Phakic intraocular lenses

Part 2: Results and complications

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The second part of a review of phakic intraocular lenses (pIOLs) addresses results and complications with current pIOL models. Phakic IOLs demonstrate reversibility, high optical quality, potential gain in visual acuity in myopic patients due to retinal magnification; correction is not limited by corneal thickness or topography. With proper anatomical conditions, pIOLs also show good results in hyperopic patients. Toric pIOL designs enable spherocylindrical correction. Complications are rare and primarily related to pIOL position and type. The main complications of angle-supported anterior chamber pIOLs are glare and halos, pupil ovalization, and corneal endothelial cell loss; of iris-fixated anterior chamber pIOLs, chronic subclinical inflammation, corneal endothelial cell loss, and dislocation or pupillary block glaucoma; and of posterior chamber pIOLs, anterior subcapsular cataract formation, pigment dispersion, and luxation or pupillary block glaucoma. No causative relationship between pIOL implantation (of any pIOL type) and retinal detachment has been established.

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Implantation of intraocular lenses in the phakic eye (pIOL) is a relatively new technique to correct high ametropia. Time between the introduction of new pIOL designs is short; thus, experience with a new pIOL is short when the pIOL is implanted. New pIOLs are presented to overcome specific complications of older pIOLs. Currently, many studies with short follow-up and various case reports addressing results and complications of pIOLs have been published, but there are few long-term studies of pIOLs that have been on the market for some time. This second part of the pIOL review reassesses the published data about results and complications of currently available pIOLs. The results of pIOLs that have been withdrawn from the market are not discussed. As in Part 1,[1] results and complications are shown for each type of pIOL: angle-supported anterior chamber, iris-fixated anterior chamber, and posterior chamber.

Journal articles were considered for this review article after a thorough literature search. A Medline (National Library of Medicine, Bethesda, Maryland, USA) search from 1994 to 2009 was performed to identify all articles describing pIOLs. The terms *intraocular lens* and *intraocular lens implantation* from the Medical Subject Headings (MeSH) and the text word "phakic" were used for a broad and sensitive search. Five other searches were performed to look for additional articles (using the text words "phakic" and "lens," "phakic" and "IOL," "anterior chamber lens," "iris fixated lens," and "posterior chamber lens." All abstracts from the Medline search were read to identify articles that were pertinent to clinical results, surgical techniques, or complications of anterior chamber, iris-fixated, and posterior chamber pIOLs. Copies of the articles were obtained and the bibliographies searched manually for additional articles published in peer-reviewed journals. Complete articles were reviewed

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to identify those that reported original clinical data or complication(s) of pIOLs. Articles that covered previously published cases were included if they added new cases or up-to-date results.

FUNCTIONAL RESULTS OF pIOLs

To provide an overview, results of published data for the pIOL types are shown in Tables 1 to 3.

Results of Angle-Supported Anterior Chamber pIOLs

Visual acuity, predictability, efficacy, and safety of the Baikoff ZB5M (Domilens Corp.), Kelman Duet ZSAL-4 (Tekia, Inc.), I-Care (Corneal Laboratories, Inc.), Vivarte (Ioltech), and AcrySof Cachet (Alcon, Inc.) pIOL models are shown in Table 1.[2–11] For the Vivarte pIOL, results of only the refractive bifocal Vivarte pIOL are included.[10] At the time this review was written, no peer-reviewed studies of the Thin-PhAc (Thin Opt-X) and Vision Membrane (Vision Membrane Technology) pIOLs had been published. Despite the long period in which anterior chamber pIOLs have been available, few long-term studies exist.[3,4] Anterior chamber pIOLs generally demonstrate good predictability, efficacy, and safety. However, there is a tendency toward undercorrection of the refractive error.

Results of Iris-Fixated Anterior Chamber pIOLs

Visual acuity, predictability, efficacy, and safety of the Artisan (Ophtec BV)/Verisyse (Abbott Medical Optics, Inc.), toric Artisan/Verisyse, and Artiflex/Veriflex iris-fixated anterior chamber pIOL models are shown in Table 2.[9,12–39] Several studies have long follow-up. The nontoric and toric models demonstrate good predictability, efficacy, and safety. With the toric pIOL models, larger amount of preoperative astigmatism can be managed successfully. Several studies address clinical outcome after toric pIOL implantation.[27,30,36,37,39] Recently, Güell et al.[27] reported a larger series with a mean follow-up of 3 years after implantation of the toric Artisan pIOL. The toric Artiflex is currently undergoing a multicenter clinical trial; it has shown excellent interim efficacy and safety results in the first 6 months of follow-up.

Results of Posterior Chamber pIOLs

Visual acuity, predictability, efficacy, and safety of the implantable Collamer Lens (ICL) (Staar Surgical Co.) and the Phakic Refractive Lens (PRL) (Carl Zeiss Meditec) posterior chamber pIOL models are shown in Table 3.[3,18,33,40–67] The safety and efficacy of these 2 pIOL models are good. In a United States Food and Drug Administration (FDA) study, the ICL pIOL showed good functional results with a low complication rate.[41] In a prospective study comparing matched populations of laser in situ keratomileusis (LASIK) and Visian ICL implantation, the ICL performed better than LASIK in almost all measures of safety, efficacy, predictability, and stability.[54] In a few case reports, results with the toric posterior chamber pIOL have been shown.[59,68,69] Schallhorn et al.[56] report better results with the toric ICL than with conventional photorefractive keratectomy in a randomized prospective comparison of safety, efficacy, predictability, and stability.

In summary, pIOLs show good refractive and clinical results. They demonstrate reversibility, high optical quality, potential gain in visual acuity in myopic patients due to retinal magnification, and correction is not limited by corneal thickness or topography. With proper anatomical conditions (especially sufficient anterior chamber depth [ACD]), pIOLs also show good refractive and clinical results in hyperopic patients.[70] Phakic IOLs preserve corneal architecture, asphericity, and accommodation. With recent innovations in the design of toric pIOLs, spherocylindrical correction is also feasible. However, pIOL implantation is not without complications. The spectrum of common and rare complications with each type of pIOL is presented in the following section.

COMPLICATIONS OF pIOLs

General Complications of Intraocular Surgery

With the increasing use of topical or parabulbar anesthesia, complications due to anesthesia such as retrobulbar hemorrhage, penetration of the globe, or life-threatening systemic side effects from accidental injection into the optic nerve are very rare. Because implantation of a pIOL is an intraocular procedure, it bears a potential risk for the development of postoperative endophthalmitis. The risk for this complication in general cataract surgery with implantation of a posterior chamber IOL is 0.1% to 0.7% with an optimal antiseptic perioperative treatment regimen.[71] Recently, a prospective randomized multicenter study by the European Society of Cataract and Refractive Surgeons[72] showed that an additional intracameral application of cefuroxime after cataract surgery significantly reduced the rate of postoperative endophthalmitis. Only one case of postoperative endophthalmitis after pIOL implantation has been reported.[73] In this case, endophthalmitis developed on the first day after anterior chamber pIOL implantation and was caused by β-hemolytic streptococci. Intraoperative sterility and meticulous postoperative follow-up examinations may help prevent this severe complication or enable early and aggressive treatment.

Table 1. Visual acuity, predictability, efficacy, and safety of angle-supported anterior chamber pIOLs.

					Efficacy				
Type of pIOL/Study*	Number of Eyes	Follow-up (Mo)	Mean Preop SE	Mean Postop SE	Postoperative ±0.5 D [%]	Postoperative ±1.0 D [%]	Postoperative UCVA ≥ 1.0 [%]	Postoperative UCVA ≥ 0.5 [%]	Efficacy Index
ZB5M									
Baikoff[2]	133	6–36	−12.5	−1.3	40	65	No data	No data	No data
Utine[3]	37	24–145	−17.45	−1.76	No data	No data	No data	No data	0.79
Javaloy[4]	225	12–144	−17.23	−1.80	No data	39.28	No data	34.69	1.26
ZSAL-4									
Pérez-Santoja[5]	23	24	−19.56	−0.55	56.5	82.6	0	54.5	1.12
Leccisotti[6]	12	12	−10.23 (keratoconus)	−1.31	67	100	0	100	0.77
Leccisotti[7]	190	12	−14.37	1.55	19	40	~7	~60	0.78
Kelman Duet									
Alió[8]	169	1–12	−14.26	−0.15	57.72	81.30	28.68	83.72	1.19
I-CARE									
Gierek-Ciacura[9]	20	12	−15.76	No data	85	100	No data	85	1.58
Vivarte Presbyopic									
Baikoff[10]	55	0.5–21	+1.8 (-5 to +5)	−0.12	No data	No data	No data	84 (≥ 0.6)	0.80
AcrySof									
Kohnen[11]	190	12	−10.38	−0.23	72.7	95.7	85.7	No data	1.04

CDVA = corrected distance visual acuity; SE = spherical equivalent; UDVA = uncorrected distance visual acuity
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Angle-Supported Anterior Chamber pIOL Complications

Loss of Corneal Endothelial Cells The main concern with anterior chamber pIOLs is the loss of corneal endothelial cells or damage to the endothelial integrity (Figure 1). Excessive corneal endothelial cell loss was, along with pupil ovalization, the main reason for recalling several anterior chamber pIOLs from the market, as described in part 1. Exact preoperative examination should exclude patients with low corneal endothelial cell counts or with shallow anterior chambers because the risk to corneal endothelial cells increases as the distance between the pIOL and the endothelium decreases. In a 7-year follow-up study, Alió et al.[74] report an early postoperative loss of corneal endothelial cells of 3.8%, gradually decreasing to about 0.5% per year after the second postoperative year. In this study, the ZB5M/ZB5MF was evaluated for the full 7 years and the ZSAL-4, for only 4 years. The percentage of corneal endothelial cell loss over 7 years was 8.4%. Other studies have confirmed the initial significant corneal endothelial cell loss and the reduction of this tendency in the second postoperative year.[2,5,75] At 2 years, the corneal endothelial cell loss was 12% for the NuVita pIOL (Bausch & Lomb) and 4.2% for the ZSAL-4; at 3 years, it was 4.8% for the ZB5M. In a study of the reasons for pIOL explantation by Alió et al.,[76] corneal endothelial cell loss was the cause in 24%. In the study with the longest follow-up (up to 12 years) after pIOL implantation, Javaloy et al.[4] report an initial reduction in corneal endothelial cells of 10.6% in the first year followed by a mean annual decrease rate of 1.8% after ZB5M pIOL implantation. The mean corneal endothelial cell loss after implantation of an I-Care pIOL was 6.1% after 1 year, as reported by Gierek-Ciaciura et al.[9] All these anterior chamber pIOLs except the I-Care were poly (methyl methacrylate) (PMMA) rigid IOLs.

In a study of the new flexible anterior chamber pIOL by Baikoff et al.,[10] the corneal endothelial cell loss 1 year after implantation of the Vivarte pIOL was less than 5.0%, but there was a difference between the loss in myopic eyes (2.3%) and that in hyperopic eyes (5.4%). For the AcrySof foldable anterior chamber pIOL, the corneal endothelial cell loss was 4.8% after a 1-year follow-up.[11] In this context, a recent study by Kohnen and Klaproth[77] reports a stable adequate central clearance distance between the AcrySof pIOL and the corneal endothelium over a period of 3 years using Scheimpflug imaging. However, meticulous long-term follow-up of each patient with an anterior chamber pIOL is necessary to detect patients who have significant damage to the endothelium and explant the pIOL whenever clinically necessary.

Pupil Ovalization/Iris Retraction Ovalization of the pupil is a specific complication of anterior chamber pIOLs (Figure 2). The position of haptics in the

Table 1. (Cont.)

Safety					
Loss of 2 or More Lines of CDVA (%)	Loss of 1 Line of CDVA (%)	No Change in CDVA	Gain of 1 Line of CDVA (%)	Gain of 2 or More Lines of CDVA (%)	Saftey Index
No data	No data	No data	No data	No data	No data
3.2	No data	No data	No data	29.8	1.45
3.5	~7	~23	~21	~25	1.50
0	No data	82.6	No data	No data	1.45
0	0	40	50	10	1.18
0	0	~25	~25	~40	1.25
0	~5	~27	~11	56.20	1.37
0	0	5	25	70	No data
No data	No data	No data	No data	No data	0.94
0	1.2	44.7	31.1	23.0	1.25

sclerocorneal angle and their size might lead to mild deformation of the iridosclerocorneal architecture, resulting in iris retraction and pupil ovalization. Alió et al.[74] report mild deformation of pupil shape in 10.3%, which did not affect the refractive, cosmetic, or optical results of surgery.

Severe ovalization causes glare and is unacceptable from a cosmetic point of view. Alió et al.[74] observed this condition in 5.9% of the eyes; it led to pIOL explantation in 2 cases. Allemann et al.[75] report 8 oval pupils in a series of 21 eyes. Pérez-Santonja et al.[5] observed 4 cases in a series of 23 eyes. Leccisotti and Fields[78] report pupil ovalization not associated with any photic phenomena in 11% of eyes after ZSAL-4 anterior chamber pIOL implantation. Javaloy et al.[4] report a cumulative incidence of 34.7% of pupil ovalization after ZB5M implantation within 12 years of follow-up. In an analysis of a series of anterior chamber pIOL explantations (ZB5M pIOL) by Alió et. al.,[76] marked pupil ovalization extending beyond the edges of the pIOL was the reason for pIOL removal in 10% of cases. For the novel AcrySof anterior chamber pIOL implanted in 190 eyes, no case of pupil ovalization was reported.[11] Iris retraction with oval pupil deformation remains primarily a concern of anterior chamber pIOLs. This together with potential damage to endothelial cells are the major objections to the anterior chamber pIOL design.

Topical use of miotic agents should be considered in the early postoperative phase if pupil ovalization associated with glare is detected. Minor pupil ovalization requires observation only, but gross ovalization indicates entrapment of the iris root and ovalization may become irreversible if the pIOL is not explanted promptly.

Optical Quality, Glare, Halos One disadvantage of anterior chamber pIOLs is that they are positioned in front of the pupil, with edge effects a potential source of optical aberrations. Furthermore, the relationship between pupil size and the center of the pIOL optic is a crucial factor that should be evaluated and discussed preoperatively. Sometimes the anterior chamber pIOL optic center and the pupil center are not coincident. If the scotopic pupil size is significantly larger than the optic of the pIOL, one should be very cautious about implanting a pIOL because it will probably result in postoperative glare and subjective discomfort. The incidence of glare is dependent on the size and position of the optic, which varies in different IOL designs and generations. A study by Maroccos et al.[79] shows that all tested types of pIOLs, in particular posterior chamber pIOLs and anterior chamber pIOLs, lead to decreased nighttime visual performance due to glare and halos.

Topical use of miotic agents should be considered in the early postoperative period if the patient is disturbed by glare and halos. A study of the effects of pIOL implantation on contrast sensitivity showed

Table 2. Visual acuity, predictability, efficacy, and safety of iris-fixated anterior chamber pIOLs.

Type of pIOL/Study*	Number of Eyes	Follow-up (Mo)	Mean SE Preop	Efficacy					
				Postop	Postoperative ±0,5 D [%]	Postoperative ±1,0 D [%]	Postoperative UCVA ≥ 1.0 [%]	Postoperative UCVA ≥ 0.5 [%]	Efficacy Index
Artisan/Verisyse									
Alexander[12]	264	6	−12.76	−0.35	No data	No data	No data	100	No data
Budo[13]	249	6–36	−12.95	−0.6	57	79	34	76.8	1.03
Landesz[14]	67	6–36	−14.70	No data	No data	67	No data	40.9	No data
Landesz[15]	78	6–24	−17.00	−2.0	50	68	30	73	No data
Maloney[16]	155	0.5–6	−12.69	−0.54	55	90	26	83	No data
Malecaze[17]	25	12	−10.19	−0.95	24	60	No data	60	0.71
Menezo[18]	137	38–154	−16.17	−0.78	No data	No data	4	81	No data
Lifshitz[19]	31	3	−11.25	−0.50	67.8	96.8	93.5	No data	0.95
Benedetti[20]	68	4–24	−11.8	−0.91	44.1	69.1	25	83.8	0.84
Benedetti[20]	25	4–24	−18.9	−1.20	32	52	8	68	0.90
Senthil[21]	60	24	−12.5	No data	73.3	90	5	75	0.93
Coullet[22]	31	12	−10.3	−1.01	No data	58	No data	51.6	0.60
Moshirfar[23]	85	6–24	−12.2	−0.50	55	84	10	84	No data
Gierek-Ciacura[9]	20	12	−15.73	No data	65	95	No data	80	1.71
Tahzib[24]	89	60	−10.36	−0.70	43.8	68.8	No data		0.80
Stulting[25]	662	12–36	−12.3	No data	71.7	94.7	34.6	88	No data
Silva[26]	26	12–60	−12.30	−0.44	74	95	74	95	No data
Güell[27]	101	12–60	−19.8	−0.50	9.9	22.8	No data	14.8	0.86
Güell[27]	173	12–60	−11.27	−0.64	37.6	57.2	2.9	42.8	0.74
Fechner[28]	67	12–120	+9.98	0.07	No data	No data	~1.5	~35	No data
Alió[29]	29	12–24	+6.06	0.1	79.3	96.6	6.9	65.5	0.83
Alió[29]	28	12–24	+5.88	0.55	50	71.4	3.6	46.4	0.70
Dick[30]	22	6	+3.25	−0.24	50	100	18	96	No data
Saxena[31]	17	3–36	+6.8	−0.03	59	81	58.8	94	No data
Pop[32]	19	1–2	+5.89	−0.03	50	78	No data	89	No data
Güell[27]	41	12–60	+4.92	−0.02	34.8	64.2	0	42.8	0.9
Boxer Wachler[33]	31	3	−12.31	−0.78	58	68	55	90	No data
Coullet[22]	31	12	−9.50	−0.58	No data	83.9	No data	77.4	0.79
Dick[34]	290	24	−7.33	−0.15	75.2	94.3	No data	97.2	1.00
Toric Artisan/ Verisyse									
Tehrani[35]	29	6	−1.9	−0.56	No data	95	No data	~85	No data
Dick[30]	70	6	−3.74	−0.7	72	100	10	88.6	1.03
Güell[36]	27	12	−3.43	No data	62.9	96.2	No data	No data	No data
Alió[37]	8	6–12	Mixed astigmatism +3.6	+0.40	75	87.5	12.5	87.5	1.0
Alió[37]	8	6–12	Myopic astigmatism --8.6	−1.1	62.5	75	12.5	62.5	1.2
Alió[37]	9	6–12	Hyperopic astigmatism +5.9	+0.50	44.4	77.8	33.3	66.6	1.0
Güell[27]	84	12–48	−0.09	No data	66.6	81.3	7.1	65.4	0.93
Toric Artisan/Verisyse post keratoplasty									
Nujits[38]	16	3–18	−6.6	−1.42	0	31.25	0	50	No data
Toric Artisan in keratoconus									
Venter[39]	18	6–12	−4.64	−0.46	No data	78	22	100	No data

CDVA = corrected distance visual acuity; SE = spherical equivalent; UDVA = uncorrected distance visual acuity
*First author

Table 2. (Cont.)

Safety					
Loss of 2 or More Lines of CDVA (%)	Loss of 1 Line of CDVA (%)	No Change in CDVA	Gain of 1 Line of CDVA (%)	Gain of 2 or More Lines of CDVA (%)	Saftey Index
6	6	6	72	22	No data
1.2	2	53		44	1.31
2.5		97.5			No data
2.6	6.4	63		28	No data
0	9.5	78.5		12	No data
0	12	64		24	1.12
0	0	14	23	62	No data
0	0	35.5	64.5	41.9	1.29
0	0	35	11	22	1.12
0	0	3	4	18	1.39
0	11.6	88.3			1.19
6.4	6.4	29.0	19.4	25.8	1.13
0	7	31	43	19	No data
0	5	20	10	65	No data
2.6	3.9	62.3		31.2	1.10
1.8	6.6	38.6	40.4	13.6	No data
0	~4	~23	~56	~17	No data
No data	No data	No data	No data	No data	1.30
No data	No data	No data	No data	No data	1.04
0	~10	~73	~9	~8	No data
0	3.4	55.1	27.5	13.7	1.1
7.2	14.3	32.1	39.3	7.2	1.05
0	0	86		14	No data
0	17.6	82.4		0	No data
0	0	73.6	21	5.2	No data
No data	No data	No data	No data	No data	1.25
3	3	66	16	6	No data
9.7	0	29.0	22.6	25.8	1.12
0	9	51	33	7	1.09
No data	No data	No data	No data	No data	No data
0	0	35	65	0	1.25
0	11	19	70	0	1.40
0	0	4	2	2	1.3
0	1	0	1	6	1.6
2	1	3	1	2	1.3
No data	No data	No data	No data	No data	1.17
0	0	31.25	18.25	50	No data
0	0	28	39	33	No data

Table 3. Visual acuity, predictability, efficacy, and safety of posterior chamber pIOLs.

Type of pIOL/Study*	Number of Eyes	Follow-up (Mo)	Mean Preop SE	Efficacy					
				Mean Postop SE	Postoperative ±0,5 D [%]	Postoperative ±1,0 D [%] ≥	Postoperative UCVA ≥ 1.0 [%]	Postoperative UCVA ≥ 0.5 [%]	Efficacy Index
ICL									
Menezo[18]	21	11–21	−16.0	−1.60	No data	No data	0	76.2	No data
Sanders[40]	258	12	−10.05	−0.56	57.4	80.2	50.9	93.3	No data
Sanders[41]	369	36	−10.06	No data	67.5	88.8	40.8	81.3	No data
Uusitalo[42]	38	6–24	−15.1	−2.0	71.1	81.6	39.5	94.7	No data
Jimenez-Alfaro[43]	20	12–24	−14.1	−1.62	No data	20	No data	60	No data
Gonvers[44]	22	3–24	−11.5	−1.19	32	45	18	68	No data
Arne[45]	58	6–24	−13.85	−1.22	No data	56.9	No data	No data	0.84
Zaldivar[46]	124	1–36	−13.38	−0.78	44	69	2	68	No data
Rosen[47]	16	3	−9.28	−0.83	56.25	No data	25	56.25	No data
Rosen[47]	9	3	−15.4	0.3	88	No data	44.4	88.9	No data
Pineda-Fernandez[48]	18	12–36	−15.27	−0.62	No data	No data	5.5	44.4	No data
Lackner[49]	65	6–48	−16.23	−1.77	No data	42	No data	No data	No data
Pesando[50]	15	6–18	+7.77	0.02	69.25	92.3	0	46.15	No data
Davidorf[51]	24	1–18	+6.51	−0.39	58	79	8	63	No data
Lackner[49]	10	6–48	+7.88	0.44	No data	73	No data	No data	No data
Chang[52]	61	1–32	−14.53	−0.10	72.5	88.2	75	100	No data
Kamiya[53]	56	48	−9.83	−0.38	79	93	70	95	0.83
Sanders[54]	164	1–6	−6.01	−0.09	85	97	63	99	No data
Boxer Wachler[33]	30	3	−11.48	−0.40	88	100	67	100	No data
Rayner[55]	116	12	−8.83	No data	No data	100	78.5	100	No data
Rayner[55]	10	12	+4.25-8.88	No data	No data	100	78.5	100	No data
Toric ICL									
Schallhorn[56]	42	1–12	−8.04	−0.17	76	100	97	100	No data
Alfonso[57]	15	24	−7.08	−0.95	66.6	80	No data	46.6	1.02
Chang[58]	44	1–12	−12.81	No data	82.9	97.1	70.6	100	No data
Park[59]	30	1–18	−10.63	0.04	70	94	67	100	No data
PRL									
Pallilkaris[60]	34	12–24	−14.7	−0.61	44	79	No data	No data	No data
Hoyos[61]	17	12	−18.46	−0.22	53	82	No data	No data	No data
Verde[62]	90	12	−11.90	+0.04	68	80	~16	~92	0.98
Donoso[63]	53	8	−17.27	−0.23	No data	71.2	60	No data	1.0
Jongsareejit [64]	50	12	−12.54	−0.23	88	96	44	82	No data
Koivula[65]	14	24	−10.28	−0.38	79	100	50	100	0.98
Hoyos[61]	14	12	+7.77	−0.38	50	79	No data	No data	No data
Gil-Cazorla[66]	16	12	+5.65	+0.07	93.75	100	12.5	100	0.8
Koivula[65]	6	24	+5.67	−0.85	67	100	17	83	0.89
Koivula[67]	40	12	+5.90	−0.46	87.5	100	17.5	82.5	0.70
Fyodorov posterior chamber pIOL									
Utine[3]	14	24–132	−15.83	−0.71	No data	No data	No data	No data	1.0

CDVA = corrected distance visual acuity; SE = spherical equivalent; UDVA = uncorrected distance visual acuity
*First author

that in comparison to posterior chamber pIOLs, anterior chamber pIOLs and iris-fixated pIOLs led to improved contrast sensitivity at all frequencies.[80] With the AcrySof Cachet pIOL, no glare has been reported during a 1-year follow-up.[11]

Surgically Induced Astigmatism Surgically induced astigmatism (SIA) is significant because patients request acceptable uncorrected visual acuity. The surgeon needs to consider the preoperative amount and axis of astigmatism to decide whether to use a larger incision with a PMMA IOL or to implant a foldable pIOL such as the AcrySof Cachet through a small incision. If significant SIA is noted, further refractive surgical procedures might be

Table 3. (Cont.)

Safety					
Loss of 2 or More Lines of CDVA (%)	Loss of 1 Line of CDVA (%)	No Change in CDVA	Gain of 1 Line of CDVA (%)	Gain of 2 or More Lines of CDVA (%)	Saftey Index
0	0	9.5	19	71.4	No data
1.6	7.8	41.2	38.5	10.9	No data
0.8	No data	No data	No data	10.8	No data
0	6.3	18.8	31.3	40.6	No data
0	0	0	0	100	No data
0	0	9.1	90.9		No data
3	5	19	35	38	1.46
0.8	7	29	28	36	No data
0	6.25	50	37.5	6.25	No data
0	11.1	44.4	22.2	22.2	No data
5.5	0	55.5	5.5	33.3	No data
13.8		1.5	84.6		1.31
7.7	0	76.9	0	15.4	No data
4	0	33	29	8	No data
60		0	40		0.98
0	~3	~27	~62	~8	No data
0	9	32	46	13	1.19
0	4	52	41	3	No data
0	0	50	40	10	No data
0	0	38	62		No data
0	0	5	92	3	No data
0	0	54	13	33	1.58
0	2.2	58.2	31	8.6	No data
0	0	No data	No data	No data	No data
2.9	0	23.5	29.4	44.1	No data
0	0	35	47	18	No data
0	0	35	33	32	1.22
5.7	1.9	15.1	41.5	35.8	1.40
0	2	40	10	14	No data
0	No data	No data	No data	No data	1.18
0	7	86	7	0	No data
0	31.25	68.75	0	0	0.9
0	No data	No data	No data	No data	0.98
5.0	No data	No data	No data	0	0.89
9.1	No data	No data	No data	No data	1.21

considered. Irregular astigmatism due to large incisions too close to the corneal center should be avoided.[81]

Pigment Dispersion or Intraocular Lens Deposits Although no incidence of pigment dispersion or deposits on the IOL are reported, these conditions are seen in clinical practice (Figure 3). However, they do not usually negatively affect visual acuity and, thus, no further procedure is required. Besides pigment dispersion, intraoperative hemorrhage (Figure 4) may lead to erythrocyte deposits on the pIOL and intraocular pressure (IOP) elevation. Bleeding originates from vessels in the scleral tunnel or from the intraoperative iridectomy.

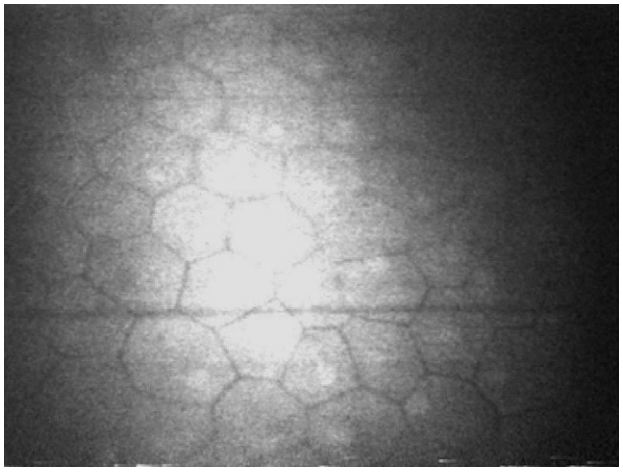


Figure 1. Confocal microscopic image of the endothelium showing endothelial cell loss after implantation of an anterior chamber pIOL (700 cells/mm^2).

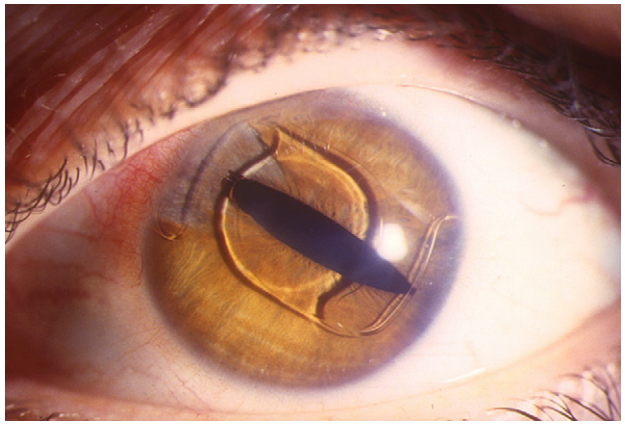


Figure 2. Severe cat-pupil ovalization following anterior chamber pIOL implantation (courtesy of J. Alió, Alicante, Spain).

Chronic Inflammation or Uveitis The first paper that described breakdown of the blood–ocular barrier was published by Alio et al. in 1993 after implantation of anterior chamber pIOLs.[82] As anterior chamber pIOLs are positioned directly in front of the iris, chronic inflammation and development of pigment dispersion is possible as pupil movement can induce some friction with the pIOL. Pérez-Santonja et al.[5] report a rate of 8.7% of eyes presenting with slight chronic inflammation during the first 6 months after ZSAL-4 IOL implantation. Allemann et al.[75] removed 1 of the 21 implanted pIOLs because of a chronic postoperative inflammatory response associated with ocular hypertension. Alió et al.[74] observed acute postoperative iritis in 4.6% of 263 anterior chamber pIOLs (ZSAL-4 and ZB5M). Leccisotti[7] reports an incidence of 3.1% of clinically significant iridocyclitis that appeared within 1 to 31 months of ZSAL-4 implantation. Van Cleynenbreugel[83] report one case of late intrapupillary membrane formation and chronic uveitis associated with corneal endothelial cell loss years after backward implantation of Vivarte anterior chamber pIOLs. Removal of the pIOL led to recovery of visual acuity. As with other complications, if conservative topical treatment does not succeed, removal of the pIOL should be considered to avoid long-term risks.

Intraocular Pressure Elevation/Pupillary Block Glaucoma The risk for acute pupillary block glaucoma is well known from aphakic anterior chamber IOLs; therefore, a peripheral iridectomy is recommended.

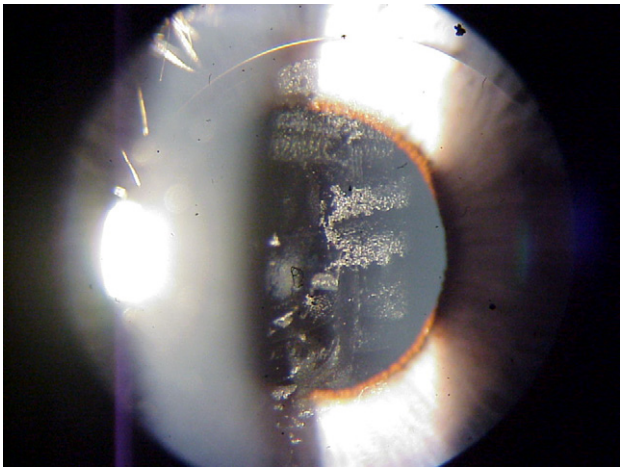


Figure 3. Protein deposits on an anterior chamber pIOL in a 34-year-old woman 1 month postoperatively.

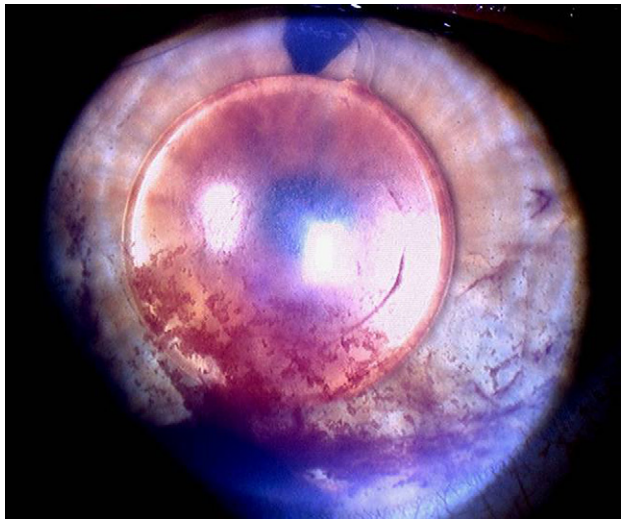


Figure 4. Anterior chamber hemorrhage after anterior chamber pIOL implantation (courtesy of E. Rosen, Manchester, United Kingdom).

Anterior chamber pIOLs have at least the same risk for acute glaucoma, primarily because the continuously growing crystalline lens is still inside the eye. Ardjomand et al.[84] observed one case of pupillary block after implantation of an anterior chamber pIOL that was successfully treated with a neodymium:YAG (Nd:YAG) iridotomy. Leccisotti and Fields[78] report a 3.0% rate of pupillary block 6 hours after anterior chamber pIOL implantation caused by incomplete iridectomy with uninterrupted pigment layer. Kohnen et al.[11] report no case of pupillary block after AcrySof foldable pIOL implantation. Moreover, increased IOP for a period of at least one month after surgery that required treatment was noted in only 3.2% of cases. Of note, iridotomy was only performed in 5 of 190 surgeries.

Two steps are recommended to prevent acute pupillary block glaucoma for angle-supported and other types of pIOLs. All the ophthalmic viscosurgical device (OVD) must be removed from the anterior segment at the end of surgery. In addition, a preoperative iridotomy using a laser or an intraoperative surgical iridectomy to forestall acute pupillary block glaucoma is mandatory. Particularly with foldable anterior chamber pIOLs, the need for a peripheral iridectomy has been discussed by experienced refractive intraocular surgeons. For the latest AcrySof pIOL, however, peripheral iridectomy does not seem to be mandatory, even though reports of acute angle-closure or pupillary block glaucoma have been published.[11] These cases might be attributed to incomplete OVD removal. Javaloy et al.[4] report a mean difference between preoperative and 12-year postoperative IOP of only 2 mm Hg. Prolonged therapy with antiglaucomatous medication was used in only 5 of 225 eyes during the complete follow-up in this study. Other factors of postoperative elevated IOP may be the steroid medication. Leccisotti and Fields[78] report steroid-related IOP elevation in 14% after ZSAL-4 implantation. Intraocular pressure elevation should be carefully observed and treated, with conversion to nonsteroidal antiinflammatory drugs and topical medication. Otherwise, if chronic IOP elevation develops, the anterior chamber angle should be examined to rule out synechiae formation and other pathologies. Removal of the pIOL should be considered, if necessary.

Phakic Intraocular Lens Rotation Rotation of an anterior chamber pIOL might occur because of undersizing. Allemann et al.[75] report that 80% of eyes showed greater than 15 degrees of rotation by 2 years; in 60% the rotation occurred between 1 year and 2 years, implying some instability in the anterior chamber. Pérez-Santonja et al.[5] observed rotation in 43.5% of 23 treated eyes. With the AcrySof pIOL, most eyes (71.1%) did not show an IOL rotation of more than 15 degrees but 28.9% did. However, IOL rotation was not associated with any clinical sequelae in these cases.[11]

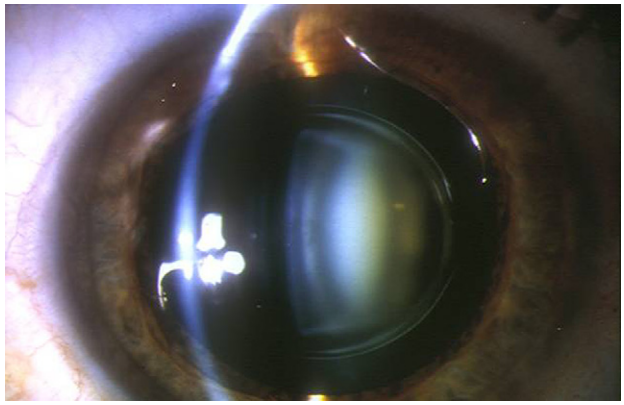


Figure 5. Nuclear cataract in an eye with an anterior chamber pIOL (courtesy of J. Alió, Alicante, Spain).

Cataractogenesis As the position of anterior chamber pIOLs is away from the lens, the formation of cataract is less significant than with a posterior chamber pIOL (Figure 5). Since cataract formation is more frequent in highly myopic patients than in the general population, discriminating between myopia-associated cataract formation and surgically triggered or hastened cataract is difficult. Alió et al.[74] report 9 cataract removals during a 7-year follow-up (3.4%). Cataracts were nuclear, and calculated survival curves for cataract development indicate that more than 90% of patients would be expected to remain free from cataract after 98 months. The same authors report that cataractogenesis seems to be increased in patients older than 40 years with an axial length longer than 29 mm.[85] A metaanalysis of cataract development after pIOL implantation reports that 15 of 1161 eyes developed new-onset cataract.[86] Of these, 9 were nuclear sclerotic, 3 were nonprogressive posterior subcapsular cataract, 2 were nonprogressive anterior subcapsular cataract, and 1 was both anterior and posterior subcapsular cataract. The total incidence of cataract formation for anterior chamber pIOLs was 1.3%. The incidence was 2.6% for the ZB5M anterior chamber pIOL and 0.6% for the ZSAL-4 anterior chamber pIOL; no cataracts were reported in eyes with the ZB, the Newlife/Vivarte Presbyopic, or the AMO multifocal prototype pIOLs.[86] With the novel AcrySof Cachet, the incidence of cataract formation was 2.6%. In 1.0% of the eyes, cataract formation was secondary to concurrent ophthalmic disease.[11] A recent study by Kohnen and Klaproth[77] using Scheimpflug imaging reports a stable distance between the AcrySof pIOL and the crystalline lens over a period of 3 years. Excessive postoperative

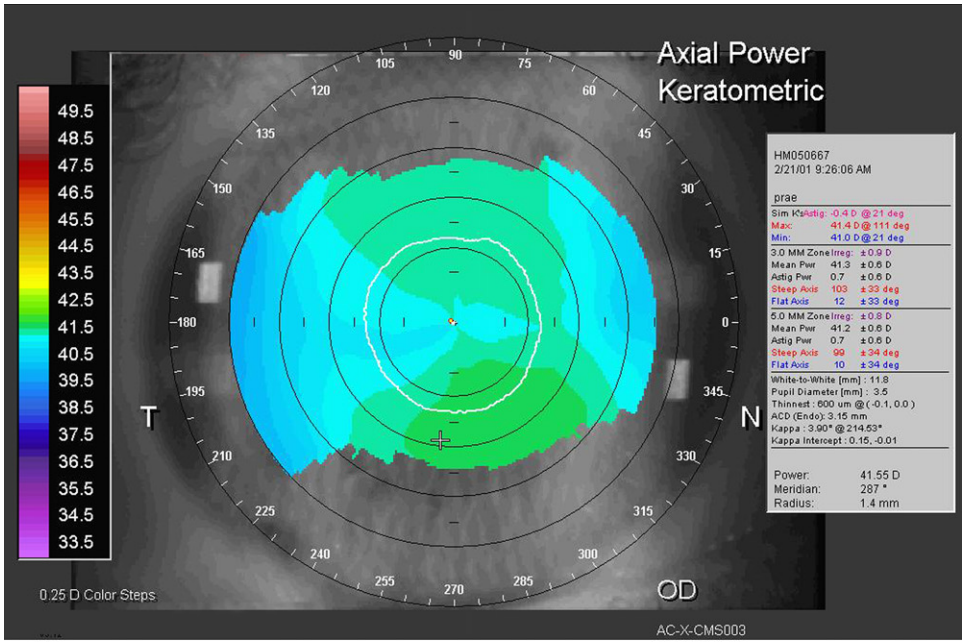
use of steroids should be avoided because of the potential risk for delayed cataract formation.[87]

Retinal Detachment Ruiz-Moreno et al.[88] report a retinal detachment (RD) rate of 4.8% 1 to 44 months after anterior chamber pIOL implantation (ZB5M and ZB5MF). In this study, no correlation between axial length and the incidence of RD was reported. The mean preoperative refraction was −18.6 diopters (D) and the mean axial length, 29.5 mm. Patients in this myopic range have been shown to have a 15 to 110 times higher risk generally than emmetropic patients for spontaneous RD.[89] Ruiz-Moreno et al.[88] also state that the time lapse between pIOL implantation and RD (mean 17.4 months) makes it difficult to infer that intraoperative hypotony with imbalance in premature degenerated vitreous structures played a role in the development of RD. In the study analyzing causes of anterior chamber pIOL explantation by Alió et al.,[76] one case of RD was noted and the pIOL had to be removed to enhance fundus visualization for retinal surgery. In a recent study reporting outcomes up to 12 years after ZB5M implantation by Javaloy et al.,[4] no case of RD was noted. For the novel AcrySof pIOL, no case of RD has been reported to date.[11]

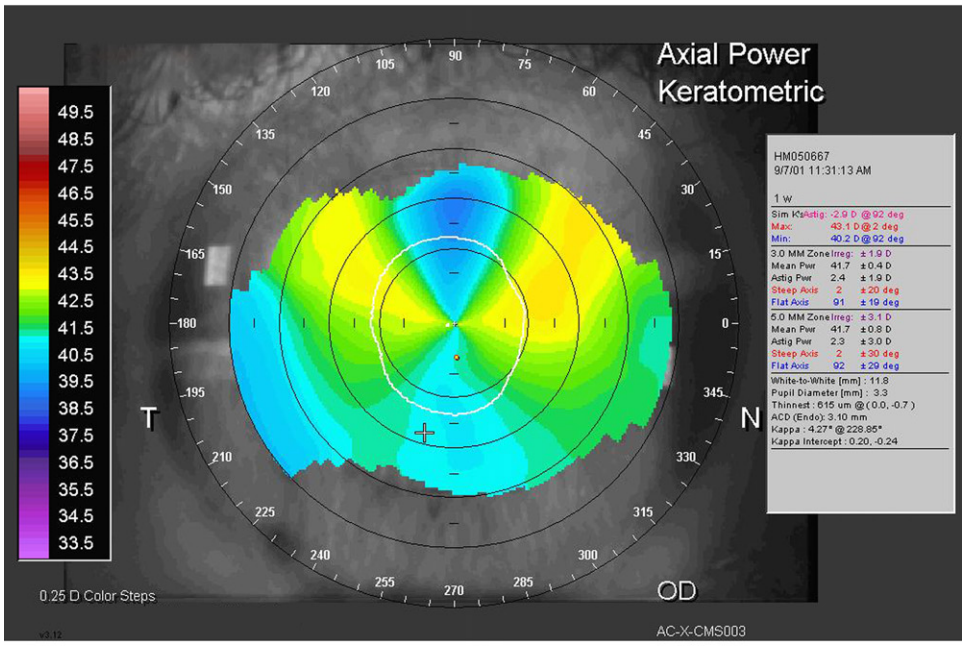
Oddities Urrets-Zavalia syndrome, fixed dilated pupil, iris ischemia, and IOP of 60 mm Hg despite a permeable surgical iridectomy after anterior chamber pIOL implantation were reported as a single case report by Yuzbasioglu et al.[90] in a 26-year-old highly myopic patient 1 day after surgery. In this case report, the type of pIOL is unfortunately not stated. Spontaneous macular hemorrhage has been reported in 2 eyes.[78] In these cases, repeat fluorescein and indocyanine angiography did not show a neovascular membrane and spontaneous improvement occurred. Also, incorrect power or upside-down placement is one possible complication that might cause secondary complications such as cataract formation. This complication is reported in 2 of 190 cases in the study by Kohnen et al.[11] after implantation of the AcrySof Cachet pIOL. A recent modification (marking) of this pIOL might prevent this complication in the future.

Iris-Fixated Anterior Chamber pIOL Complications

Optical Quality, Glare, Halos A pIOL can be implanted in eyes with large scotopic pupil diameters because of the mean age of preponderantly young patients. This can result in glare phenomena if the pupil is larger than the IOL optic. Glare and halos affect night vision and driving and are therefore important considerations in pIOL implantation. A study by Maroccos et al.[79] shows significantly less glare and halos with the Artisan pIOL than with other pIOLs (anterior chamber pIOL NuVita and posterior chamber pIOL ICL), especially the 6.0 mm optic. This was attributed to the larger optic (6.0 mm versus 5.0 mm) and the fixation of the IOL to the iris, which causes less pupil dilation. Therefore, the 6.0 mm optic iris-fixated pIOL seems to be preferable to the 5.0 mm optic. However, it is not always possible to implant this optic because of the greater thickness of the optic with higher corrections and the possible damage to the corneal endothelium in a given ACD. The power of the 6.0 mm optic has an upper limit of –15.5 D for myopia. The range of the 5.0 mm optic is +1.0 to +12.0 D for hyperopia. Menezo et al.[91]describe a case of permanent wide dilation of the pupil, causing decreased postoperative visual acuity because of glare. Landesz et al.[14] report 2 of 38 patients that required pilocarpine eyedrops because of halos after implantation of the 5.0 mm optic Artisan IOL. Maloney et al.[16] report mild to moderate glare in 18 eyes (13.8%) and severe glare in 1 eye (0.8%) of 130 eyes. In 3 eyes, an IOL with a 5.0 mm optic was exchanged for an IOL with a 6.0 mm optic, with no glare noticed afterward. Senthil et al.[21] report no glare and halos after implantation of the Artisan pIOL in 60 myopic eyes, probably because Indian eyes generally have smaller pupils than white eyes. Moshirfar et al.[23] report an incidence of 6.0% of glare and halos 1 month after Artisan/Verisyse implantation, which decreased to 2.7% at 2 years follow-up. In a recent study by Stulting et al.[25] analyzing the 3-year results of the Artisan/Verisyse pIOL, no contrast sensitivity decrease was seen. In this prospective study, patients with a mesopic pupil greater than the pIOL optic were not included; 80% of the pIOLs had a 6.0 mm optic and only 20% had a 5.0 mm optic. A study by Chung et al.[92] shows that Artisan pIOLs do not alter higher-order aberrations (HOAs) significantly, a finding comparable to that of Chandhrasri et al.,[93] who report a small increase in HOAs under photopic conditions after Verisyse pIOL implantation. One study investigating HOAs shows that after Artiflex pIOL implantation, postoperative trefoil increased and spherical aberration decreased.[94] The authors report a significant correlation between pIOL decentration and postoperative spherical aberration and coma. However, both trefoil and spherical aberration increased in the Artisan pIOL group postoperatively. Different incision sizes may explain differences in trefoil, whereas the different optic design of the two pIOLs seems to affect spherical aberration.[93,94] Bühren and Kohnen[81] report slightly increased HOAs after Artisan pIOL implantation,with induction of trefoil as a result of the incision and increase in spherical aberration from the pIOL. Cisneros-Lanuza et al.[95] report some degree of lenticular glistenings in 20% of the eyes



A



B

Figure 6. Induction of corneal SIA due to a 6.0 mm superior limbal incision (35-year-old man). *A*: Preoperative topography. *B*: Corneal topography 6 months postoperatively.

after Artiflex IOL implantation. Glistenings were noted from 6 days to 6 months after surgery, and neither decreased over time nor affected visual acuity or caused complaints.

Surgically Induced Astigmatism Because the PMMA iris-claw IOL (Artisan/Verisyse) is not foldable, it requires an incision that approximately equals the optic diameter (5.0 or 6.0 mm), which may induce SIA (Figure 6). According to the literature, SIA after the 5.0 to 6.0 mm incisions is less than one might expect. Menezo et al.[91] report no significant increase in postoperative astigmatism. Alió et al.[29] report a mean SIA of 1.48 D ± 0.89 (SD) for the hyperopic Artisan IOL with correction of primary hyperopia and 1.85 ± 1.19 D with correction of secondary hyperopia after corneal refractive surgery. Maloney et al.[16] report a mean decrease in astigmatism of 0.3 D after 6 months. Stulting et al.[25] report a change of more than 2.0 D cylinder in 3.5% of eyes 3 years after Artisan/Verisyse implantation and secondary refractive procedures had to be performed in 6.9% of eyes during the

follow-up. The foldable Artiflex/Veriflex further reduces SIA. In a prospective randomized study comparing the Artisan pIOL in one eye and the Artiflex pIOL in the other eye, the mean refractive cylinder power of the Artiflex pIOL was significantly lower than that of the Artisan pIOL, −0.56 ± 0.47 D and −1.02 ± 0.63 D, respectively.[22] The mean SIA was 0.29 ± 1.67 D and 0.73 ± 2.9 D, respectively, which was close to statistical significance ($P = .07$). In another study, SIA after Artiflex implantation was 0.42 D.[96] In a later report, the mean SIA 2 years after Artiflex pIOL implantation was only 0.33 D.[34]

Loss of Corneal Endothelial Cells Damage to the corneal endothelium may be due to direct contact between the pIOL and the inner surface of the cornea during implantation or from postoperative changes in pIOL position. Moreover, subclinical inflammation may cause direct toxicity to the endothelium and lead to further damage. In 1991, Fechner et al.[97] described the first results of this type of pIOL with a follow-up of more than 12 months: Five of 109 eyes experienced corneal endothelial cell loss by surgical trauma and 5 eyes showed progressive corneal endothelial cell loss that caused corneal edema in one eye. In a prospective study that included 111 eyes with a follow-up of 4 years, Menezo et al.[98] report that the largest percentage of corneal endothelial cell loss was noticed during the first 6 months after implantation and conclude that the main cause for corneal endothelial cell loss is surgical trauma. Corneal endothelial cell pleomorphism and polymegathism did not change significantly after surgery. One pIOL that was placed too superiorly caused corneal edema and had to be removed. Other studies have shown similar results.[14,29,99,100] Maloney et al.[16] report no difference in corneal endothelial cells between preoperatively and 6 months postoperatively. Budo et al.[13] report a corneal endothelial cell loss of 0.7% 3 years after Artisan/Verisyse implantation. Pop and Payette[32] report no significant change in corneal endothelial cells 2 years after Artisan implantation. Senthil et al.[21] did not find significant corneal endothelial cell loss 24 months after Artisan surgery. Moshirfar et al.[23] report a 6.2% decrease in corneal endothelial cells 2 years after Artisan/Versisyse implantation. A similar rate, 6.8%, was reported by Gierek-Ciaciura et al.[9] 1 year after Verisyse implantation. A recent study by Stulting et al.[25] shows a mean corneal endothelial cell change of 4.8% 3 years after surgery. Another recent study by Güell et al.[27] reports a significant decrease in corneal endothelial cells after myopic Verisyse implantation, whereas corneal endothelial cell loss was not significant in the hyperopic Verisyse and toric Verisyse groups 3 years after implantation. Overall, corneal endothelial cell loss in this study was 5.11% at 4 years. Natural loss of corneal endothelial cells is about 0.6% per year, as reported by Bourne et al.[101] One study has shown that corneal endothelial cell loss following combined pIOL explantation after Artisan implantation was only 3.5% 6 months after surgery.[102] Dick et al.[34] report corneal endothelial cell loss of only 1.1% 2 years after Artiflex implantation.

In contrast to these findings, Pérez-Santonja et al.[103] report continuous corneal endothelial cell loss with a decrease of 17.6% 24 months after surgery and Saxena et al.[104] report a corneal endothelial cell loss of 8.3% with a mean follow-up of 35.3 months. Saxena et al.[104] report a significant negative correlation between ACD and corneal endothelial cells. Benedetti et al.[105] report a continuous decrease in corneal endothelial cells after Artisan pIOL implantation; at 5 years, the decrease was 9.0%. Silva et al.[26] report a decrease of 14.05% corneal endothelial cells 5 years after Artisan implantation. In a recent study of factors leading to corneal endothelial cell loss after pIOL implantation,[106] the authors report a yearly corneal endothelial cell loss of 1.0% for a mean minimum distance of 1.43 mm between the edge of the pIOL and the corneal endothelium; the loss was 1.7% for a mean minimum distance of 1.20 mm and 0.2% for a mean minimum distance of 1.66 mm. In this study, according to a linear mixed model analysis, patients with preoperative corneal endothelial cells of 3000, 2500, or 2000 cells/mm^2 and an edge-distance of 1.43 mm, a critical corneal endothelial cell level of 1500 cells/mm^2 would be reached 56, 37, and 18 years after Artisan/Artiflex implantation.

All authors agree that preoperative endothelial microscopy is mandatory. Patients with endothelial damage or corneal endothelial cells below 2000/mm^2 should therefore not receive a pIOL. The height of the Artisan IOL and therefore the potential closeness to the cornea increases with its dioptric power. Therefore, a sufficient ACD for the calculated pIOL is necessary so the distance between the pIOL and the corneal endothelium is not less than 1.5 mm.[107,108]

Pigment Dispersion/Lens Deposits The optic of the iris-claw pIOL has an anterior vault to prevent iris chafing. Pop et al.[109,110] performed postoperative ultrasonic biomicroscopy of the haptics of myopic and hyperopic pIOLs and found no evidence of irritation of the iris pigment epithelium by the pIOL haptics during a follow-up of 24 to 371 days. Pigment cells are occasionally visible on the pIOL optic in the early postoperative period from surgical trauma. Figure 7 shows iris pigment defects at the site of enclavation as a possible source of pigment dispersion. Stulting et al.[25]

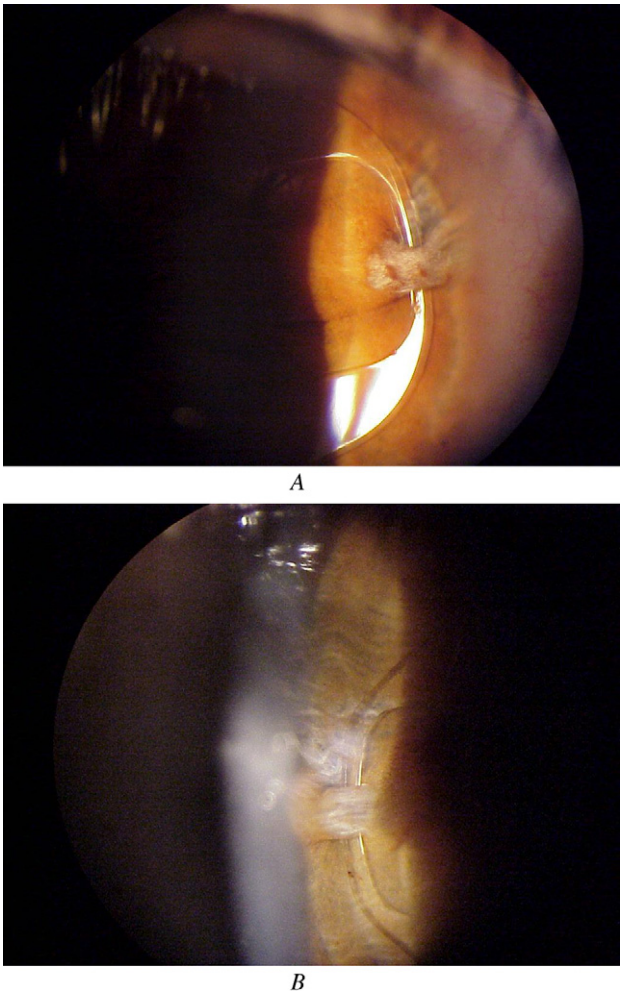


Figure 7. Iris pigment defects at the site of enclavation may be one source of dispersed iris pigment (30-year-old man [*A*] and 47-year-old woman [*B*]; both 3 months postoperatively).

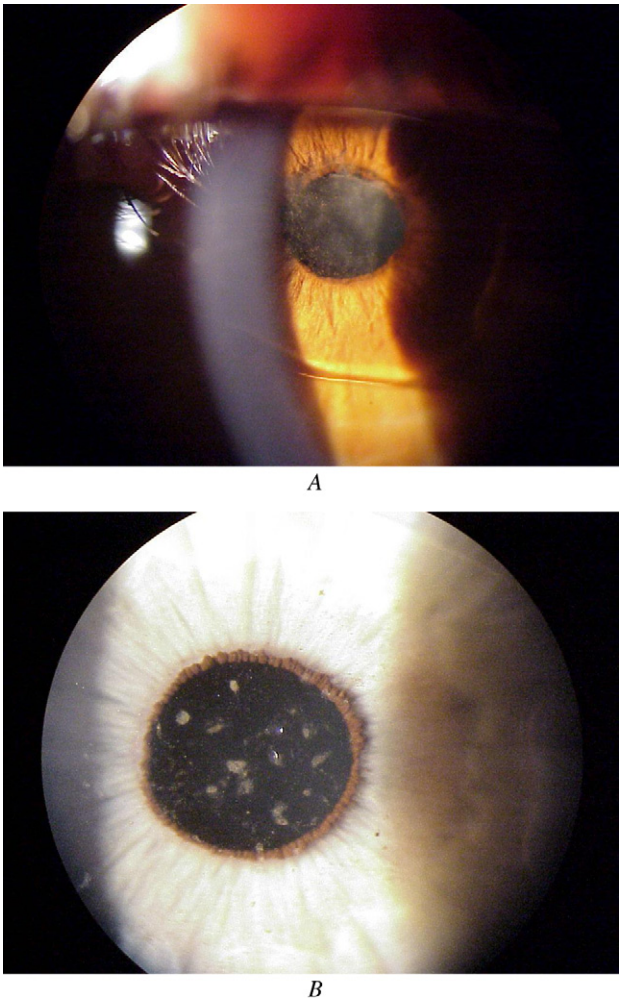


Figure 8. Inflammatory reaction after iris-claw IOL implantation. *A*: Dense fibrin coating on the pIOL 1 week postoperatively (34-year-old woman). *B*: Persistent deposits 3 months after implantation (37-year-old man).

report iris pigment precipitates with an incidence of 6.9% at 4 to 6 months follow-up and no case at the 3-year follow-up. Menezo et al.[18] report a long-term incidence of 6.6% pigment dispersion with the longest mean follow-up of 10 years after Artisan implantation. However, in the phase III trial for the hyperopic iris-claw pIOL, there are reports of 3 patients who had pigment dispersion or pupillary membrane formation due to iris touch.[111] Baikoff et al.[112] consider crystalline lens rise as a risk factor for developing pigment dispersion after iris-fixated pIOL implantation. In their study, 67% of eyes with a rise of more than 600 µm developed pupillary pigment dispersion after implantation of the Artisan pIOL. Nearly all eyes were hyperopic. For the Artiflex pIOL, pigment precipitates were reported in 4.8% of eyes, nonpigment precipitates in 1.4%, and synechiae formation in 1.4% 2 years after surgery.[34]

Chronic Inflammation/Uveitis Chronic inflammation has been a major concern with the iris-claw IOL because this pIOL is fixated directly to the iris tissue and causes pressure or shear forces when the eye is moving or patients rub their eyes (Figure 8). This may lead to injury or increased permeability of the iris vessels with breakdown of the blood–aqueous barrier and chronic release of inflammatory mediators. This has been repeatedly examined using different technologies. Two studies using iris angiography show no leakage of the iris vessels,[91,97] whereas studies conducted using a laser-flare cell meter show different results. Fechner et al.[97] report no elevated flare levels in 109 eyes with at least 12 months of follow-up. Pérez-Santonja et al. (Perez-Santonja JJ, Iradier MT, Benıtez del Castillo JM, Serrano JM, Zato MA. Chronic subclinical inflammation in phakic eyes with intraocular lenses to correct myopia. J Cataract Refract Surg 1996;

22:183–187) report elevated flare levels in 30 eyes compared with the levels in a normal population at 12, 18, and 24 months after surgery. Groβ et al.[100] report no significantly elevated flare after 6 months in a study with 44 eyes. In all studies, clinically relevant inflammation could be detected in individual cases only. In a case report by Koss et al.,[113] posterior synechias developed 2 weeks after Artiflex implantation and required surgical reenclavation. However, 2 years after surgery, posterior synechiae did not change. A similar case report by Tahzib et al.[114] describes development of severe cell deposition 1 week after Artiflex implantation. After pIOL exchange, inflammation in the anterior chamber disappeared completely. Senthil et al.[21] report postoperative iritis in 3% of the eyes after Artisan implantation that resolved completely. Moshirfar et al.[23] report an incidence of 1.2% of cells and flare for 1 month after Artisan/Verisyse surgery. Moshirfar et al.[115] describe a case of toxic anterior segment syndrome (TASS), also known as sterile endophthalmitis, in a patient who presented with severe corneal edema 1 day after Verisyse pIOL surgery. The TASS resolved after a 2-month course of topical steroids. However, corneal endothelial cells decreased by 69% 1 year after surgery. Careful postoperative monitoring of inflammatory signs is generally necessary. If persistent intraocular inflammation occurs and is not sufficiently treatable with drugs, pIOL removal must be considered.

Pupil Ovalization/Iris Retraction Pupil ovalization or irregularity can occur if fixation of the pIOL haptics is performed asymmetrically. No progressive pupil ovalization has been reported. Maloney et al.[16] report pupil irregularities in 14.0% of eyes on the first day after surgery and 1.2% after 6 months. Moshirfar et al.[23] report a pupil ovalization incidence of 2.4% after Artisan/Verisyse implantation. Stulting et al.[25] report an incidence of 13.0% of asymptomatic oval pupil 1 day after Artisan/Verisyse pIOL implantation, which decreased to 0.4% at 3 years. As enclavation is performed in the peripheral iris, pupil dilation is limited after pIOL implantation. Artisan/Verisyse pIOLs are centered on the middle of the pupil. This can lead to difficulties if the pupil itself is decentered and the optical axis is not in the middle of the pupil (Figure 9). Postoperative decentration is possible if the enclavation is not sufficient. Menezo et al.[91] report an incidence of 13.5% decentration, but in only one case was a second intervention necessary because of double vision. Pérez-Santonja et al.[103] report a decentration greater than 0.5 mm in 43% of the examined eyes. Pérez-Torregrosa et al.[116] report a mean decentration of 0.47 with respect to the pupil center in 22 eyes using a digital imaging system. If the pIOL is fixated properly, no postoperative decentration or rotation of the optic should occur.

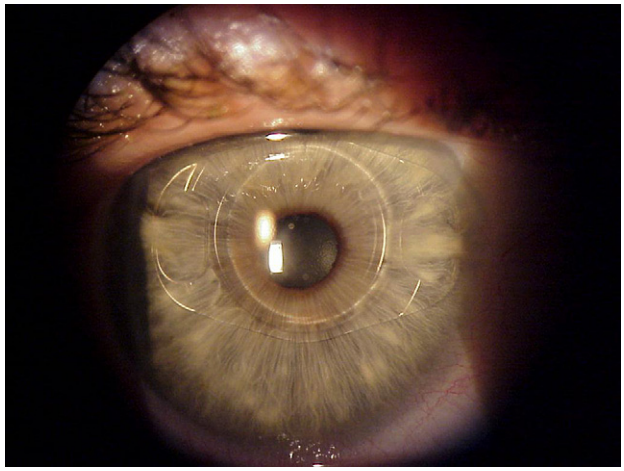


Figure 9. First generation iris-claw pIOL (Worst-Fechner) in an aphakic eye 11 years after implantation (61-year-old woman). Note slight decentration.

Intraocular Pressure Elevation The anterior chamber angle is not generally thought to be affected by the haptics of the iris-claw pIOL. Coullet et al.[22] report that within 1 year of surgery, IOP did not significantly change after Artisan or Artiflex pIOL implantation. However, Yamaguchi et al.[117] report that after implantation of an Artisan/Verisyse pIOL, partial narrowing of the anterior chamber angle of more than 5 degrees occurred in the area where the pIOL haptics pinched the iris. This did not affect IOP. A peripheral iridectomy or iridotomy is necessary to prevent acute pupillary block glaucoma. In several studies, cases of elevated IOP in the early postoperative period resolved without further damage and were probably related to retained OVD or steroid medication.[4,16,21,25,61,118,119]

Phakic Intraocular Lens Rotation Photographic analysis after implantation of toric Artisan pIOLs showed no rotation greater than 2 degrees at 6 months follow-up in a report by Tehrani et al.[35] Using Scheimpflug photography, Baumeister et al.[120] examined the postoperative stability of pIOLs and report that the iris-fixated pIOL had the best positional stability compared with anterior chamber and posterior chamber pIOLs. Therefore, the iris-fixated pIOL is particularly interesting for toric pIOL designs. However, spontaneous postoperative dislocations or dislocations due to blunt ocular trauma have been described (Figure 10).[16,91,103,121]

Cataractogenesis Formation of cataract due to the iris-claw pIOL is unlikely because the pIOL is inserted over a miotic pupil without contact with the crystalline lens. Menezo et al.[122] report a nuclear cataract rate of 3% after implantation of an iris-fixated pIOL. In this study, the implanted IOL was the older

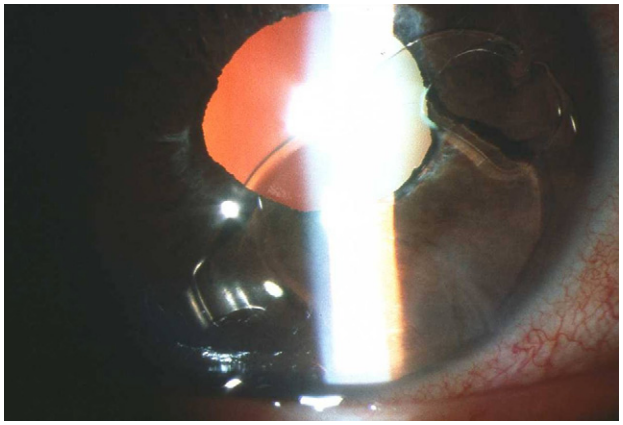


Figure 10. Traumatic dislocation of an iris-claw anterior chamber pIOL (courtesy of D. J. Annen, Winterthur, Switzerland).

Worst-Fechner pIOL. Patient age older than 40 years and axial length greater than 30.0 mm were factors related to nuclear cataract formation. However, new-onset nuclear cataracts were not ascribed to pIOL surgery. Clinically relevant cataract formation associated with the iris-claw IOL has also been reported by Stulting et al.[25] Most lens opacities were nuclear and unlikely to be related to the implanted pIOL. Lens opacities that required cataract extraction developed in 0.25% of patients. Very few were anterior subcapsular opacities, which were expected to be caused by surgical trauma. A metaanalysis of cataract development after pIOL surgery reported that 20 of 2781 eyes developed new-onset cataract.[86] Of these, 10 were nuclear sclerotic, 8 were cortical vacuoles, and 1 was anterior subcapsular cataract (data for 1 eye was not clear.) The incidence of cataract formation was 1.1% for the iris-fixated pIOL; it was 2.2% for the Worst-Fechner biconcave pIOL, 1.1% for the myopic Artisan/Verisyse pIOL, and 0.3% for the hyperopic Artisan/Verisyse pIOL. No cataracts have been reported to date with the Artiflex pIOL.[86] As for anterior chamber pIOLs, an excessive postoperative use of steroids should be avoided because of the potential long-term risk for cataract formation.[87]

Retinal Detachment Thorough examination of the posterior segment to rule out vitreoretinal pathologies is mandatory, although no vitreoretinal complications have been shown to be causally related to iris-fixated pIOL implantation to date. In the European multicenter study of the Artisan pIOL over 8 years, retinal detachment (RD) occurred in 2 eyes.[13] Stulting et al.[25] report an RD rate of 0.3% per year after Artisan/Verisyse implantation in eyes with a mean spherical equivalent between −11.50 D and −18.6 D. This is similar to RD rates that have been reported in the highly myopic population that did not have refractive surgery.[123–125] Güell et al.[27] report one case of RD in a series of 399 eyes with the Artisan/Verisyse pIOL. Retinal detachment was not thought to be related to the pIOL implantation. A recent report describes a bilateral giant tear RD following Artisan pIOL implantation in a 39-year-old patient with an axial length of 25.5 mm in the right eye and 25.8 mm in the left eye.[126] In this report, RD was attributed to a combination of inflammatory response and perioperative IOP fluctuations as a causative pathophysiological mechanism based on the time between the RD and the pIOL implantation.

Oddities Other complications of iris-fixated pIOL implantation are Urrets-Zavalia syndrome, early postoperative hyphema, and ischemic optic neuropathy.[127] Hyphema in the early postoperative phase from iris trauma is occasionally described.[14,16,91] Iris bleeding can also be caused by preoperative argon or Nd:YAG laser treatment of the iris to mark fixation points for pIOL enclavation. Iris perforation by the claw haptic of a pIOL is reported by Benedetti et al.[20] Another rare complication is implantation of a pIOL with incorrect power. Due to the aim of the surgery—to correct ametropia as precisely as possible—this complication should not occur with current formulas, as described in the first part of this review. Kohnen et al.[128] report a myopic shift of 4.0 D 10 days after Artisan pIOL implantation. They postulate that this event happened because of secondary movement of the ciliary body inwardly or forwardly or irritation of iris innervation by induction of ciliary body contraction.

Posterior Chamber pIOL Complications

The complication spectrum is similar for the ICL and PRL and is related to the position of the pIOL between the rear surface of the iris and the front surface of the crystalline lens. Differences in the incidence of most common complications such as cataractogenesis, pupillary block, and glaucoma are due to the different pIOL designs and materials.

Optical Quality, Glare, Halos Consequences of a small optic diameter (ICL up to 5.5 mm; PRL up to 5.0 mm) and decentration of posterior chamber pIOLs in relation to the pupil size are glare and halos, especially at night. Therefore, patients with larger pupils have increased difficulties driving at night, which, in extreme cases, may lead to an actual inability to drive at night. Menezo et al.[119] report a high incidence of visual disturbances after implantation of an ICL, which may be due to decentration of the posterior chamber pIOL and/or an optic diameter that is too small relative to the pupil size. Several studies report glare and diplopia in eyes with decentration of the ICL greater

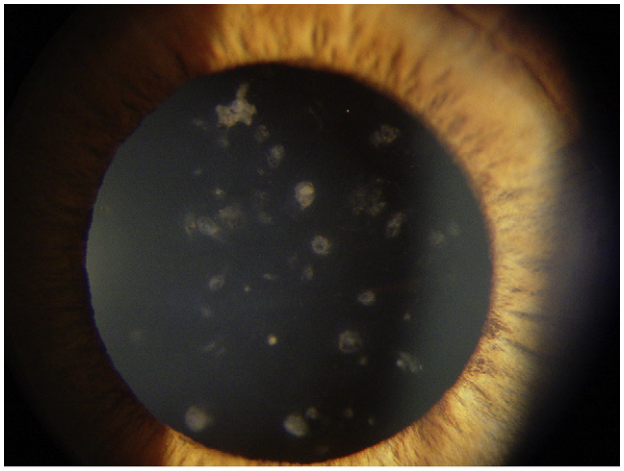
than 1.0 mm.[129,130] Maroccos et al.[79] report a greater increase in postoperative glare and halos after ICL implantation than after Artisan pIOL implantation in the anterior chamber. These findings were thought to be due to the edge effects of the small diameter of the whole ICL and the small optic diameter (4.5 to 5.5 mm) in relation to the pupil size (5.3 to 7.4 mm). With the PRL, which has an optic of 4.5 to 5.0 mm, glare and halos are also a concern. After PRL implantation, 25% of 31 patients reported halos and night glare.[61] To avoid this complication, a preoperative mesopic pupil larger than 5.0 mm should be considered a limitation. In large-pupil cases, a larger optic pIOL should be implanted. For example, an iris-fixated pIOL with a 6.0 mm optic should be used in patients with large scotopic pupils. In a study of the 3-year results of ICL implantation, patients were asked about their optical quality of vision. Improvement of glare and halos was reported in 11.9% of cases and 9.6% of cases, respectively, and worsening in 9.6% and 11.5%, respectively.[41] After PRL implantation, 26% to 28% of patients complained of glare and halos at night.[60,61] Some of the patients had scotopic pupils of 6.0 to 7.0 mm so the difference between the pupil size and the 5.0 mm PRL optic seemed responsible for the problems.[60] A recent report by Koivula and Zetterström[67] shows glare and halos after hyperopic PRL implantation in 2 of 40 eyes, requiring PRL explantation.

Surgically Induced Astigmatism Surgically induced astigmatism has not been reported to be a major concern of posterior chamber pIOLs because of the small-incision surgical procedure. In one study, the SIA after ICL implantation in 73 eyes through a 3.0 mm horizontal clear cornea incision was 0.45 D using a keratometer and 0.49 D using corneal topography.[53]

Loss of Corneal Endothelial Cells Loss of corneal endothelial cells can be divided into direct trauma loss caused by surgery and long-term loss. In various studies of the ICL, immediate corneal endothelial cell loss of 5.2% to 5.5% was documented after 12 months. However, the pace of corneal endothelial cell loss slowed down substantially from 1 year to 2 years (6.6% to 7.9%).[131,132] Researchers therefore considered surgery to be the cause of the early corneal endothelial cell loss. Four years postoperatively, corneal endothelial cell counts showed further decrease in cell density, which may be due to the implanted ICL, the learning curve of the surgeon, or natural cell loss, which is in the range of 0.5% in the normal population.[132] A recent study by Kamiya et al.[133] reports corneal endothelial cell loss of 3.7% 4 years after ICL implantation. Another study shows a cumulative corneal endothelial cell loss of 8.5% 3 years after surgery and 8.4% 4 years after surgery.[41,129] These figures also suggest that corneal endothelial cell density stabilizes over time. Alfonso et al.[57] show corneal endothelial cell loss of 8.1% 2 years after toric ICL implantation in eyes after penetrating keratoplasty. In a report by Koivula et al.,[65] no significant corneal endothelial cell loss was noted between 1 week and 1 or 2 years after implantation of a hyperopic PRL. In a report by Koivula and Zetterström,[67] corneal endothelial cell loss was 3.8% 1 year after hyperopic PRL implantation. Verde et al.[62] did not find a significant reduction in corneal endothelial cells 12 months after PRL implantation in 90 myopic eyes. Jongsareejit[64] reports corneal endothelial cell loss of 5.4% after a short follow-up of 6 months.

Pigment Dispersion/Intraocular Lens Deposits/Intraocular Pressure Elevation Using ultrasound biomicroscopy (UBM), contact between posterior chamber pIOLs (ICL, PRL) and the posterior surface of the iris has been shown.[131,134–137] Pigment dispersion and consecutive pigment accumulation in the anterior chamber angle is one possible consequence (Figure 11).[51,61,119,137] However, development of secondary glaucoma has not been observed. Nevertheless, eyes with pigment dispersion must be kept under observation to spot any increase in IOP. Menezo et al.[18] report a not statistically significant IOP increase of 1.5 mm Hg over 3 years after ICL implantation. Park et al.[59] did not find an IOP increase over 1 to 18 months after toric ICL implantation. In contrast, other studies of ICLs or PRLs have reported significantly increased IOP in rare cases 1 month after implantation. Kamiya et al.[133] did not find an increase of IOP 4 years after ICL implantation. Zaldivar et al.[46] report that 2 of 124 eyes showed IOL-related IOP spikes. One of these eyes with a decentered ICL had excessive pigment deposition on the pIOL surface. It remained unclear whether the pigment dispersion was related to the decentration or to the pIOL itself. In both eyes, the ICL had to be removed and phacoemulsification with capsular bag IOL implantation was performed. The IOP was subsequently well controlled without medication. Sanchez-Galeana et al.[138] report a case of refractory IOP increase due to pigment dispersion after ICL implantation. Despite medical therapy and ICL removal, this patient needed a trabeculotomy to control IOP.

Although Jiménez-Alfaro et al.[131] observed contact of the ICL and posterior iris with UBM in all cases, they did not find pigment dispersion. The authors suggest that the similarity between the Collamer and the anterior capsule of the crystalline lens could prevent mechanical pigment loss. Davidorf et al.[51] report that the pigment deposition on the pIOL surface remained stable over time in all eyes, with no occurrence of pigment dispersion glaucoma. They suggest that pigment



A

B

Figure 11. *A*: Pigment deposits on anterior surface of a PRL posterior chamber pIOL. *B*: Pigment dispersion in the anterior chamber angle after implantation of an ICL posterior chamber pIOL, gonioscopic view, 3 months after implantation (53-year-old man).

dispersion was probably surgically related. Hoyos et al.[61] report a case of window defects of the iris and increased angular pigmentation without increased IOP after PRL implantation in hyperopic eyes. They propose that a too shallow ACD of 2.8 mm was the cause and suggest a minimum ACD of 3.0 mm for posterior chamber pIOL implantation. Donoso and Castillo[63] report no change in IOP after PRL implantation with a mean follow-up of 8 months. Koivula and Zetterström[67] also report no change in IOP 1 year after PRL implantation. Verde et al.[62] report an increase in mean postoperative IOP compared with the preoperative values; the mean IOP was within normal limits in the follow-up. Only 1 of 90 eyes required antiglaucomatous medication. Some authors have reported incidents of secondary induced glaucoma due to the use of topical steroids. However, IOP normalized after a postoperative treatment regimen with steroids and

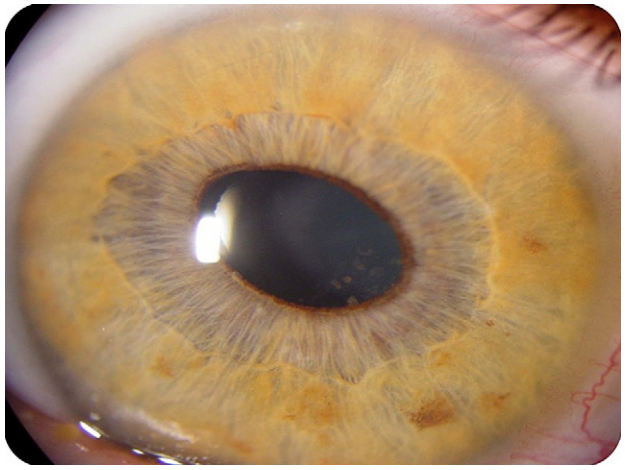


Figure 12. Pupil ovalization after PRL implantation.

was stopped in all eyes.[46,48,61,131] Davidorf et al.[51] report increasing vascularization of the anterior chamber angle and development of secondary glaucoma after ICL implantation in a hyperopic eye. Rosen and Gore[47] also report the development of secondary glaucoma after implantation of a hyperopic ICL. In both cases, the IOL had to be explanted as IOP could not be controlled by repeated iridotomy and topical medication.

Chronic Inflammation/Uveitis To detect intraocular inflammation, laser flare photometry was performed 6 months after ICL implantation. All eyes showed normal aqueous flare values.[42] Another study did not detect any long-term inflammation 2 to 3 years after ICL implantation.[139]

Pupil Ovalization/Iris Retraction In contrast to anterior chamber pIOLs, no cases of pupil ovalization or iris retraction have been reported to date with posterior chamber pIOLs. However, in our experience, they can still occur (Figure 12).

Pupillary Block/Malignant Glaucoma Due to the position of the posterior chamber pIOL, the iris may be pushed forward and cause acute pupillary block glaucoma, especially in hyperopic eyes.[46,50,51,131,140] The diameter of posterior chamber pIOLs is involved in this pathophysiological process. To prevent pupillary block glaucoma, preoperative or intraoperative iridotomies or iridectomies should be performed.[46,47,51] In some cases, preoperative iridotomies become nonpermeable over time because they are too small or the haptic of the posterior chamber pIOL blocks them. This may cause acute pupillary block glaucoma. A second iridotomy has to be performed in these cases.[129,141,142] In one case, pupillary block appeared 1.5 years after PRL implantation because the iridectomy was

obstructed by the PRL haptic.[61] After treatment with a second iridectomy, the IOP in all eyes normalized. Especially in the case of the PRL, which may rotate, 2 iridotomies in an angle of 90 degrees are required.[61] For hyperopic treatment, preoperative iridotomy is even more important to prevent early pupillary block. In such cases, it is necessary to make 2 peripheral and sufficiently sized iridotomies preoperatively with the Nd:YAG laser or during implantation surgery using the vitrectome or scissors.[51] In a recent report by Koivula and Zetterström,[67] 7 of 40 eyes developed pupillary block by a mean of 6 days after hyperopic PRL implantation. All eyes were treated successfully with laser iridotomy.

Malignant glaucoma after posterior chamber pIOL implantation is rare and has only been described by Kodjikian et al.[118] in a myopic eye that had an IOP of 54 mm Hg 3 days after ICL implantation. Both preoperatively performed laser iridotomies were patent and seemed large enough. The iris was not bowed forward, and the posterior segment did not show any pathology. Acute glaucoma due to pupillary block was ruled out. Despite medical treatment, the IOP remained 50 mm Hg; 5 days after implantation, ICL explantation had to be performed. Thereafter, IOP normalized without medical treatment and the corrected distance visual acuity was 20/25.

Decentration/Incorrect size /Phakic Intraocular Lens Rotation Preoperatively, it is mandatory to properly measure the white-to-white (WTW) distance to choose a pIOL with sufficient length to prevent decentration or rotation, even though limitations regarding the WTW distance relative to the sulcus diameter are well-known.[119,130] Although in few cases, Menezo et al.[119] report decentration with an adequate IOL length relative to the corneal diameter. The consequences of decentration are diplopia, glare, and pigment dispersion syndrome because of mechanical trauma.[46,51]

Trindade and Pereira[143] report the exchange of an ICL because of oversized length. Malpositioning with a very large vault and undercorrection occurred because the ICL was too long. The ICL was exchanged for a smaller ICL with higher power. This procedure was uneventful, and the patient was satisfied with the final visual outcome. In a study with a 12-month follow-up, UBM showed ICL rotation in 11% of eyes.[134] Although there was no decentration of the optic, the authors suggest that the diameter of the ICL was too small.[134] In another study,[61] decentration occurred after implantation of a PRL with a diameter that was too small. After the small PRL was exchanged for a newer generation PRL with a larger diameter, no decentration was observed. A recent study by Koivula et al.[65] showes a median PRL rotation 18.5 degrees during the first year after implantation and 0 degree during the second year. Centration of the PRL was good in all eyes for up to 1 year. Minor decentration of a PRL was observed in 5 of 90 eyes in a study by Verde et al.[62] The ICL length has to be calculated on the basis of the horizontal WTW diameter (addition of 0.5 mm to WTW measure). Baumeister et al.[144] report that a most accurate value of horizontal WTW diameter is determined by the IOLMaster (Carl Zeiss Meditec). In this study, the mean rotation of the ICL was 0.9 degrees. A recent study reports that postoperative rotation after toric ICL implantation was less than 5 degrees in 74% of eyes and less than 11% after 8 months.[59]

Cataractogenesis A metaanalysis of cataract development after posterior chamber pIOL surgery found that 223 of 1210 eyes developed new-onset cataract.[86] Of these, 195 were anterior subcapsular (Figure 13), 5 nuclear sclerotic, and 4 anterior subcapsular and cortical opacities. The overall incidence of cataract formation for posterior chamber pIOLs was 9.60%, which is significantly higher than the incidence for anterior chamber pIOLs and iris-fixated pIOLs. The incidence was 25.7% for the Adatomed pIOL, 8.5% for the ICL pIOL, and 3.6% for the PRL.[86] Because of this incidence, the Adatomed is no longer in use. Cataracts after ICL and PRL implantation often remain stable over a long period of time and rarely lead to a reduction in visual acuity. The most common type of cataract after posterior chamber pIOL implantation is anterior subcapsular.[145,146] Possible reasons are operative trauma, continuous or intermittent contact of the posterior chamber pIOL with the crystalline lens, insufficient nutrition through anterior chamber flow between the posterior chamber pIOL and the crystalline lens, or chronic subclinical inflammation with disruption of the blood–aqueous barrier due to friction between the pIOL and posterior iris or the haptic on the ciliary sulcus.[49,145,147] Studies with UBM and Scheimpflug-imaging techniques (Figure 14) have shown a central gap between the ICL and the crystalline lens but contact in the midperiphery.[131,134,137,143] Moreover, anteroposterior movement of the ICL during iris contraction or accommodation have led to intermittent central contact.[131,134] However, if the distance between the crystalline lens and posterior chamber pIOL is increased, the posterior chamber pIOL is closer to the iris with the consequent risk for pigment dispersion and development of pigment-induced secondary glaucoma.

In a study by Zaldivar et al.,[46] none of 124 eyes developed lens opacities due to ICL implantation. Nevertheless, one eye developed peripheral lens opacification at the position where Nd:YAG iridotomy

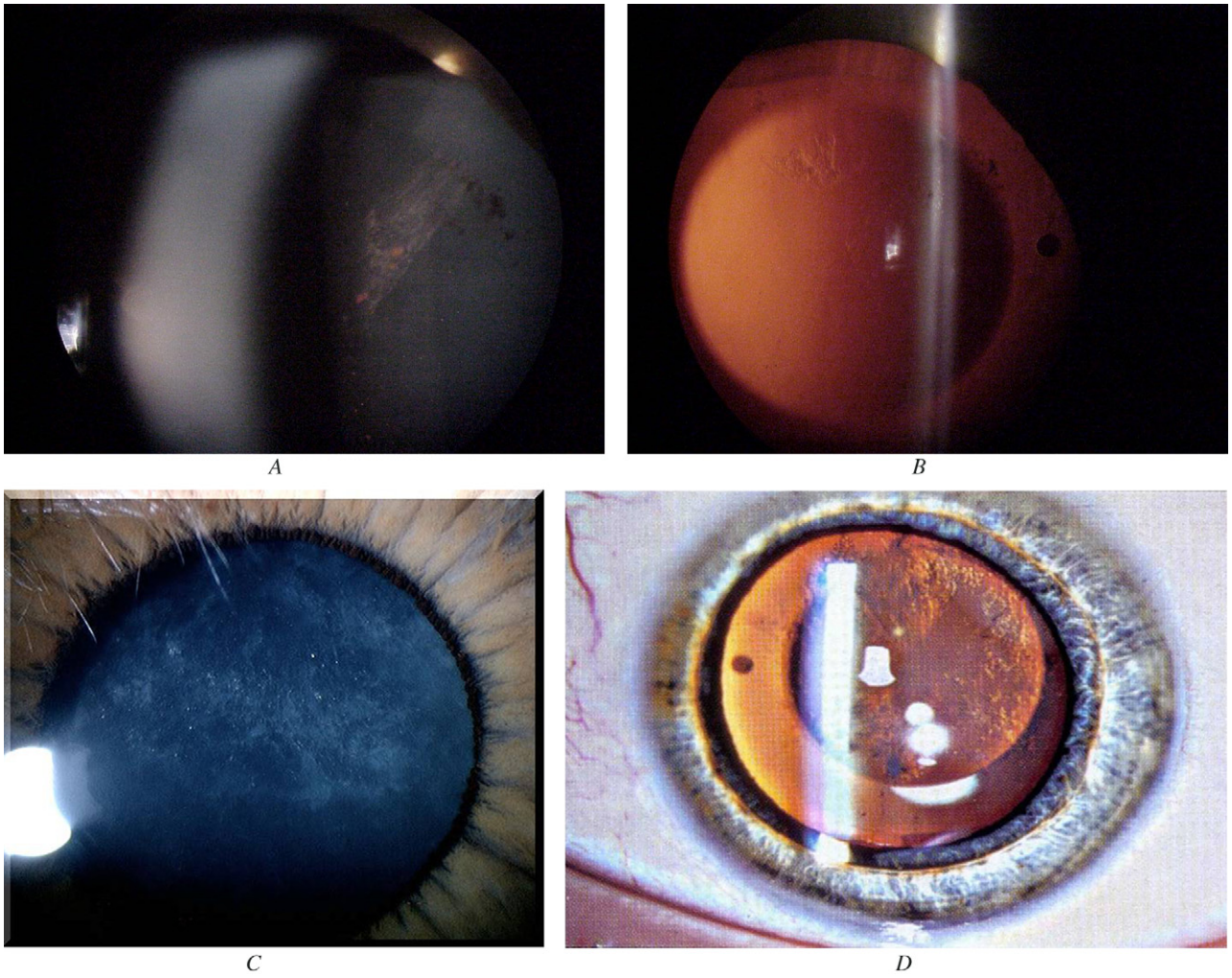
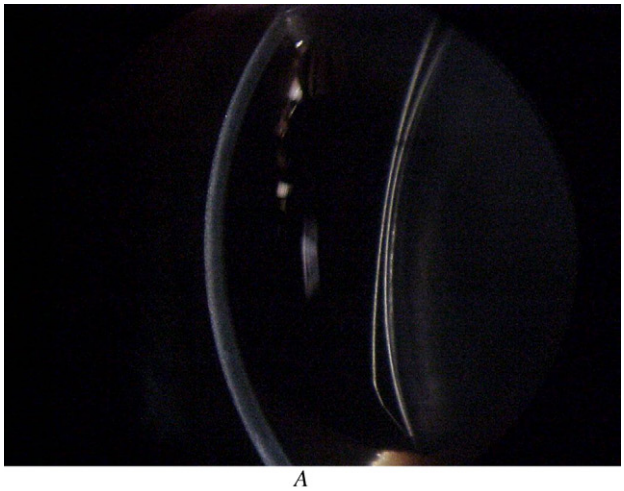


Figure 13. Cataract formation after implantation of posterior chamber pIOL. *A*: Faint anterior subcapsular opacities, 12 months after implantation (45-year-old woman). *B*: Same eye, retroillumination. *C*: Distinct anterior subcapsular cataract in an eye with posterior chamber pIOL. *D*: Retroillumination of anterior subcapsular cataract in an eye with posterior chamber pIOL (*C*: Courtesy of E. Rosen, Manchester, United Kingdom; *D*: Courtesy of J. Alió, Spain).

was performed preoperatively. Zadok and Chayet[148] report a case of focal lens opacification under the Nd:YAG laser iridotomy site, which did not enlarge after ICL implantation. Another study reports 2 eyes in one patient with anterior subcapsular cataractogenesis 1.5 years after ICL implantation.[42] Also, Trindade and Pereira[137] observed anterior subcapsular cataract formation in the eye of a 59-year-old patient 6 months after ICL implantation. The surgery was uneventful and atraumatic. With UBM, they were able to measure a central vault between the ICL and the natural lens, whereas contact was present in the midperiphery. Anterior subcapsular lens opacities developed in the noncontact area. Therefore, the authors surmised that both the proximity of the ICL to the natural lens, which may lead to metabolic disturbances, and pressure from the posterior chamber pIOL on the anterior surface of the crystalline lens could trigger cataract formation. In an FDA trial with a mean follow-up of 4.7 years, a cumulative probability estimate of 6% to 7% of anterior subcapsular opacities was found 7+ years after implantation of the Visian ICL.[149] However, only 1% to 2% progressed to a clinically significant cataract.

With various generations of the ICL, appearance of cataract formation is different. The less vaulted model V3 caused a higher incidence of cataract formation than the newer V4 and V5 models.[119] With the V4 model, the recently published FDA study showed an incidence of 2.1% anterior subcapsular opacities.[150] To prevent cataract formation, a sufficient vault between the posterior chamber pIOL and the lens seems to be important. With UBM, it was possible to measure central vault after implantation of ICL; in



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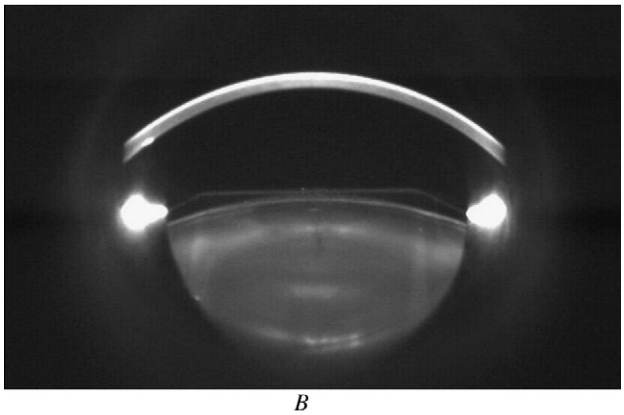

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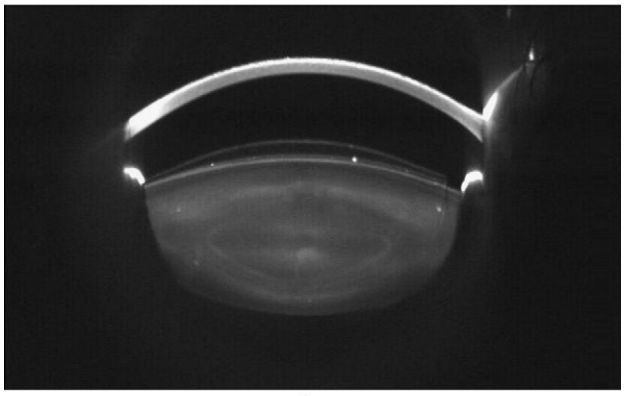

C

Figure 14. Contact between posterior chamber pIOL and crystalline lens. *A*: Myopic ICL, slitlamp image. Note delicate opacities in the lower hemisphere (40-year-old man). *B*: Myopic ICL, Scheimpflug image. *C*: Hyperopic ICL, Scheimpflug image.

the midperiphery, lens–IOL contact existed in most cases.[131,134,137] Also, size changes, the loss of the central vault, as well as changes in the location and extension of the contact zone were measured (Figure 14).[131,134] These findings would indicate anteroposterior shifts in the position of the ICL. Such shifts may be due to the flexibility of the pIOL material,

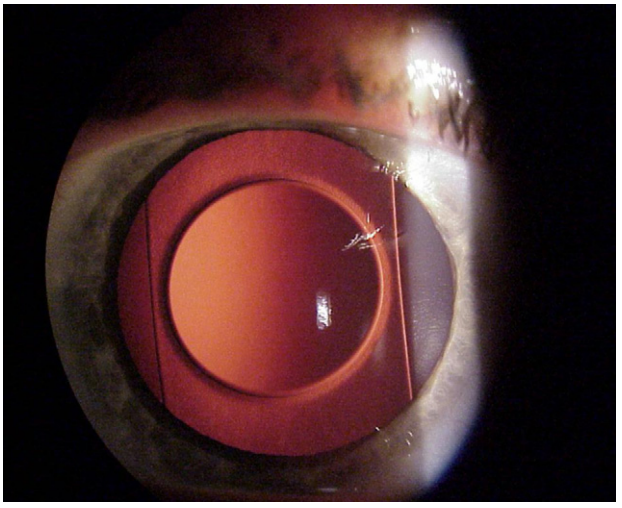

Figure 15. Residual OVD substance between a hyperopic ICL and the crystalline lens 1 week postoperatively (23-year-old woman).

which would allow the ICL to become deformed, perhaps while iris movements or accommodation occurred. Nevertheless, lens opacities did not effect visual acuity in any examined eye. One study evaluated the dynamics of the PRL in myopic and hyperopic eyes during accommodation with Visante OCT.[151] The PRL moved forward during accommodation in all eyes, with preserved distance between the anterior surface of the crystalline lens and the smaller PRL 100 model. However, with other PRL models, 101 in myopic eyes and 200 in hyperopic eyes, this distance decreased significantly. The authors conclude that this finding combined with the floating design of the PRL could permit aqueous humor circulation to the anterior surface of the crystalline lens, resulting in a less cataractogenic effect than with the ICL. After PRL implantation, Hoyos et al.[61] observed anterior cortical opacification in the immediate postoperative examination in one eye. This opacification remained stable up to 2 years of follow-up. The authors suggest that the touch of the natural lens during surgery was the trigger. Koivula and Zetterström[67] report no case of cataract formation one year after hyperopic PRL implantation. Other risk factors are experience of the surgeon, older patient age, and preexisting lens opacities.[49] As a differential diagnosis of lens opacities, residues of OVD substances (Figure 15) should be considered, particularly if the opacity is seen in the early postoperative period. If cataract formation progresses and leads to a decrease in visual acuity, posterior chamber pIOL explantation, phacoemulsification, and posterior chamber IOL implantation are indicated.[137,152]

Administration of pilocarpine in eyes with posterior chamber pIOLs should be considered carefully since

a case report demonstrated posterior chamber flattening and resulting crystalline lens opacification after instillation of pilocarpine eyedrops in a 46-year-old hyperopic patient who had ICL implantation.[153] As for all pIOLs, one should also consider that excessive use of steroids postoperatively is a potential cause of cataract formation.[87]

Retinal Detachment As for all intraocular surgeries, implantation of a posterior chamber pIOL carries a potential risk for vitreoretinal complications and RD. Most implantations of posterior chamber pIOLs are performed in patients with high myopia and long axial length, who therefore have a predisposition for spontaneous RD, as discussed previously. Thorough preoperative and postoperative fundoscopic investigation is mandatory to rule out retinal changes and to perform prophylactic laser photocoagulation, if required. Zaldivar et al.[46] report a single case of RD after implantation of a posterior chamber pIOL in 124 eyes. In this myopic patient, no causal relationship to pIOL surgery was noted. Panozzo and Parolini[154] describe 4 cases of RD after posterior chamber pIOL implantation in a consecutive case series. Two of the 4 cases had giant retinal tears. One case of bilateral giant retinal tear was reported 4 months after posterior chamber pIOL implantation. The patient had a history of RD.[155] Another case of RD as a late postoperative complication was reported after PRL implantation.[63] In a prospective study comprising 61 eyes, one eye developed RD 15 months after Visian ICL implantation.[52] This case was attributed to the pre-existing axial length of 31.0 mm and not to the pIOL surgery. The largest clinical trial reporting results in 526 eyes after Visian PIOL implantation found only 3 RDs.[41] The largest series of RD after posterior chamber pIOL surgery was published by Martőnez-Castillo et al.[156] and included 16 eyes after ICL implantation (ICMV2, ICMV3, and ICMV4). In this retrospective study, RD occurred from 1 to 70 months after lens surgery (mean 29 months) and no giant retinal tear or retinal dialysis was noted. As mean axial length of the 16 eyes was 30.1 mm, the authors conclude that these RDs were part of the natural history of RD in high myopia.

Oddity: Zonular Dehiscence There are some reports of serious complication with PRL luxation into the vitreous cavity. Eleftheriadis et al.[157] report a spontaneous dislocation of PRL 2 months after uneventful implantation into the vitreous cavity. Luxation was attributed to preexsisting zonular defect in the highly myopic eye and unrecognized ocular trauma. In a case report by Martőnez-Castillo et al.,[158] 2 patients had PRL luxation into the vitreous cavity after normal surgery, 4 and 22 months postoperatively. Hoyos et al.[159] report 2 cases of zonular dehiscence 2 years after PRL implantation in highly myopic eyes. Donoso and Castillo[63] report 2 cases of subluxation of PRL inferotemporally through the zonules with no predisposing factors. The authors speculate that an altered position and rotation of this type of pIOL and/or preoperative or undetected intraoperative trauma might contribute to this rare but potentially severe complication. For posterior chamber pIOL implantation, selection of a pIOL with an incorrect power is an avoidable complication that should not occur using current biometric formulas.

In summary, the main complications of anterior chamber pIOLs are glare and halos, pupil ovalization, and corneal endothelial cell loss; the main complications of iris-fixated pIOLs are chronic subclinical inflammation, corneal endothelial cell loss, dislocation or pupillary block glaucoma; and the main complications of posterior chamber pIOLs are anterior subcapsular cataract formation, pigment dispersion, pupillary block glaucoma, or luxation of pIOL (PRL). For all types of pIOLs, there is no established direct relationship between pIOL and RD.

DISCUSSION

According to Charles Kelman,[160] learning from complications of former and current pIOL models, a pIOL to be developed should fit the following requirements: The haptics should not damage the anterior chamber angle and haptics should not be in touch with peripheral corneal endothelium; the pIOL should not be in contact with any part of the iris that moves during pupillomotoric reflexes; the pIOL should be flexible if the assumed internal diameter might be smaller than the diameter of the pIOL; the pIOL should be placed in the largest diameter of the eye to avoid rotation; and the edges of pIOL should be smooth. Additionally, there should be sufficient space between the pIOL and the corneal endothelium and between the pIOL and the crystalline lens.

REFERENCES

1. Güell JL, Morral M, Kook D, Kohnen T. Phakic intraocular lenses. Part 1: historical overview, current models, selection criteria, and surgical techniques. J Cataract Refract Surg 2010; 36:1976–1993
2. Baikoff G, Arne JL, Bokobza Y, Colin J, George JL, Lagoutte F, Lesure P, Montard M, Saragoussi JJ, Secheyron P. Angle-fixated anterior chamber phakic intraocular lens for myopia of −7 to −19 diopters. J Refract Surg 1998; 14:282–293
3. Utine CA, Bayraktar S, Kaya V, Eren H, Prente I, Kucuksumer Y, Kevser MA, Yilmaz ÖF. ZB5M anterior chamber and Fyodorov's posterior chamber phakic intraocular lenses: long-term follow-up. J Refract Surg 2006; 22:906–910
4. Javaloy J, Alio JL, Iradier MT, Abdelrahman AM, Javaloy T, Borrás F. Outcomes of ZB5M angle-supported anterior chamber phakic intraocular lenses at 12 years. J Refract Surg 2007; 23:147–158

5. Pérez-Santonja JJ, Alió JL, Jiménez-Alfaro I, Zato MA. Surgical correction of severe myopia with an angle-supported phakic intraocular lens. J Cataract Refract Surg 2000; 26:1288–1302
6. Leccisotti A, Fields SV. Angle-supported phakic intraocular lenses in eyes with keratoconus and myopia. J Cataract Refract Surg 2003; 29:1530–1536
7. Leccisotti A. Iridocyclitis associated with angle-supported phakic intraocular lenses. J Cataract Refract Surg 2006; 32:1007–1010
8. Alió JL, Piñero D, Bernabeu G, Galal A, Vargas JM, Ismail MM. The Kelman Duet phakic intraocular lens: 1-year results. J Refract Surg 2007; 23:868–879
9. Gierek-Ciaciura S, Gierek-Lapinska A, Ochalik K, Mrukwa-Kominek E. Correction of high myopia with different phakic anterior chamber intraocular lenses: ICARE angle-supported lens and Verisyse iris-claw lens. Graefes Arch Clin Exp Ophthalmol 2007; 245:1–7
10. Baikoff G, Matach G, Fontaine A, Ferraz C, Spera C. Correction of presbyopia with refractive multifocal phakic intraocular lenses. J Cataract Refract Surg 2004; 30:1454–1460
11. Kohnen T, Knorz MC, Cochener B, Gerl RH, Arné J-L, Colin J, Alió JL, Bellucci R, Marinho A. AcrySof phakic angle-supported intraocular lens for the correction of moderate-to-high myopia: one year results of a multicenter European study. Ophthalmology 2009; 116:1314–1321. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642009001031.pdf. Accessed June 30, 2010
12. Alexander L, John M, Cobb L, Noblitt R, Barowsky RT. U.S. clinical investigation of the Artisan myopia lens for the correction of high myopia in phakic eyes. Report of the results of phases 1 and 2, and interim phase 3. Optometry 2000; 71:630–642
13. Budo C, Hessloehl JC, Izak M, Luyten GPM, Menezo JL, Sener BA, Tassignon MJ, Termote H, Worst JGF. Multicenter study of the Artisan phakic intraocular lens. J Cataract Refract Surg 2000; 26:1163–1171
14. Landesz M, Worst JGF, van Rij G. Long-term results of correction of high myopia with an iris claw phakic intraocular lens. J Refract Surg 2000; 16:310–316
15. Landesz M, van Rij G, Luyten G. Iris-claw phakic intraocular lens for high myopia. J Refract Surg 2001; 17:634–640
16. Maloney RK, Nguyen LH, John ME. Artisan phakic intraocular lens for myopia; short-term results of a prospective, multicenter study; the Artisan Lens Study Group. Ophthalmology 2002; 109:1631–1641. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642002011703.pdf. Accessed June 30, 2010
17. Malecaze FJ, Hulin H, Bierer P, Fournié P, Grandjean H, Thalamas C, Guell JL. A randomized paired eye comparison of two techniques for treating moderately high myopia; LASIK and Artisan phakic lens. Ophthalmology 2002; 109:1622–1630
18. Menezo JL, Peris-Martínez C, Cisneros AL, Martínez-Costa R. Phakic intraocular lenses to correct high myopia: Adatomed, Staar, and Artisan. J Cataract Refract Surg 2004; 30:33–44
19. Lifshitz T, Levy J, Aizenman I, Klemperer I, Levinger S. Artisan phakic intraocular lens for correcting high myopia. Int Ophthalmol 2004; 25:233–238
20. Benedetti S, Casamenti V, Marcaccio L, Brogioni C, Assetto V. Correction of myopia of 7 to 24 diopters with the Artisan phakic intraocular lens: two-year follow-up. J Refract Surg 2005; 21:116–126
21. Senthil S, Reddy KP, Ravisankar, Srilakshmi. A retrospective analysis of the first Indian experience on Artisan phakic intraocular lens. Indian J Ophthalmol 2006; 54:251–255. Available at: http://www.ijo.in/article.asp?issn=0301-4738;year=2006;volume=54;issue=4;spage=251;epage=255;aulast=Senthil. Accessed June 30, 2010
22. Coullet J, Guëll J-L, Fournié P, Grandjean H, Gaytan J, Arné J-L, Malecaze F. Iris-supported phakic lenses (rigid vs foldable version) for treating moderately high myopia: randomized paired eye comparison. Am J Ophthalmol 2006; 142:909–916
23. Moshirfar M, Holz HA, Davis DK. Two-year follow-up of the Artisan/Verisyse iris-supported phakic intraocular lens for the correction of high myopia. J Cataract Refract Surg 2007; 33:1392–1397
24. Tahzib NG, Nuijts RM, Wu WY, Budo CJ. Long-term study of Artisan phakic intraocular lens implantation for the correction of moderate to high myopia: ten-year follow-up results. Ophthalmology 2007; 114:1133–1142
25. Stulting RD, John ME, Maloney RK, Assil KK, Arrowsmith PN, Thompson VM. Three-year results of Artisan/Verisyse phakic intraocular lens implantation; results of the United States Food and Drug Administration Clinical Trial; the U.S. Verisyse Study Group. Ophthalmology 2008; 115:464–472. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642007009578.pdf. Accessed June 30, 2010
26. Silva RA, Jain A, Manche EE. Prospective long-term evaluation of the efficacy, safety, and stability of the phakic intraocular lens for high myopia. Arch Ophthalmol 2008; 126:775–781. Available at: http://archopht.ama-assn.org/cgi/reprint/126/6/775. Accessed June 30, 2010
27. Güell JL, Morral M, Gris O, Gaytan J, Sisquella M, Manero F. Five-year follow-up of 399 phakic Artisan-Verisyse implantation for myopia, hyperopia, and/or astigmatism. Ophthalmology 2008; 115:1002–1012
28. Fechner PU, Singh D, Wulff K. Iris-claw lens in phakic eyes to correct hyperopia: preliminary study. J Cataract Refract Surg 1998; 24:48–56
29. Alió JL, Mulet ME, Shalaby AMM. Artisan phakic iris claw intraocular lens for high primary and secondary hyperopia. J Refract Surg 2002; 18:697–707
30. Dick HB, Alió J, Bianchetti M, Budo C, Christiaans BJ, El-Danasoury MA, Güell JL, Krumeich J, Landesz M, Loureiro F, Luyten GPM, Marinho A, Rahhal MS, Schwenn O, Spirig R, Thomann U, Venter J. Toric phakic intraocular lens; European multicenter study. Ophthalmology 2003; 110:150–162
31. Saxena R, Landesz M, Noordzij B, Luyten GPM. Three-year follow-up of the Artisan phakic intraocular lens for hypermetropia. Ophthalmology 2003; 110:1391–1395
32. Pop M, Payette Y. Initial results of endothelial cell counts after Artisan lens for phakic eyes; an evaluation of the United States Food and Drug Administration Ophtec Study. Ophthalmology 2004; 111:309–317. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642003011850.pdf. Accessed June 30, 2010
33. Boxer Wachler BS, Scruggs RT, Yuen LH, Jalali S. Comparison of the Visian ICL and Verisyse phakic intraocular lenses for myopia from 6.00 to 20.00 diopters. J Refract Surg 2009; 25:765–770
34. Dick HB, Budo C, Malecaze F, Güell JL, Marinho AAP, Nuijts RMMA, Luyten GPM, Menezo JL, Kohnen T. Foldable Artiflex phakic intraocular lens for the correction of myopia: two-year follow-up results of a prospective European multicenter study. Ophthalmology 2009; 116:671–677. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642008013596.pdf. Accessed June 30, 2010
35. Tehrani M, Dick HB, Schwenn O, Blom E, Schmidt AH, Koch H-R. Postoperative astigmatism and rotational stability after Artisan toric phakic intraocular lens implantation. J Cataract Refract Surg 2003; 29:1761–1766

36. Güell JL, Vazquez M, Malecaze F, Manero F, Gris O, Velasco F, Hulin H, Pujol J. Artisan toric phakic intraocular lens for the correction of high astigmatism. Am J Ophthalmol 2003; 136:442–447
37. Alió JL, Mulet ME, Gutiérrez R, Galal A. Artisan toric phakic intraocular lens for correction of astigmatism. J Refract Surg 2005; 21:324–331
38. Nuijts RMMA, Abhilakh Missier KA, Nabar VA, Japing WJ. Artisan toric lens implantation for correction of postkeratoplasty astigmatism. Ophthalmology 2004; 111:1086–1094
39. Venter J. Artisan phakic intraocular lens in patients with keratoconus. J Refract Surg 2009; 25:759–764
40. Implantable Contact Lens in Treatment of Myopia (ITM) Study Group. U.S. Food and Drug Administration clinical trial of the Implantable Contact Lens for moderate to high myopia. Ophthalmology 2003; 110:255–266
41. ICL in Treatment of Myopia (ITM) Study Group. United States Food and Drug Administration clinical trial of the Implantable Collamer Lens (ICL) for moderate to high myopia; three-year follow-up. Ophthalmology 2004; 111:1683–1692. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642004005597.pdf. Accessed June 30, 2010
42. Uusitalo RJ, Aine E, Sen NH, Laatikainen L. Implantable contact lens for high myopia. J Cataract Refract Surg 2002; 28: 29–36
43. Jiménez-Alfaro I, Gómez-Tellería G, Bueno JL. Puy P: Contrast sensitivity after posterior chamber phakic intraocular lens implantation for high myopia. J Refract Surg 2001; 17: 641–645
44. Gonvers M, Othenin-Girard P, Bornet C, Sickenberg M. Implantable contact lens for moderate to high myopia; short-term follow-up of 2 models. J Cataract Refract Surg 2001; 27:380–388
45. Arne JL, Lesueur LC. Phakic posterior chamber lenses for high myopia: functional and anatomical outcomes. J Cataract Refract Surg 2000; 26:369–374
46. Zaldivar R, Davidorf JM, Oscherow S. Posterior chamber phakic intraocular lens for myopia of −8 to −19 diopters. J Refract Surg 1998; 14:294–305
47. Rosen E, Gore C. Staar Collamer posterior chamber phakic intraocular lens to correct myopia and hyperopia. J Cataract Refract Surg 1998; 24:596–606
48. Pineda-Fernández A, Jaramillo J, Vargas J, Jaramillo M, Jaramillo J, Galíndez A. Phakic posterior chamber intraocular lens for high myopia. J Cataract Refract Surg 2004; 30: 2277–2283
49. Lackner B, Pieh S, Schmidinger G, Hanselmayer G, Dejaco-Ruhswurm I, Funovics MA, Skorpik C. Outcome after treatment of ametropia with implantable contact lenses. Ophthalmology 2003; 110:2153–2161. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642003008303.pdf. Accessed June 30, 2010
50. Pesando PM, Ghiringhello MP, Tagliavacche P. Posterior chamber collamer phakic intraocular lens for myopia and hyperopia. J Refract Surg 1999; 15:415–423
51. Davidorf JM, Zaldivar R, Oscherow S. Posterior chamber phakic intraocular lens for hyperopia of +4 to +11 diopters. J Refract Surg 1998; 14:306–311
52. Chang JS, Meau AY. Visian Collamer phakic intraocular lens in high myopic Asian eyes. J Refract Surg 2007; 23:17–25
53. Kamiya K, Shimizu K, Aizawa D, Igarashi A, Komatsu M. Surgically induced astigmatism after posterior chamber phakic intraocular lens implantation. Br J Ophthalmol 2009; 93: 1648–1651
54. Sanders DR. Matched population comparison of the Visian Implantable Collamer Lens and standard LASIK for myopia of −3.00 to −7.88 diopters. J Refract Surg 2007; 23:537–553
55. Rayner SA, Bhikoo R, Gray T. Spherical implantable collamer lenses for myopia and hyperopia: 126 eyes with 1-year follow up. Clin Exp Ophthalmol 2010; 38:21–26
56. Schallhorn S, Tanzer D, Sanders DR, Sanders ML. Randomized prospective comparison of Visian Toric Implantable Collamer lens and conventional photorefractive keratectomy for moderate to high myopic astigmatism. J Refract Surg 2007; 23:853–867
57. Alfonso JF, Lisa C, Abdelhamid A, Montés-Micó R, Poo-López A, Ferrer-Blasco T. Posterior chamber phakic intraocular lenses after penetrating keratoplasty. J Cataract Refract Surg 2009; 35:1166–1173
58. Chang J, Lau S. Toric Implantable Collamer Lens for high myopic astigmatic Asian eyes. Ophthalmology 2009; 116:2340–2347
59. Park SC, Kwun YK, Chung E-S, Ahn K, Chung T-Y. Postoperative astigmatism and axis stability after implantation of the STAAR Toric Implantable Collamer Lens. J Refract Surg 2009; 25:403–409
60. Pallikaris IG, Kalyvianaki MI, Kymionis GD, Panagopoulou SI. Phakic refractive lens implantation in high myopic patients: one-year results. J Cataract Refract Surg 2004; 30:1190–1197
61. Hoyos JE, Dementiev DD, Cigales M, Hoyos-Chacón J, Hoffer KJ. Phakic refractive lens experience in Spain. J Cataract Refract Surg 2002; 28:1939–1946
62. Verde CM, Teus MA, Arranz-Marquez E, Cazorla RG. Medennium posterior chamber phakic refractive lens to correct high myopia. J Refract Surg 2007; 23:900–904
63. Donoso R, Castillo P. Correction of high myopia with the PRL phakic intraocular lens. J Cataract Refract Surg 2006; 32: 1296–1300
64. Jongsareejit A. Clinical results with the Medennium phakic refractive lens for the correction of high myopia. J Refract Surg 2006; 22:890–897
65. Koivula A, Taube M, Zetterström C. Phakic refractive lens: two-year results. J Refract Surg 2008; 24:507–515
66. Gil-Cazorla R, Teus MA, Arranz-Marquez E, Marina-Verde C. Phakic refractive lens (Medennium) for correction of +4.00 to +6.00 diopters: 1-year follow-up. J Refract Surg 2008; 24:350–354
67. Koivula A, Zetterström C. Phakic intraocular lens for the correction of hyperopia. J Cataract Refract Surg 2009; 35: 248–255
68. Gimbel HV, Ziémba SL. Management of myopic astigmatism with phakic intraocular lens implantation. J Cataract Refract Surg 2002; 28:883–886
69. Mertens EL, Sanders DR, Vitale PN. Custom-designed toric phakic intraocular lenses to correct high corneal astigmatism. J Refract Surg 2008; 24:501–506
70. Baikoff G. Anterior segment OCT and phakic intraocular lenses: a perspective. J Cataract Refract Surg 2006; 32:1827–1835
71. Endophthalmitis Vitrectomy Study Group. Results of the Endophthalmitis Vitrectomy Study; a randomized trial of immediate vitrectomy and of intravenous antibiotics for the treatment of postoperative bacterial endophthalmitis. Arch Ophthalmol 1995; 113:1479–1496. Available at: http://archopht.ama-assn.org/cgi/reprint/113/12/1479. Accessed June 30, 2010
72. ESCRS Endophthalmitis Study Group. Prophylaxis of postoperative endophthalmitis following cataract surgery: results of the ESCRS multicenter study and identification of risk factors. J Cataract Refract Surg 2007; 33:978–988
73. Pérez-Santonja JJ, Ruíz-Moreno JM, de la Hoz F, Giner-Gorriti C, Alió JL. Endophthalmitis after phakic intraocular

lens implantation to correct high myopia. J Cataract Refract Surg 1999; 25:1295–1298
74. Alió JL, de la Hoz F, Pérez-Santonja JJ, Ruiz-Moreno JM, Quesada JA. Phakic anterior chamber lenses for the correction of myopia: a 7-year cumulative analysis of complications in 263 cases. Ophthalmology 1999; 106:458–466. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642099901033.pdf. Accessed June 30, 2010
75. Allemann N, Chamon W, Tanaka HM, Mori ES, Campos M, Schor P, Bäikoff G. Myopic angle-supported intraocular lenses; two-year follow-up. Ophthalmology 2000; 107:1549–1554. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642000002219.pdf. Accessed June 30, 2010
76. Alió JL, Abdelrahman AM, Javaloy J, Iradier MT, Ortuño V. Angle-supported anterior chamber phakic intraocular lens explantation; causes and outcome. Ophthalmology 2006; 113:2213–2220. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642006007524.pdf. Accessed June 20, 2010
77. Kohnen T, Klaproth OK. Three-year stability of an angle-supported foldable hydrophobic acrylic phakic intraocular lens evaluated by Scheimpflug photography. J Cataract Refract Surg 2010; 36:1120–1126
78. Leccisotti A, Fields SV. Clinical results of ZSAL-4 angle-supported phakic intraocular lenses in 190 myopic eyes. J Cataract Refract Surg 2005; 31:318–323
79. Maroccos R, Vaz F, Marinho A, Guell J, Lohmann CP. Blendempfindlichkeit und Halos nach "phakic IOL"-Operation zur Behandlung einer hohen Myopie [Glare and halos after "phakic IOL" surgery for the correction of high myopia]. Ophthalmologe 2001; 98:1055–1059
80. Yu A, Wang Q, Xue A, Zhu S, Wang S, Chen Y, Sun J. Comparison of contrast sensitivity after angle-supported, iris-fixated and posterior chamber phakic intraocular lens implantation for high myopia. Ophthalmologica 2008; 222:53–57
81. Bühren J, Kohnen T. Anwendung der Wellenfrontanalyse in Klinik und Wissenschaft Vom irregulären Astigmatismus zu Aberrationen höherer Ordnung – Teil II: Beispiele [Application of wavefront analysis in clinical and scientific settings. From irregular astigmatism to aberrations of a higher order – Part II: examples]. Ophthalmologe 2007; 104:991–1006; CME-Fragebogen 1007–1008
82. Alio JL, De La Hoz F, Ismail M. Subclinical inflammatory reaction induced by phakic anterior chamber lenses for the correction of high myopia. Ocul Immunol Inflamm 1993; 1,3:219–224
83. Van Cleynenbreugel H. Late postoperative complications of backward implantation of a Vivarte phakic intraocular lens. J Cataract Refract Surg 2007; 33:1474–1476
84. Ardjomand N, Kölli H, Vidic B, El-Shabrawi Y, Faulborn J. Pupillary block after phakic anterior chamber intraocular lens implantation. J Cataract Refract Surg 2002; 28:1080–1081
85. Alió JL, de la Hoz F, Ruiz-Moreno JM, Salem TF. Cataract surgery in highly myopic eyes corrected by phakic anterior chamber angle-supported lenses. J Cataract Refract Surg 2000; 26:1303–1311
86. Chen L-J, Chang Y-J, Kuo JC, Rajagopal R, Azar DT. Metaanalysis of cataract development after phakic intraocular lens surgery. J Cataract Refract Surg 2008; 34:1181–1200
87. Lovisolo CF, Reinstein DZ. Phakic intraocular lenses. Surv Ophthalmol 2005; 50:549–587
88. Ruiz-Moreno JM, Alió JL, Pérez-Santonja JJ, de la Hoz F. Retinal detachment in phakic eyes with anterior chamber intraocular lenses to correct severe myopia. Am J Ophthalmol 1999; 127:270–275
89. Kaluzny J. Myopia and retinal detachment. Pol Med J 1970; 9:1544–1549
90. Yuzbasioglu E, Helvacioglu F, Sencan S. Fixed, dilated pupil after phakic intraocular lens implantation. J Cataract Refract Surg 2006; 32:174–176
91. Menezo JL, Aviño JA, Cisneros A, Rodriguez-Salvador V, Martinez-Costa R. Iris claw phakic intraocular lens for high myopia. J Refract Surg 1997; 13:545–555
92. Chung S-H, Lee SJ, Lee HK, Seo KY, Kim EK. Changes in higher order aberrations and contrast sensitivity after implantation of a phakic artisan intraocular lens. Ophthalmologica 2007; 221:167–172
93. Chandhrasri S, Knorz MC. Comparison of higher order aberrations and contrast sensitivity after LASIK, Verisyse phakic IOL, and Array multifocal IOL. J Refract Surg 2006; 22:231–236
94. Tahzib NG, MacRae SM, Yoon G, Berendschot TTJM, Eggink FAGJ, Hendrikse F, Nuijts RMMA. Higher-order aberrations after implantation of iris-fixated rigid or foldable phakic intraocular lenses. J Cataract Refract Surg 2008; 34:1913–1920
95. Cisneros-Lanuza A, Hurtado-Sarrió M, Duch-Samper A, Gallego-Pinazo R, Menezo-Rozalén JL. Glistenings in the Artiflex phakic intraocular lens. J Cataract Refract Surg 2007; 33:1405–1408
96. Coullet J, Gontran E, Fournié P, Arné JL, Malcaze F. Efficacité réfractive et tolérance de l'implant phaque myopique souple à fixation irienne Artiflex® dans la correction chirurgicale de la myopie forte: résultats à deux ans [Refractive efficacy and tolerance of the foldable phakic iris-supported Artiflex® lens in the surgical correction of moderately high myopia: a 2-year follow-up]. J Fr Ophtalmol 2007; 30:335–343
97. Fechner PU, Strobel J, Wichmann W. Correction of myopia by implantation of a concave Worst-iris claw lens into phakic eyes. Refract Corneal Surg 1991; 7:286–898
98. Menezo JL, Cisneros AL, Rodriguez-Salvador V. Endothelial study of iris-claw phakic lens: four year follow-up. J Cataract Refract Surg 1998; 24:1039–1049
99. Krumeich JH, Daniel J, Gast R. Closed-system technique for implantation of iris-supported negative-power intraocular lens. J Refract Surg 1996; 12:334–340
100. Groß S, Knorz MC, Liermann A, Troost R, Weigold S, Jendritza B, Liesenhoff H. Ergebnisse nach Implantation einer Worst Iris Claw Lens zur Korrektur der hohen Myopie [Results of implantation of a Worst Iris Claw lens for correction of high myopia]. Ophthalmologe 2001; 98:635–638
101. Bourne WM, Nelson LR, Hodge DO. Central corneal endothelial cell changes over a ten-year period. Invest Ophthalmol Vis Sci 1997; 38:779–782. Available at: http://www.iovs.org/cgi/reprint/38/3/779. Accessed June 3, 2010
102. de Vries NE, Tahzib NG, Budo CJ, Webers CAB, de Boer R, Hendrikse F, Nuijts RMMA. Results of cataract surgery after implantation of an iris-fixated phakic intraocular lens. J Cataract Refract Surg 2009; 35:121–126
103. Pérez-Santonja JJ, Bueno JL, Zato MA. Surgical correction of high myopia in phakic eyes with Worst-Fechner myopia intraocular lenses. J Refract Surg 1997; 13:268–281; discussion 281–284
104. Saxena R, Boekhoorn SS, Mulder PGH, Noordzij B, van Rij G, Luyten GPM. Long-term follow-up of endothelial cell change after Artisan phakic intraocular lens implantation. Ophthalmology 2008; 115:608–613. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642007005933.pdf. Accessed June 30, 2010
105. Benedetti S, Casamenti V, Benedetti M. Long-term endothelial changes in phakic eyes after Artisan intraocular lens implantation to correct myopia; five-year study. J Cataract Refract Surg 2007; 33:784–790

106. Doors M, Berendschot TTJM, Webers CAB, Nuijts RMMA. Model to predict endothelial cell loss after iris-fixated phakic intraocular lens implantation. Invest Ophthalmol Vis Sci 2010; 51:811–815
107. Baikoff G, Lutun E, Ferraz C, Wei J. Analyse du segment antérieur de l'oeil avec un tomographe à cohérence optique. Étude statique et dynamique [Analysis of the eye's anterior segment with optical coherence tomography. Static and dynamic study]. J Fr Ophtalmol 2005; 28:343–352
108. Doors M, Cals DWJK, Berendschot TTJM, de Brabander J, Hendrikse F, Webers CAB, Nuijts RMMA. Influence of anterior chamber morphometrics on endothelial cell changes after phakic intraocular lens implantation. J Cataract Refract Surg 2008; 34:2110–2118
109. Pop M, Mansour M, Payette Y. Ultrasound biomicroscopy of the iris-claw phakic intraocular lens for high myopia. J Refract Surg 1999; 15:632–635
110. Pop M, Payette Y, Mansour M. Ultrasound biomicroscopy of the Artisan phakic intraocular lens in hyperopic eyes. J Cataract Refract Surg 2002; 28:1799–1803
111. Comaish IF, Lawless MA. Phakic intraocular lenses. Curr Opin Ophthalmol 2002; 13:7–13
112. Baïkoff G, Bourgeon G, Jodai HJ, Fontaine A, Viera Lellis F, Trinquet L. Pigment dispersion and Artisan phakic intraocular lenses; crystalline lens rise as a safety criterion. J Cataract Refract Surg 2005; 31:674–680
113. Koss MJ, Cichocki M, Kohnen T. Posterior synechias following implantation of a foldable silicone iris-fixated phakic intraocular lens for the correction of myopia. J Cataract Refract Surg 2007; 33:905–909
114. Tahzib NG, Eggink FAGJ, Frederik PM, Nuijts RMMA. Recurrent intraocular inflammation after implantation of the Artiflex phakic intraocular lens for the correction of high myopia. J Cataract Refract Surg 2006; 32:1388–1391
115. Moshirfar M, Whitehead G, Beutler BC, Mamalis N. Toxic anterior segment syndrome after Verisyse iris-supported phakic intraocular lens implantation. J Cataract Refract Surg 2006; 32:1233–1237
116. Pérez-Torregrosa VT, Menezo JL, Harto MA, Maldonado MJ, Cisneros A. Digital system measurement of decentration of Worst-Fechner iris claw myopia intraocular lens. J Refract Surg 1995; 11:26–30
117. Yamaguchi T, Negishi K, Yuki K, Saiki M, Nishimura R, Kawaguchi N, Tsubota K. Alterations in the anterior chamber angle after implantation of iris-fixated phakic intraocular lenses. J Cataract Refract Surg 2008; 34:1300–1305
118. Kodjikian L, Gain P, Donate D, Rouberol F, Burillon C. Malignant glaucoma induced by a phakic posterior chamber intraocular lens for myopia. J Cataract Refract Surg 2002; 28: 2217–2221
119. Menezo JL, Peris-Martínez C, Cisneros A, Martínez-Costa R. Posterior chamber phakic intraocular lenses to correct high myopia: a comparative study between Staar and Adatomed models. J Refract Surg 2001; 17:32–42
120. Baumeister M, Bühren J, Kohnen T. Position of angle-supported, iris-fixated, and ciliary sulcus-implanted myopic phakic intraocular lenses evaluated by Scheimpflug photography. Am J Ophthalmol 2004; 138:723–731
121. Yoon H, Macaluso DC, Moshirfar M, Lundergan M. Traumatic dislocation of an Ophtec Artisan phakic intraocular lens. J Refract Surg 2002; 18:481–483
122. Menezo JL, Peris-Martínez C, Cisneros-Lanuza AL, Martínez-Costa R. Rate of cataract formation in 343 highly myopic eyes after implantation of three types of phakic intraocular lenses. J Refract Surg 2004; 20:317–324
123. Coonan P, Fung WE, Webster RG Jr, Allen AW Jr, Abbott RL. The incidence of retinal detachment following extracapsular cataract extraction; a ten-year study. Ophthalmology 1985; 92:1096–1101
124. Tielsch JM, Legro MW, Cassard SD, Schein OD, Javitt JC, Singer AE, Bass EB, Steinberg EP. Risk factors for retinal detachment after cataract surgery; a population-based case-control study. Ophthalmology 1996; 103:1537–1545
125. Beijing Rhegmatogenous Retinal Detachment Study Group. Incidence and epidemiological characteristics of rhegmatogenous retinal detachment in Beijing, China. Ophthalmology 2003; 110:2413–2417
126. Georgalas I, Petrou P, Papaconstantinou D, Koutsandrea C, Ladas I. Bilateral giant tear-associated retinal detachment following Artisan® phakic intraocular lens implantation for correction of moderate myopia [letter]. Acta Ophthalmol (Oxf) 2010; 88(4):e143–e144
127. Park SH, Kim SY, Kim HI, Yang SW. Urrets-Zavalia syndrome following iris-claw phakic intraocular lens implantation. J Refract Surg 2008; 24:959–961
128. Kohnen T, Cichocki M, Bühren J, Baumeister M. Intermittent myopic shift of 4.0 diopters after implantation of an Artisan iris-supported phakic intraocular lens. J Cataract Refract Surg 2005; 31:1444–1447
129. Edelhauser HF, Sanders DR, Azar R, Lamielle H. Corneal endothelial assessment after ICL implantation; the ICL in Treatment of Myopia Study Group. J Cataract Refract Surg 2004; 30:576–583
130. Fechner PU. Cataract formation with a phakic IOL [letter]. J Cataract Refract Surg 1999; 25:461; reply by Trindade F, Pereira F, 461–462
131. Jiménez-Alfaro I, Benítez del Castillo JM, García-Feijoó J, Gil de Bernabé JG, Serrano de la Iglesia JM. Safety of posterior chamber phakic intraocular lenses for the correction of high myopia; anterior segment changes after posterior chamber phakic intraocular lens implantation. Ophthalmology 2001; 108:90–99; discussion by SM MacRae, 99 Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642000004036.pdf. Accessed June 20, 2010
132. Dejaco-Ruhswurm I, Scholz U, Pieh S, Hanselmayer G, Lackner B, Italon C, Ploner M, Skorpik C. Long-term endothelial changes in phakic eyes with posterior chamber intraocular lenses. J Cataract Refract Surg 2002; 28:1589–1593
133. Kamiya K, Shimizu K, Igarashi A, Hikita F, Komatsu M. Four-year follow-up of posterior chamber phakic intraocular lens implantation for moderate to high myopia. Arch Ophthalmol 2009; 127:845–850
134. García-Feijoó J, Jiménez Alfaro I, Cuiña-Sardiña R, Méndez-Hernandez C, Benítez del Castillo JM, García-Sánchez J. Ultrasound biomicroscopy examination of posterior chamber phakic intraocular lens position. Ophthalmology 2003; 110:163–172. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642002014495.pdf. Accessed June 30, 2010
135. García-Feijoó J, Hernández-Matamoros JL, Castillo-Gómez A, Lázaro C, Méndez-Hernández C, Martín T, Martínez de la Casa JM, García-Sánchez J. High-frequency ultrasound biomicroscopy of silicone posterior chamber phakic intraocular lens for hyperopia. J Cataract Refract Surg 2003; 29:1940–1946
136. García-Feijoó J, Hernández-Matamoros JL, Méndez-Hernández C, Castillo-Goméz A, Lázaro C, Martín T, Cuiña-Sardiña R, García-Sánchez J. Ultrasound biomicroscopy of silicone posterior chamber phakic intraocular lens for myopia. J Cataract Refract Surg 2003; 29:1932–1939

137. Trindade F, Pereira F. Cataract formation after posterior chamber phakic intraocular lens implantation. J Cataract Refract Surg 1998; 24:1661–1663
138. Sanchez-Galeana CA, Zadok D, Montes M, Cortés MA, Chayet AS. Refractory intraocular pressure increase after phakic posterior chamber intraocular lens implantation. Am J Ophthalmol 2002; 134:121–123
139. ICL in Treatment of Myopia (ITM) Study Group. Postoperative inflammation after implantation of the implantable contact lens. Ophthalmology 2003; 110:2335–2341. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642003008261.pdf. Accessed June 30, 2010
140. Smallman DS, Probst L, Rafuse PE. Pupillary block glaucoma secondary to posterior chamber phakic intraocular lens implantation for high myopia. J Cataract Refract Surg 2004; 30:905–907
141. Featherstone KA, Bloomfield JR, Lang AJ, Miller-Meeks MJ, Woodworth G, Steinert RF. Driving simulation study: bilateral Array multifocal versus bilateral AMO monofocal intraocular lenses. J Cataract Refract Surg 1999; 25:1254–1262
142. Findl O, Kriechbaum K, Menapace R, Koeppl C, Sacu S, Wirtitsch M, Buehl W, Drexler W. Laserinterferometric assessment of pilocarpine-induced movement of an accommodating intraocular lens; a randomized trial. Ophthalmology 2004; 111: 1515–1521. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642004003409.pdf. Accessed June 30, 2010
143. Trindade F, Pereira F. Exchange of a posterior chamber phakic intraocular lens in a highly myopic eye. J Cataract Refract Surg 2000; 26:773–776
144. Baumeister M, Terzi E, Ekici Y, Kohnen T. Comparison of manual and automated methods to determine horizontal corneal diameter. J Cataract Refract Surg 2004; 30:374–380
145. Gonvers M, Bornet C, Othenin-Girard P. Implantable contact lens for moderate to high myopia; relationship of vaulting to cataract formation. J Cataract Refract Surg 2003; 29:918–924
146. Sanchez-Galeana CA, Smith RJ, Sanders DR, Rodríguez FX, Litwak S, Montes M, Chayet AS. Lens opacities after posterior chamber phakic intraocular lens implantation. Ophthalmology 2003; 110:781–785. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642002019735.pdf. Accessed June 30, 2010
147. Fechner PU, Haigis W, Wichmann W. Posterior chamber myopia lenses in phakic eyes. J Cataract Refract Surg 1996; 22:178–182
148. Zadok D, Chayet A. Lens opacity after neodymium:YAG laser iridectomy for phakic intraocular lens implantation. J Cataract Refract Surg 1999; 25:592–593
149. Sanders DR. Anterior subcapsular opacities and cataracts 5 years after surgery in the Visian Implantable Collamer Lens FDA trial. J Refract Surg 2008; 24:566–570
150. Implantable Contact Lens in Treatment of Myopia (ITM) Study Group. U.S. Food and Drug Administration clinical trial of the Implantable Contact Lens for moderate to high myopia. Ophthalmology 2003; 110:255–266
151. Koivula A, Kugelberg M. Optical coherence tomography of the anterior segment in eyes with phakic refractive lenses. Ophthalmology 2007; 114:2031–2037. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642007007178.pdf. Accessed June 30, 2010
152. Wiechens B, Winter M, Haigis W, Happe W, Behrendt S, Rochels R. Bilateral cataract after phakic posterior chamber top hat-style silicone intraocular lens. J Refract Surg 1997; 13:392–397
153. Maldonado MJ, García-Feijoó J, Benítez Del Castillo JM, Teutsch P. Cataractous changes due to posterior chamber flattening with a posterior chamber phakic intraocular lens secondary to the administration of pilocarpine. Ophthalmology 2006; 113:1283–1288. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642006005513.pdf. Accessed June 20, 2010
154. Panozzo G, Parolini B. Relationships between vitreoretinal and refractive surgery. Ophthalmology 2001; 108:1663–1668. discussion by A Rodriguez, 1668–1669. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642001006728.pdf. Accessed June 20, 2010
155. Navarro R, Gris O, Broc L, Corcóstegui B. Bilateral giant retinal tear following posterior chamber phakic intraocular lens implantation. J Refract Surg 2005; 21:298–300
156. Martínez-Castillo V, Boixadera A, Verdugo A, Elíes D, Coret A, García-Arumí J. Rhegmatogenous retinal detachment in phakic eyes after posterior chamber phakic intraocular lens implantation for severe myopia. Ophthalmology 2005; 112:580–585. Available at: http://download.journals.elsevierhealth.com/pdfs/journals/0161-6420/PIIS0161642004015234.pdf. Accessed June 30, 2010
157. Eleftheriadis H, Amoros S, Bilbao R, Teijeiro M-A. Spontaneous dislocation of a phakic refractive lens into the vitreous cavity. J Cataract Refract Surg 2004; 30:2013–2016
158. Martínez-Castillo V, Elies D, Boixadera A, García-Arumí J, Mauricio J, Cavero L, Coret A. Silicone posterior chamber phakic intraocular lens dislocated into the vitreous cavity. J Refract Surg 2004; 20:773–777
159. Hoyos JE, Cigales M, Hoyos-Chacón J. Zonular dehiscence two years after phakic refractive lens (PRL) implantation. J Refract Surg 2005; 21:13–17
160. Kelman C. Phakic intraocular lenses. Are the sirens singing again? [editorial] J Refract Surg 1998; 14:273–274

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