Recently Acquired Diplopia in Adults With Long-standing Strabismus

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Background: The evaluation and management of recent-onset diplopia in an adult with a history of long-standing strabismus can be perplexing and challenging. No guidelines exist, to my knowledge, for the examination of such patients.

Design: A retrospective medical record review.

Subjects: Patients seen in my practice with a history of recently acquired diplopia and a history of strabismus dating back to childhood.

Results: One hundred fifty-two patients who met the enrollment criteria were identified. Using the treatment approach outlined herein, 132 patients were relieved of their symptoms of diplopia. In most cases, the onset of the diplopia could be correlated with a change in the patient’s ocular alignment, refractive needs, or refractive management. Returning patients to their motor status before the onset of symptoms or addressing the change in refractive needs or management usually resulted in relief of symptoms.

Conclusion: In most cases, adult patients with a history of long-standing strabismus and a recent onset of diplopia can be effectively treated.


The symptom of diplopia can usually be attributed to 1 of 2 causes. Most frequently, it occurs in patients with no prior history of strabismus and, hence, no abnormal sensory adaptation who develop an ocular misalignment after visual maturity.1,2 This commonly occurs in patients with Graves disease or with acute paralytic strabismus or after an orbital fracture.3 Treatment of these patients usually involves correcting the misalignment of the visual axes, with surgery, botulinum toxin type A (Botox Allergen Inc, Irvine, Calif), or prisms. A second common cause of diplopia is the surgical overcorrection of patients with long-standing strabismus. For example, patients with intermittent exotropia or superior oblique muscle palsy will predictably develop diplopia if they have experienced a surgical overcorrection.

Less commonly, adult patients with a history of strabismus dating back to childhood may experience a short-term or gradual onset of diplopia when they had been previously diplopia free. Such patients comprise a heterogeneous group who pose a challenge to the clinician. The cause of the diplopia may be difficult to determine, and its treatment may not be obvious. There is a need for published guidelines for the examination and treatment of such patients. In my experience, most of these patients can be made symptom free if a systematized approach to examination and treatment is followed. This report describes my experience in the examination and treatment of this patient population.

Results

This series consists of 152 patients whose age ranged between 18½ and 76 years. Ninety-five were women, and 57 were men. In 132 (87%) of the patients, the symptom of diplopia was effectively eliminated without resorting to fogging or occluding one eye. In most of the remaining 20 patients, fogging in the form of a plus-powered contact lens (between 5 and 25 diopters [D]) was used to blur the second image in the nondominant eye. In some patients, occlusion in the form of a Bangerter filter or a contact lens with an opaque center was used.

A summary of the clinical findings and treatment of the 152 patients who comprise this series is listed in the Table.
SUBJECTS AND METHODS

A retrospective medical record review was conducted of all patients I had examined between January 1, 1978, and December 31, 1998, who met the following enrollment criteria: A history of strabismus was present dating back to childhood. The patients had been diplopia free before the age of 18 years; however, they now had symptoms of troublesome diplopia (either intermittent or constant) that were not precipitated by ocular surgical intervention. They were at least aged 18 years at the time of my examination for diplopia.

Because this is a heterogeneous group of patients, they were not treated with one specific therapeutic modality. My examination and treatment of them was tailored to their specific problem, and my treatment approach evolved over the years as my experience with these patients increased. A list of, and definitions for, the different categories of patients in this series are provided in the “Results” section and in the Table.

The total number of patients per cause listed in the Table exceeds 152, because many patients were included in more than 1 category. For example, patients may have experienced a change in their angle of strabismus as a result of a change in their refractive management. In that situation, the patients would represent 2 entries in the Table. In general, I attempted to only include a patient in a given category if I believed that cause represented a major cause of the patient’s symptoms. I recognize that this is a somewhat subjective criterion and, consequently, believe the data in the Table are only intended to give a gestalt of this patient population; they are not to be considered exactly quantitative. Of the 95 patients who experienced a documented change in their angle of strabismus, in 5 it was attributed to monovision; 4, to contact lens–related problems; 9, to premature presbyopia; and 6, to a refraction being performed without cycloplegia (resulting in a substantially incorrect spectacle prescription). Overall, 71 patients in this series needed to undergo strabismus surgery to achieve symptomatic relief. As previously stated, some patients are listed in more than 1 category in the Table. Consequently, the number of patients listed in the Table as requiring surgery exceeds 71. For most patients undergoing surgery, an adjustable suture technique was used. The Table also lists the 20 patients in whom treatment was considered a failure. Three of them were included among the 71 patients who required strabismus surgery. Thus, the success rate for surgery in eliminating symptoms in this series was 96% (68 of 71 patients) for those in whom surgery was performed.

The systematized steps in examining and treating these patients are provided, with representative detailed examples of some of the different entities encountered in this series.

<table>
<thead>
<tr>
<th>Cause of Symptoms</th>
<th>Underwent Strabismus Surgery</th>
<th>Treatment Unsuccessful</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monocular diplopia</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Corneal irregularity</td>
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<td>0</td>
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<tr>
<td>Cataract</td>
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<td>0</td>
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<tr>
<td>IOL dislocation</td>
<td>4</td>
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<tr>
<td>Macular irregularity</td>
<td>52</td>
<td>4</td>
</tr>
<tr>
<td>Change in angle of strabismus</td>
<td>95</td>
<td>52</td>
</tr>
<tr>
<td>Change in refractive management</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Intentional monovision causing loss of control</td>
<td>18</td>
<td>2</td>
</tr>
<tr>
<td>Fixation switch diplopia (iatrogenic—includes some monovision)</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>Accommodative esotropia or amblyopia—manifest refraction</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Switch from “line” to “no-line” bifocal with vertical in comittance</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Change in the optical center</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

*Data are given as number of patients. IOL indicates intraocular lens.

**STEP 1: IS THE DIPLOPIA MONOCULAR OR BINOCULAR?**

Of the 152 patients, 9 had monocular diplopia. A list of the different causes for their monocular diplopia is provided in the Table, and some common characteristics of this patient group are represented by case 1.

Case 1 was a 62-year-old woman with a history of strabismus since childhood. She had undergone 2 strabismus surgical procedures before the age of 10 years and reports that her eyes have been misaligned for as long as she can remember. For the past year, she has noted intermittent vertical diplopia when she reads. On ocular examination, she was found to have 30 prism diopters (PD) of esotropia and 20 PD of hypertropia in her left eye. She had a low hyperopic refractive error that was accurately corrected by her spectacles. With correction, her visual acuity was 20/20 OD and 20/25 OS. At the time of her initial examination by me, she was free of diplopia. The rest of her general ophthalmologic examination was unremarkable. She indicated that she typically would need to read for approximately 45 to 60 minutes before her symptoms of diplopia would appear. She was, therefore, instructed to sit in the waiting room and read a book until she noticed diplopia. After 1 hour, she indicated that she was seeing double. On reexamination, she was asked if the double vision could be eliminated by closing 1 eye. She then closed her left eye and reported that the double vision was gone. It was not until she was asked if she could eliminate the diplopia by closing her
right eye that it became evident that her diplopia was monocular (limited to the left eye). Fluorescein staining of her left cornea then revealed a horizontal line of punctate stain just above the lower eyelid margin. It was subsequently observed that she manifested incomplete blinking in her left eye. This resulted in a corneal epithelial defect when she was holding her eyes in the down-gaze reading position for a prolonged period. A successful trial of artificial teardrops followed by lacrimal punctal plugs eliminated her symptoms.

In many of the other patients who had monocular diplopia, it was not immediately evident that the double vision was in fact monocular. Typically, when asked if the double vision could be eliminated by closing 1 eye, the patients would respond yes. It was only after asking if the diplopia was eliminated by closing the other eye that the monocular nature of the diplopia was apparent. Case 1 illustrates the importance of determining if the diplopia is monocular vs binocular, even in patients with manifest strabismus. Case 1 also indicates the importance of duplicating the circumstances that elicit the symptoms in patients with intermittent diplopia (in her case, by reading for approximately 1 hour). In 5 of the 9 patients with monocular diplopia, the underlying cause could be corrected and the symptom eliminated. The remaining 4 patients had untreatable macular abnormalities.

**STEP 2: WHAT CHANGED AT THE ONSET OF THE SYMPTOMS (ALIGNMENT, REFRACTIVE MANAGEMENT, OR REFRACTIVE NEEDS)?**

**Changes in Alignment**

For most patients, the onset of diplopia could be traced to a change in their clinical situation. Often this was a change in the angle of strabismus. In some individuals, there was a change in the direction of the deviation (eg, a person with esotropia who drifted and developed exotropia or a person with exotropia who drifted and developed esotropia); in others, it was simply a change in the magnitude of the tropia (eg, a small esotropia becoming a larger one). In still other patients, the precipitating change was in their refractive needs or management. As seen in the Table, 95 patients in this series experienced a change in motor alignment as one of the major precipitating causes of their problem. Case 2 is representative of the patients who experienced a change in the direction of their misalignment.

Case 2 was a 20-year-old woman with a history of amblyopia and strabismus since young childhood. For the past 1½ years, she had noted horizontal diplopia. At the time of my first examination, she was found to have 14 PD of exotropia of her left eye. Her spectacles and visual acuity when first seen were as follows: +2.50 +2.50 axis 100 and 20/20 OD; +3.50 +3.50 axis 100 and 20/20 OS, respectively. Sensory testing with prisms in free space showed that she would suppress the diplopic image with 14 PD or more of base-in prism over her left eye. This optically simulated the state of esotropia. A cycloplegic refraction revealed +2.50 +2.50 axis 100 and 20/20 OD; and +3.50 +3.00 axis 70 and 20/40 OS.

A review of past ocular records indicated that she had been esotropic; however, quantitative measurements had never been obtained. My treatment consisted of changing the left spectacle lens to contain her proper correction. After she obtained the new spectacle lens, she was still diplopic. Subsequently, a 16-PD Fresnel prism was applied base in over the left eye, which eliminated her diplopia. During the subsequent 8 months, the strength of the prism was gradually decreased to 6 PD base in, after which she could not tolerate any further reduction. Then, an attempt to further decrease her angle of exotropia was made by trying to reduce the amount of hyperopic correction in her spectacles. The intention was to stimulate accommodative convergence. This was unsuccessful, however, because she had abnormally low accommodative amplitudes and did not tolerate the reduction in hyperopic correction. At this point, spectacles were ordered with a total of 6 PD of base-in prism ground in the spectacle lenses. She has remained asymptomatic for the subsequent 11 years. Had she been desirous of eliminating the need of spectacles by using contact lenses, either a small left lateral rectus muscle recession or chemodenervation with botulinum toxin type A would have been treatment options available to her to eliminate her need of a prism.

Case 2 illustrates the importance of determining the situation that existed before the onset of diplopia (in this case, an esotropic alignment) and returning the patients to their prior situation; reviewing records of prior eye examinations can be helpful in this regard. It also indicates the importance of performing an objective refraction (retinoscopy) using cycloplegia in eyes that are amblyopic. In my experience, amblyopic eyes that are refracted subjectively and without cycloplegia (as in case 2) frequently receive an incorrect spectacle correction. This occurs because individuals with amblyopia have a decreased ability to subjectively discriminate between lens choices provided to their ambylopic eye during a refraction.

**Changes in Refractive Management**

Changes in refractive management precipitated the symptoms in 46 patients, in 15 of whom they resulted in a substantial change in their angle of strabismus. The Table subdivides these patients into 6 different categories for the specific refractive management problem that was implicated. The entity most frequently encountered in this subset of patients is that of “fixation switch diplopia.” This phenomenon occurs because patients with nonalternating strabismus may experience diplopia if they fixate with their nonpreferred eye. It has been suggested that because the suppression that accompanies strabismus is facultative, the suppression scotoma may not transfer to the dominant eye when the nondominant eye is fixating. Fixation switch diplopia can occur if the normal shift toward myopia that occurs secondary to growth results in the fixating eye being more myopic; patients are optically encouraged to fixate with the nondominant eye in the distance if they are not prescribed spectacles. This phenomenon occurred in the 2 patients listed in the Table as having idiopathic fixation switch diplo-
pia. Also, inaccurate refractive correction of patients with amblyopia may result in their fixation with the nondominant eye at some viewing distance. This occurred in 6 of the 18 patients listed in the Table as having iatrogenic fixation switch diplopia. Finally, the practice of monovision to treat presbyopia (optical correction of one eye for distance and the other for near, described in detail later) may result in fixation with the nondominant eye at either distance or near viewing with subsequent diplopia. My experience with fixation switch diplopia before 1995 has been described in detail.9

The second most frequently encountered entity in this subset of patients was the intentional creation of monovision (eg, optically placing one eye in focus for distance and the other for near) to treat presbyopia.7 In some patients, the institution of monovision immediately resulted in fixation switch diplopia. To simplify data analysis, those patients are included in the iatrogenic fixation switch category in the Table (12 of the 18 patients listed) and are not included in the monovision category. However, in 7 other patients, monovision resulted in a slower breakdown of the stability of a previously well-controlled strabismus; they are listed in the Table under “intentional monovision causing loss of fusion.” In these 7 patients, the monovision was created with spectacles (n=1), contact lenses (n=2), intraocular lens choice (n=1), and refractive surgery (n=3). Case 3 is representative.

Case 3 was a 50-year-old woman with a documented history of intermittent exotropia since childhood. She was myopic and successfully wore contact lenses since the age of 15 years. The exotropia had been asymptomatic until 5 years before my first examination. When she had begun experiencing symptoms of presbyopia at the age of 45 years, she was fit with monovision contact lenses to put her nondominant left eye in focus for near. The power of the contact lenses was chosen to give her an effective 2-D add for reading by undercorrection of the myopia in the left eye. Shortly thereafter, she noted intermittent diplopia for driving and a deterioration in her control over the exotropia. She underwent strabismus surgery by another ophthalmologist at the age of 46 years. This took the form of bilateral lateral rectus muscle recessions. Initially, she had the desired small-angle overcorrection that resolved within several weeks. However, by 6 months after surgery, she was again experiencing diplopia and manifesting a poorly controlled intermittent exotropia of 20 PD. Further strabismus surgery was recommended, at which time she saw me in consultation. When I first examined her, I found her to have a poorly controlled intermittent exotropia of 20 PD at distance and near. I advised her to discontinue monovision and obtain bifocals, in the form of either spectacles or contact lenses. Bifocals resulted in an elimination of her symptoms of diplopia, and she again returned to exerting good control over her intermittent exotropia. She has been asymptomatic for 4 years.

Four patients had vertically noncomitant strabismus, in the form of either an A or a V pattern, or hypertropia that increased in down gaze. They were all well aligned in the primary position; however, they developed a more substantial vertical or horizontal deviation in down gaze. Each of them experienced diplopia when they switched from a flattop “line” bifocal to a “no-line” progressive bifocal. Because progressive bifocals have a wide transition zone between the distance and near segment, they generally force patients to put their eyes further into down gaze for reading at 0.33 m than is necessary with the standard flattop segment. This can result in diplopia in patients with vertically noncomitant strabismus.3 Each of the 4 patients in this series was perplexed that their new spectacles caused symptoms, because they were presumably the same prescription as the spectacles they had replaced.

Contact lenses created their own unique set of problems for 8 patients in this series for several different reasons. Four patients had unilateral amblyopia but previously compensated strabismus. They were subsequently advised by other eye care practitioners to discontinue wearing the contact lens in their amblyopic eye because they “did not see with that eye anyway.” This resulted in a deterioration of their previously stable alignment with the development of diplopia. Four other patients developed a deterioration of their ocular alignment with diplopia when they switched from spectacles to contact lenses. In all 4, the contact lens over their amblyopic eye left a substantial amount of the refractive error uncorrected. This occurred in some patients because they were not astigmatic in their dominant eye and were fit with soft contact lenses bilaterally (which left a substantial residual uncorrected cylinder in the amblyopic eye). In other cases, deterioration occurred because a noncycloplegic subjective refraction was performed for an amblyopic eye. For the reasons outlined in the description of case 2, this resulted in an inaccurate correction of the refractive error. In one 27-year-old patient with a history of accommodative esotropia as a child, a recurrence of the esotropia with diplopia occurred when she inadvertently reversed the contact lenses between her right and left eyes. She had become myopic as a teenager and had 2 D of spheroic myopic anisometropia. When she put the more myopic contact lens in her less myopic eye, she needed to accommodate 2 D more than normal. This resulted in an esotropia that was symptomatic for several months, because she was unaware she had reversed the contact lenses. The necessary diagnostic workup included a complete refraction for spectacles and another refraction over her existing contact lenses to diagnose the contact lens reversal. Finally, there was one 48-year-old woman with a history of type 3 Duane syndrome since birth. She had always assumed a face turn to the left because her eyes were properly aligned in right gaze. When her head was held straight, she had a 40-PD esotropia and a 12-PD hypertropia in her right eye. She had recently obtained a new pair of spectacles that incorporated the identical prescription as her former pair. She noted constant vertical and horizontal diplopia with her new spectacles. An examination of the optical center of the lenses revealed that they were positioned with an interpupillary distance of 70 mm and a vertical offset of 7 mm (right eye higher). Although this positioning resulted in the optical centers lining up with her eyes when her head was held straight, there was an induced base-in prism and base-down prism in the right eye when she was assuming her preferred head posture of a face turn left. New spectacles were ordered...
for her with the optical centers positioned in front of her pupils when she assumed her preferred head posture (which were similar to her older spectacles), and she was again asymptomatic.

**Changes in Refractive Needs**

Many patients with a history of accommodative esotropia in childhood may have decreased accommodative amplitudes and may experience premature presbyopia.\(^8\) It is not clear whether this is a result of prolonged wearing of hyperopic spectacles in childhood or if decreased accommodative amplitudes initially may have contributed to the development of the accommodative esotropia. I have the clinical impression that many patients with previously well-compensated accommodative esotropia experience a deterioration in their ocular alignment as they approach presbyopia. This appeared to be the cause of diplopia in 9 patients in this series. Presumably, the increased accommodative effort necessary to overcome the early presbyopia caused the esotropia to increase. In some patients, early recognition of this phenomenon and the institution of bifocals at a younger age than is typically necessary may prevent the deterioration of ocular alignment. Case 4 is representative.

Case 4 was a 37-year-old woman with a history of accommodative esotropia in childhood. I had examined her at the age of 30 years and found her to have 5 PD of esotropia at distance and near in her right eye while wearing her hyperopic spectacles. She reported that between the ages of 32 and 37 years, her esotropia had been gradually increasing; she was experiencing diplopia with reading. On ocular examination, she was found to have 12 PD of esotropia at 6 m and 25 PD of esotropia at 0.33 m in her right eye, associated with diplopia. Her cycloplegic refraction and best-corrected visual acuity were essentially the same as her current spectacles and measured as follows: +1.50 +0.75 axis 180 and 20/25 OD, respectively; and +0.75 +0.50 axis 180 and 20/20 OS, respectively.

Her near point of accommodation was found to be 45 cm OD and 50 cm OS, both of which were decreased for her age. She also had reported blurred vision for reading. Bifocals were ordered that incorporated a 2.00-D add bilaterally. Three months after she obtained those spectacles, she was found to have an 8-PD esotropia at 6 m and at 0.33 m in her right eye (through her bifocal segment) and was free of diplopia.

Many patients with a history of accommodative esotropia appear to have difficulty relaxing their accommodation if they are refracted without cycloplegia. This phenomenon is not limited to amblyopic eyes. Although many adult patients with normal motility can be accurately refracted without cycloplegia, problems can occur when spectacles are prescribed based on a manifest refraction (eg, without cycloplegia) in patients with a history of accommodative esotropia; similar problems can occur when refracting patients with amblyopia. Reliance on a manifest refraction appeared to be the cause of symptoms for 8 patients in this series, as illustrated by case 5.

Case 5 was a 48-year-old woman with a history of accommodative esotropia in childhood. She indicated that during the past 10 years her esotropia had been gradually increasing, and she had been experiencing asthenopia and diplopia. She was referred to me for strabismus surgery. A review of the past clinical records indicated that 10 years earlier she had less than 10 PD of esotropia at 6 m and at 0.33 m. On ocular examination, I found the following values for her spectacles and visual acuity to be +3.00 sphere and 20/20 OD, respectively; and +3.50 sphere and 20/30 OS, respectively.

With those spectacles, she had 18 PD of esotropia at 6 m with intermittent diplopia and 30 PD of esotropia at 0.33 m with constant diplopia. A cycloplegic refraction revealed +5.00 sphere and 20/20 OD, respectively; and +6.00 sphere and 20/30 OS, respectively.

I prescribed spectacles incorporating her cycloplegic refraction, cutting her hyperopic correction by 0.5 D bilaterally. She immediately noted a resolution of her symptoms of diplopia, and her esotropia again decreased to less than 10 PD. Although 4 different ophthalmologists had examined her during the previous 10 years, and each did dilate her eyes for ophthalmoscopy, they had all performed the refraction before the use of cycloplegia.

As stated earlier, many patients with vertically incomitant horizontal strabismus (eg, an A or a V pattern or a hypertropia that increases in down gaze) may have a small angle of misalignment in the primary position and be asymptomatic. When they become presbyopic and must use down gaze for reading through a bifocal, they may experience symptoms related to their strabismus. Eight patients in this series were of this type. I previously published a detailed description\(^3\) of my experience in managing diplopia limited to down gaze.

**STEP 3: IS THE DIPLOPIA CONSTANT OR INTERMITTENT? IF INTERMITTENT, WHAT IS THE NATURE OF THE INTERMITTENCY?**

The cause of the diplopia was considered by me to be unknown (or idiopathic) in only 18 of the 152 patients in this series after my examination and testing. In some of the 18 patients, unavailability of past records combined with uncertainties in the patients’ histories made the determination of “what had changed” unclear. In others, accurate past records and a detailed history were available; however, the cause of the onset of diplopia was still unclear. In these patients, it was often helpful to determine if the diplopia was constant or intermittent. If the diplopia was constant, a simple trial with prisms frequently proved helpful in determining appropriate treatment. In 8 patients in this group, prisms exactly equal to the full angle of misalignment as determined with the alternate prism and cover test eliminated the diplopia. In another 3 patients, a prism that was smaller than the full angle of strabismus was sufficient to eliminate the diplopia. For example, one such patient manifested 25 PD of esotropia but was free of diplopia with only 15 PD of base-out prism. In these latter 3 patients, I assumed that the prism that eliminated the diplopia placed the object of regard within a suppression scotoma that was eccentric to the macula. In both groups of patients (the 8 needing fully correcting prisms and the 3 requiring less
than fully correcting prisms), successful treatment consisted of either long-term use of the prism dose that was found to be successful during the office examination or surgery to duplicate that same angle of reduction of the deviation. Presumably, the 11 patients who comprise these 2 subsets of the “unknown cause” category had experienced a change in their motor alignment that subsequently caused diplopia, although accurate historical documentation of this conjecture was not available. In 2 patients in the unknown cause category with constant diplopia, I was only able to eliminate symptoms by occluding or fogging 1 eye. Treatment was considered to have failed in these patients.

When a patient reported intermittent symptoms of diplopia, it was useful to determine if there was a motor intermittency or a sensory intermittency. Patients experiencing diplopia associated with a motor intermittency would report that as the diplopia appeared, the single image of regard would separate into 2, and the distance between the 2 images would increase until a stable separation distance was achieved. As the diplopia resolved, the 2 images blended together. These patients appeared to have intermittent symptoms because their alignment only broke down intermittently. There were 4 patients of this type in this series. Their examination consisted of determining the maximum size of their deviation that could be found, after either 1 hour of monocular occlusion or prism adaptation. Then, appropriate treatment (surgery or prisms) was effective in eliminating symptoms when used to address this larger angle of misalignment. On the other hand, patients with a sensory intermittency would report different symptoms and were more difficult to treat. Such patients would indicate that with the onset of the diplopia, the second image would suddenly appear in some eccentric position referable to the primary object of regard. When the double vision resolved, the second image faded away in place. These patients were not experiencing a change in their motor alignment, but instead were experiencing a change in the way the brain perceived the images from the 2 eyes. The only treatment modality I have found effective for patients with a sensory intermittency is to provide the most careful correction of even the most seemingly insignificant refractive error. In 3 patients this proved helpful. For 6 other patients, the only option was to fog or occlude the nondominant eye.

**COMMENT**

This series confirms that adults with a history of long-standing strabismus and a recent onset of diplopia are a heterogeneous population for whom a systematized approach to treatment can be fruitful. Most of these patients can be made sensorially comfortable.

The most common single cause of diplopia in this series was a change in the motor alignment. Many adult patients with long-standing strabismus have a suppression scotoma in the deviating eye that represents an adaptation to a long-standing angle of misalignment. If the angle of strabismus changes, the object of regard in the deviating eye may be shifted out of the suppression scotoma, and diplopia develops. For many of the patients in this report, symptoms could be traced to unequal vision inputs from the 2 eyes. Jampolsky has elegantly shown how this can destabilize ocular alignment. Such was the situation in the patients who had inadequate refractive correction (either with spectacles or contact lenses) and in some of the patients who were optically corrected to monovision. It has been shown that monovision causes a reduction in binocular visual acuity, near and distance stereopsis, binocular contrast sensitivity, and horizontal vergence amplitudes. It also causes a central suppression scotoma. It is, therefore, not surprising that for patients with unstable binocular cooperation, monovision may result in a deterioration of their alignment.

For this study, I considered patients who needed to resort to occlusion or fogging of the nondominant eye to eliminate diplopia as “treatment failures.” In fact, many of the patients were quite accepting of this option. Patients who comprise this series are individuals who have gone most of their life without normal binocularity; many believed that they never really used their eyes together normally. It was, therefore, less disturbing for these patients to occlude or fog 1 eye than it would be for patients who were accustomed to normal single binocular vision. Similarly, none of the patients in this series needed to resort to surgical intervention to intentionally misalign their eyes to get rid of double vision. In my experience, that treatment is usually not necessary, because these patients are accepting of monocular fogging or occlusion. For patients who are able to wear contact lenses, a fogging or centrally opaque contact lens is a satisfactory option. For those who are contact lens intolerant, Bangerter filters seem preferable to opaque occlusion, because they are more cosmetically acceptable. There was one patient in this series in whom the history and sensory findings suggested that diplopia began with a spontaneous drift from a larger angle of esotropia to a smaller one; however, prior records were unavailable to confirm this. This patient chose to wear a fogging contact lens rather than have her eyes surgically returned to the prior larger angle of esotropia.

One other category of patient deserves mention in any series dealing with acquired diplopia. Often, patients with a convergence insufficiency type of exotropia or well-controlled vertical heterophorias may lose control as they get older and may develop diplopia. Although such patients may constitute a substantial percentage of adults who are seen with recent-onset diplopia, they are outside the inclusion criteria for the present series and, hence, are not included herein. This study was limited to patients with a known history of asymptomatic strabismus or amblyopia in childhood and an onset of symptoms after the age of 18 years. In my experience, patients with convergence insufficiency exotropia or compensated heterophorias are usually unaware they had the problem in childhood; the age of onset is typically unknown. Alternatively, but less frequently, they may have been symptomatic in childhood. In either case, they would not have met the inclusion criteria of this report.

The patients with sensory intermittency were the most difficult to treat and constituted the highest individual pool of treatment failures. It is not clear to me why some patients benefited from optical correction of small refractive errors; however, empirically, this seems to work in some patients.
Useful guidelines for examining these patients include the following: (1) Determine if the diplopia is monocular or binocular. If it is monocular, look for corneal surface problems, lenticular problems, or macular problems. (2) Determine if the symptoms began with a change in the angle of misalignment. If so, duplicate the prior asymptomatic angle with prisms, surgery, or botulinum toxin type A. (3) Determine if there was a change in the patient’s refractive management as a cause of the symptoms. This includes recent switching to a no-line bifocal, institution of monovision, an iatrogenic fixation switch, inaccurate refractive correction with contact lenses, and problems with optical centration. (4) Determine if there was a change in the patient’s refractive needs. This includes looking for idiopathic fixation switch diplopia, premature presbyopia, undercorrected hyperopia (or overcorrected myopia), and the onset of bifocal use in patients with vertically noncomitant strabismus. (5) Determine if intermittent diplopia was associated with a motor intermittency or a sensory intermittency. If the former, determine the maximum angle of misalignment with either monocular occlusion or prism adaptation and then treat that angle of misalignment. If a sensory intermittency is present, careful optical correction may be beneficial.

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