurs during more than 50% of waking hours or causes asthenopic problems, surgery should be performed. Other signs of progression are development of a secondary convergence insufficiency, an increase in size of the basic deviation, development of suppression as evidenced by absence of diplopia during the manifest phase of the strabismus, or decrease of stereoacuity. If one or several of these signs or symptoms are present when the patient is first examined or if they develop while the patient is under observation, surgery must be considered. An asthenopic patient with exophoria may require surgery if the deviation cannot be controlled with prisms.

The most desirable age at which surgery should be performed for intermittent exodeviations has been a matter of some dispute. Jampolsky⁹⁰ prefers to delay surgery in visually immature infants to

avoid overcorrection. In the interval, he reinforces fusion with minus lenses or prevents development of suppression by means of alternating occlusion. On the other hand, Knapp⁹⁸ is an advocate of early surgery for treatment of intermittent exotropia, a view shared by other authors.^{3, 50, 146, 151, 156} More recently, however, Baker and coworkers⁸ found in a comparative study that patients operated on after the age of 4 years had better functional results.

We prefer to delay surgical intervention for *intermittent* exotropia in young children, since we share Jampolsky's concern about the effects of a consecutive esotropia in a visually immature child. Unfortunately, in such patients, good preoperative visual acuity in each eye with normal stereoacuity may have been exchanged for persistent monocular esotropia with amblyopia, loss of stereopsis, and the development of anomalous retinal correspondence. Edelman and coworkers⁵² reported that 5 of 24 children who developed consecutive esotropia after surgery for exotropia before the age of 4 years became amblyopic. Even when surgery was delayed until the age of 4 to 6 years, amblyopia still occurred in 3 of 39 patients.

Although the prevalence of consecutive constant esotropia in patients under 5 years of age has been reported to be only 10%,^{96, 120, 151} we have witnessed this unfortunate event in a sufficient number of our patients to advocate delaying surgery until the child has reached at least 4 years of age. In the interim, binocular vision should be reinforced with prisms base-in or minus lenses. Surgery at an earlier age should be considered only if there is a rapid functional deterioration of fusional control in spite of nonsurgical therapy or if the deviation is constant.

Finally, the size of the deviation determines the decision to operate. The angle of primary exodeviations generally exceeds 20^Δ, and unlike the situation in esotropia, small angle exodeviations are rare. If for functional reasons surgery is indicated, the deviation should measure at least 15^Δ at distance or near fixation before a procedure is carried out. Patients are seldom self-conscious or embarrassed by strabismus of this magnitude and surgery usually is not performed unless the deviation measures at least 20^Δ to 25^Δ.

GOALS OF SURGERY. Although the aim of most operations for strabismus is to align the eyes as nearly as possible, many ophthalmologists have proposed that for intermittent exodeviations a small surgical overcorrection is desirable, since it

appears that functional results will be more stable.^{12; 41; 53; 64; 81, p. 645; 90; 98; 152; 154; 164; 185} Raab and Parks¹⁵² proposed that the surgeon should strive for an overcorrection of 10^Δ to 20^Δ. Lesser degrees of overcorrection have been associated with recurrence of the exodeviations after some time has elapsed. A higher degree of overcorrection will necessitate further surgery for consecutive esotropia. Proponents of deliberate overcorrection cite the therapeutic value of postoperative diplopia in stimulating development of fusional vergences and thus in stabilizing eventual alignment of the eyes. On the other hand, it has also been shown that an initial overcorrection does not guarantee a desirable final outcome.¹⁵⁹ Dunlap⁵¹ observed that the difficult element in striving for overcorrection is knowing how to produce some, but not too much of it. In one series of his patients the prevalence of unintended overcorrection was 40%, whereas Cooper,⁴¹ who deliberately attempted to overcorrect his patients, reported a prevalence of only 37%. Since introduction of adjustable sutures this unpredictability has become less of a problem.

We are in agreement with those who believe that a small angle of consecutive esotropia in the immediate postoperative phase is desirable and tends to stabilize a functional result, even though such deviations occasionally may persist for a long time and cause problems of management (see Case 17-4). However, we have been unable to accomplish this goal other than by pure chance or by postoperative suture adjustment.

As pointed out above, surgical overcorrection, as beneficial as it may be in the older child or adult, must be avoided under all circumstances in visually immature children in view of the disastrous consequences of a small angle esotropia in this age group. On the other hand, Schlossman and coworkers¹⁶² concluded from their data that adult patients do better with slight undercorrection rather than overcorrection after surgery, provided the residual exodeviation remains under 15^Δ.

Although it may be difficult or even impossible to plan surgery to achieve a small, beneficial amount of overcorrection, there may be ways to avoid overcorrection in cases in which it is undesirable. The importance of including lateral gaze incomitance into the surgical planning has been mentioned.

CHOICE OF PROCEDURE. Burian^{26, 31} emphasized that correct differentiation between the true and simulated divergence excess patterns is essential

for proper choice of surgical procedures. We have followed his suggestions and still advocate for exotropia of the *true divergence excess type* a recession of both lateral rectus muscles and for *basic exotropia* or the *simulated divergence excess pattern* a recession of the lateral rectus muscle with resection of the ipsilateral medial rectus muscle on the nondominant eye. This approach has worked well in our hands.¹³⁶ However, a recent study by Kushner¹⁰⁸ has shown that bilateral lateral rectus recessions may be equally effective in simulated divergence excess and in basic exotropia. Others have reported that there are no differences between the results of asymmetrical (recession of the lateral rectus muscle and resection of the medial rectus muscle of one eye) and symmetrical (recession of both lateral rectus muscles) surgery in intermittent exotropia even though the immediate postoperative results seemed better with asymmetrical surgery.¹⁸⁷ Some authors¹¹⁴ prefer a resection of both medial rectus muscles for most forms of exotropia. Others have reported satisfactory results after recession of only one lateral rectus muscle.^{56, 133, 143, 176} We reserve this procedure for patients with a dissociated exodeviation (see Chapter 18).

Further long-term prospective studies comparing these different surgical methods in the treatment of the various manifestations of exotropia are necessary to define the real advantages of one procedure over the other. Until the time that such data become available we see no reason to deviate from Burian's recommendations, which have served us well thus far.

For an exodeviation larger at near than at distance fixation (*convergence insufficiency type*) we resect both medial rectus muscles, a procedure that is often followed by a temporary overcorrection of which the patient must be apprised. The amount of resection ranges from 3 to 6 mm, depending on the size of the deviation. Others have proposed asymmetrical surgery for this condition, placing the emphasis of the operation on the resection of one medial rectus and performing lesser amounts of recession on its antagonist, the lateral rectus muscle.¹⁰⁴ This method has been reported to collapse the near-distance differences and as having a low risk of postoperative diplopia. A different approach was taken by Snir and coworkers,¹⁶⁶ who use slanted recessions of the lateral rectus muscles in patients with an exodeviation greater at distance than at near. The upper edge of the muscle insertion was recessed according to the distance exode-

viation and the lower edge according to the near deviation. These authors claim that this procedure is superior to standard recessions in reducing the exodeviation at distance and near fixation and in collapsing the difference between them.

A special surgical approach has been proposed for *intermittent exotropia with a high AC/A ratio*.²¹ It consists of a bilateral lateral rectus muscle recession combined with a posterior fixation suture on both medial rectus muscles. Although theoretically interesting, this procedure may not be without its risks in the long term for patients with normal binocularity.

Weakening procedures on all four oblique muscles, which are frequently found to be apparently overacting in large angle exotropia have been advocated⁹² but are not used by us. We find that such overaction of all oblique muscles is not a true overaction and often disappears after treating the exotropia by conventional surgery of the horizontal rectus muscles.

In the case of asymmetrical surgery we prefer to operate on the nondominant eye. It has been suggested by Mitsui and coworkers¹²⁵ that better surgical results are obtained when the operation is done on the dominant eye. However, Lennerstrand¹¹⁶ was unable to confirm the superiority of this over the conventional approach to do surgery on the nondominant eye or on both lateral rectus muscles.

An adult patient with a *large angle exotropia* of an amblyopic eye may require special management. Rayner and Jampolsky¹⁵⁴ recommended surgery on the amblyopic eye consisting of recession of the lateral rectus muscle to the equator, recession or T-closure of the temporal conjunctiva to release its restrictions, and maximal resection of the medial rectus muscle up to 14 mm to hold the eye in alignment. According to these authors, the disadvantage of postoperative limitation of abduction, created by the excessive amount of resection of the medial rectus muscle, may be viewed as an advantage in such cases since it prevents recurrence of the deviation.

Surgery on one eye consisting of recession of the lateral rectus muscle and resection of the medial rectus muscle has been supplemented with intraoperative injection of 10 units of botulinum toxin, type A (Botox) into the lateral rectus muscle.¹⁴⁵ More cases and longer follow-up are required before this procedure can be recommended. Botulinum toxin injection into the lateral rectus muscles has also been advocated as an alternative

to surgery in intermittent exotropia.¹⁶⁷ The long-term stability of the results seem questionable to us.

For reasons that are discussed further in Chapter 26, we are not convinced of the values of dose-response curves and tables. However, provided there is no incomitance in lateral gaze and visual acuity is equal in both eyes, we use amounts of recession of the lateral rectus muscles that are similar to those used by other strabismologists here and abroad^{12, 147, 157} (Table 17-1). In large angle exotropia (greater than 50^Δ) it may be necessary to recess both lateral rectus muscles maximally and resect one or both medial rectus muscles in one session.^{6, 13, 68, 89, 171} The *prism adaptation test* (PAT) is of little help in deciding how much surgery to do to each muscle for exodeviations since no differences in surgical results were found between responders to this test, in whom the surgical dosage was increased, and non-responders.¹⁴²

We have found *adjustable sutures* helpful in patients with large angle exotropia but rarely use them in intermittent exotropia. In this condition motor fusion may tend to mask a residual deviation during adjustment. The result is a patient with surgical undercorrection who would have benefited from a postoperative adjustment. Intraoperative adjustment has also been suggested.³³ However, we believe that the eye position under general anesthesia is too variable to rely on this information for modification of the original surgical plan.

RESULTS OF SURGERY. Surgical results, in terms of restoration of binocular function and conversion of a deviation from constant heterotropia to heterophoria, vary according to the binocular state before surgery. Table 17-2 lists results reported

TABLE 17-2. Effect of Surgery on Binocular Function in Exodeviations

Authors	n	Satisfactory Results* (%)
Beneish & Flanders ¹⁰	67	60
Burian & Spivey ³¹	200	40
Hardesty et al ⁷⁵	100	78
von Noorden ¹³⁶	49	77
Pratt-Johnson & Wee ¹⁵⁰	100	41
Raab & Parks ¹⁵²	145	52
Richard & Parks ¹⁵⁶	111	95
Windsor ¹⁸¹	115	58
Winter et al ¹⁸²	85	82

*Defined by most authors listed as fusion at near and distance.

by different authors. The variance of success rates shown in the table can be explained by different lengths of follow-up and criteria used for a cure. We define cure as restoration of stable fusion at near and distance fixation in an asymptomatic patient. As may be expected, in patients without suppression and preoperative diplopia in whom strabismus is manifest only occasionally, the prognosis is better than in those with constant exotropia of long duration. Although it is sufficient for practical purposes to define a cure as reestablishment of fusion, it is of interest and underlines the complex nature of exodeviations that more refined testing will reveal minor defects of normal binocular vision in a high percentage of patients with intermittent exodeviations after treatment. Baker and Davies⁷ reported defective stereopsis in most of their patients before and after surgical alignment of the eyes. Stimulated by this report, Haase and de Decker⁶⁹ studied 156 patients with intermittent exotropia in whom a wide array of sensory tests had been performed. Their findings are astonishing indeed. Microexotropia occurred in 32%, subnormal binocular vision (see Chapter 16) in 50%, and a complete sensory cure in only 17% of their patients.

Since becoming aware of these studies, we have reexamined a group of patients who were orthotropic or slightly esophoric at 33 cm and 6 m fixation distances and had formerly been classified as surgical cures. The examination involved fixation maintained on a red light at the end of a 25-m-long corridor.¹³⁹ In most instances a small constant exotropia was present under these circumstances, a finding that is incompatible with a complete cure. These observations have reinforced our opinion that complete restoration of normal

TABLE 17-1. Surgical Dosage of Recession of Both Lateral Rectus Muscles*

Deviation (Δ)	Recession (mm)
15	4
20	5
25	6
30	7
35	7
40	8
≥50	7 + Resection of one medial rectus

*Modified from Parks MM, Mitchell P: Concomitant exodeviations. In Duane TD ed: *Clinical Ophthalmology*, Vol 1. Philadelphia, JB Lippincott, 1988, p 1.

and stable binocular vision in patients with intermittent exotropia presents a major challenge and that the results of treatment are often frustrating for the ophthalmologist.

In conclusion, exotropia is a condition which can be improved and, in many instances, controlled by surgery. However, the prognosis for a long-term cure must be guarded since recurrences are common. In this respect the condition is quite different from normosensorial esotropia where timely diagnosis and treatment may result in a complete and permanent cure.

MANAGEMENT OF UNDERCORRECTIONS.

Most patients with persistent intermittent exotropia require additional surgery. The residual deviation is apparent in some of them immediately after the operation. In others it does not appear until months or even years after an initially satisfactory result. Use of base-in membrane prisms of a power greater than the residual deviation has been advocated to provoke convergence and thus lessen the exodeviation.^{73, 74, 101, 129} Hardesty and coworkers⁷⁵ emphasized the need to restore fusion with prisms that equal the deviation as a means of improving fusional amplitudes before the second operation.

MANAGEMENT OF OVERCORRECTIONS (CONSECUTIVE ESOTROPIA).

The reported prevalence of surgical overcorrections in patients with exodeviations varies according to different authors (6%,⁷⁵ 8%,⁵⁸ 10%,³¹ 11%,¹³⁶ 17%,¹¹⁵ and 20%⁵¹). A large overcorrection with gross limitation of ocular motility of the surgically treated eye on the day after surgery may require immediate surgical intervention. Mechanical factors, such as excessive resection of the medial rectus or disinsertion of the lateral rectus, may be responsible for causing this complication, which is discussed further in Chapter 26.

Therapy for smaller degrees of esotropia, which are usually comitant in nature, is one of watchful waiting. A postoperative esodeviation of 10^Δ to 15^Δ may disappear completely with time and is, as stated above, desirable, but larger deviations tend to increase. In any case, a second operation should not be performed until at least 6 months have elapsed, except when there are significant limitations of ductions that cause incomitance in lateral gaze.¹²¹ During the waiting period several nonsurgical therapeutic measures may be carried out that will decrease the postoperative deviation

or, if this is not possible, maintain fusion and keep the patient comfortable.

No therapy is advocated for the first 2 weeks after surgery for small degrees of overcorrection. Should diplopia persist after this time, miotics or a temporary prescription for a hypermetropic refractive error may decrease the deviation to the point where the patient will fuse. Patients with a high AC/A ratio will respond well to slight overcorrection of the hypermetropic refractive error; if the deviation is larger at near fixation, the prescription of additional plus lenses in the form of bifocals may be beneficial.

If this therapy is unsuccessful, alternating occlusion not only will eliminate diplopia but also will tend to decrease the angle of the consecutive esotropia. A great deal of patience is required by the physician in treating persistent consecutive esodeviations since spontaneous reduction of the postoperative angle may require a considerable length of time, as illustrated by case 17-4.

CASE 17-4. Age: 9 years

August 1964

Distance: 35^Δ XT

Near: 18^Δ X(T)

Visual acuity: OD 6/7.5

OS 6/7.5

Refraction: OD - 1.50 D sph + 0.50 cyl ax 180°

OS - 1.50 D sph + 0.62 cyl ax 180°

After 1 hour of occlusion: 50^Δ XT at near

Diagnosis: simulated divergence excess

October 1964

Operation: 7 mm recession lateral rectus muscle
OU

December 1964

Distance: 6^Δ ET

Near: 7^Δ ET

Complains about uncrossed diplopia at distance
and near fixation

Rx: 0.125% echthiophate iodide (Phospholine
Iodide) OU every other night

June 1965

Distance: 5^Δ ET

Near: 8^Δ E(T)

Complains about diplopia or distance fixation
interfering with school work

November 1965

Measurements unchanged

Still has diplopia at distance

Rx: discontinue miotics

Start alternating occlusion (OD one day, OS one
day)

December 1966

Distance: 2^Δ exophoria

Near: 4^Δ exophoria
 Rx: Discontinue alternating occlusion
 April 1967
 Distance: orthophoria
 Near: orthophoria
 Stereoacuity: 80 seconds of arc

When fusion must be maintained under all circumstances, as in visually immature children or for occupational reasons in adults, prisms base-out are the preferable treatment.^{72, 115} Press-on Fresnel membrane prisms have eliminated many difficulties previously encountered with this form of therapy. Frequent adjustments to adapt the prismatic correction to the changing postoperative angle can now be made at nominal cost to the patient. Hardesty and coworkers⁷⁵ reported that a consecutive esotropia of less than 15^Δ can be cured with prism therapy alone, whereas surgery usually becomes necessary for larger esodeviations. Our criteria for reoperation depend on the following factors: nonacceptance of conservative treatment by a patient, lack of improvement of the basic deviation in spite of prisms, increase of the deviation in spite of prisms, persistence of diplopia because of incomitance, and limitation of ductions.¹²¹ In fact, persistent limitation of ductions during the postoperative period in an overcorrected patient mitigates against delay of reoperation for a 6-month period, since, for example, a surgical overcorrection caused by a tight medial rectus or excessively recessed lateral rectus muscle does not improve with time. Botulinum toxin injections in the medial rectus muscle have also been shown to be effective in treating consecutive esotropia in patients with retained motor fusion.⁴⁶

Dissociated Exodeviations

Dissociated exodeviations are discussed together with dissociated vertical deviations in Chapter 18.

Secondary Exodeviations

Sensory Exotropia

Sensory exotropia occurs as a result of primary sensory deficit such as anisometropia, unilateral aphakia, and unilateral visual impairment brought about by organic causes, followed by partial or complete disruption of fusion. Recently, vitreous hemorrhage has been reported as a cause of sen-

sory exotropia.⁶³ Development of a sensory exotropia or esotropia under these circumstances is discussed on page 346. Characteristically, the deviation is unilateral and involves the amblyopic eye. Surgery is usually required to restore normal facial configuration; surgical management is discussed on page 369.

Consecutive Exotropia

Consecutive exotropia arises either spontaneously in a formerly esotropic patient or iatrogenically after surgical overcorrection. Spontaneous change from esotropia to exotropia usually can be associated with poor vision of the deviating eye (sensory exotropia), even though all cases cannot be explained on this basis. High hypermetropia in an esotropic patient may be another contributing factor since consecutive exotropia is not uncommon in this group of patients (see Chapter 16). Treatment is surgical and indications for surgery are cosmetic.

Consecutive exotropia after surgical overcorrection of an esodeviation is discussed in Chapter 16.

REFERENCES

1. Abraham SV: Nonparalytic Strabismus, Amblyopia and Heterophoria. Los Angeles, Pan American, 1966, p 155.
2. Altizer LB: The nonsurgical treatment of exotropia. *Am Orthopt J* 22:71, 1972.
3. Asbury T: The role of orthoptics in the evaluation and treatment of intermittent esotropia. In Arruga A, ed: *International Strabismus Symposium*, University of Gies-sen, Germany 1966. Basel, S Karger, 1968, p 331.
4. Awaya S, Nozaki H, Itoh T, Hanada K: Studies of suppression in alternating constant exotropia and intermittent exotropia with reference to fusional background. In Moore S, Mein J, Stockbridge L, eds: *Orthoptics: Past, Present, Future*. Miami, Symposia Specialists, 1976, p 531.
5. Awaya S, Sugawara M, Komiyama K, Ikeyama K: Studies on stereoacuity in four constant exotropes with good stereoacuity, with a special reference to the Titmus stereo test and EOG analysis. *Acta Soc Ophthalmol Jpn* 83:425, 1979.
6. Azar RF: Surgical management of exotropia exceeding 70 prism diopters. *Ann Ophthalmol* 3:159, 1971.
7. Baker JD, Davies GT: Monofixational intermittent esotropia. *Arch Ophthalmol* 97:93, 1979.
8. Baker JD, Schweers M, Petrunak J: Is earlier surgery a sensory benefit in the treatment of intermittent exotropia? In Lennerstrand G, ed: *Advances in Strabismology*. Proceedings of the Eighth Meeting of the International Strabismological Association, Maastricht, Dept. 10-12, 1998. Buren, The Netherlands, Aeolus Press, 1999, p 289.
9. Ball A, Drummond GT, Pearce WG: Unexpected stereoacuity following surgical correction of long-standing horizontal strabismus. *Can J Ophthalmol* 28:217, 1993.
10. Beneish R, Flanders M: The role of stereopsis and early postoperative alignment in long-term results of intermittent exotropia. *Can J Ophthalmol* 29:119, 1994.

11. Bérard PV: Prisms—their therapeutic use in concomitant strabismus with normal correspondence. In Feels P, ed: First International Congress of Orthoptists. St Louis, Mosby—Year Book, 1968, p 77.
12. Berland JE, Wilson ME, Saunders RA: Results of large (8–9 mm) bilateral lateral rectus muscle recession for exotropia. *Binocular Vision Strabismus Q* 13:97, 1998.
13. Berke R: Principles, technique and complications of horizontal nonparalytic nonaccommodative strabismus. In *Strabismus. Symposium of the New Orleans Academy of Ophthalmology*. St Louis, Mosby—Year Book, 1962, p 178.
14. Bielschowsky A: Divergence excess. *Arch Ophthalmol* 12:157, 1934.
15. Bielschowsky A: Exophorie und “Divergenzexzess.” *Klin Monatsbl Augenheilkd* 92:11, 1934.
16. Biglan AW, Davis JS, Cheng KP, Pettapiece MC: Infantile exotropia. *J Pediatr Ophthalmol Strabismus* 33:79, 1996.
17. Binder HF, Arndt CL: Binocularity in anomalous retinal correspondence. *Acta Ophthalmol* 41:653, 1963.
18. Blodi FC, Van Allen M: Electromyography in intermittent esotropia; recordings before, during and after corrective operation. *Doc Ophthalmol* 26:21, 1962.
19. Breinin GM: The nature of vergence revealed by electromyography. *Arch Ophthalmol* 58:623, 1957.
20. Breinin GM, Moldaver J: Electromyography of the human extraocular muscles. *Arch Ophthalmol* 54:200, 1955.
21. Brodsky MC, Fray KJ: Surgical management of intermittent exotropia with high AC/A ratio. *J Am Assoc Pediatr Ophthalmol Strabismus* 2:330, 1998.
22. Brown HW: Aids in the diagnosis of strabismus. In *Strabismus. Symposium of the New Orleans Academy of Ophthalmology*. St Louis, Mosby—Year Book, 1962, p 231.
23. Brown HW: Discussion of paper by Burian HM, Franceschetti AT: Evaluation of diagnostic methods for the classification of exodeviations. *Trans Am Ophthalmol Soc* 68:56, 1970.
24. Burian HM: Intermittent (facultative) divergent strabismus. Its influence on visual acuity and the binocular visual act. *Am J Ophthalmol* 28:525, 1945.
25. Burian HM: The sensorial retinal relationship in comitant strabismus. *Arch Ophthalmol* 37:336,504,648, 1947.
26. Burian HM: Selected problems in the diagnosis and treatment of the neuromuscular anomalies of the eyes. In *II Curso Internacional de Oftalmología*. Barcelona, Publicaciones del Instituto Barranquer, 1958, p 25.
27. Burian HM: Exodeviations: Their classification, diagnosis, and treatment. *Am J Ophthalmol* 62:1161, 1966.
28. Burian HM: Pathophysiology of exodeviations. In Manley DR, ed: *Symposium on Horizontal Ocular Deviations*. St Louis, Mosby—Year Book, 1971, p 119.
29. Burian HM, Franceschetti AT: Evaluation of diagnostic methods for the classification of exodeviations. *Trans Am Ophthalmol Soc* 68:56, 1970.
30. Burian HM, Smith DR: Comparative measurement of exodeviations at twenty and one hundred feet. *Trans Am Ophthalmol Soc* 69:188, 1971.
31. Burian HM, Spivey BE: The surgical management of exodeviations. *Am J Ophthalmol* 59:603, 1965.
32. Caldeira JA: Lateral gaze incomitance in surgical exodeviations: Clinical features. *Binocular Vision Strabismus Q* 7:75, 1992.
33. Caldeira JAF: Stage I intraoperative adjustment of eye muscle surgery for exodeviations based on eye position during general anesthesia: A prospective study of 35 patients. *Binocular Vision Strabismus Q* 12:253, 1997.
34. Caltrider N, Jampolsky A: Overcorrecting minus lens therapy for treatment of intermittent exotropia. *Ophthalmology* 90:1160, 1983.
35. Campos EC: Binocularity in comitant strabismus: Binocular visual field studies. *Doc Ophthalmol* 53:249, 1982.
36. Campos EC, Cipolli C: Binocularity and photophobia in intermittent exotropia. *Percept Mot Skills* 74:1168, 1992.
37. Cass EE: Divergent strabismus. *Br J Ophthalmol* 21:538, 1937.
38. Chamberlain W: Discussion of paper by Burian HM, Franceschetti AT: Evaluation of diagnostic methods for the classification of exodeviations. *Trans Am Ophthalmol Soc* 68:56, 1970.
39. Chavasse FE: *Worth’s Squint or the Binocular Reflexes and the Treatment of Strabismus*. London, Baillière, Tindall & Cox, 1939.
40. Ciancia AO, Melek N: A new treatment of anomalous retinal correspondence in intermittent esotropia. In Fells P, ed: *The First Congress of the International Strabismological Association*. St Louis, Mosby—Year Book, 1971, p 106.
41. Cooper EL: Purposeful overcorrection in exotropia. In Arruga A, ed: *International Strabismus Symposium*, University of Giessen, Germany, 1966. Basel, S Karger, 1968, p 311.
42. Cooper EL, Leyman IA: The management of intermittent exotropia. A comparison of the results of surgical and nonsurgical treatment. In Moore S, Mein J, Stockbridge L, eds: *Orthoptics: Past, Present, Future*. Transactions of the Third International Orthoptic Congress, New York, Stratton Intercontinental, 1976, p 563.
43. Chew E, Remaley NA, Tamboli A, et al: Risk factors for esotropia and exotropia. *Arch Ophthalmol* 112:1349, 1994.
44. Costenbader FD: The physiology and management of divergent strabismus. In Allen JH, ed: *Strabismic Ophthalmic Symposium I*. St Louis, Mosby—Year Book, 1950.
45. Dannheim E: *Panoramasehen bei Strabismus divergens*. *Orthopt Pleopt* 6:17, 1978.
46. Dawson ELM, Marshman WE, Lee JP: Role of botulinum toxin A in surgically overcorrected exotropia. *J Am Assoc Pediatr Ophthalmol Strabismus* 3:269, 1999.
47. de Decker W: Heterotropie (manifeste Strabismus). In Kaufmann H, ed: *Strabismus*. Stuttgart, Ferdinand Enke Verlag, 1986, p 139.
48. Donders FC: An essay on the nature and the consequences of anomalies of refraction. Oliver CA, ed: Philadelphia, P Blakiston’s Son & Co, 1899, p 59.
49. Duane A: A new classification of the motor anomalies of the eyes based upon physiological principles, together with their symptoms, diagnosis and treatment. *Ann Ophthalmol Otolaryngol* 5:969, 1896; 6:84,247, 1897.
50. Dunlap EA: Overcorrections in esotropia surgery. In Arruga A, ed: *International Strabismus Symposium*, University of Giessen, Germany, 1966. Basel, S Karger, 1968, p 319.
51. Dunlap EA: Overcorrections in horizontal strabismus surgery. In *Symposium on Strabismus*. Transactions of the New Orleans Academy of Ophthalmology. St Louis, Mosby—Year Book, 1971, p 255.
52. Edelman PM, Brown MH, Murphree AL, Wright KW: Consecutive esotropia . . . then what? *Am Orthopt J* 38:111, 1988.
53. Eino D, Kraft SP: Postoperative drifts after adjustable-suture strabismus surgery. *Can J Ophthalmol* 32:163, 1997.
54. Eustace P, Wesson ME, Druby DJ: The effect of illumination on intermittent divergent squint of the divergence excess type. *Trans Ophthalmol Soc UK* 93:559, 1973.
55. Fahle M: Naso-temporal asymmetry of binocular inhibition. *Invest Ophthalmol Vis Sci* 28:1016, 1987.
56. Feretis D, Mela E, Vasilopoulos G: Excessive single lateral rectus muscle recession in the treatment of inter-

- mittent exotropia. *J Pediatr Ophthalmol Strabismus* 27:315, 1990.
57. Fletcher M: Natural history of idiopathic strabismus. In Symposium on Strabismus. Transactions of the New Orleans Academy of Ophthalmology. St Louis, Mosby-Year Book, 1971, p 15.
 58. Fletcher MC, Silverman SJ: Strabismus. I. A summary of 1110 consecutive cases. *Am J Ophthalmol* 61:86, 1966.
 59. Flynn JT, McKenney S, Rosenhouse M: Eine Behandlungsform des intermittierenden Begleitschielens. *Klin Monatsbl Augenheilkd* 167:185, 1975.
 60. Forrer HT: Retinal correspondence in intermittent exotropia. In Moore S, Mein J, Stockbridge L, eds: *Orthoptics: Past, Present, Future*, Transactions of the Third International Orthoptic Congress. New York, Stratton Intercontinental, 1976, p 547.
 61. Frandsen AD: Occurrence of squint. A clinical-statistical study on the prevalence of squint and associated signs in different groups and ages of the Danish population. *Acta Ophthalmol (kbb) Suppl* 62:1, 1960.
 62. Friedman Z, Neumann E, Hyams SW, Peleg B: Ophthalmic screening of 38,000 children, age 1 to 2½ years, in child welfare clinics. *J Pediatr Ophthalmol Strabismus* 17:261, 1980.
 63. Fujikado T, Ohmi G, Ikeda T, et al: Exotropia secondary to vitreous hemorrhage. *Graefes Arch Clin Exp Ophthalmol* 235:143, 1997.
 64. Graefe A von: Über musculäre Asthenopie. *Graefes Arch Clin Exp Ophthalmol* 8:314, 1862.
 65. Graefe E: Messungen von Heterophorien unter verschiedenen Umfeldleuchtdichten. Dissertation, University Eye Clinic, Hamburg-Eppendorf, Hamburg, 1967.
 66. Graham PA: Epidemiology of strabismus. *Br J Ophthalmol* 58:224, 1974.
 67. Gregersen E: The polymorphous exo patient. Analysis of 231 consecutive cases. *Acta Ophthalmol* 47:579, 1969.
 68. Guibor G: The surgical treatment of exotropia resulting from anterior internuclear ophthalmoplegia. *Am J Ophthalmol* 33:1840, 1950.
 69. Haase W, Decker W de: Binokulare sensorische Defekte beim Strabismus divergens intermittens. *Klin Monatsbl Augenheilkd* 179:81, 1981.
 70. Hall IB: Primary divergent strabismus. Analysis of aetiological factors. *Br Orthopt J* 18:106, 1961.
 71. Hardesty HH: Treatment of recurrent intermittent exotropia. *Am J Ophthalmol* 60:1036, 1965.
 72. Hardesty HH: Treatment of overcorrected intermittent exotropia. *Am J Ophthalmol* 66:80, 1968.
 73. Hardesty HH: Treatment of under- and over-corrected intermittent exotropia with prism glasses. *Am Orthopt J* 19:110, 1969.
 74. Hardesty HH: Prisms in the management of intermittent exotropia. *Am Orthopt J* 22:22, 1972.
 75. Hardesty HH, Boynton JR, Keenan JP: Treatment of intermittent exotropia. *Arch Ophthalmol* 96:268, 1978.
 76. Helveston EM: The use and "abuse" of the +3.00 D lenses (editorial). *J Pediatr Ophthalmol Strabismus* 11:175, 1974.
 77. Herzau V: Untersuchungen über das binokulare Gesichtsfeld Schielender. *Doc Ophthalmol* 49:221, 1980.
 78. Hiles DA, Davies GT, Costenbader FD: Long-term observations on unoperated intermittent exotropia. *Arch Ophthalmol* 80:436, 1968.
 79. Holland G: Häufigkeit und Vorkommen der anomalen Netzhautkorrespondenz. *Graefes Arch Clin Exp Ophthalmol* 166:559, 1964.
 80. Holland G: Über Zeitpunkt und Ursache des frühkindlichen Schielens. *Klin Monatsbl Augenheilkd* 147:498, 1965.
 81. Hugonnier R, Crayette-Hugonnier S: Strabismus, Hetero-
 - phoria, Ocular Motor Paralysis. *Clinical Ocular Muscle Imbalance*. Translated and edited by Véronneau-Troutman S. St Louis, Mosby-Year Book, 1969, p 201.
 82. Hunter DG, Ellis FJ: Prevalence of systemic and ocular disease in infantile exotropia. *Ophthalmology* 106:1951, 1999.
 83. Hurtt J: The selection of cases for orthoptics and pleoptics in the United States. In *First International Congress of Orthoptists*. St Louis, Mosby-Year Book, 1968, p 83.
 84. Iacobucci I, Henderson JW: Occlusion in the preoperative treatment of exodeviations. *Am Orthopt J* 15:42, 1965.
 85. Ikeyama K, Awaya S: Studies on stereoacuity by the Titmus stereotest with alternate presentation to each eye through the liquid crystal shutter. *Acta Soc Ophthalmol Jpn* 83:2021, 1979.
 86. Inagaki Y, Awaya S, Yagasaki T, Sato M: Effect of the checkerboard pattern stimulator on sensory fusion in exotropia. *Folia Ophthalmol Jpn* 44:825, 1993.
 87. Ing MR, Stephan W, Pang L: The racial distribution of strabismus. In Reinecke RD, ed: *Strabismus*. Third Congress of the International Strabismological Association, Kyoto, Japan. New York, Grune & Stratton, 1978, p 107.
 88. Jampolsky A: Characteristics of suppression in strabismus. *Arch Ophthalmol* 54:683, 1955.
 89. Jampolsky A: Surgical management of exotropia. *Am J Ophthalmol* 46:646, 1958.
 90. Jampolsky A: Management of exodeviations. In *Strabismus*. Symposium of the New Orleans Academy of Ophthalmology. St Louis, Mosby-Year Book, 1962.
 91. Jampolsky A: Ocular deviations. *Int Ophthalmol Clin* 4:567, 1964.
 92. Jampolsky A: Ocular divergence mechanisms. *Trans Am Ophthalmol Soc* 65:730, 1970.
 93. Jampolsky A, Flom BC, Weymouth FS, Moses LE: Unequal corrected visual acuity as related to anisometropia. *Arch Ophthalmol* 54:893, 1955.
 94. Jenkins R: Demographics: Geographic variations in the prevalence and management of exotropia. *Am Orthopt J* 42:82, 1992.
 95. Joose MV, Simonsz HJ, van Minderhout EM, et al: Quantitative visual fields under binocular viewing conditions in primary and consecutive divergent strabismus. *Graefes Arch Clin Exp Ophthalmol* 237:535, 1999.
 96. Keech RV, Stewart SA: The surgical overcorrection of intermittent exotropia. *J Pediatr Ophthalmol Strabismus* 27:218, 1990.
 97. Kii T, Nakagawa T: Natural history of intermittent exotropia—statistical study of preoperative strabismic angle in different age groups. *Acta Soc Ophthalmol Jpn* 96:904, 1992.
 98. Knapp P: Divergent deviations. In Allen JH, ed: *Strabismic Ophthalmic Symposium II*. St Louis, Mosby-Year Book, 1958, p 354.
 99. Knapp P: Treatment of divergent deviations. In Allen JH, ed: *Strabismic Ophthalmic Symposium II*. St Louis, Mosby-Year Book, 1958, p 364.
 100. Knapp P: Management of exotropia. In Symposium on Strabismus. Transactions of the New Orleans Academy of Ophthalmology. St Louis, Mosby-Year Book, 1971, p 233.
 101. Knapp P: Use of membrane prisms. *Trans Am Acad Ophthalmol Otolaryngol* 79:718, 1975.
 102. Kommerell G: In discussion of Mitsui Y, Tamura O, Bérard PV, Reydy R: Optomotor effect in esotropia. In Reinecke RD, ed: *Strabismus II*. Proceedings of the Fourth Meeting of the International Strabismological Association, Asilomar. New York, Grune & Stratton, 1982, p 439.
 103. Kornder LD, Nursey JN, Pratt-Johnson JA, Beattie A:

- Detection of manifest strabismus in young children. 2. A retrospective study. *Am J Ophthalmol* 77:211, 1974.
104. Kraft SP, Levin AV, Enzenauer RW: Unilateral surgery for exotropia with convergence weakness. *J Pediatr Ophthalmol Strabismus* 32:183, 1995.
 105. Krzystkowska K, Pajakowa J: The sensorial state in divergent strabismus. In *Orthoptics. Proceedings of the Second International Orthoptics Congress. Amsterdam, Excerpta Medica, 1972, p 72.*
 106. Kushner BJ: Exotropic deviations: A functional classification and approach to treatment. *Am Orthopt J* 38:81, 1988.
 107. Kushner BJ: The distance angle to target in surgery for intermittent exotropia. *Arch Ophthalmol* 116:189, 1998.
 108. Kushner BJ: Selective surgery for intermittent exotropia based on distance/near differences. *Arch Ophthalmol* 116:324, 1998.
 109. Kushner BJ: Distance/near differences in intermittent exotropia. *Arch Ophthalmol* 116:478, 1998.
 110. Kushner BJ: Diagnosis and treatment of exotropia with a high accommodation convergence-accommodation ratio. *Arch Ophthalmol* 117:221, 1999.
 111. Kushner BJ: Does overcorrecting minus lens therapy for intermittent exotropia cause myopia? *Arch Ophthalmol* 117:638, 1999.
 112. Landolt H: Behandlung der Divergenz durch überkorrigierende Konkavgläser. *Klin Monatsbl Augenheilkd* 51:47, 1913.
 113. Lang J: *Strabismus. Diagnostik, Schielformen, Therapie.* Bern, Hans Huber, 1971, p 129.
 114. Lange W, Decker W de: Two therapeutic concepts in intermittent divergent squint. *Doc Ophthalmol* 84:187, 1993.
 115. Laws HW: An evaluation of the use of prisms in the postoperative orthoptic care of divergence strabismus. In Arruga A, ed: *International Strabismus Symposium. University of Giessen, Germany, 1966. Basel, S Karger, 1968, p 324.*
 116. Lennerstrand G: Effects of surgery on the dominant eye in exodeviations. *Acta Ophthalmol* 64:391, 1986.
 117. Luke NE: Antisuppression techniques in exodeviations. *Am Orthopt J* 20:100, 1970.
 118. Manley DR: Classification of the exodeviations. In Manley D, ed: *Symposium on Horizontal Ocular Deviations. St Louis, Mosby-Year Book, 1971, p 128.*
 119. Marlow FW: Prolonged monocular occlusion as a test for the muscle balance. *Am J Ophthalmol* 4:238, 1921.
 120. McDonald RJ: Secondary esotropia. *Am Orthopt J* 20:91, 1970.
 121. Meyer E, Noorden GK von, Avilla CW: Management of consecutive esotropia. In Mein J, Moore S, eds: *Orthoptics: Research and Practice. London, H Kimpton, 1981, p 236.*
 122. Miller JE: The electromyography of vergence movement. *Arch Ophthalmol* 62:790, 1959.
 123. Mitsui Y: Etiology and treatment of strabismus. *Ophthalmic Pract* 49:1151, 1978.
 124. Mitsui Y: *Strabismus and the Sensorimotor Reflex.* Amsterdam, Excerpta Medica, 1986.
 125. Mitsui Y, Tamura O, Hirai K, et al: Effect on master eye surgery in exodeviations. *Jpn J Ophthalmol* 24:221, 1980.
 126. Moore S: Orthoptic treatment for intermittent exotropia. *Am Orthopt J* 13:14, 1963.
 127. Moore S: The prognostic value of lateral gaze measurements in intermittent exotropia. *Am Orthopt J* 19:69, 1969.
 128. Moore S, Cohen RL: Congenital exotropia. *Am Orthopt J* 35:68, 1985.
 129. Moore S, Stockbridge L: An evaluation of the use of Fresnel press-on prisms in childhood strabismus. *Am Orthopt J* 25:62, 1975.
 130. Nakagawa T, Kii T: Natural history of exodeviation. In Kaufmann H, ed: *Transactions of the 21st Meeting of the European Strabismological Association, Salzburg, Gies-sen, Gahmig Press, 1993, p 241.*
 131. Nawratzki I, Jampolsky A: A regional hemiretinal difference in amblyopia. *Am J Ophthalmol* 46:339, 1958.
 132. Neitker B: Effects of diagnostic occlusion of the deviated and the dominant eye in intermittent exotropia. *Strabismus* 3:1, 1995.
 133. Nelson LB, Bacal DA, Burke MJ: An alternative approach to the surgical management of exotropia—the unilateral lateral rectus recession. *J Pediatr Ophthalmol Strabismus* 29:357, 1992.
 134. Nordlow W: Squint—the frequency of onset at different ages and the incidence of some defects in a Swedish population. *Acta Ophthalmol Scand* 42:1015, 1964.
 135. Noorden GK von: Some aspects of exotropia. Presented at Wilmer Residents' Association, Johns Hopkins Hospital, Baltimore, April 26, 1966.
 136. Noorden GK von: Divergence excess and simulated divergence excess: Diagnosis and surgical management. *Ophthalmologica* 26:719, 1969.
 137. Noorden GK von: The patch test and plus lenses in the diagnosis of exodeviation. *The Prism, October-November 1982.*
 138. Noorden GK von: Discussion: Oculomotor control and strabismus. In Lennerstrand G, Noorden GK von, Campos E, eds: *Strabismus and Amblyopia. London, Macmillan, 1988, p 147.*
 139. Noorden GK von: Unpublished observations, 1987.
 140. Noorden GK von: Unpublished observations, 1988.
 141. Noorden GK von, Avilla, CW: Accommodative convergence in hypermetropia. *Am J Ophthalmol* 110:287, 1990.
 142. Ohtsuki H, Hasebe S, Okano M, Furuse T: Comparison of surgical results of responders and non-responders to the prism adaptation test in intermittent exotropia. *Acta Ophthalmol Scand* 75:528, 1997.
 143. Olitsky SE: Early and late postoperative alignment following unilateral lateral rectus recession for intermittent exotropia. *J Pediatr Ophthalmol Strabismus* 35:146, 1998.
 144. O'Neal TD, Rosenbaum AL, Stathacopoulos RA: Distance stereoacuity improvement in intermittent exotropic patients following strabismus surgery. *J Pediatr Ophthalmol Strabismus* 32:353, 1995.
 145. Owens PL, Strominger MB, Rubin PA, Veronneau-Troutman S: Large-angle exotropia corrected by intraoperative botulinum toxin A and monocular recession resection surgery. *J Am Assoc Pediatr Ophthalmol Strabismus* 2:144, 1998.
 146. Parks MM: Comitant exodeviations in children. In *Strabismus. Symposium of the New Orleans Academy of Ophthalmology. St Louis, Mosby-Year Book, 1962, p 45.*
 147. Parks MM, Mitchell P: Concomitant exodeviations. In Duane TD, ed: *Clinical Ophthalmology, vol 1. Philadelphia, JB Lippincott, 1988, p 1.*
 148. Pigassou R: Prisms in strabismus. *Int Ophthalmol Clin* 6:519, 1966.
 149. Piper HF: Über verschiedene Formen des Schielens, ihre Entstehung und Behandlung. In Vehlgen K, ed: *Abhandlungen auf dem Gebiet der Augenheilkunde, vol 26. Leipzig, Georg Thieme VEB 1961.*
 150. Pratt-Johnson J, Wee HS: Suppression associated with exotropia. *Can J Ophthalmol* 4:136, 1969.
 151. Pratt-Johnson JA, Barlow JM, Tilton G: Early surgery for intermittent exotropia. *Am J Ophthalmol* 84:689, 1977.
 152. Raab EL, Parks MM: Recession of the lateral recti. *Arch Ophthalmol* 82:203, 1969.
 153. Ravault AP, Bongrand M, Bonamour G: The utilization of prisms in the treatment of divergent strabismus. In

- Orthoptics. Proceedings of the Second International Orthoptics Congress. Amsterdam, Excerpta Medica, 1972, p 77.
154. Rayner JW, Jampolsky A: Management of adult patients with large angle exotropia. *Ann Ophthalmol* 5:95, 1973.
 155. Repka MX, Arnoldi KA: Lateral incomitance in exotropia: Fact or artifact? *J Pediatr Ophthalmol Strabismus* 28:125, 1991.
 156. Richard JM, Parks MM: Intermittent exotropia: Surgical results in different age groups. *Ophthalmology* 90:1172, 1983.
 157. Romano PE, Wilson MF, Robinson JA: World-wide surveys of current management of intermittent exotropia by MD strabologists. *Binocular Vision Strabismus Q* 8:167, 1993.
 158. Rubin SE, Nelson LB, Wagner RS, et al: Infantile exotropia in healthy children. *Ophthalmic Surg* 19:792, 1988.
 159. Ruttum MS: Initial versus subsequent postoperative motor alignment in intermittent exotropia. *J Am Assoc Pediatr Ophthalmol Strabismus* 1:88, 1997.
 160. Sanfilippo S, Clahane AC: The immediate and long-term results of orthoptics in exodeviations. In *First International Congress of Orthoptists*. St Louis, Mosby-Year Book, 1968, p 300.
 161. Schlossman A, Boruchoff SA: Correlation between physiologic and clinical aspects of exotropia. *Am J Ophthalmol* 40:53, 1955.
 162. Schlossman A, Muchnick RS, Stern K: The surgical management of intermittent exotropia in adults. *Ophthalmology* 90:1166, 1983.
 163. Scobee RG: *The Oculorotary Muscles*, ed 2. St Louis, Mosby-Year Book, 1952.
 164. Scott WE, Mash AJ: The postoperative results and stability of exodeviations. *Arch Ophthalmol* 99:1814, 1981.
 165. Seaber JH: Pseudomyopia in exodeviations. *Am Orthopt J* 16:67, 1966.
 166. Snir M, Axer-Siegel R, Shalev B, et al: Slanted lateral rectus recession for exotropia with convergence weakness. *Ophthalmology* 106:992, 1999.
 167. Spencer RF, Tucker MG, Choi RY, McNeer KW: Botulinum toxin management of childhood intermittent exotropia. *Ophthalmology* 104:1762, 1997.
 168. Stathacopoulos RA, Rosenbaum AL, Zanoni D, et al: Distance stereoacuity—Assessing control in intermittent exotropia. *Ophthalmology* 100:495, 1993.
 169. Swan KC: Problems of exotropia. *J Pediatr Ophthalmol* 2:25, 1965.
 170. Tamlar E, Jampolsky A: Is divergence active? An electromyographic study. *Am J Ophthalmol* 63:452, 1967.
 171. Urist MJ: Right angle exotropia. *Am J Ophthalmol* 58:987, 1964.
 172. Van den Berg AV, van Rijn LJ, de Faber JT: Excess cyclovergence in patients with intermittent exotropia. *Vision Res* 35:3265, 1995.
 173. Velez G: Results in intermittent exotropia. In Moore S, Mein J, Stockbridge L, eds: *Orthoptics: Past, Present, Future*. Transactions of the Third International Orthoptic Congress. New York, Stratton Intercontinental, 1976, p 559.
 174. Véronneau-Troutman S: Fresnel prism membrane in the treatment of strabismus. *Can J Ophthalmol* 6:249, 1971.
 175. Wang FM, Chryssanthou G: Monocular eye closure in 172. Van den Berg AV, van Rijn LJ, de Faber JT: Excess 176. Weakley DR, Stager DR: Unilateral lateral rectus recession *Res* 35:3265, 1995.
 177. Weiss JB, Rouchy JP, Ruellan YM, Teliques JP: Utilisation des prismes compensateurs provisoires pour rompre le cercle vicieux “déviation diplopie.” *Bull Soc Ophthalmol Fr* 69:303, 1969.
 178. Weiss L: Upon the relation between the internal and external recti as affected by increasing divergence of the orbits. *Arch Ophthalmol* 25:341, 1896.
 179. White JW: Discussion of paper by Bielschowsky A: Divergence excess. *Arch Ophthalmol* 12:157, 1934.
 180. Wiggins RE, Noorden GK von: Monocular eye closure in sunlight. *J Pediatr Ophthalmol* 27:16, 1990.
 181. Windsor CE: Surgery, fusion, and accommodative convergence in esotropia. *J Pediatr Ophthalmol Strabismus* 8:166, 1971.
 182. Winter R, Winter M, de Decker W: Langzeitergebnisse nach operativer Behandlung des Strabismus divergens intermittens. *Ber Vers Dtsch Ophthalmol Ges* 76:683, 1979.
 183. Wirtschafter JD, Noorden GK von: The effect of increasing luminance on exodeviations. *Invest Ophthalmol* 3:549, 1964.
 184. Wittebol-Pol D, Graaf M de: Early onset exotropia. In Kaufmann H, ed: *Transactions of the 20th Meeting of the European Strabismological Association*, Brussels, 1992, p 203.
 185. Wynnanski-Jaffe T, Wyanbeek Y, Bessler E, Spierer A: Strabismus surgery using the adjustable suture technique. *J Pediatr Ophthalmol Strabismus* 36:184, 1999.
 186. Yildirim C, Mutlu FM, Chen Y, Altinsoy HI: Assessment of central and peripheral fusion and near and distance stereoacuity in intermittent exotropic patients before and after strabismus surgery. *Am J Ophthalmol* 128:222, 1999.
 187. Yuksel D, Spiritus M, Vandannoitte S: Chirurgie symétrique comme traitement initial de l'exotropie intermittente de base. *Bull Soc Belge Ophthalmol* 268:195, 1998.