Intractable Diplopia After Strabismus Surgery in Adults

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Objectives: To investigate the incidence of persistent intractable diplopia in adults undergoing surgery for long-standing, constant strabismus and to define tests that may be useful for identifying patients at risk for developing this complication.

Methods: A retrospective medical record review of adults without diplopia undergoing surgery for constant strabismus.

Results: Medical records of 424 adult patients undergoing strabismus surgery were studied. Of these patients, 143 (34%) experienced diplopia when tested preoperatively with prisms to simulate the desired surgical outcome. Only 40 patients (9%) had temporary diplopia after surgery, which resolved in all cases by 6 weeks postoperatively. Three patients (0.8%) developed persistent intractable diplopia. Experiencing diplopia with preoperative prism testing was significantly more likely to result in postoperative diplopia than if diplopia was not present preoperatively (P<.001 and P=.04 for temporary and persistent postoperative diplopia, respectively). Preoperative testing had a sensitivity and negative predictive value for temporary postoperative diplopia of 100%, a specificity of 73%, and a positive predictive value of 28%. Similar values were found for persistent diplopia after surgery with the exception of the positive predictive value, which was only 2%. Patients who did not see double during preoperative testing with prisms never developed diplopia after surgery. However, the presence of preoperative diplopia with prism testing (including a prolonged trial with Fresnel prisms) was infrequently predictive of postoperative diplopia.

Conclusions: Intractable diplopia after strabismus surgery in adults without previous diplopia is very rare. The diagnostic use of prisms prior to surgery may identify some patients who have little or no risk of postoperative diplopia, as well as a group of patients with a small but definite risk of intractable postoperative diplopia.

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The possibility of diplopia occurring after strabismus surgery has been recognized since its description by von Graefe1 in 1854. If diplopia persists after surgery and particularly if it was not anticipated preoperatively, it can be disconcerting to both the patient and the surgeon. In some circumstances, diplopia after strabismus surgery is a predictable occurrence. For example, if a visually mature patient with acquired strabismus has diplopia prior to surgery and if the surgery is not successful in realigning the eyes, one would expect that diplopia would persist postoperatively. Also, there are some situations in which a patient without previous diplopia may predictably see double after strabismus surgery if the postoperative motor alignment is not satisfactory. Common examples include persistent surgical overcorrection in an adult with exotropia2-5 or persistent overcorrection in an adult patient with a decompensated paresis of the fourth cranial nerve; such patients typically do not have strong vertical vergence to compensate for a hypotropia of the previously hypertropic eye.6 In both of these circumstances, the presence of diplopia after surgery can be attributed to an unsuccessful attempt to surgically realign the eyes. Much less commonly, adults with a constant strabismic deviation (but no preoperative diplopia) may experience diplopia after surgery despite successful realignment of the eyes to the desired position (whatever that position may be for the particular patient). With this troublesome type of diplopia, the patient cannot fuse the images using prisms, nor can the second image be hidden in a super-
pression scotoma, unless the diplopic image is shifted optically to its original preoperative location on the retina of the deviating eye. This differs from the more common type of diplopia, which is simply due to an unsatisfactory postoperative alignment. In this latter situation, the use of prisms to simulate the desired outcome or further surgery to accomplish the desired ocular alignment will eliminate the diplopia; in the former, the diplopia cannot be eliminated.

In theory, the use of prisms prior to surgery to optically simulate the desired postoperative alignment should be helpful in identifying patients who are at risk for intractable postoperative diplopia. However, in my experience, the use of prisms is not as predictive of postoperative diplopia as one might intuitively expect. I have also found that intractable diplopia after surgery is relatively infrequent. To my knowledge, the literature does not contain tested guidelines or risk stratification for predicting which patients may potentially develop intractable diplopia after strabismus surgery. The purpose of this article is to present my experience in assessing adult patients without prior diplopia who had constant strabismus preoperatively and to outline some simple steps for identifying patients at risk for developing intractable diplopia after strabismus surgery.

## METHODS

This series was compiled by doing a retrospective medical record review of all patients I operated on for a constant (but perhaps variable angle) vertical or horizontal tropia between January 1, 1978, and January 1, 2001, who were 18 years or older at the time of surgery. The objective of this study was to investigate patients who developed intractable diplopia after surgery that could not be eliminated with prisms or further strabismus surgery to achieve a more desired motor alignment. Consequently, patients were excluded if they reported diplopia prior to surgery because postoperative double vision in such patients merely represents a failure of the surgery. Similarly, patients who had postoperative diplopia that could be eliminated by using prisms to exactly correct their deviation, or with further surgery to attain a more desirable ocular alignment, were excluded for the same reason. The outcome determination date was approximately 6 weeks after surgery for patients who did not develop postoperative diplopia. Because this symptom usually appears almost immediately after surgery, it was assumed that if it had not occurred by 6 weeks, it would not do so later. For patients in whom diplopia did occur after surgery, the data were analyzed up to the last postoperative visit to determine if the finding was long lasting.

In addition to the usual testing of ocular motility and sensory status, I routinely test all adult patients with constant strabismus in the following manner. While wearing an appropriate optical correction, the patient fixates on a Snellen optotype close to the vision threshold in their better-seeing eye. Then, neutralize the deviation by placing prisms over the deviating eye and ask the patient if he or she sees double. Regardless of the response to this test, I then remove the prism and reintroduce prisms of increasing power using either a prism bar or rotary prism. I begin with 0 prism diopters (PD) and stop when the amount of prism overcorrects the deviation by 5 to 10 PD. The patient is instructed to report if and when diplopia occurs as the prism power is increased. If the patient reports seeing double, the prism power is successively decreased and again increased (perhaps several times) to determine at what point the diplopia can be eliminated and if the findings are reproducible. In addition, if patients report diplopia, they are asked to indicate if it is crossed or uncrossed and to estimate the distance between the two images. Finally, patients are asked to describe if the second image was sharp and intense or more of a shadowy ghost image. As an additional exclusion criterion, patients were eliminated from this study if they did not undergo preoperative testing with prisms as outlined previously.

If patients did not see double with any of these tests, it was assumed that they would not see double after surgery, and no further testing was done. Patients who did report diplopia were told that there was a small risk of their having persistent postoperative diplopia. Most patients elected to proceed with surgery despite that admonition. For those who were concerned about developing diplopia yet were still interested in pursuing the option of strabismus surgery, a prolonged prism trial was carried out. To neutralize the deviation, Fresnel prisms were applied to patients' spectacles, which they wore until the double vision resolved or for a period of up to 2 weeks. This was accomplished by placing the Fresnel prism over the habitually deviating eye or placing more prisms over the deviating eye if the size of the deviation mandated bilateral Fresnel prisms. After this trial, patients were assessed for the presence of double vision. If the double vision had subsided, it was assumed that postoperative diplopia would not occur if satisfactory alignment was obtained. If diplopia persisted with prisms, patients were told that they had a small but definite risk of developing persistent postoperative diplopia. Most decided to undergo surgery despite that risk.

For the purpose of data analysis, patients were arbitrarily considered to have temporary double vision if the symptom lasted more than several consecutive minutes during the first 6 weeks after surgery yet subsided by the 6-week outcome date. Patients who reported only momentary diplopia lasting several seconds—with shift of gaze direction, with fatigue, or on first arising—were considered not to have postoperative diplopia. In these patients, it was impossible to determine if their symptoms were truly abnormal or were just a manifestation of physiologic diplopia. Also, for the purpose of this study's inclusion criteria, patients who manifested a small, constant tropia (≤ 8 PD) but had undergone surgery for an intermittent, larger deviation were considered to have intermittent strabismus and were excluded. Although technically such patients do have a constant tropia, they are sensorially different from the patients who were the subject of this investigation; many of the former type have monofixation syndrome.

Patients were categorized as having congenital esotropia if their medical records and history both confirmed that the esotropia was present prior to 1 year of age. Patients were assumed to have probable congenital esotropia if their history suggested an onset prior to 1 year of age and if either dissociated vertical divergence (DVD) or optokinetic asymmetry was present but confirmatory medical records were not available. Similarly, patients were considered to have a known consecutive esotropia if medical records confirmed that diagnosis; patients were labeled as having probable consecutive esotropia if their history suggested the diagnosis but it could not be confirmed with medical records. Similar reasoning was used for the diagnosis of known and probable consecutive exotropia. Patients were considered to have both a vertical and horizontal deviation only if the surgical procedure specifically addressed both components. For the purpose of data analysis, no attempt was made to separate the type of esotropia (eg, congenital or acquired) initially present in patients with consecutive exotropia, the initial type of exotropia in patients with consecutive esotropia, or the type of esotropia or exotropia in
The medical record review identified 424 patients who met the entry criteria for this study. Of these patients, 221 were women and 203 were men. The mean ± SD age at the time of surgery was 31.4 ± 10.4 years (range, 18-64 years). An additional 29 patients were eliminated from the initial review because they did not meet the inclusion criteria of having undergone preoperative testing with prisms as outlined previously. In most cases, the failure to perform that testing was due to lack of patient cooperation or intelligence that made the testing impossible.

Clinical details of the patients are presented in Table 1. Of the 424, 281 patients (66%) did not see double with preoperative prism testing during the initial office examination. Although none of them developed temporary or permanent diplopia after surgery, some reported momentary episodes of diplopia lasting several seconds. The remaining 143 patients (34%) did experience diplopia with prism testing during the initial office examination; in general, these patients showed consistency with this testing. When testing was carried out repeatedly as indicated in the “Methods” section, diplopia typically appeared and disappeared with a reproducible amount of prism. However, many patients exhibited a moderate difference between the amount of prism needed to induce diplopia and the lesser amount at which the diplopia disappeared.

Of the 143 patients who had diplopia with preoperative prism testing, 40 experienced temporary postoperative diplopia, and 3 additional patients experienced persistent diplopia following surgery. The difference in the incidence of postoperative diplopia for patients who did vs did not see double with preoperative prism testing was significant (for temporary postoperative diplopia, P < .001 using the χ² test; for persistent postoperative diplopia, P = .04 using the Fisher exact test). The sensitivity, specificity, and predictive values with preoperative prism testing for both temporary and persistent postoperative diplopia are presented in Table 2. As the table indicates, the absence of diplopia with preoperative prism testing is excellent assurance that neither patients undergoing surgery for horizontal and vertical deviations simultaneously, because of the small numbers involved. Patients undergoing simultaneous symmetric oblique muscle surgery and horizontal muscle surgery were categorized as having surgery only to correct the horizontal deviation because they did not undergo surgery to address a hypertropia in the primary position.

Finally, several patients who had unusual manifestations of postoperative diplopia came to my attention during the medical record review, although they did not meet the inclusion criteria of this study. Their findings are anecdotally reported because they shed light on the subject of diplopia after strabismus surgery.
temporary nor persistent double vision will occur after surgery. The presence of diplopia with preoperative prism testing indicates only a small risk of temporary postoperative diplopia (28%) and a much smaller risk of persistent diplopia (2%). The overall incidence of temporary or persistent postoperative diplopia in this series of 424 patients was 9.4% and 0.7%, respectively.

The mean ± SD age of the 43 patients who did experience diplopia after surgery was 31.4 ± 8.9 years, which was almost identical to that for the 381 patients who did not see double postoperatively (31.6 ± 10.6 years). Although the exact onset time of the strabismus cannot be determined with certainty for the patients in this series, all of them had strabismus dating back to childhood. Consequently, older age at surgery was a general reflection of a longer duration of strabismus. It does not appear that duration of the deviation had a substantial influence on the development of postoperative diplopia.

Of the 143 patients who experienced diplopia during the initial office testing with prisms, 103 (72%) were not sufficiently concerned about postoperative diplopia to undergo a Fresnel prism trial preoperatively. Of these 103 patients, only 3 had persistent diplopia after surgery. One was a 35-year-old man with probable congenital esotropia. He underwent bilateral medial rectus resections and lateral rectus resections to correct a 70°PD esotropia. After surgery he had a 5°PD residual esotropia and was diplopic. He could neither fuse the images nor suppress the second one with any amount of base-in or base-out prism less than 50 PD. When last seen 2½ years after surgery, he indicated that he was still seeing double; however, he was able to easily ignore the second image.

The second patient with persistent diplopia was a 32-year-old woman who underwent a left lateral rectus recession and left medial rectus advancement to correct a consecutive exotropia. Postoperatively, she had less than 5 PD of residual exotropia with constant diplopia and could neither fuse the images nor suppress the second one with any amount of prism. When last seen 1½ years after surgery, she was still diplopic and was fogging one eye with a high plus-powered contact lens.

The third patient was a 23-year-old woman who had undergone surgery at 21 months of age for a known congenital esotropia. At age 14 years, she underwent further surgery to treat a left hypertropia. Then, at age 23 years, she underwent a right inferior rectus recession and a small re-recession of the right medial rectus muscle to treat a residual esotropia and left hypertropia. At the time of her preoperative examination, she would see double if either the vertical or horizontal deviation were overcorrected with prisms. After surgery she immediately developed a consecutive exotropia with underaction of the right medial rectus muscle and had constant diplopia. A month later the right medial rectus was advanced, which left her with a 3°PD esotropia at 6.0 m and a 5°PD esotropia at 0.3 m combined with 5 PD of left hypertropia at both fixation distances. She continued to be bothered by intermittent diplopia despite satisfactory alignment. Multiple attempts to improve her symptoms with prisms, to secure the second image in a scotoma, or to manipulate her angle of strabismus by altering her hyperopic spectacle correction were not successful. Her symptoms of diplopia persisted for approximately 2 years after surgery, then gradually became less bothersome. When I spoke to her by telephone 3 years after surgery, she indicated that she rarely experienced diplopia, most commonly when she drove a motor vehicle at night. With the exception of limiting her night driving, diplopia was no longer keeping her from any other usual activity.

All 3 of these patients described their postoperative symptoms in a similar manner. Specifically, the patients were unable to clearly describe the location of the second image with respect to the primary image, and they could not determine whether they had crossed or uncrossed diplopia. Also, when attempts were made to move the second image with prisms, the patients had a hard time perceiving the movement of the image and gave inconsistent descriptions of what they were observing. The amount of movement they perceived was not commensurate with the amount of prism introduced. Jampolsky described this phenomenon as “confused or lost localization” of the diplopic image.

Eighteen of the 143 patients who experienced diplopia with prism testing during the office examination elected to undergo preoperative testing with Fresnel prisms. Their clinical findings are summarized in Table 3. Of these patients, 8 reported that the diplopia disappeared after several days to 2 weeks of using the prism.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Sample Size</th>
<th>Underwent Preoperative Fresnel Prism Trial</th>
<th>Persistent Diplopia With Fresnel Prism Trial</th>
<th>Temporary Diplopia After Surgery</th>
<th>Persistent Diplopia After Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Known congenital ET</td>
<td>22</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Probable congenital ET</td>
<td>29</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Acquired ET</td>
<td>121</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Primary XT†</td>
<td>89</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Known consecutive XT</td>
<td>43</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Probable consecutive XT</td>
<td>26</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Miscellaneous vertical deviation‡</td>
<td>13</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>343</td>
<td>18</td>
<td>10</td>
<td>3</td>
<td>0</td>
</tr>
</tbody>
</table>

*Data are presented as number of patients. ET indicates esotropia; XT, exotropia.
†Includes decompensated intermittent XT and probable congenital or idiopathic XT.
‡Includes patients with hypertropia not fitting any other category.

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Data are presented as number of patients. ET indicates esotropia; XT, exotropia.
None of them had temporary or persistent diplopia after surgery.

The remaining 10 patients continued to see double after the Fresnel prism trial. Nevertheless, they elected to undergo surgery despite the risk of postoperative diplopia. Three of them experienced temporary postoperative diplopia that disappeared by 6 weeks after surgery; the other 7 experienced no postoperative diplopia. Notably, some of these patients continued to wear the Fresnel prisms and to see double right up until the time of their strabismus surgery. Although they had diplopia with the preoperative prism testing, they never experienced diplopia afterward, from the moment the eye patch was removed immediately following surgery.

Several other interesting observations were made in this series of patients, who tended to describe the quality of their diplopia during the office prism testing in 1 of 3 manners. Some patients described the second image as a shadowy ghost image in the far periphery of their visual field when the deviation was neutralized with prisms prior to surgery. This type of response is indicative of anomalous retinal correspondence (ARC). These patients rarely had temporary diplopia after surgery and never had permanent diplopia. Other patients described the 2 images as being intense and close together; a higher percentage of these patients had temporary diplopia after surgery. Finally, some patients were unable to subjectively localize the second image and exhibited what was described previously as lost or confused localization.

This group constituted most patients who had temporary diplopia and all patients with permanent diplopia. Many patients reported that diplopia would appear during preoperative office testing when only a fraction of the deviation was corrected with prisms. For example, a patient with a 40-PD exotropia might experience diplopia when 20 PD or more of base-in prism was introduced. This did not seem predictive of whether the patient would experience diplopia after surgery. Even if the vision was surgically undercorrected and patients were left with a deviation matching an angle that would have caused diplopia if simulated with prisms preoperatively, they did not have postoperative diplopia. Many other patients would see double only when the testing prisms came very close to offsetting the deviation; however, they continued to experience diplopia as the amount of prism was decreased. For example, a patient with 40 PD of exotropia might not see double until 35 PD of base-in prism was introduced, but the diplopia would persist until the amount of prism was decreased to less than 15 PD. This observation confirms the statement by Jampolsky that many patients with strabismus may give inconsistent answers in successive trials of the same sensory test. This finding also seemed to have no predictive value regarding postoperative diplopia.

The presence of poor vision in the deviating eye (either due to amblyopia or to other causes) did not necessarily prevent the occurrence of temporary or permanent diplopia. Of the 40 patients who experienced temporary double vision, 14 had a best-corrected visual acuity in their deviating eye of worse than 20/40. Three had visual acuities that ranged from 20/800 to 20/200. One of the patients who experienced permanent double vision after surgery had a best-corrected visual acuity of 20/200 in the deviating eye.

During the time span covered by this study, I cared for an additional 19 patients who would have met the inclusion criteria but decided not to undergo surgery owing to the risk of postoperative diplopia. Fourteen made the decision after experiencing diplopia during the initial office testing with prisms. The other 5 decided against surgery after experiencing persistent diplopia with the Fresnel prism trial.

In the course of compiling this series, I reviewed the records of several other patients who did not meet the inclusion criteria yet provided further insight into the subject of diplopia after strabismus surgery. Three were patients who had undergone extensive vision therapy including antisuppression exercises prior to seeing me. One was a 28-year-old woman with a congenital esotropia that previously had not been surgically treated, the second was a 38-year-old man with an esotropia that was probably acquired, and the third was a 36-year-old man with a constant exotropia that previously had not been surgically treated. After receiving preoperative vision therapy, each patient developed constant diplopia that persisted after strabismus surgery. Although they each attained satisfactory alignment (within 10 PD of orthotropia), they were unable to either fuse the images or suppress the second image with or without prisms. Their sensory responses were similar to the description of horror fusion, which has been characterized as an “active aversion to fusion” or “magnetic repulsion” of the 2 images if an attempt is made to superimpose them with prisms.

Although these patients did have permanent intractable diplopia after strabismus surgery, they were excluded from the formal data analysis of this study because they had diplopia prior to surgery.

Only 4 patients in this series underwent surgery for DVD. Most adult patients I operated on for DVD during the study time span were excluded because their deviation was intermittent; a constant deviation was an inclusion criterion. Nevertheless, I noted during my medical record review that several of these excluded patients did experience temporary diplopia after surgery if the hypertropia was overcorrected. One such patient, a 19-year-old woman with a history of surgery for infantile esotropia, maintained a 4-PD overcorrection after a unilateral superior rectus recession for DVD. She developed persistent diplopia after surgery and required long-term use of a base-up prism to optically simulate a hypertropia of the surgically treated eye for relief of the condition. Thus, the presence of DVD does not rule out the possibility of postoperative diplopia. An additional 3 patients who were visually mature but younger than 18 years had temporary intractable diplopia that lasted up to 1 year after surgery. One was an 11-year-old boy undergoing surgery to correct a residual esotropia that was probably congenital. Two were 17-year-old boys undergoing surgery for consecutive exotropia. One had had a prior congenital esotropia, and the other had probable congenital esotropia. The diagnosis of prior congenital esotropia does not rule out the possibility of postoperative diplopia.
This study shows that intractable diplopia after strabismus surgery is quite rare if the desired alignment is obtained surgically and that the use of prisms prior to surgery may help in identifying patients at risk. Most prior reports of intractable diplopia after strabismus surgery have been anecdotal case reports\(^\text{1,3,6-10}\) that do not provide predictive factors or incidence figures. One study that did address the incidence of postoperative diplopia was published by Scott et al.\(^\text{11}\) That study, however, included patients who had diplopia prior to surgery as well as those who did not. It also included patients in whom postoperative diplopia was related to unsatisfactory ocular alignment after surgery. Consequently, Scott and colleagues intermingled patients in whom surgery was unsuccessful with those who experienced diplopia despite good postoperative alignment. Only the latter type of patient is the subject of my article.

The data show that the use of prisms preoperatively to predict postoperative diplopia is not as helpful as one might intuit. If patients do not see double with preoperative prism testing, they will probably not see double after surgery. If patients do see double with preoperative testing (including a prolonged trial with Fresnel prisms), there is only a small chance of persistent postoperative diplopia. In this series, the incidence of intractable diplopia was just 0.8% of all patients undergoing surgery, 2.0% of those who reported diplopia during the office examination with prisms, and 7.5% of those with persistent diplopia after a trial with Fresnel prisms. Why prisms do not provide better predictive information about postoperative diplopia deserves discussion. It may seem surprising that a patient can go into surgery experiencing diplopia while wearing Fresnel prisms yet not see double after the procedure, from the moment the patch is removed from the surgically treated eye. It often appeared that there was no need for an adaptation period in such patients. However, the use of prisms to simulate a change in alignment is not the same as performing surgery to effect that change. In the former, there is no change in the proprioceptive afferent input to the system responsible for the localization of an image in space; in the latter, there is such a change. Nevertheless, the instantaneous adaptation exhibited by patients in this series speaks for a remarkable fluidity in most patients’ ARC.

This attribute of ARC has been noted by others.\(^\text{12-14}\) Flom et al\(^\text{13}\) described a woman with an intermittent exotropia and ARC who had an excellent ability to voluntarily change the magnitude of her deviation. As she changed the angle of her deviation, the angle of anomaly would immediately and synchronously vary to keep the ARC harmonious. Similarly, many patients with harmonious ARC and an A or V pattern will automatically alter their angle of anomaly as they look into upgaze or downgaze to keep the ARC harmonious. This fluidity is a hallmark of ARC that “makes squint surgery possible without postoperative diplopia,” according to Herzau.\(^\text{12}\) The existence of this plasticity led Verhoeff\(^\text{15}\) to suggest that there is no true correspondence in ARC. He reasoned that because normal retinal correspondence is so stable, ARC should be similarly stable because it is simply a shift from normality. However, Flom and colleagues indicated (and cited Hallidén and Kerr as concurring\(^\text{13}\)) that the same phenomenon is not observed with the introduction of prisms if the motor angle does not actually change. They believed that Hallidén’s “sensory-fusion” mechanism, which is based on diplopia resulting from the stimulation of disparate retinal points, might be a more useful explanation of these sensory findings. Similarly, the concept of a “response shift” as an alternative to a truly anomalous correspondence of the retinal elements, developed by Boeder,\(^\text{16,17}\) may be more appropriately descriptive of this phenomenon.

The data also show that the presence of amblyopia may not preclude postoperative diplopia. This is not surprising when one considers that unlike visual confusion, which is caused by interaction between the fovea of the two eyes, diplopia is a result of the fovea of one eye and the retinal periphery of the other perceiving the same object. Consequently, decreased visual acuity, which is a function of the resolving ability of the fovea, does not necessarily preclude diplopia. Finally, vision therapy in the form of antisuppression exercises before surgery may result in horror fusionis despite otherwise successful strabismus surgery.\(^\text{9,10}\)

For patients who do develop persistent postoperative diplopia, several treatment options are possible. Theoretically, they can undergo a reoperation to re-create their initial preoperative deviation, which might eliminate the diplopia. In my own experience, this has never been necessary. I find that such patients usually accept either a high plus-powered contact lens (typically \(>20\) diopters) to fog 1 eye or an occluder contact lens. Because these patients did not have binocular vision prior to surgery, they generally do not have a problem functioning monocularly after surgery. By using a contact lens to blur or occlude 1 eye, they retain the aesthetic benefit of the surgery, which would be lost if the preoperative deviation were re-created. Fortunately, intractable diplopia is rare and usually does not incapacitate adult patients with long-standing strabismus, as opposed to the type of diplopia that occurs in acquired paretic or restrictive strabismus.

Like all studies, this one needs to be viewed in light of certain limitations. Being retrospective in nature, the data reflect only those tests and questions that I routinely use in my practice. Perhaps more rigorous sensory testing to determine the nature and depth of retinal correspondence in these patients might have proved useful. On the other hand, Jampolsky\(^\text{2}\) pointed out that many commonly used tests of suppression give superfluous or misleading information because they do not mimic everyday visual circumstances. This study provides useful risk stratification guidelines that can be easily adhered to by any physician without requiring sophisticated testing modalities.

In conclusion, most adult patients without diplopia who have constant strabismus will not develop intractable diplopia after strabismus surgery if the eyes are successfully aligned. The preoperative use of prisms can identify patients who have little cause for concern.
about postoperative diplopia and other patients who have a small but definite risk. It cannot accurately identify patients who will have intractable diplopia postoperatively.

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