Pseudophakic negative dysphotopsia: Surgical management and new theory of etiology

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PURPOSE: To evaluate the benefit of various surgical methods to address pseudophakic negative dysphotopsia.

SETTING: Private practice, Los Angeles, California, USA.

DESIGN: Interventional case series.

METHODS: The following 4 surgical methods were used to treat negative dysphotopsia: secondary piggyback intraocular lens (IOL) implantation, reverse optic capture, in-the-bag IOL exchange, and iris suture fixation. Ultrasound biomicroscopy (UBM) was used to analyze posterior chamber anatomy. The primary outcome was partial or complete resolution of the negative dysphotopsia symptoms 3 months postoperatively.

RESULTS: Twelve eyes of 11 patients with negative dysphotopsia had surgical treatment. All 10 patients who had piggyback IOL implantation or reverse optic capture had partial or complete resolution of symptoms by 3 months. No patient who had in-the-bag IOL exchange (n = 3) or iris suture fixation of the capsular bag–IOL complex (n = 1) improved despite alteration of IOL material or edge design in the case of IOL exchange or UBM confirmation of posterior chamber collapse in the case of iris suture fixation of the capsular bag–IOL complex.

CONCLUSIONS: Consistent with a new hypothesis, resolution of negative dysphotopsia symptoms depended on IOL coverage of the anterior capsule edge rather than on collapse of the posterior chamber alone. Furthermore, negative dysphotopsia was not attributed to a particular IOL material or edge design.

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Negative dysphotopsia, first reported by Davison, is a poorly understood clinical condition that may follow cataract surgery with in-the-bag implantation of a posterior chamber intraocular lens (PC IOL). Negative dysphotopsia is manifested as patient-reported observation of a dark crescent in the temporal field of vision. Patients commonly state that the symptoms are similar to wearing horse blinders. This condition is distinguished from positive dysphotopsia, which is characterized by undesired optical images, typically light streaks, arcs, and halos. The latter is likely induced by properties of the IOL, whereas the cause of negative dysphotopsia has remained speculative. Osher suggests that the temporal clear corneal incision (CCI) may be the cause of early postoperative negative dysphotopsia; however, there have been reports of negative dysphotopsia after superiorly placed sclerocorneal tunnel incisions. Negative dysphotopsia has been reported to occur with a variety of IOLs. Although an understanding of the genesis of negative dysphotopsia has been elusive, we have recognized certain clinical manifestations as follows:

1. Negative dysphotopsia may be associated with many types of PC IOLs that are confined to the capsular bag and well centered after uneventful cataract surgery.
2. Negative dysphotopsia has not been reported after surgery complicated by malpositioned IOLs.
3. Negative dysphotopsia has not been reported with ciliary sulcus–fixated PC IOLs or anterior chamber IOLs.
4. Symptoms of negative dysphotopsia can be stimulated by a light source in the temporal field of the patient’s view and are relieved by occluding the...
temporal field with a hand, temple piece of spectacles, and so forth.
5. Symptoms of negative dysphotopsia are generally reduced with pharmacologic pupil dilation and increased with pupil constriction.
6. Negative dysphotopsia has not been associated with astigmatic corneal incisions, radial corneal incisions, laser in situ keratomileusis flaps, or penetrating keratoplasty.
7. Negative dysphotopsia is not related to preoperative or postoperative ametropia.
8. No clinical test has been useful in corroborating negative dysphotopsia self-reported symptoms. In our experience, Humphrey visual field testing of symptomatic individuals in the past has resulted in normal examinations.
9. Negative dysphotopsia symptoms that are present early after surgery generally remit; however, chronic cases, with symptoms persisting several months after surgery, albeit rare, are problematic.
10. No medical therapy is seemingly beneficial; however, surgical management may be successful.

Given the above, there has been interest in identifying the anatomic characteristics of symptomatic patients. Understandably, the posterior chamber is deeper in the pseudophakia eye than in the phakic eye. Based on the concept that the expanded posterior chamber is a potential cause of negative dysphotopsia, Ernest9 implanted a secondary plano piggyback IOL in the ciliary sulcus of a patient, leaving the initial IOL undisturbed. Surgery resulted in elimination of the negative dysphotopsia symptoms. In a more recent study, Vamosi et al.10 used ultrasound biomicroscopy (UBM) to evaluate a series of 5 patients. Although the results in their study showed a deepened posterior chamber in pseudophakic eyes, they did not find a difference in posterior chamber depth between symptomatic patients and asymptomatic patients. In their report, patients with negative dysphotopsia were aided by IOL exchange only if the secondary IOL was placed in the ciliary sulcus. The patients in their series had no improvement if the new IOL was placed in the capsular bag; improvement occurred only if the IOL was placed anterior to the edge of the capsule.

Treatment of negative dysphotopsia has been empirical and based on the theory that the increased posterior chamber depth in the pseudophakic eye or the material or design of the IOL is the cause. We report our clinical and surgical experience with negative dysphotopsia. Ultrasound biomicroscopy was used as it became available to evaluate symptomatic patients before and after a variety of surgical strategies. The surgical outcomes strongly suggest that neither enhanced posterior chamber depth nor IOL material or design is the cause. The results in the study led us to consider a new theory for the etiology of negative dysphotopsia.

PATIENTS AND METHODS

This retrospective case series comprised patients with negative dysphotopsia symptoms after uncomplicated cataract extraction with IOL implantation in the capsular bag, underneath the anterior capsule. Thirteen of the corrective procedures were performed by the same surgeon (S.M.) and 1 by another surgeon. Negative dysphotopsia was defined as the subjective spontaneous description of a dark crescent temporally in the operative eye that improved with dilation and persisted for more than 6 months in an otherwise anatomically normal pseudophakic eye. No patient in the study had corneal, macular, peripheral retinal, or optic nerve abnormalities.

The primary outcome measure was resolution of negative dysphotopsia symptoms 3 months postoperatively. The secondary outcome measure was evaluation of the posterior chamber anatomy and the correlation with negative dysphotopsia symptoms in selected patients as the technology became available in the practice. No attempt to discern the overall frequency or the rate of spontaneous resolution of symptoms was made. Patients were not age or sex matched, and there were no controls because this was a retrospective review. There were no deviations from the follow-up schedule.

Patients with negative dysphotopsia were evaluated in a private practice setting (Advanced Vision Care, Los Angeles, California, USA) between 1998 and 2010. All incisions for primary cataract or secondary cataract surgery were created using the temporal clear corneal approach under topical anesthesia. Incision sizes varied from 2.2 to 3.0 mm, and sutures were placed when appropriate.

Symptomatic complaints of negative dysphotopsia were recorded at the preoperative examination as well as postoperatively at 1 day, 3 days, and 3 months.

Ultrasound biomicroscopy (Accutome, Inc.) was performed using a 48 MHz probe preoperatively and postoperatively in selected patients as the technology became
available in the private practice setting. The light intensity at the patient’s head was between 60 and 70 foot-candles. The distance between the posterior surface of the iris and the anterior surface of the IOL was measured at the 3, 6, 9, and 12 o’clock positions. The mean of the 4 measurements was considered to be the iris–IOL distance. The position of the IOL in relation to the anterior capsular bag was also evaluated.

Surgical methods used to address negative dysphotopsia were secondary piggyback IOL implantation, reverse optic capture, in-the-bag IOL exchange, and iris suture fixation of the capsular bag–IOL complex.

Secondary Piggyback Intraocular Lens

This strategy involved placement of a 3-piece IOL in the ciliary sulcus, anterior to the IOL–capsular bag complex, which was left undisturbed. The piggyback IOL power was between −1.00 diopter (D) and +1.50 D, varying with any associated ametropia.

Reverse Optic Capture

Patients in this category had negative dysphotopsia symptoms after implantation of a 1-piece acrylic IOL (AcrySof IQ Toric SN60T3, Alcon Laboratories, Inc.). At surgery in these 3 cases, the capsule edge was freed from the anterior surface of the previously placed IOL by blunt dissection, aided by an ophthalmic viscosurgical device (OVD). The optic edge was partially elevated above the anterior capsule with a spatula; however, the portion of the optic adjacent to the haptic remained in the confines of the capsular bag.

In-the-Bag Intraocular Lens Exchange

Case 1 was an earlier patient who had severe negative dysphotopsia symptoms for more than 6 months after implantation of a 1-piece acrylic IOL (AcrySof MA30BA, Alcon Laboratories, Inc.). The IOL was exchanged for an SI-40NB IOL (Abbott Medical Optics). Case 2 was markedly symptomatic for 10 months after implantation of a 1-piece acrylic IOL (AcrySof SA60AT, Alcon Laboratories, Inc.). At surgery, the capsular bag was opened by blunt dissection under protection of an OVD and the IOL was folded within the anterior chamber and removed through a corneal incision. A 3-piece silicone IOL (AQ2010V, Staar Surgical Co.) was implanted in the capsular bag. The last patient in this category (Case 3) had an IOL exchange by a previous surgeon in similar fashion to Case 2.

Iris Suture Fixation of Capsule Bag–Intraocular Lens Complex

Case 3 was a patient with negative dysphotopsia symptoms after implantation of a 1-piece acrylic IOL (AcrySof SA60AT). A second surgeon exchanged the IOL for a 3-piece silicone IOL (AQ2010V), noted zonular weakness, and placed a capsular tension ring (CTR); however, the symptoms were unchanged. Given a deep posterior chamber on UBM and the question of zonular stability to sustain positioning of a piggyback IOL, the IOL–capsular bag complex was fixated to the iris with 10-0 polyester sutures, incorporating the haptics of the IOL. Surgery was successful as significantly reducing posterior chamber depth (Figure 1).

RESULTS

The study included 14 procedures in 12 eyes of 11 patients. Secondary piggyback IOL implantation was performed in 7 cases (AQ5010V IOL, Staar Surgical, MA; Clariflex IOL, Abbott Medical Optics, Inc., in 1 case), reverse optic capture in 3 cases, in-the-bag IOL exchange in 3 cases, and iris suture fixation of the capsular bag–IOL complex in 1 case. All patients having secondary piggyback IOL implantation or reverse optic capture reported partial or complete resolution of symptoms by 3 months postoperatively. Patients receiving in-the-bag IOL exchange or iris suture fixation of the capsular bag–IOL complex had no improvement despite UBM-confirmed collapse of the posterior chamber (Table 1).

Representative Cases

Case 4 A 69-year-old man had uneventful phacoemulsification cataract extraction in the left eye. In-the-bag implantation of a PC IOL (+22.50 D AcrySof SN60WF, Alcon Laboratories, Inc.) was performed through a 2.2 mm temporal CCI. Fourteen days postoperatively, the patient reported a dark shadow temporally in the left eye. His description was consistent with negative dysphotopsia. Furthermore, the symptoms improved with dilation and persisted for more than 6 months. Slitlamp examination showed a well-centered PC IOL; the optic edge was covered by the anterior capsule for 360 degrees, and the posterior
capsule was clean. A secondary piggyback IOL (+0.00 D AQ5010V) was implanted in the ciliary sulcus in the left eye without complication. Negative dysphotopsia symptoms were improved at all postoperative visits. Ultrasound biomicroscopy showed a well-positioned piggyback IOL and a mean iris-IOL distance of 0.0 mm with complete anterior capsule coverage for 360 degrees (Figure 2).

In the fellow right eye, phacoemulsification with prophylactic reverse optic capture technique was performed. A 3-piece PC IOL (+21.50 D MN60AC, Alcon Laboratories, Inc.) was implanted with the haptics in the bag and the optic deliberately prolapsed anterior to the edge of the capsulorhexis. In addition, a 13.0 mm CTR (Abbott Medical Optics, Inc.) was placed in the capsular bag. Postoperatively, the patient was asymptomatic. Negative dysphotopsia symptoms were not described at any point during the postoperative period. Slitlamp examination showed a well-positioned IOL with reverse optic capture and transient capsular bag distention syndrome (capsular block) (Figure 3). The patient was asymptomatic at the last follow-up visit.

**Case 5** An 83-year-old woman had uneventful cataract extraction and multifocal PC IOL implantation (+15.00 D SN60D3 Restor, Alcon Laboratories, Inc.) through a 2.2 mm temporal CCI in the right eye.

<table>
<thead>
<tr>
<th>Case</th>
<th>Initial Procedure</th>
<th>Sex</th>
<th>Eye</th>
<th>Surgical Method 1</th>
<th>Symptoms Improved?</th>
<th>Surgical Method 2</th>
<th>Symptoms Improved?</th>
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<tr>
<td>1</td>
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<td>IOL exchange, SI-40NB; bag/bag</td>
<td>No</td>
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<td>—</td>
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<td>2</td>
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<td>M</td>
<td>L</td>
<td>IOL exchange, AQ2010V; bag/bag</td>
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<td>Piggyback, AQ5010V</td>
<td>Yes</td>
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<td>3</td>
<td>AcrySof SN60AT IOL</td>
<td>M</td>
<td>L</td>
<td>IOL exchange, AQ2010V/CTR; bag/bag</td>
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<td>Iris-suture fixation (capsular bag/IOL complex)</td>
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<td>L</td>
<td>Piggyback, AQ5010V</td>
<td>Yes</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
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<td>F</td>
<td>R</td>
<td>Piggyback, AQ5010V</td>
<td>Yes</td>
<td>—</td>
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<td>Piggyback, Clariflex</td>
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<td>Crystalens IOL</td>
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<td>L</td>
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**Table 1.** Patient descriptions and surgical method.

IOL = intraocular lens

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**Figure 2.** Ultrasound biomicroscopy shows a piggyback secondary IOL in the ciliary sulcus anterior to an in-the bag PC IOL (IOL = intraocular lens; PC = posterior chamber).

**Figure 3.** Ultrasound biomicroscopy shows reverse optic capture with the optic deliberately placed in the sulcus and anterior to the capsulorhexis edge and the haptics in the capsular bag (CTR = capsular tension ring; IOL = intraocular lens).
At the 2-week postoperative visit, she noted darkness in her peripheral visual field. The symptoms persisted for more than 6 months and improved with pharmacologic pupil dilation. Anterior and posterior segment examinations were unremarkable. In addition, Humphry visual field testing was normal. Slitlamp examination showed a well-positioned PC IOL and clean capsule with 360-degree coverage of the optic edge by the anterior capsule. A secondary piggyback IOL (+0.0 D AQ5010V) was implanted, and the symptoms of negative dysphotopsia were significantly improved by the 2-week follow-up examination.

A prophylactic reverse optic capture technique was performed in the fellow left eye (+16.00 D MN60D3 Restor, Alcon Laboratories, Inc.); the optic was left in the sulcus with the haptics confined to the capsular bag. However, by 1 month postoperatively, there was rapid fibrotic posterior capsule opacification (PCO) (Figure 4). A neodymium:YAG posterior capsulotomy was performed 2 months after surgery. The patient has remained asymptomatic in both eyes as of the last follow-up visit.

Cases 10 and 11 A 54-year-old woman had uneventful bilateral phacoemulsification with in-the-bag placement of 1-piece IOL (AcrySof Toric SN60T4, Alcon Laboratories, Inc.) in March 2007 by a previous surgeon. She was referred with complaints of peripheral darkness in both eyes for more than 6 months. Anterior and posterior examinations were unremarkable. A reverse optic capture was performed in both eyes, freeing the anterior capsule edge from the optic and elevating the optic anterior to the capsule edge while leaving the haptics in the capsular bag. The patient’s symptoms of negative dysphotopsia were resolved at the last postoperative visit.

Postoperatively, the posterior chamber depth and relationship of the optic to the anterior capsule were evaluated by UBM. The negative dysphotopsia symptoms in the right eye were alleviated with reverse optic capture; both nasal and temporal edges remained anterior to the capsulorhexis (Figure 5, A). Of note, in the left eye, the nasal edge of the optic remained anterior to the edge of the capsulorhexis (Figure 5, B) whereas the temporal edge of the optic slipped back into the capsular bag, causing the optic to tilt (Figure 5, C). Despite optic tilt and only partial reverse optic capture, the symptoms of negative dysphotopsia were fully resolved.

Case 3 A 69-year-old man had uneventful cataract surgery in the left eye in April 2008 with placement of a 1-piece acrylic IOL (AcrySof SA60AT) and developed negative dysphotopsia, which he described as a dark blur temporally in the operative eye. His symptoms were minimally relieved with dilation. A previous surgeon performed an in-the-bag IOL exchange (+19.00 D, AQ2010V) and placed a CTR because of the marked zonular weakness. The in-the-bag IOL exchange did not relieve the negative dysphotopsia symptoms. Examination showed a temporal CCI, healed limbal relaxing incisions at 12 o’clock and 6 o’clock, modest iridodonesis, and a 3-piece silicone IOL in a clean capsular bag. Because there was evidence of zonular instability and a deep posterior chamber, consideration was given to bringing the capsular–bag complex anteriorly by suture fixating the haptics to the iris, effectively reducing posterior chamber depth. Iris suture fixation of the capsular bag-IOL complex was performed; however, despite a significant reduction in posterior chamber depth (Figure 1), negative dysphotopsia symptoms persisted unchanged.

DISCUSSION

At present, there are no clinical testing devices to determine the presence or extent of negative dysphotopsia symptoms; therefore, we are fully reliant on patient-reported observations to evaluate the benefit of any form of therapy. Although not previously reported in the literature, pharmacologic alteration of pupil size yields seemingly paradoxical results, with symptoms increasing with miotic agents and decreasing with mydriatic agents. Our clinical experience strongly suggests that only surgery is beneficial in reducing symptoms because chronic pupil dilation is not
a satisfactory long-term treatment option in pseudophakia. Surgical strategies, as discussed here, have included IOL exchange, secondary piggyback IOL placement, reduction in posterior chamber depth, and reverse optic capture. The success or failure of any of these modes is determined purely from patient-reported observations of their negative dysphotopsia symptoms. In this study, we report success with secondary piggyback IOL implantation and with reverse optic capture. However, in-the-bag IOL exchange for a different IOL material or design and reducing the depth of the posterior chamber failed to improve patient-reported symptoms of negative dysphotopsia.

As mentioned, it has been suggested that exchanging the existing and "offending" IOL for an IOL of different material or edge design would reduce negative dysphotopsia symptoms. However, in a study by Vamosi et al., IOL exchange failed to alleviate symptoms when the alternate new IOL was implanted in the capsular bag; the symptoms improved only if the new IOL was placed in the ciliary sulcus. This finding is in keeping with the present case series in that negative dysphotopsia symptoms were not alleviated (Cases 1, 2, and 3) when the exchanged second IOL optic was placed in the capsular bag. As such, the findings in the present study and in the Vamosi et al. study are in disagreement with the report by Cooke. In his single-case study, a patient had relief of bilateral negative dysphotopsia when acrylic IOLs were exchanged for in-the-bag silicone IOLs. But in contrast to Cooke’s experience, our Case 2 is particularly revealing because 2 procedures were needed to alleviate the symptoms. At the first attempt to eliminate negative dysphotopsia symptoms, an acrylic IOL with a square-edged design (AcrySof SA60AT) was removed and a silicone IOL with rounded edges (AQ2010V) was implanted fully in the capsular bag. The patient’s symptoms were unchanged; however, after sulcus placement of a secondary piggyback IOL (AQ5010V), the negative dysphotopsia symptoms were eliminated. Therefore, our experience suggests that in-the-bag IOL exchange is not a useful method for alleviating negative dysphotopsia symptoms.

Given that the posterior chamber is usually deeper in pseudophakia than in the phakic state, surgical strategies to alleviate negative dysphotopsia by reducing posterior chamber volume have been developed. The results in our study do not agree with this theory, and Case 3 is helpful to our understanding the absence of a relationship between posterior chamber depth and negative dysphotopsia. This patient had...
negative dysphotopsia after uneventful cataract surgery in which a 1-piece acrylic IOL (AcrySof SA60AT) was placed in the capsular bag. A second surgeon exchanged the IOL for a 3-piece silicone IOL (AQ2010V); however, similar to Case 2, the patient’s symptoms were unchanged. In addition, the surgeon noted that zonular integrity was poor, necessitating placement of a CTR concomitant with IOL exchange. After referral to our practice, anterior segment UBM (Figure 1) evaluation suggested a very deep posterior chamber. Given the history of weakened zonules, a piggyback IOL was contraindicated. Instead, the capsular bag–IOL complex was successfully sutured to the iris, significantly reducing the depth of the posterior chamber (Figure 1). Nevertheless, negative dysphotopsia symptoms were unchanged, indicating that posterior chamber depth in and of itself is unrelated to negative dysphotopsia.

Another example is a patient who had significant negative dysphotopsia symptoms after uneventful cataract surgery, despite a very shallow posterior chamber. The anterior segment UBM (Figure 6, A) showed a markedly shallow posterior chamber, consistent with high hyperopia before surgery; +30.00 D IOLs (AcrySof SN60WF) were required for emmetropia. Although symptomatic, the patient has not had additional surgery. Thus, her case was not included in the present study’s data analysis. However, her case highlights the concept that expanded posterior chamber depth is not a cause of negative dysphotopsia symptoms.

Finally, Case 9 also had a shallow posterior chamber after uneventful cataract surgery during which a Crystalens IOL (Bausch & Lomb) was implanted. Despite a shallow posterior chamber (Figure 6, B), the patient was highly symptomatic with respect to negative dysphotopsia. Therefore, our findings are in keeping with those of Vamosi et al. and clearly show that exaggerated posterior chamber depth alone is not a cause of negative dysphotopsia.

Furthermore, it is generally held that negative dysphotopsia is associated with hydrophobic acrylic IOLs rather than those of other materials. Nevertheless, and in keeping with the Vamosi et al.’s study, our Cases 1, 2, and 3 were not improved when square-edged hydrophobic acrylic IOLs were exchanged for round-edged silicone IOLs. Moreover, in 3 eyes of 2 patients (Cases 10, 11, and 12), negative dysphotopsia symptoms were alleviated when the edge of the optic was secondarily elevated above the anterior capsulorhexis, creating a reverse optic capture. In these 3 eyes, the negative dysphotopsia symptoms were improved despite retention of the acrylic IOLs. These cases confirm that acrylic IOL material in and of itself is not a cause of negative dysphotopsia.

The outcomes in the present study, in keeping with the existing literature, show that negative dysphotopsia symptoms can be improved by sulcus placement of the IOL (primarily or after IOL exchange), with secondary piggyback IOL implantation, and by reverse optic capture of the IOL optic, suggesting that negative dysphotopsia can be alleviated when the anterior capsulotomy edge is covered by the IOL optic. In all cases in our series treated in that manner, the negative dysphotopsia symptoms decreased. In light of these observations, we considered using reverse optic capture as a prophylactic measure at primary cataract surgery in fellow eyes of patients with significant negative dysphotopsia symptoms after cataract surgery in the first eye. We considered this a more stable alternative than pure sulcus fixation. Although one

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**Table 2.** Cases in which prophylactic reverse optic capture was performed in the asymptomatic eye.

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Eye</th>
<th>IOL</th>
<th>Symptoms of Negative Dysphotopsia?</th>
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<tr>
<td>2</td>
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<td>R</td>
<td>CQ2015A/CTR</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>R</td>
<td>MN60AC/CTR</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>L</td>
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</tr>
<tr>
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<td>F</td>
<td>L</td>
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<td>No</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>R</td>
<td>CQ2015A/CTR</td>
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</table>

CTR = capsular tension ring; IOL = intraocular lens
cannot be certain that these eyes would have sustained negative dysphotopsia symptoms, all were fully free from symptoms after surgery. We used this strategy in 5 eyes of 5 patients; Table 2 shows the results. We observed rapid fibrotic PCO and capsule contraction in the first eye in which we used this technique (Figure 4). In subsequent cases, we carefully removed the anterior subcapsular lens epithelial cells and placed a CTR concomitant with prophylactic reverse optic capture to reduce capsular striae and reduce fibrosis (video; available at http://jcrsjournal.org).11,12

The current study strongly suggests that negative dysphotopsia is prevented or reversed when the IOL optic covers the anterior capsulotomy edge. On the other hand, in our investigation simply reducing posterior chamber depth was not a successful strategy, nor was in-the-bag IOL exchange for an IOL of different material. Given these observations, it would appear that negative dysphotopsia is likely induced at the interface of the anterior capsulotomy and the front surface of the PC IOL, suggesting that a reflection of the anterior capsulotomy edge is projected onto the nasal peripheral retina. Another example is illustrative; Cases 10 and 11 (2 eyes of the same patient) had reverse optic capture of toric IOLs for negative dysphotopsia. After the secondary surgery, the temporal edge of the optic fell behind the anterior capsular edge while the nasal aspect of the optic remained anterior to the nasal edge of the capsule. These findings are shown in Figure 5, B and C. The UBM showed that IOL tilt was induced, yet anterior capture of the nasal edge of the optic was sufficient to eliminate the patient’s symptoms. This example backs the theory that the anterior capsulotomy is the root cause of negative dysphotopsia symptoms. This concept could account for some clinical observations associated with negative dysphotopsia as follows:

1. Negative dysphotopsia has been reported with a variety of IOLs.
2. Symptoms increase with pupil constriction and decrease with dilation.
3. Negative dysphotopsia is stimulated by a point source of light in the patient’s temporal field.
4. Negative dysphotopsia has not been reported with radial keratotomy, penetrating keratoplasty, or other forms of corneal surgery.3

The findings in the current study, therefore, suggest that Osher’s “incision shadow” is an unlikely cause of chronic negative dysphotopsia. However, of interest is that Osher reported that negative dysphotopsia symptoms were common early after surgery but were less frequent with passage of time. It remains unclear, other than for neuroadaptation, why patients would report reduced negative dysphotopsia symptoms over time.

In a preliminary study,13 nonsequential ray tracing was used to test the proposed etiology of negative dysphotopsia and the theoretical benefit of a piggyback IOL. The analyses appear to confirm both hypotheses. It appears, therefore, that negative dysphotopsia can be considered a complication of anterior capsulorhexis with in-the-bag implantation of an IOL. Although the latter has been the preferred method for anterior capsulotomy and IOL implant surgery over many years and offers advantages over noncontinuous capsulotomy methods, complications have been reported with this method.14 In view of the findings in the present study, new IOL designs might be considered to preclude negative dysphotopsia symptoms.

The current study appears to be the largest reported clinical series of cases treated for negative dysphotopsia. This study simply proposes a new hypothesis for the etiology of negative dysphotopsia. The hypothesis implicates the anterior capsulorhexis as causal and provides further evidence that negative dysphotopsia is not relieved by collapsing posterior chamber depth alone, exchanging IOL material or design, or a combination. However, there are limitations of this study. It was not case-controlled or a prospective study, there was a small sample size, there was no age or sex matching, and although the strategies have been successful and a new understanding of the cause is apparent, the study is greatly limited by the absence of any objective means to test for negative dysphotopsia. Given that we are limited to subjective patient responses, many additional cases must be evaluated and other theories of causation considered.

REFERENCES

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