Diplopia After Refractive Surgery

ARCHIVES EXPRESS

Occurrence and Prevention

Burton J. Kushner, MD; Lionel Kowal, FRANZCO

Objectives: To report the occurrence of persistent diplopia manifesting after refractive surgery, to describe the different causes of this complication, to provide risk stratification for its occurrence, and to outline minimal screening techniques for its prevention.

Methods: A retrospective medical record review of patients seen in 2 private strabismus practices who experienced persistent diplopia after refractive surgery.

Results: A total of 28 patients were identified who met the inclusion criteria. The causes of postoperative diplopia could be traced to 1 of 5 mechanisms. These included technical problems, prior need of prisms, anisekonia, iatrogenic monovision, and improper control of accommodation in patients with strabismus. The recommended screening techniques would have identified all patients in this series as being at risk for postoperative diplopia with the exception of those in whom technical problems were responsible.

Conclusions: Diplopia can become manifest after refractive surgery. With proper attention paid to risk stratification and recommended screening criteria, the incidence of this complication can be minimized.

Arch Ophthalmol. 2003;121:315-321

From the Department of Ophthalmology and Visual Sciences, University of Wisconsin, Madison (Dr Kushner); the Department of Ophthalmology, University of Melbourne, Melbourne, and the Royal Victorian Eye and Ear Hospital, East Melbourne, Australia (Dr Kowal).
Drs Kushner and Kowal have no relevant financial interest in this article.

REFRACTIVE SURGERY is becoming increasingly popular with patients who seek an alternative to wearing spectacles. It has been estimated that every year, approximately 1.5 million patients worldwide undergo laser-assisted in situ keratomileusis (LASIK).[1] Recent reviews of complications of refractive surgery have discussed infection, scarring, cataract formation, strabismus, and corneal decompensation.[1,2] In addition, there have been several small case series of diplopia after refractive surgery.[3-10] Some of these tudies described temporary diplopia, attributed to an interim period of iatrogenic anisometropia or monocular occlusion that occurred between surgical stages in both eyes of patients in whom a 2-stage procedure was planned.[3,7,10] There have also been patients with long-lasting symptoms.[4-6,8] Little has been written on the different causative mechanisms of diplopia after refractive surgery, stratification of patient characteristics that constitute different levels of risk for its occurrence, or recommended screening criteria.[11] To our knowledge, an incidence figure for this complication has not been published. In the last several years, we have increasingly been referred patients who experience diplopia after refractive surgery. The purpose of this article is to review our experience with this complication, analyze the different causative mechanisms of its occurrence, outline relative risk stratification criteria, and provide specific screening guidelines for patients undergoing refractive surgery.

METHODS

A retrospective review was conducted of patients seen in our clinical practices who were evaluated because they experienced persistent monocular or binocular diplopia (did not resolve spontaneously) after undergoing refractive surgery, either in the form of LASIK, photorefractive keratectomy, or radial keratotomy. The series consists of all such patients seen consecutively between 1987 and 2002; there were no exclusion criteria. All patients were personally seen by 1 of the 2 of us and were evaluated and treated in our usual manner. This included a complete ophthalmologic evaluation with particular attention to ocular motility and sensory status. In all patients, measurement of any angle of misalignment with the alternate prism and cover test, assessment of the potential for elimination of diplopia if the deviation was offset with prisms, and stereopsis testing with

Table 1. Etiology of Diplopia*

Etiology	No. of Patients
Technical problems	
Scarring	1†
Ablation zone too small	1†
Decentered treatment zone	1
Astigmatic axis or power change	2
Residual hyperopia in patient with accommodative esotropia	3
Residual hyperopia in patient with anisometropic exotropia	1
Overcorrection	
Induced aniseikonia	1
Overcorrected myopia with esotropia	2
Prior prism	3
Aniseikonia	1
Monovision	
Intermittent strabismus, unreliable fusional mechanisms	3
Fixation switch diplopia	3
Fourth cranial nerve paresis, secondary deviation	1
Incorrect targeted outcome: accommodation problems	
Residual accommodation in patient with esotropia and a high accommodative convergence-to-accommodation ratio	1
Unrecognized bifocal lens in patient with prepresbyopia	1
Intermittent exotropia previously treated with overcorrecting minus lenses	2
Difference between absolute and manifest hyperopia	1
Prior undercorrected hyperopia with intermittent exotropia	2

*The total exceeds 28 because some patients are listed in more than 1 category.
†Diplopia was monocular.

the Titmus test were performed. All patients underwent a cycloplegic refraction and, when indicated, a manifest refraction. When appropriate, fusional amplitudes were measured with a prism bar or rotary prism, cyclotorsion was assessed objectively by viewing the fundus with the indirect ophthalmoscope and subjectively with the double Maddox rod test, and aniseikonia was quantified using the Awaya New Aniseikonia Test.[12] In some patients, an additional orthoptic evaluation in the form of testing with the synoptophore was carried out. To understand the causative mechanism of diplopia in some of the patients in this series, we reviewed records from the ophthalmologist who had performed the refractive surgery or from ophthalmologists or optometrists who had cared for the patient years earlier.

RESULTS

Our review identified 28 patients who met the inclusion criteria. Their ages ranged from 20 years to 57 years (mean±SD, 37.2±9.3 years). The sex distribution was 15:13 for females to males, respectively. Five general mechanisms accounted for the complication of postoperative diplopia in this series. An overview of the different causes we identified appears in **Table 1**.

TECHNICAL PROBLEMS

Technical problems in the form of either scarring or failure to achieve the desired optical outcome were responsible for diplopia in 11 of our patients. The only 2 patients in the series who developed monocular diplopia did so as a result of technical problems while undergoing LASIK. In 1 patient this condition occurred because of a buttonhole through the center of the flap, resulting in a scar near the center of the visual axis. In the other it occurred because the ablation zone was too small in a patient with relatively large pupils.

Another patient, previously described in detail,[9] had approximately 27 diopters (D) of myopia preoperatively (spherical equivalent) and developed postoperative binocular diplopia after undergoing LASIK, which had attempted to correct 23 D of myopia. Because he had difficulty maintaining fixation during the procedure, the treatment zone was inadvertently decentered upward. This induced a vertical prismatic effect, which resulted in 16 prism diopters (PD) of hypertropia. The mechanism by which decentration of the flap can have the same effect as "grinding" a large amount of prism in the cornea has previously been described in detail.[9] In general, vertical fusional amplitudes are much smaller than horizontal ones. Consequently, inadvertent horizontal decentration of the treatment zone is more forgiving than vertical decentration and less likely to cause diplopia.

One patient, a 24-year-old man, developed diplopia secondary to a series of bilateral radial keratotomy procedures to treat myopia. After 2 surgical procedures on his right eye and 4 on the left, which were unsuccessful in reducing the myopia in his left eye, he still had a substantial amount of anisometropia. Although he had only 5 PD of exotropia after his last refractive surgical procedure, he could not maintain fusion when the deviation was offset with prisms secondary to a 7% aniseikonia.

Two patients developed diplopia because of inaccurate correction of an astigmatic refractive error. One was a 34-year-old man who had a preoperative cycloplegic refraction of –4.00+2.00, axis 100, OD and –5.00+2.50, axis 85, OS. After undergoing radial keratotomy surgery in his left eye only, he had a residual refractive error of –2.00+3.25, axis 55, OS. This 30° rotation in the axis of astigmatism resulted in an optically induced 7° excyclotropia of the operated eye, as measured subjectively with the double Maddox rod test. Although he had normal ocular motility (no shift on the cover test) and no objective fundus torsion, he remained symptomatic when last seen 4 years after refractive surgery. He was able to fuse the torsionally rotated image and, when tested with the synoptophore, had fusional amplitudes if the torsional misalignment was optically corrected. The patient declined strabismus surgery to treat his torsional diplopia. Rubin[13] has explained the mechanism by which a shift in the axis of astigmatism can result in a tilting of the image. Another patient, a 40-year-old woman, had a preoperative refractive error of –4.00+4.00, axis 90, OD. As a result of a mathematical error in converting her refractive error from plus-cylinder to minus-cylinder format, the refractive error that was programmed into the computer placed the cylinder axis 90° from its correct location. After surgery she had a final refractive error of –8.00+8.50, axis 170, OD, which resulted in binocular diplopia. Although she was ortho-

phoric, she could not fuse the 2 images because she had aniseikonia of 9% and distortion of the image in the operated eye.

Given the current technology, refractive surgery to correct hyperopia is less precise and more likely to fluctuate postoperatively than surgery to correct myopia. It is important for the refractive surgeon to realize that hyperopia is not the mirror image of myopia. The treatment of myopia always centers on a relatively fixed target: the cycloplegic refractive error. In the treatment of hyperopia, the target is less stable as a result of the variable influence of accommodation in each eye. The clinical status and comfort of the patient are dependent on the subtleties of the interaction of accommodation and refractive error as well as the technical accuracy of the refractive surgery. Four patients in this series had unintentional residual hyperopia, which resulted in decompensation of a previously well-controlled accommodative esotropia. One such patient was a 32-year-old woman who had a previously well-controlled accommodative esotropia when wearing her cycloplegic refractive correction of +3.50+2.50, axis 115, OD, and +3.50+3.50, axis 60, OS. After undergoing bilateral LASIK surgery, she had a cycloplegic refractive correction of +1.75+1.50, axis 115, OD, and +1.25+1.75, axis 60, OS. She had an uncorrected visual acuity of 20/25 OU after surgery despite the residual uncorrected astigmatism. Consequently, she did not wear an optical correction after surgery and had a 15-PD esotropia. She had continual diplopia along with the esotropia. Similarly, the overcorrection of myopia in 2 patients with a history of accommodative esotropia resulted in postoperative diplopia.

PRIOR NEED OF PRISM

Refractive surgery can allow a preexisting diplopia that has been controlled with prisms to become manifest. Three patients experienced binocular diplopia after refractive surgery because they had required prisms in their spectacles to eliminate diplopia preoperatively. In 1 case, both the refractive surgeon and the patient were unaware that the patient had been wearing prisms in his spectacles. In another, the refractive surgeon was unaware of the patient's need for prisms even though the patient was aware. The patient had not mentioned it to the refractive surgeon because she did not realize that it was relevant. In the third case, the surgeon had hoped that a reduction in the patient's astigmatic refractive error might eliminate his need for prisms after surgery. This turned out not to be accurate.

ANISEIKONIA

Aniseikonia can produce a peculiar form of binocular diplopia in which the 2 images are not displaced in space but one image is larger than the other. There is disagreement regarding the exact amount of aniseikonia that healthy subjects can tolerate. Katsumi et al[14] reported that aniseikonia of 3% or more can impair binocular function. Others have suggested that aniseikonia of up to 5% can be tolerated.[15,16] Knapp's rule states that if anisometropia is axial, aniseikonia should not be present with spectacle correction.[17] Similarly, if a patient with an axial anisometropia (and without aniseikonia) has the refractive error corrected at the corneal plane, aniseikonia is likely to be induced. This occurs because a shift in location of the optical correction from the spectacle plane to the corneal plane results in a change in the perceived image size, even if there is no image size difference between the 2 eyes when the optical correction is made with spectacles. To the extent that Knapp's rule is correct, if a patient has substantial anisometropia (we estimate that to be > approximately 4 D based on Linksz and Bannon's rule[18]) and normal fusion, it would follow that the anisometropia is axial and that significant aniseikonia is not present. With such a patient, refractive surgery can result in aniseikonia of at least 4%, which may cause diplopia.[8,18] One patient in this series had myopia of 2 D OD and 8 D OS with good fusion; he had 60 seconds of stereopsis prior to refractive surgery. After undergoing bilateral refractive surgery, which left him with a negligible refractive error in both eyes, he had intractable diplopia secondary to aniseikonia, which measured 7%.

MONOVISION

Monovision (the intentional optical correction of one eye for distance focus and the other eye for near focus to treat presbyopia) is becoming increasingly common with the rising popularity of refractive surgery. Monovision is successful in approximately 73% of patients with presbyopia.[19] If, however, a patient has strabismus, certain important factors render monovision a less satisfactory treatment option.[20,21] We have observed 3 different mechanisms by which monovision can cause diplopia in patients with previous strabismus. It has been shown that long-standing monovision in adults results in the absence of foveal fusion and reduced stereoacuity.[22] Thus, patients who have an intermittent strabismus (eg, intermittent exotropia) and hence an unreliable fusional mechanism may experience decompensation of a previously well-compensated strabismus as a result of the iatrogenic anisometropia produced by monovision.[7,20,21,23] This mechanism was the cause of diplopia in 3 of our patients. Similarly, patients with paretic strabismus such as a fourth cranial nerve paresis may lose control of a previously well-compensated strabismus because of monovision.[4,6] With monovision, the patient is required to fixate with the paretic eye either at distance or near focus. When fixating with the paretic eye, a larger tropia (a secondary deviation) is present because of Hering's law. This may exceed the previously established fusional vergence amplitudes and result in a deterioration of control of the ocular alignment. This mechanism was responsible for diplopia in 1 patient in this series. Finally, patients with a constant nonalternating strabismus (either a microtropia or larger deviation) may not be accustomed to suppressing the image from their dominant eye when they fixate with their nondominant eye. In such patients, the suppression scotoma that is typically present in their nondominant eye may not transfer to their dominant eye when they fixate with the nondominant one. This entity, known as fixation switch dip-

lopia, has been previously described in detail.[20] It was the cause of diplopia in 3 of our patients.

Of the 7 patients in whom monovision appeared responsible for the diplopia, it was intentional in 6 and unplanned in 1. In the 7 patients with monovision, the interocular difference ranged between 1.50 D and 2.50 D after refractive surgery.

CONTROL OF ACCOMMODATION IN PATIENTS WITH STRABISMUS

The relationship between accommodation and convergence is crucial to the understanding of esotropia and exotropia. In the "Technical Problems" section, we described 6 patients with previously well-controlled esotropia in whom failure to achieve the targeted optical end point resulted in a recurrence of esotropia and diplopia. There are also many circumstances in which the surgeon may attain his or her targeted optical outcome but because of certain nuances in the relationship between accommodation and horizontal strabismus, that targeted outcome may not be ideal. Such was the case in 9 additional patients in this series who are listed in Table 1 under the heading "Incorrect Targeted Outcome: Accommodation Problems." One patient was a 32-year-old woman with myopia who had a preoperative refractive error of approximately –2.50 sphere bilaterally. She had always taken her glasses off to read at near focus. After undergoing LASIK surgery, which successfully eliminated her myopia, she had a 12-PD intermittent esotropia at 0.33 m with diplopia. Review of her prior ophthalmologic records revealed that she had a preexisting esophoria at near focus, which was probably the reason she chose to remove her spectacles for reading. She was unaware of the esophoria and merely felt that her vision was more comfortable without her glasses for near viewing. Another patient, a 24-year-old woman, had a history of accommodative esotropia with a high accommodative convergence-to-accommodation ratio. Her strabismus was well controlled with a progressive bifocal lens. The refractive surgeon was unaware that her spectacles contained a bifocal segment because he did not expect a 24-year-old patient to need a bifocal. After undergoing LASIK, she manifested esotropia and diplopia during near viewing.

Two patients had intermittent exotropia that had previously been controlled with intentional optical overcorrection of their myopia. The refractive surgery successfully targeted and corrected their cycloplegic refraction but did not take into account that the patients were accustomed to having an additional minus correction in their glasses. This resulted in a deterioration of their exotropia. In 1 patient with hyperopia and accommodative esotropia, diplopia occurred because he normally required substantially more plus correction than his absolute hyperopia (the minimum amount of plus correction for threshold visual acuity) to control his deviation. The surgery targeted his absolute hyperopia rather than the most plus correction he could accept and still have threshold visual acuity. Finally, 2 patients had intermittent exotropia with hyperopic astigmatic refractive error. Prior to refractive surgery, their spectacles did not fully correct their hyperopia; they needed to exert some accommodative convergence to control their horizontal deviation. After refractive surgery, which corrected much of their hyperopia, they were no longer able to use this accommodative convergence to control their strabismus. The exotropia deteriorated, resulting in diplopia.

Another theoretical mechanism deserves mention. The increased accommodation necessary for near focus after surgical correction of myopia might cause the deterioration of a previously well-controlled accommodative esotropia. This would be similar to the sudden manifestation of presbyopic symptoms that can occur in patients with myopia who switch from spectacles to contact lenses. Although this mechanism did not account for the diplopia of any of our patients, the refractive surgeon should be aware that it could possibly complicate an otherwise successful surgical result.

In all 28 patients in this series, diplopia was persistent unless or until some optical or surgical measures were taken to correct the underlying cause. It did not resolve spontaneously in any of the patients.

COMMENT

This study shows that diplopia can occur after refractive surgery. The mechanisms we identified as causative include technical problems, the existence of prior prisms, aniseikonia, monovision, and other accommodative issues in patients with previous strabismus.

From this series, we have generated minimum screening criteria to identify patients at risk for diplopia after refractive surgery. These recommendations are summarized in **Table 2**. When neutralizing a patient's current spectacles, the lenses should be marked while overlying the patient's pupil, and a lensometer reading should be obtained at that point to see if the patient is currently wearing prisms. Also, particular attention should be paid to identifying the presence of a progressive bifocal lens because this type of lens is not grossly visible. Otherwise the examiner may not notice that the patient requires a bifocal lens. The cover-uncover test should be performed at 6 m and 0.33 m while the patient is wearing the targeted optical correction at both distances. Thus, if monovision is the desired end point, testing for strabismus should be performed while the patient is wearing a monovision correction. Similarly, if a patient habitually wears spectacles only for viewing at either far distance or 0.33 m (but not both), strabismus testing should be carried out with the desired optical outcome in place at both distances. All patients should have a manifest refraction (without cycloplegia) and a cycloplegic refraction, with cyclopentolate hydrochloride being the preferred cycloplegic agent. For the manifest refraction, the end point in patients with myopia should be the weakest minus correction that provides threshold visual acuity. For patients with hyperopia, the absolute hyperopia (minimum amount of plus correction needed for threshold visual acuity) and manifest hyperopia (maximum hyperopic correction accepted that permits threshold visual acuity) should both be noted prior to cycloplegia. For the cycloplegic refraction in those with myopia, the least amount of minus correction needed for threshold visual acuity should again be determined, and the dif-

Table 2. Screening Criteria

Screening Procedure	Comments
Minimal screening criteria	
History	Strabismus, diplopia, prism in spectacles, bifocal lens in patient with prepresbyopia, prior eye exercises, patching.
Check current spectacles including assessment for prisms and no-line bifocal lens	
Cover-uncover test and alternate prism and cover test, distance and near focus	Performed while patient is wearing targeted optical correction.
Refraction	
Manifest	For patients with myopia, target least minus correction for threshold acuity. For patients with hyperopia, least plus correction for threshold acuity = absolute hyperopia; most plus correction accepted for threshold acuity = target.
Cycloplegic	Note difference between cycloplegic and manifest refractions. Difference between manifest maximum plus correction and cycloplegic refraction = latent hyperopia.
Additional tests	
Fusional divergence and convergence amplitudes	Perform if there is a history or findings of diplopia, strabismus, or prisms in spectacles, or a moderate-sized phoria.
Optical trial of monovision with spectacles or contact lenses	Perform if monovision is the desired outcome and patient has substantial phoria, prisms in spectacles, or history or findings of strabismus.
Trial with neutralizing prisms	Perform if patient is wearing prisms in spectacles.
Measure astigmatic axis with monocular and binocular viewing	If substantially different in patients with strabismus, measure again during operation.

ference between that value and the one found with the manifest refraction should be noted. For patients with hyperopia, the maximum amount of hyperopic correction that permits threshold visual acuity with cycloplegia should be determined. One should then note the difference between that value and the manifest refraction, which represents the amount of latent hyperopia.

Patients with strabismus but no diplopia as a result of suppression were not included in this study. However, the principles presented in this article should be equally useful to help prevent strabismus from manifesting after refractive surgery in patients in whom this condition was previously well controlled.

Tjon-Fo-Sang et al[24] recently observed that there can be a surprising amount of cyclotorsion of either eye when a patient changes from binocular viewing to monocular viewing. This is particularly likely to occur in patients with fourth nerve paresis, latent nystagmus, a latent cyclotropia, or dissociated vertical divergence. Because most patients undergoing corneal topography prior to refractive surgery have that test performed with binocular viewing and because refractive surgery is performed with monocular viewing, their axis of astigmatism may rotate as much as 31° when they fixate monocularly during the refractive surgical procedure. It has also been reported that only a 15° angle of error in the axis of astigmatism can result in a 50% error in the magnitude of the surgically corrected astigmatism.[25] Tjon-Fo-Sang and colleagues recommend that corneal astigmatism be measured prior to surgery, both with binocular and monocular viewing conditions. If there is a substantial difference between the 2 readings, the axis of astigmatism should again be checked during the operation. Patients who demonstrate this rotation of the astigmatic axis and have 1 of the previously mentioned strabismus conditions are at risk for postoperative diplopia. They should undergo a detailed sensorimotor evaluation.

Additional testing may be indicated for patients who are designated at moderate or high risk for diplopia. These additional tests are listed in Table 2 along with suggested indications. If a patient is currently wearing prisms in his or her spectacles, assessment of fusional amplitudes with a prism bar or rotary prism may be helpful in determining if the patient will be comfortable without prisms. If office testing shows a safe range of fusional amplitude, refractive surgery should be safe. However, if there is uncertainty about the quality of the patient's fusional amplitudes, the definitive test is for the patient to wear spectacles (or contact lenses) without prisms for 1 to several weeks prior to undergoing refractive surgery. This can easily be accomplished by applying a Fresnel prism to a patient's existing spectacles in a direction that will neutralize or subtract the existing prisms. Such a trial would be an acceptable alternative to measuring fusional amplitudes for all patients wearing prisms. Similarly, if monovision is the desired outcome in a patient with moderate or high risk, a preoperative trial of monovision with spectacles or contact lenses is recommended.

As a result of our experience derived from this series, we have developed a relative risk stratification for avoiding diplopia after refractive surgery, which is summarized as follows. Some of the specific recommendations are arbitrary and cannot be supported with firm data. However, our combined experience in treating adults with diplopia suggests that these recommendations are generally sound and sufficiently inclusive to identify most patients who are at risk for this complication. With the exception of the patients whose diplopia was caused by technical problems, these screening criteria would have identified all remaining patients in this series as being at risk.

LOW RISK

Patients should be considered to have low risk if they meet all of the following criteria: myopia, less than 4 D of anisometropia, no history of strabismus or diplopia, no prisms currently in their spectacles, no more than a mini-

mal phoria on the alternate prism and cover test, and current spectacles, manifest refraction, and cycloplegic refraction all within 0.5 D of each other. Patients in the low-risk group may still develop diplopia if major technical problems result in an undesired optical outcome or if scarring occurs. However, patients with accommodative esotropia but good fusional reserve (amplitudes >10 PD) while wearing their absolute hyperopic correction should also be at low risk, irrespective of the prescription usually worn. Patients who have had prior strabismus surgery and now have a good range of fusion while wearing their absolute hyperopic or proper myopic correction should be able to safely undergo refractive surgery.

MODERATE RISK

Patients who do not meet all of the low-risk criteria are considered to have at least a moderate risk. We recommend the additional tests listed in Table 2 (depending on the specific risk factor) for these patients. For patients undergoing testing of fusional amplitudes because of any reason outlined in Table 2, values greater than 10 PD can generally be considered safe. If values of less than 5 PD are found, the patient is at some risk for postoperative diplopia. If patients wear prisms in their spectacles and a trial of optical treatment without prisms results in comfortable binocular vision without diplopia, the risk is moderate. Similarly, if monovision is the desired outcome of refractive surgery in a patient with strabismus, and a trial of contact lenses or spectacles to achieve monovision does not result in diplopia, these patients are still at moderate risk. Patients with accommodative esotropia and poor fusional reserves (<5 PD) have a moderate risk of postoperative diplopia because of the less precise nature of refractive surgery for hyperopia. In addition, the presence of more than 2 D of latent hyperopia (eg, the difference between the most plus power accepted for threshold visual acuity and the cycloplegic refraction) may pose a theoretical risk for late-occurring diplopia. Because latent hyperopia will inevitably manifest with time, such patients will need larger ranges of fusional divergence to compensate for the esophoria that may subsequently develop. Finally, patients who undergo surgery to correct a substantial astigmatic refractive error and have a considerable difference between the axis of astigmatism with binocular and monocular fixation are at risk for an inaccurate correction of their astigmatism. Such patients typically have a strabismus such as a fourth cranial nerve paresis, latent cyclotropia, or dissociated vertical divergence. Hence, they are also at risk for developing diplopia if they undergo refractive surgery.

HIGH RISK

Any patient who fails the additional testing needed to qualify for moderate risk should be considered at high risk. Also, patients are at high risk for postoperative diplopia if they have accommodative esotropia and require substantially more plus correction than their absolute hyperopia to control their deviation. In addition, patients with more than 4 D of anisometropia and good fusion are at high risk for developing symptomatic aniseikonia. A trial with a contact lens may be helpful in determining how these patients will respond to a shift of the optical correction closer to the corneal plane. We recommend that any patient who is designated at high risk be assessed by someone with particular expertise in strabismus (eg, a strabismologist or certified orthoptist) before undergoing refractive surgery. In some situations, a moderate or high risk of diplopia is not an absolute contraindication for refractive surgery. Many patients may elect to undergo refractive surgery and treat diplopia, if it occurs, with strabismus surgery. In this case, we recommend that the strabismus surgery be performed after the refractive surgery. It is important to note that this option will not be successful for patients in whom the diplopia is caused by iatrogenic aniseikonia or if it results from monovision.

Because this series was compiled from the practices of 2 strabismologists, we do not have a basis for estimating an incidence figure for diplopia after refractive surgery. Given the large number of refractive surgical procedures that are performed, we assume that this complication occurs infrequently. Nevertheless, any practical measures that can be taken to minimize the risk of this complication are useful. We have attempted to outline the most common causes of diplopia after refractive surgery and have provided guidelines for screening patients to assess risk. We realize that many of these guidelines are arbitrary and cannot be supported by firm data at this time. Because of the relatively uncommon occurrence of this complication, it is unlikely that studies in the near future will be able to statistically support recommendations for the prevention of this problem. In the meantime, we feel that these guidelines, which are based on our combined experience in treating patients with strabismus, seem reasonable. Although our guidelines may not identify every at-risk patient, they are useful tools to minimize postoperative diplopia.

Submitted for publication August 15, 2002; final revision received December 5, 2002; accepted December 20, 2002.

This study was presented in part as the 33rd Jules Stein Lecture (Dr Kushner), Los Angeles, Calif, March 2, 2002.

Corresponding author and reprints: Burton J. Kushner, MD, 2870 University Ave, Suite 206, Madison, WI 53705 (e-mail: bkushner@facstaff.wisc.edu).

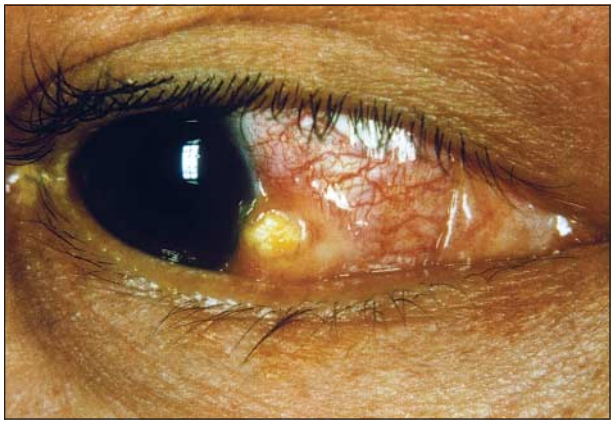
REFERENCES

1. Melki SA, Azar DT. LASIK complications: etiology, management, and prevention. *Surv Ophthalmol.* 2001;46:95-116.
2. Davis EA, Hardton DR, Lindstom RL. LASIK complications. *Int Ophthalmol Clin.* 2000;40:67-75.
3. Marmer RH. Ocular deviation induced by radial keratectomy. *Ann Ophthalmol.* 1987;19:451-452.
4. Mandava N, Donnenfeld ED, Owens PL, Kelly HS, Haight DH. Ocular deviations following excimer laser photorefractive keratectomy. *J Cataract Refract Surg.* 1996; 22:504-505.
5. Zwaan J. Strabismus induced by radial keratotomy. *Mil Med.* 1996;161:630-631.
6. Schuler E, Silverberg M, Beade P, Moadel K. Decompensated strabismus after laser in situ keratomileusis. *J Cataract Refract Surg.* 1999;25:1552-1553.
7. Kim SK, Lee JB, Han SH, Kim EK. Ocular deviation after unilateral laser in situ keratomileusis. *Yonsei Med J.* 2000;41:404-406.

8. Holland D, Amm M, de Decker W. Persisting diplopia after bilateral laser in situ keratomileusis. *J Cataract Refract Surg.* 2000;26:1555-1557.
9. Yap EY, Kowal L. Diplopia as a complication of laser in situ keratomileusis surgery. *Clin Experiment Ophthalmol.* 2001;29:268-271.
10. Furr BA, Archer SM, Del Monte MA. Strabismus misadventures in refractive surgery. *Am Orthopt J.* 2001;51:11-15.
11. Kowal L. Refractive surgery and diplopia. *Clin Experiment Ophthalmol.* 2000;28: 344-346.
12. Awaya S, Sugawara M, Horibe F, Miura M. *Studies on Aniseikonia and Stereopsis With the "New Aniseikonia Tests": Proceedings of the 4th Meeting of the International Strabismological Association.* New York, NY: Grune & Stratton; 1984: 549-560.
13. Rubin M. Astigmatism. In: *The Fine Art of Prescribing Glasses.* Gainesville, Fla: Triad Scientific Publishers; 1978:81-85.
14. Katsumi O, Tanino T, Hirose T. Effect of aniseikonia on binocular function. *Invest Ophthalmol Vis Sci.* 1986;27:601-604.
15. Crone RA, Leuridan OMA. Tolerance for aniseikonia, I: diplopia thresholds in the vertical and horizontal meridians of the visual field. *Albrecht Von Graefes Arch Klin Exp Ophthalmol.* 1973;188:1-16.
16. Hosaka A. Asthenopia and aniseikonia. *Nippon Ganka Gakkai Zasshi.* 1954;58: 792-797.
17. Benjamin WJ. Patients with anisometropia and aniseikonia. In: *Borish's Clinical Refraction.* Philadelphia, Pa: WB Saunders; 1998:1134-1159.
18. Linksz A, Bannon R. Aniseikonia and refractive problems. *Int Ophthalmol Clin.* 1965;5:515-534.
19. Jain S, Arora I, Azar DT. Success of monovision in presbyopes: review of the literature and potential applications to refractive surgery. *Surv Ophthalmol.* 1996; 40:491-499.
20. Kushner BJ. Fixation switch diplopia. *Arch Ophthalmol.* 1995;113:896-899.
21. Kushner BJ. Recently acquired diplopia in adults with long-standing strabismus. *Arch Ophthalmol.* 2001;119:1795-1801.
22. Fawcett SL, Herman WK, Alfieri CD, Castleberry KA, Parks MM, Birch EE. Stereoacuity and foveal fusion in adults with long-standing surgical monovision. *J AAPOS.* 2001;5:342-347.
23. Jampolsky AJ. Unequal vision inputs and strabismus management: a comparison of human and animal strabismus. In: *Symposium on Strabismus: Transactions of the New Orleans Academy of Ophthalmology.* St Louis, Mo: CV Mosby Co; 1978:358-492.
24. Tjon-Fo-Sang MJ, de Faber JT, Kingma C, Beekhuis WH. Cyclotorsion: a possible cause of residual astigmatism in refractive surgery. *J Cataract Refract Surg.* 2002;28:599-602.
25. Vajpayee RB, McCarty CA, Taylor HR. Evaluation of the axis alignment system for correction of myopic astigmatism with the excimer laser. *J Cataract Refract Surg.* 1998;24:911-916.

ARCHIVES Web Quiz Winner

Congratulations to the winner of our December quiz, Anmar M. A. Rahman, MD, registrar, Department of Ophthalmology, Dunedin Hospital, Dunedin, New Zealand. The correct answer to our December challenge was conjunctival keratoacanthoma. For a complete discussion of this case, see the Clinicopathologic Reports, Case Reports, and Small Case Series section in the January ARCHIVES (Kifuku K, Yoshikawa H, Sonoda KH, Kawano YI, Miyazaki K, Ishibashi T. Conjunctival Keratoacanthoma in an Asian. *Arch Ophthalmol*. 2003;121:118-119).



Be sure to visit the *Archives of Ophthalmology* Web site (http://www.archophthalmol.com) and try your hand at our Clinical Challenge Interactive Quiz. We invite visitors to make a diagnosis based on selected information from a case report or other feature scheduled to be published in the following month's print edition of the ARCHIVES. The first visitor to e-mail our Web editors with the correct answer will be recognized in the print journal and on our Web site and will also be able to choose one of the following books published by AMA Press: *Clinical Eye Atlas, Clinical Retina*, or *Users' Guides to the Medical Literature.*