massage from orbicularis mediated lid closure for some time in the postoperative period. Indeed, these cases present recalcitrant, recurrent epithelial ingrowth occurring on the side with weaker eyelid closure, one from weak orbicularis tone associated with previous Bell's palsy and the other from previous floor fracture and orbital surgery.

The right eye in case 1 had the additional risk factor of an enhancement surgery. There are several studies suggesting the rate of epithelial ingrowth is higher after flap lifting enhancement surgery. 5,6 However, a study of 3786 eyes found the incidence of epithelial ingrowth was not statistically different between primary and enhancement LASIK.7 Furthermore, the epithelial ingrowth in case 1 initially was noted prior to enhancement. The literature also suggests higher hyperopic corrections are a risk factor for epithelial ingrowth due to a larger exposed gutter from flap-stromal bed mismatch.8 In case 1, however, primary epithelial ingrowth occurred in the eye with the much lower hyperopic correction, whereas no epithelial ingrowth was noted in the contralateral eye that had a higher hyperopic correction. This finding seems to implicate another mechanism for ingrowth besides flap-bed mismatch.

These cases of recurrent epithelial ingrowth suggest compromised eyelid closure may be a predisposing factor to poor flap adhesion and subsequent epithelial ingrowth, presumably by allowing an epithelial fistula to develop in the immediate postoperative period from insufficient closure of the potential space between the flap and the bed. Close attention to lid function may be of importance in deciding between LASIK and photorefractive keratectomy, particularly in patients with other risk factors for epithelial ingrowth.

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Long-term Endothelial Cell Loss After Traumatic Dislocation and Repositioning of Artisan Phakic IOL

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ABSTRACT

PURPOSE: To evaluate long-term endothelial cell loss after traumatic dislocation and repositioning of an Artisan phakic intraocular lens (PIOL).

METHODS: Traumatic PIOL dislocation occurred in the patient's left eye 4 months after uneventful implantation for unilateral congenital myopia. Using the Konan semi-automated analysis method, endothelial cell density was measured preoperatively, before Artisan repositioning, and 1, 2, and 4 years after primary implantation.

RESULTS: Endothelial cell density was 2770 cells/mm² preoperatively and 2634 cells/mm² before Artisan repositioning. After successful repositioning, endothelial cell density progressively decreased—1, 2, and 4 years from primary implantation, endothelial cell density was 2582, 2524, and 2538 cells/mm², respectively, corresponding to losses of 6.8%, 8.9%, and 8.4%, respectively.

CONCLUSIONS: Progressive and long-term endothelial loss after traumatic dislocation and repositioning of the Artisan PIOL may be comparable to that reported after uneventful implantation. [J Refract Surg. 2008;24:546-548.]

rtisan phakic intraocular lenses (Ophtec, Groningen, Netherlands [PIOL]) are iris-supported lenses implanted in the anterior chamber in

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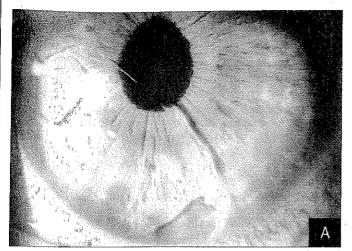
The authors have no financial or proprietary interest in the materials presented herein.

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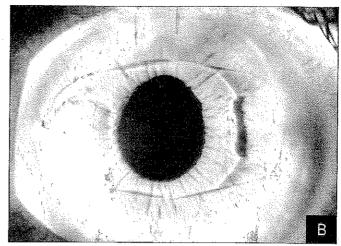


Figure. A) Dislocation of Artisan PIOL in the left eye after trauma. The lower edge of the optic occupies the angle in the inferonasal sector, and the upper edge of the optic is at the edge of the pupillary area. B) Artisan PIOL in situ after successful repositioning.

front of the pupillary area, for the refractive correction of high myopia, hyperopia, and astigmatism. Iris fixation is achieved with two pincer-like haptics (claws), through which a fold of the peripheral iris is drawn during implantation, in a process known as enclavation.

In eyes successfully implanted with the Artisan PIOL, enclavation may be lost spontaneously⁴ or after blunt ocular trauma.^{2,5} Suggested management of this complication includes PIOL removal,^{2,5} exchange,⁶ and repositioning.^{7,8} Although visual outcome after Artisan PIOL repositioning may be good,^{7,8} long-term effects on endothelial cell density have not been determined. A case of traumatic disenclavation with consequent dislocation within the anterior chamber of an Artisan PIOL is described and the long-term endothelial loss that occurred after repositioning of the lens is reported.

CASE REPORT

An Artisan 206 PIOL was implanted in the left eye of a 22-year-old man at the University of Turin Ophthalmic Clinic in November 2002 to correct unilateral congenital myopia ($-16.50 -1.00 \times 80^{\circ}$). Preoperatively, uncorrected visual acuity (UCVA) and best spectacle-corrected visual acuity (BSCVA) in the left eye were 20/2000 and 20/30, respectively. Before surgery, ophthalmic evaluation included examination of the central corneal endothelium with the Konan CC7000 center method of analysis (Konan Medical Inc. Nishinomiya, Japan). 9,10 Applying this method on a single, clear endothelial image and counting more than 100 contiguous cells, cell density, coefficient of variation, and hexagonality were 2770 cells/mm², 23%, and 78%, respectively. No postoperative complications were noted.

One month postoperatively, the Artisan PIOL was well-centered and enclavated. Uncorrected visual

acuity and BSCVA had improved to 20/40 and 20/25, respectively, with refraction of $-0.50 - 1.00 \times 110^{\circ}$. Three months later, the patient presented to our institution the day after severe ocular trauma (hit by an elbow while skiing) with complaint of monocular diplopia and decreased vision in the left eye. Neurological examination and orbital computed tomography scans were normal. The surgical wound was intact in the left eye and the cornea showed slight edema. The Artisan's temporal claw was no longer enclavated, and the PIOL was dislocated within the anterior chamber without touching the corneal endothelium (Fig A). Uncorrected visual acuity was 20/100, improving to 20/50 with a pinhole. The intraocular pressure was 10 mmHg and no traumatic alterations of the vitreous or retina were observed. Analysis of the corneal endothelium (with the method described above) revealed that cell density, coefficient of variation, and hexagonality were 2634 cells/mm², 37%, and 61%, respectively.

The PIOL was repositioned the day after presentation. After peribulbar anesthesia, a 3-mm scleral tunnel was prepared at 12 o'clock and acetylcholine chloride and Healon GV (Advanced Medical Optics Inc, Santa Ana, Calif) were injected into the anterior chamber through a paracentesis of 1.2 mm at 2 o'clock. The PIOL was grasped and enclavated in its original position using implantation forceps and a blunt 30-G blended needle. Finally, the scleral tunnel was sutured with two interrupted 10.0 nylon sutures.

No postoperative complications occurred. One week after repositioning (Fig B), UCVA and BSCVA were 20/60 and 20/25, respectively, with $-1.00-1.50\times100^{\circ}$. The patient was then re-examined and specular microscopy was repeated using the same analysis method at 1, 2, and 4 years after primary PIOL implantation. At

these intervals, cell density was 2582, 2524, and 2538 cells/mm², respectively, coefficient of variation was 31%, 28%, and 27%, respectively, and hexagonality was 64%, 67%, and 70%, respectively. At last follow-up, UCVA and BSCVA were 20/80 and 20/25, respectively, with $-0.75-1.75\times115^\circ$.

DISCUSSION

Despite adequate positioning of the Artisan PIOL, blunt ocular trauma may lead to complete freeing of the iris from the claw haptics with consequent PIOL disenclavation and dislocation. This complication has been reported in two eyes, ^{7,8} which were successfully managed with Artisan repositioning. However, due to the absence of preoperative endothelial cell density values and because follow-up was short, these studies did not investigate the long-term endothelial loss that occurred after PIOL repositioning. This is an important issue as progressive endothelial loss has been reported after Artisan primary implantation, ^{1,5} and in eyes with posttraumatic dislocation it may be further increased by the trauma itself and/or by the surgery required for PIOL repositioning.

This report describes traumatic disenclavation of the Artisan PIOL and assesses the long-term endothelial loss that occurred after repositioning. Artisan repositioning was scheduled as neither iris tissue nor claw haptics had been damaged by the trauma or by disenclavation. Moreover, the trauma, which occurred 4 months after primary implantation, had not caused severe endothelial damage. The endothelial loss (4.9%), reduction of hexagonal cells (pleomorphism), and greater variability of cell size (polymegatism) were presumably due partly to the initial surgery. Larger drop in endothelial cell density and variation of endothelial morphometric parameters (hexagonality and coefficient of variation) typically occur in the early postoperative period (6 months) after implantation of Artisan PIOL, and mean loss of endothelial cells between 3.8% and 4.8% may be expected. 1,5 Endothelial damage caused by the trauma was probably limited because the nasal claw haptic of the PIOL remained enclavated to the iris, thereby reducing PIOL movement and avoiding prolonged contact between the PIOL and corneal endothelium. Visual recovery was good after Artisan repositioning, but further reduction of endothelial cell density occurred. However, the endothelial loss did not show long-term pathological progression, and endothelial morphometric parameters improved over time. At more than 4 years after primary implantation, endothelial loss was 8.4%. This is comparable or even below the loss reported in some large-series studies,1,5 in which 3 to 4 years after implantation of myopic Artisan PIOL, mean endothelial cell loss ranged from 9.4% to 13.4%.

This report shows progressive and long-term endothelial cell loss after repositioning of a traumatically dislocated Artisan PIOL to be comparable to eyes with uneventful implantation.

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Spontaneous Bilateral, Recurrent, Late-onset Diffuse Lamellar Keratitis After LASIK in a Patient With Cogan's Syndrome

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ABSTRACT

PURPOSE: To report a patient with severe and progressive bilateral deafness who suffered recurrent episodes of diffuse lamellar keratitis (DLK) in both eyes ≥4 months after undergoing uneventful LASIK for myopia.

METHODS: Four months after LASIK, the patient presented with blurred vision in the left eye. Best spectacle-corrected visual