Intraocular Lens Complications Requiring Removal or Exchange

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Abstract. Intraocular lens (IOL)-related complications are caused primarily by mechanical trauma, inflammatory or infectious complications, or optical problems. Complications may occur at the time of surgery or be the result of an ongoing postoperative process. Mechanical and inflammatory injury may produce corneal decompensation, cystoid macular edema, hyphema, uveitis, and glaucoma, causing reduced vision and in some cases chronic pain. Optical problems may be due to a wrong power of the IOL or to postoperative decentration or dislocation of the lens. Ophthalmologists should be aware of the indications for IOL removal or exchange in those patients who have ongoing IOL-induced injury or impairment. Removal or exchange of an IOL frequently involves a complex decision-making process and is often associated with immense technical challenge. Various medical and surgical treatments may be tried to correct IOL problems before the decision is made to remove or exchange the lens. (Surv Ophthalmol 42:417–440, 1998. © 1998 by Elsevier Science Inc. All rights reserved.)

Key words. bullous keratopathy • cataract surgery • complications of intraocular lenses • corneal edema • cystoid macular edema • glaucoma • hyphema • malposition of intraocular lenses • removal or exchange of intraocular lenses • uveitis

Although complications from intraocular lenses (IOLs) were common in the early days of cataract surgery,8,215 improvements in surgical technique and IOL design and quality have reduced the problems. Mechanisms for IOL-related complications fall into four groups: mechanical trauma, chronic inflammation, infectious disease, and optical problems (Table 1).

Mechanical trauma from an IOL may occur at the time of original surgery or it may be the result of ongoing intraocular injury. Surgical contact with the corneal endothelium causes endothelial cell loss, which may cause transient or persistent dysfunction with corneal decompensation. Anterior chamber (AC)-IOLs also may produce ongoing mechanical trauma to the cornea, iris, ciliary body, or the anterior chamber angle. Mechanical injury to the iris may produce atrophy and chronic uveitis, potentially compromising aqueous outflow and leading to glaucoma. Erosion of the IOL into the ciliary body also may cause ocular discomfort.

A more subtle form of injury may result from chronic inflammation produced by ongoing mechanical trauma. The IOL may erode into uveal tissue, liberating inflammatory mediators that damage other ocular structures; this is especially common with oversized or older AC-IOLs with a closed-loop design. These inflammatory products may contribute to progressive corneal endothelial cell loss, cystoid macular edema (CME), and glaucoma.

A more serious form of inflammatory injury occasionally results from infectious endophthalmitis. Acute infectious endophthalmitis usually is not di-
Mechanisms for IOL-Related Complications

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prosthesis and IOL exchange.

TABLE 1
Mechanisms for IOL-Related Complications

rectly attributable to IOL implantation, although epidemiologic data suggest that polypropylene loops are a risk factor for endophthalmitis. Menikoff et al reported that one-piece polymethylmethacrylate lenses appear less likely to be associated with infectious endophthalmitis than multi-piece design lenses with polypropylene loops. Successful treatment of endophthalmitis seldom requires IOL removal, although some patients with localized endophthalmitis are resistant to treatment until the IOL and the capsular bag are removed.

Optical problems may be caused by improper power selection or abnormal IOL position. Improper IOL power may produce an unacceptable refractive error, which may require IOL exchange. Malposition may produce symptoms associated with the optic edge, IOL tilt, or other IOL structures, such as positioning holes, lens loops, or laser ridges.

Failure of an IOL represents a great disappointment to the patient, who is usually aware of many successful outcomes of cataract/IOL surgery among friends. The patient often blames the surgeon and seeks a new ophthalmologist for follow-up care of the complications; thus, the original surgeon may not even be aware of the eventual need for IOL explantation. The consulting physician can help the patient to understand that the original surgeon shared a high level of optimism and that, although an IOL may currently be recognized as a poor design, it may have been acceptable or even the state-of-the-art IOL at the time of the original surgery.

Corneal Complications

Corneal complications were the leading indication for IOL explantation before the development of newer IOLs and surgical techniques. The corneal endothelium consists of a monolayer of nonregenerative cells responsible for maintaining corneal clarity by its pump function. The cells that make up this layer are among the most delicate type in the human body.

Corneal endothelial dysfunction after cataract surgery may be mild-to-moderate and transient (resulting in temporary microcystic edema and visual impairment), or it may be severe and permanent. Persistent endothelial dysfunction with corneal decompensation is currently the most common indication for penetrating keratoplasty.

Surgical trauma can cause corneal decompensation, but the condition may be hastened by pre-existing corneal disease (e.g., Fuchs' dystrophy). Endothelial cell loss related to the IOL may be caused by direct mechanical trauma, including excessive vaulting of an AC-IOL, lens mobility (pseudophakodonesis) of an undersized or poorly fixated IOL (tilting, rocking, or rotating [propellering]), or IOL malposition (e.g., migration through a peripheral iridectomy). Even peripheral corneal-IOL contact has the potential to cause central endothelial dysfunction. Peripheral cell loss may stimulate endothelial migration and reduce endothelial cell density centrally, causing corneal decompensation.

Erosion of an IOL into uveal tissue may incite inflammation and cause the liberation of inflammatory mediators, such as prostaglandins, which may damage the corneal endothelium. Inflammation is often the mechanism for late corneal decompensation associated with closed-loop AC-IOL designs that erode into peripheral iris, anterior chamber angle, and ciliary body. This mechanism can explain the late corneal decompensation that may occur in patients who appear to have stable IOLs that are not directly traumatizing the corneal endothelium. Because the inflammation often is very mild, careful examination is required to reveal subtle cell and flare in the anterior chamber and fine inflammatory precipitates on the IOL surface. Inflammation associated with the semiflexible, closed-loop style AC-IOLs contributed to their removal from the USA market. Corneal failure may also result from continuing postoperative endothelial cell loss associated with aging, ocular disease, laser treatment, or iris or vitreous contact with the endothelium.

PSEUDOPHAKIC CORNEAL EDEMA

Intraocular Lens Design

We prefer pseudophakic corneal edema over the term pseudophakic bullous keratopathy to describe patients with corneal decompensation, because not all patients develop bullae. Pseudophakic corneal edema is the leading indication for penetrating keratoplasty. Kraff et al reported that 36% of IOL removals are performed because of pseudophakic corneal edema. In a series of 101 eyes of 98 consecutive patients, Lyle and Jin reported bullous keratopathy as the causative indication in 86.7% of patients who underwent combined penetrating keratoplasty and IOL exchange.
When corneal decompensation is associated with the IOL design (e.g., the semiflexible, closed-loop design [Fig. 1]), the IOL should always be exchanged or removed when penetrating keratoplasty is performed. Such designs include the Leiske Style 10 (Surgical Corp., Goleta, CA), Azar 91Z (IOLAB Corp., Covina, CA), Optical Radiation Corp. model 11 Stableflex (Azusa, CA), the Intermedics Hessburg model 024 (Intraocular Inc., Pasadena, CA), and the Pannu AC-IOL (Allergan Medical Optics, Irvine, CA).8 Also to be avoided are AC-IOL designs featuring positioning holes or eyelets, which may erode into the angle and act as a smaller version of the closed-loop designs.8,155 Flexible, open, thin-loop lenses, such as the Dubroff-style (Intermedics Pharmacia, Monrovia, CA) AC-IOL, may also erode into the peripheral angle and create low-grade, chronic inflammation with concomitant corneal endothelial damage and CME.147

Surgical results of penetrating keratoplasty after removal or exchange of closed-loop AC-IOLs have been good. Kornmehl et al reported an average recovery of corrected visual acuity to 20/44 in 40 consecutive patients who underwent exchange of a closed-loop AC-IOL (30 Leiske, 10 Stableflex) for a Kelman Omnifit AC-21 AC-IOL (Allergan Medical Optics) at the time of penetrating keratoplasty.135 Koenig et al reported good results in 20 consecutive patients with corneal decompensation associated with a Stableflex-style AC-IOL who underwent penetrating keratoplasty, anterior vitrectomy, and IOL exchange for a Kelman Multiflex-style, open-loop AC-IOL (Cooper Vision Cilco Inc., Pomona, CA).135 Thirty-five percent (7 of 20) attained a spectacle correction of 20/40 or better, and all grafts remained clear during a follow-up period of 4–45 months.

Zaidman and Goldman reported a 90% chance of allograft clarity at the end of 12–18 months in patients undergoing penetrating keratoplasty and IOL exchange with a Kelman-style, open-loop AC-IOL or posterior chamber (PC)-IOL.285

Price et al noted that 20.6% (7 of 34) patients developed allograft failure with retained AC-IOLs compared with 5.1% (4 of 78) in patients with retained PC-IOLs at the time of keratoplasty.205 In contrast, Speaker et al reported a large number of graft failures in patients who retained closed-loop AC-IOLs at the time of keratoplasty.245 Allograft failure was noted in 60% of these patients at 1 year, 75% at 2 years, and 100% at 5 years. Smith et al, Insler et al,118 and Cohen et al reported series in which allograft outcomes were compromised by retention of closed-loop AC-IOLs.

Removal of iris-supported lenses also should be considered at the time of penetrating keratoplasty, particularly if the IOL appears poorly fixated or excessively mobile. Despite initial reports suggesting a favorable surgical outcome in patients who retained these lenses, subsequent longer-term studies invariably demonstrated a higher rate of complications.12,40,74,145,167,226,258,270 Speaker et al reported that 75% of the allograft failures in patients with retained iris-supported IOLs occurred after the second postoperative year.213 Exchange did not appear to offer significant advantage over retention in their study. Rao et al found endothelial cell loss to occur at a greater rate with iris-supported IOLs than with Choyce-style AC-IOLs.207 Several additional authors noted that patients with retained iris-supported IOLs had the worst prognosis for maintenance of a clear graft.157,191,226

Reports by Meyer and Sugar,167 Kozarsky et al,137 and Waring et al257 support leaving a properly positioned, secure PC-IOL or modern flexible open-loop AC-IOL at the time of keratoplasty. Notably, Price et al reported that grafts for pseudophakic bullous keratopathy with retained posterior chamber lenses had a significantly higher failure rate from rejection and endothelial decompensation, 5.1%, compared with less than 1.0% in keratoconus, Fuchs’ dystrophy, or bullous keratopathy with secondary implants.205

Several authors have addressed the clinical and technical strategies associated with IOL exchange during penetrating keratoplasty.27,54,59,62,92,99,114,133,135,146,199,205,207,241,272,281,285

Instability of the Intraocular Lens

Excessive IOL mobility (pseudophakodonesis) jeopardizes allograft success. An unstable IOL should be removed at the time of keratoplasty unless it can be safely stabilized in a secure position and is considered a safe IOL design. Even modern, acceptable IOLs should be removed if they cannot be properly
fixated, e.g., because of inadequate anatomic support, improper sizing, etc.

Clinically, the best indication of unacceptable pseudophakodonesis is “shimmering” from mobile Purkinje-Sanson images III and IV from the anterior and posterior IOL surfaces, which can be observed during examination of subtle eye movements at the slit lamp (Holladay J: personal communication, 1996). Miller and Doane\textsuperscript{169} and Jacobs et al\textsuperscript{121} used high-speed cinematography to demonstrate pseudophakodonesis with corneal touch associated with iris-supported IOLs. Rao et al identified progressive corneal endothelial cell loss over time.\textsuperscript{207}

**PERIPHERAL CORNEAL EDEMA**

Corneal decompensation due to ongoing mechanical injury may be caused by central and/or peripheral endothelial contact with the IOL. Intermittent or constant central corneal touch may cause accelerated loss of endothelial cells.\textsuperscript{106,107,112–121} Corneal touch is more commonly associated with AC-IOLs and may be caused by improper IOL positioning, IOL migration due to improper IOL size, ocular trauma (including eye rubbing), excessive IOL vaulting, or prolonged head-down position.\textsuperscript{60} Migration of an AC-IOL through a wound dehiscence or a peripheral iridectomy (Fig. 2A) may also cause IOL-corneal touch (Fig. 2B).

Patients with peripheral IOL-corneal touch may initially have no signs or symptoms of corneal decompensation. However, although the central cornea may appear clear, the peripheral contact may cause local cell loss, which can stimulate cell migration from the central cornea to the periphery. Eventually, continued endothelial cell loss will result in central corneal decompensation unless the source of damage is removed.

To undergo IOL repositioning, removal, or exchange, the cornea must have adequate endothelial reserve. Clinical signs of inadequate endothelial reserve may include a low cell density, (e.g., less than 500 cells/mm$^2$), abnormal cell morphology (e.g., polymegathism or pleomorphism), or increased corneal thickness by pachymetry. Clinical symptoms may include decreased vision in the morning secondary to microcystic corneal epithelial edema. Microcystic corneal epithelial edema is frequently worse in the morning because of reduced evaporation and slight endothelial hypoxia from eyelid closure during sleep (Fig. 3). Patients with reduced endothelial cell density who develop microcystic corneal epithelial edema with normal intraocular pressure (IOP) are at high risk for corneal decompensation after intraocular surgery and may require penetrating keratoplasty. Patients who have IOL-corneal touch should be considered for IOL repositioning, removal, or exchange despite a clear central cornea provided that adequate endothelial cell reserve exists, as this may prevent the need for penetrating keratoplasty.\textsuperscript{106,107,160}

![Fig. 2](image1.png)

**Fig. 2.** *Left:* Migration of a Kelman-style II AC-IOL through a peripheral iridectomy. *Right:* This migration results in IOL tilt and corneal endothelial contact inferiorly. Intraocular lens exchange was performed to decrease the risk of corneal endothelial decompensation.

![Fig. 3](image2.png)

**Fig. 3.** Microcystic corneal epithelial edema is best seen under high magnification using indirect retroillumination.
CLEAR CORNEA WITH INTRAOCULAR LENS-INDUCED ACCELERATED ENDOTHELIAL CELL LOSS

The decision to exchange an IOL is controversial when a patient has normal vision and no IOL-corneal touch but shows evidence of IOL pseudophakodone-sis and/or chronic low-grade inflammation. Such patients may be at risk for corneal decompensation because of their IOL design, e.g., a closed-loop AC-IOL.

When accelerated endothelial cell loss (i.e., cell loss at a much more rapid rate than could be attributed to aging) can be documented by serial specular cell photomicrography in a patient with a closed-loop–style AC-IOL, removal or exchange of the IOL should be considered. Coli et al reported a series of 102 eyes with anterior chamber or iris-plane IOLs and signs of progressive endothelial damage and concluded that corneal decompensation may be prevented if the IOLs are removed before a critical degree of endothelial cell loss or dysfunction occurs. This requires serial evaluation with documentation of a reliable trend that considers the accuracy and reproducibility of each test. Evaluation should include assessment of changes in cell density, morphology, or pachymetry, as well as examination for factors that would suggest IOL-induced corneal compromise (IOL design, position, stability, proper size, evidence of erosion, or inflammatory precipitates on the IOL surface or corneal endothelium). If a patient has adequate endothelial reserve but demonstrates a clinical course that indicates likelihood of corneal decompensation, an early exchange procedure may forestall corneal transplantation. This spares the patient from potential problems, such as allograft rejection, corneal astigmatism, and prolonged corneal wound healing.

Considering IOL exchange in these patients requires an assessment of the risks and benefits of observation compared with intervention. A patient with a closed-loop–style AC-IOL who continues to demonstrate good vision and a stable cell count is certainly at less risk with continued observation than a patient with documented progression of endothelial cell loss with marginal cell reserve (e.g., 500 cell/mm² centrally [Fig. 4]). Intervention carries the risk of making the patient acutely worse, through surgically induced corneal decompensation, hemorrhage, iridodialysis, and failed or incomplete IOL removal. This is because the closed-loop AC-IOL designs may erode into the angle and ciliary body. Removal techniques must respect this erosion to preserve the integrity of the anterior segment and to avoid complications.

The goal of IOL exchange in the presence of a clear cornea is to avoid the need for a more involved procedure later, which will be more expensive, require a longer recovery, and be associated with more potential complications. However, patients with low endothelial cell counts (less than 500 cells/mm²) often demonstrate increased stromal thickness by pachymetry and are likely to develop acute corneal decompensation after surgical intervention. These patients are better managed with IOL exchange combined with penetrating keratoplasty after visually significant corneal decompensation occurs.

Patients with pre-existing progressive corneal disorders, such as Fuchs’ corneal endothelial dystrophy, would obviously continue to demonstrate progression associated with their underlying corneal disorder despite IOL removal.

It appears that all AC-IOLs accelerate endothelial cell loss to some degree. This may be an acceptable situation in older patients receiving modern versions of the Kelman-style implant (e.g., Multiflex style) that are properly sized and do not have positioning holes at the end of the haptic loop that can cause synechiae.

Retinal Indications

RETINAL DETACHMENT

Retinal detachment rarely requires explantation of the IOL. Lens removal should be considered to repair retinal detachment in patients 1) who have concomitant pathology creating other indications for IOL removal, 2) whose IOL obscures visualization of the detachment (which is more common in eyes with AC-IOLs and severe anterior proliferative vitreoretinopathy) or mechanically obstructs the surgery, 3) whose IOL may create specific intraoperative or postoperative management problems (e.g., condensation of a gas bubble on a silicone optic or
the creation of silicone oil “droplets” on the surface of a silicone optic, 17,65,76,91,144,235,253 or 4) who have significant capsular pathology (localized endophthalmitis, severe capsular contraction, or limited IOL support) that might contribute to IOL-related intraoperative or postoperative complications.

CYSTOID MACULAR EDEMA

Cystoid macular edema is a well-recognized complication of intraocular surgery with or without IOL implantation. 3,55,64,68,74,81,83,96–98,100,101,104,245 Potential mechanisms of pathogenesis include increased permeability of the perifoveolar capillaries, an ischemic tissue injury, a secondary response to intraocular inflammation, and direct traction on the macula after vitreous shifts. 8

Incidence and Mechanisms

Clinically, CME can be divided into three groups: 1) angiographic CME, in which vision is usually unaffected; 2) clinically significant CME, in which vision is decreased; and 3) chronic CME, defined as chronic or recurrent CME of more than 6 month’s duration. The presence of angiographic CME alone is of uncertain importance. In a study by Stark et al, 78% of patients with fluorescein-positive angiographic evidence of CME had 20/20 vision, and the rest had 20/25 vision. 245 Clinically significant CME is associated with transient decrease in vision in up to 8% of patients after uncomplicated cataract extraction and is associated with chronic decrease in 1–2% of patients. 49 A clinical study at the Wilmer Eye Institute found that clinically significant CME developed in a smaller percentage of cases (2% total incidence and 0.3% chronic) if the IOL implantation was uncomplicated. 245 Visual potential probably correlates best with thickness of edema rather than size or severity of angiographic leakage. 184

The peak incidence of CME after cataract surgery is approximately 4–16 weeks postoperatively, but the onset may be delayed months or even years after surgery. 49,175 Spontaneous resolution occurs in 75% of cases within 6 months. 40,79,92,95,104,105,171,221,284 Cystoid macular edema that causes permanent structural damage to the macula is an important cause of poor vision that persists despite IOL removal or exchange. 26,153

Chronic CME may be associated with photoreceptor atrophy, lamellar hole formation, and reactive retinal pigment epithelium (RPE) changes in the fovea. 40 Histopathologic examination supports the role of inflammation in the development of CME by documenting the presence of inflammation in the iris, ciliary body, vitreous, and retinal blood vessels. Breakdown of the blood-retinal barrier can be seen with iris angiography and vitreous fluorophotometry. 156,171 The degree and duration of breakdown of the blood-retinal barrier may correlate with the amount of intraoperative damage to the iris and the extent of the posterior synechiae. 71 For this reason some authors have suggested that IOLs placed in the capsular bag may be better tolerated than those placed in the ciliary sulcus. 73,172

Epidemiologic studies suggest that surgical techniques and IOL design play an important role in the development of CME. Jaffe et al found that only 3 patients in a series of 103 who underwent extracapsular cataract extraction (ECCE) with posterior chamber IOL implantation developed angiographic CME. 125 The lower incidence of CME after ECCE versus intracapsular cataract extraction (ICCE) is supported by studies by Binkhorst, 23 Kraff et al, 139 Taylor et al, 259 Stark et al, 245 and Jaffe et al. 124,125 Iris-supported IOLs are associated with a higher incidence of CME. In a retrospective study by Taylor et al, clinical CME developed in only 2% of 630 eyes that underwent ICCE alone, versus 9.9% of 850 eyes that underwent ICCE combined with iris-supported IOL implantation. 259 Rigid closed-loop AC-IOLs have also been associated with an increased incidence of CME. In a study by Maynor, CME developed in 1.9% of 207 patients who underwent ICCE and implantation of a Leiske-style IOL and in 5% of 302 patients undergoing ICCE and implantation of an Azar 91Z lens. 162 Choyce found a 3% incidence of CME in a series involving 1,000 Mark IX rigid AC-IOLs (Coburn, Clearwater, FL). 41

Maintaining the integrity of the posterior capsule and vitreous appears to reduce the risk of CME. 37 Niki et al reported CME in 35.7% of cases of extracapsular cataract extraction complicated by posterior capsular rupture requiring anterior vitrectomy and insertion of an AC-IOL. 183 This compares with a 1.5% incidence of CME in cases of uncomplicated extracapsular cataract extraction with insertion of a PC-IOL. Kraff et al randomly performed primary capsulotomies in 152 patients and left the capsule intact in 136 patients. 138 Cystoid macular edema was evident, by angiography, in 5.6% of those with an intact capsule, as opposed to 21.5% of those with capsulotomy. However, overall visual acuity results were virtually identical in the two groups. Angiographic findings in patients with intact capsules did not correlate with visual acuity.

Winslow and colleagues found angiographic evidence of CME in 48% of patients with vitreous prolapse after capsulotomy, whereas edema was demonstrable in only 10% of patients without vitreous loss and with intact capsules. 280 Jaffe found that if capsulotomy is delayed for a year or longer after cataract extraction, CME is less likely to occur. 126
Management

Cystoid macular edema per se is only a relative indication for IOL removal or exchange, and it often occurs in the presence of other ocular inflammatory conditions, such as pseudophakic corneal edema, IOL-induced uveitis, and localized or generalized endophthalmitis. Therefore, chronic CME is infrequently the sole indication for IOL removal or exchange.\textsuperscript{158,254,259} The true incidence of CME as an associated finding in patients undergoing IOL exchange is unknown because of coexisting conditions, such as pseudophakic corneal decompensation. Isolated studies report the overall incidence of CME before IOL removal or exchange to be 3.3–5.5%.\textsuperscript{46,135,153,191,227,254}

Although many authors report improvement in clinically observed CME and visual acuity after IOL removal or exchange, it is difficult to predict the response in an individual patient. Visual outcome may be limited by permanent structural changes of the fovea or from perpetuation of CME by mechanisms that are not directly attributed to the IOL. Smith reported significant improvement in visual acuity after IOL removal or exchange in some patients with chronic CME attributed to an IOL.\textsuperscript{259} In their study, 50% of the patients with CME achieved a significant improvement in vision after removal of the IOL. Lyle and Jin reported resolution of CME in 7 of 14 patients (50%) after IOL exchange; however, in their series of 101 eyes in 98 patients, the overall incidence of postoperative CME was higher than the preoperative incidence.\textsuperscript{153} In that study, the incidence of postoperative CME was significantly associated with the preoperative CME. Lyle and Jin also found that new CME was more likely to develop after penetrating keratoplasty combined with IOL exchange. They identified CME and glaucoma as the cause of a poor visual outcome in patients undergoing IOL exchange without penetrating keratoplasty. This is consistent with the findings of Busin et al, who identified glaucoma and CME as the most common cause of a final visual acuity worse than 20/60 after IOL exchange.\textsuperscript{26} In a retrospective analysis by Mamalis et al of 102 patients who underwent IOL removal or exchange, the postoperative vision improved in 39%, remained unchanged in 46%, and worsened in 15%.\textsuperscript{158} The most common cause for worsening of vision postoperatively was corneal decompensation, then glaucoma and CME, respectively.

Coli et al studied 102 patients with AC-IOL–induced corneal endothelial damage who underwent IOL exchange.\textsuperscript{48} This group contained 47 patients (46%) with concomitant CME before IOL exchange. Patients with low endothelial cell counts (less than 900 cell/mm\textsuperscript{2}) and either a rigid-loop– or closed-loop–style AC-IOL had a higher incidence (71%) of preoperative CME. Posterior chamber-IOLs were substituted for AC-IOLs in all cases, with 87 sutured to the iris, 3 sutured to the sclera, and 12 inserted into the ciliary sulcus. Among all patients with CME, visual acuity improved in 30 eyes (64%), remained unchanged in 6 eyes (13%), and worsened in 11 eyes (23%) after exchange for an iris-sutured PC-IOL and vitrectomy.

In another study, penetrating keratoplasty, AC-IOL removal, anterior vitrectomy, and insertion of an iris-sutured PC-IOL resulted in angiographic resolution of CME in 18 of 25 patients diagnosed with CME and pseudophakic corneal edema.\textsuperscript{255} Improvement of visual acuity to 20/40 or better was achieved in 16 patients, with visual recovery usually occurring within a year of surgery.

Stark et al reported three patients who underwent IOL exchange because of pain with CME.\textsuperscript{246} Exchange of an AC-IOL for a sutured PC-IOL in the absence of capsular support relieved the pain in all the patients, and the CME improved in two of them.\textsuperscript{246} Two additional patients who underwent IOL exchange because of CME alone also demonstrated improved vision and reduced CME.

Insertion of a three-piece–C-loop PC-IOL in the anterior chamber has been associated with inflammatory complications, including CME and epimacular membrane formation. Liu et al reported improvement in visual acuity and reduced CME in two patients who underwent exchange of a three-piece–C-loop PC-IOL that had been inserted into the anterior chamber.\textsuperscript{152}

Many of the data addressing CME and visual outcome after IOL exchange are retrospective, and there are little data from prospective controlled interventions. Schein et al compared IOL exchange techniques in a randomized prospective study of 176 consecutive patients undergoing penetrating keratoplasty with IOL exchange.\textsuperscript{227} They found that the cumulative risk of CME with iris-fixated PC-IOLs was significantly less than that with AC-IOLs or traditionally sutured PC-IOLs. However, the number of resolved cases and the number of newly developed cases of CME were not reported. Also, the cumulative risk of CME after penetrating keratoplasty and IOL removal alone was not determined.

Three retrospective uncontrolled studies have been reported, two comparing open-loop–Kelman-style AC-IOLs with iris-fixated lenses and one comparing open-loop–Kelman-style AC-IOLs, iris-fixated PC-IOLs, and transscleral fixated PC-IOLs.\textsuperscript{54,98,146} None of these studies reported any differences in visual outcome or complication rates after IOL exchange among the different techniques.

Harbour et al treated 24 consecutive patients with chronic pseudophakic CME with pars plana vitrectomy without IOL removal.\textsuperscript{94} Preoperatively, the pa-
patients had evidence of either vitreous adhesions to anterior segment structures (23 patients) or iris capture of the IOL (1 patient), and all had responded poorly to medical therapy. In all patients, pars plana vitrectomy with removal of vitreous adhesions improved the visual acuity, with a mean improvement of 4.6 Snellen lines.

The surgeon performing IOL removal or exchange for the treatment of visually significant CME should direct meticulous attention toward alleviating potential causes of chronic inflammation, such as vitreocorneal or iridocorneal adhesions, iris incarceration, IOL capture, IOL-induced iris chafing, or any vitreous traction (Figs. 5–8). This may reduce the sources of inflammation responsible for CME. Cystoid macular edema is a potential complication of any intraocular procedure, and not all pseudophakic patients with CME benefit from IOL removal or exchange. Patients with concomitant inflammation and clinical signs that suggest that the IOL is the cause of ongoing CME are the patients most likely to benefit from IOL removal or exchange. Surgical intervention should be reserved for cases of CME refractory to medical treatment. Cystoid macular edema may also be complicated by the development of an epimacular membrane or foveal hole formation.

**Inflammatory (Noninfectious) Indications**

**MICROHYPHEMA**

Microhyphema has been noted in approximately 2–5% of anterior chamber and iris-fixated lenses after cataract extraction. Microhyphema associated with increased IOP and uveitis in the presence of an anterior segment lens (uveitis-glaucoma-hyphema syndrome) is discussed in the next section.
Before the development of modern surgical techniques, recurrent hyphema was more common and was associated with vascular ingrowth of the incision (Fig. 9). Microhyphema also has been reported after PC-IOL implantation. The incidence of late bleeding in the anterior segment after PC-IOL implantation has been reported to be 0.4%. The patients may have a history of floaters or intermittent visual decrease lasting for several hours. Some of these patients are on chronic coumadin therapy. Bleeding usually results from IOL trauma to the iris (Fig. 10) or ciliary body. Iris fluorescein angiography may demonstrate hyperfluorescent leakage at the bleeding site.

On examination, the microhyphema may be observed inferiorly in the anterior chamber by slit-lamp biomicroscopy or may require gonioscopy to be visualized. Suspended red blood cells may be observed in the aqueous. The IOP associated with a microhyphema may be elevated. Magargal et al reported that an increased IOP occurred after a microhyphema in two of five cases.

Treatment of microhyphema involves preventing recurrent hemorrhage by chronic topical cycloplegics, argon laser photoocoagulation, or Nd:YAG laser photoocoagulation to the bleeding site. Aqueous suppressants (e.g., -adrenergic blockers, carbonic anhydrase inhibitors, apraclonidine) may be prescribed to treat an elevated IOP. Removal of the IOL has rarely been required to treat recurrent uncontrolled vitreous hemorrhage associated with chronically reduced vision.

**UVEITIS AND GLAUCOMA**

Postimplantation uveitis was formerly associated with the toxic lens syndrome, which was related to IOL polishing and sterilization and the uveitis-glaucoma-hyphema syndrome, which resulted from a poorly designed and finished IOL that caused mechanical injury to the iris and breakdown of the blood-aqueous barrier. Improvements in IOL design and finish have made both of these syndromes largely extinct. Intraocular lens-induced inflammation may still occur in association with excessive uveal contact resulting from IOL chafing or erosion, IOL malposition, or improper IOL sizing (particularly with AC-IOLs).

Ocular inflammation resulting from IOL touch to the iris or the ciliary body may increase the IOP by several mechanisms. Inflammatory cells or debris may occlude the trabecular meshwork. Ocular inflammation resulting from IOL touch to the iris or the ciliary body may increase the IOP by several mechanisms. Inflammatory cells or debris may occlude the trabecular meshwork. Uveitis-induced adherence of the iris to the IOL optic may cause pupillary block. Glaucoma may be caused by the topical steroids used to treat the uveitis. In addition to inflammatory changes, outflow may be reduced by direct mechanical injury to the trabecular meshwork or peripheral anterior synchiae. The incidence of secondary glaucoma resulting from uveitis after lens implantation is 2.3% with an AC-IOL, 1.7% with an iris-fixated lens, and 0.8% with a PC-IOL.

Evidence of uveitis on clinical examination includes anterior chamber cell and flare, keratitic precipitates, and pigment debris in the inferior trabecular meshwork, as noted by gonioscopy. Gonioscopic findings in the uveitic eye may be compared with those in the uninvolved eye to determine increased angle pigment.

If the increased IOP is secondary to inflammation, it should be controlled when the inflammation is controlled by topical steroids. The minimum dose of topical steroids required to control the inflammation should be used, because a steroid-induced IOP increase may occur after 3–4 weeks in sensitive individuals.

Aqueous suppressants may reduce the IOP. Treatment methods that depend on altering outflow facility, such as topical miotics and argon laser trabecu-
plasty, may also be tried, but might be less effective in eyes with uveitis. When IOP cannot be controlled by these methods, filtration surgery may be required. Occasionally, persistent uveitis that is causing secondary complications may necessitate removal of the IOL.

**INTRAOCULAR LENS-INDUCED PAIN**

Intraocular lens-induced pain may be caused by local irritation or from a more generalized inflammatory response. It is not uncommon for patients with AC-IOLs to experience low-grade discomfort and tenderness with local pressure. The tenderness is often noted in the region of IOL erosion into the ciliary body and is more apparent with IOLs that have a diameter that is too large.

Patients with PC-IOLs rarely have such discomfort, although Legler et al reported a patient with chronic, vague complaints of discomfort that improved after PC-IOL repositioning. They hypothesized that the PC-IOL loop was causing local irritation.

An analysis of 6,521 explanted IOLs evaluated at the Center for IOL Research (Charleston, SC) revealed that pain was clinically present in 32.9% of patients who underwent AC-IOL removal compared with 17.7% of patients who underwent PC-IOL removal. Pain is subjective and usually not identified separately from inflammatory complications in series that address indications for IOL exchange.

**Infectious Indications**

**GENERALIZED ENDOPHTHALMITIS**

**Incidence**

Infectious endophthalmitis is an uncommon complication after modern cataract surgery with IOL insertion. An IOL is essentially a foreign body and, like any prosthetic device, it requires additional surgical manipulation for implantation. This could potentially create a higher incidence of infectious endophthalmitis in the pseudophakic eye compared with the aphakic eye; however, most studies show that this is not the case.

Kattan et al reported that the risk of endophthalmitis after a standard ECCE with or without primary IOL implantation is 0.072%, but that it increases to 0.3% with a secondary IOL. Javitt et al reported an incidence of 0.12% in a national multicenter study. Gaping wounds are a major risk factor for infection and are present in 65% of all cases of postsurgical endophthalmitis. Stonecipher et al reported three cases after sutureless cataract surgery. Williams and Gills reported 27,181 consecutive cases of sutureless cataract surgery with an incidence of infection of 0.015%.

Driebe et al reported one of the largest series of pseudophakic endophthalmitis. A bacterial source was isolated in 57 of 83 cases. Only one case required IOL removal to clear the infection. In this series, the causative organisms differed little from isolates responsible for infections in patients without IOL implantation.

**Risk Factors**

Several highly publicized clusters of endophthalmitis have been associated with contamination of solutions used for sterilizing and irrigating. Clayman et al reported the potential for bacterial contamination of the phacoemulsification tip. Filtration of irrigating solutions along with the perioperative use of antibiotics may reduce postoperative endophthalmitis.

The potential for contaminating the IOL intraoperatively was demonstrated by Vafidis et al, who reported that IOLs placed on the periocular surface before being implanted were contaminated 26% of the time. Most surgeons acknowledge the importance of avoiding IOL contact with the eyelids and lashes to reduce the risk of infectious complications. The importance of meticulous, sterile surgical technique and the appropriate preoperative treatment of pre-existing infections, such as blepharitis or conjunctivitis, is generally accepted.

Menikoff et al reported that multipiece IOLs with polypropylene loops represent an independent risk factor for the development of postoperative microbial endophthalmitis. Additional work by Raskin et al suggested a mechanism of greater bacterial adherence to polypropylene compared with polymethylmethacrylate.

**Clinical Presentation and Signs**

Acute onset, generalized endophthalmitis characteristically develops within a few days after intraocu-
lar surgery. Patients typically experience the rapid development of severe ocular pain associated with reduced vision, redness, tearing, and photophobia. Occasionally, they may present without pain.56

Clinical signs (Fig. 11) include conjunctival inflammation with chemosis; episcleral inflammation; eyelid edema with blepharospasm and secondary ptosis; corneal stromal edema secondary to endothelial dysfunction; anterior chamber inflammation, including hypopyon, cell, flare, keratic precipitates, and IOL precipitates; secondary glaucoma; uveal inflammation, including engorged iris vessels, iris nodules, posterior synechiae, and peripheral anterior synechiae; and inflammation of the posterior segment, including vitreous inflammation, inflammatory membranes, macular edema, and retinal necrosis.4,72,170

Patients who develop postoperative endophthalmitis with virulent organisms tend to have a generalized inflammatory process that is severe in nature and occurs rapidly after surgery. Occasionally, a virulent organism may create severe endophthalmitis that is delayed in onset if inoculation occurs after surgery owing to a suture tract or wound abnormality (Fig. 12).192

Management

Successful treatment of generalized endophthalmitis requires prompt recognition and initiation of antimicrobial therapy. It usually does not require removal of the IOL, unlike infections in other parts of the body that may require removal of any associated prosthetic device to adequately clear the infection. Removal of the IOL may be required in patients who worsen while on maximum medical therapy. This is more likely to occur in patients with forms of endophthalmitis involving mycotic infections50 or localized anaerobic infections sequestered within the capsular bag.

LOCALIZED ENDOPTHALMITIS

Virtually all microbial organisms have the potential to become intraocular pathogens. Less virulent organisms may result in infectious endophthalmitis that has a delayed onset. Indolent microbial organisms may sequester within the capsular sac, creating a localized, smoldering infection.34,61,164,165,190,197,219,257,260,261 Apple et al coined the term localized endophthalmitis to describe this condition, indicating that it should be an important consideration in the differential diagnosis of otherwise unexplainable inflammation after cataract surgery.8,10,197

Clinical Features

Patients with localized endophthalmitis tend to develop inflammation that is characteristically delayed in onset, chronic, and often low grade; however, severity may be variable. Inflammation typically

Fig. 12. Delayed onset, generalized endophthalmitis associated with an infected filtering bleb.

Fig. 13. A: Intraoperative photo of a localized plaque within the capsular bag surrounding the polypropylene IOL loop in a patient undergoing partial capsulectomy with removal of the sequestered infection, IOL exchange, and endocapsular antibiotic inject. B: Histology (Brown and Brenn stain) showing Propionibacterium acnes densely sequestered within the capsular bag. There is an absence of inflammatory cells.
improves with corticosteroids, and this response may further delay the diagnosis. The patient’s symptoms of pain, reduced vision, redness, tearing, and photophobia are often less intense than in patients with acute generalized endophthalmitis. Signs of inflammation may also be less severe than those of acute generalized endophthalmitis. An endocapsular plaque (Figs. 13 and 14) may be present, representing sequestered organisms. Stefansson et al reported an endocapsular hypopyon as a clinical sign of localized bacterial endophthalmitis. Meisler et al were the first to recognize the importance of Propionibacterium acnes as a cause of delayed-onset endophthalmitis after cataract extraction. They reported six patients with chronic P. acnes endophthalmitis who previously would have been classified as having toxic-lens syndrome. The initial cataract surgery was routine and without complication, and each patient achieved good visual acuity before the onset of inflammation. On average, inflammation developed 4 months after surgery and improved with topical corticosteroids. The etiologic diagnosis was delayed an average of 10 additional months. These cases developed despite the use of perioperative antibiotic prophylaxis and were not specific to an individual surgeon or IOL. Although not specific to an individual IOL, reported cases of localized endophthalmitis have occurred almost exclusively in patients with IOLs with polypropylene loops.

Piest et al studied the development of localized endophthalmitis in patients clinically diagnosed with toxic-lens syndrome. In each case, the differential diagnosis included biomaterial-related inflammation and phacoanaphylaxis. Each patient underwent removal of the IOL and lens capsule with particular care to ensure that no lens material remained in the eye. Specimens submitted for histopathologic evaluation revealed Gram-positive organisms, suggestive of P. acnes, that were sequestered in the residual lens capsule. Piest et al reported an additional case in a patient who did not have an IOL, verifying that only the presence of the capsular sac is necessary for this condition to develop. The IOL may play a role, however, in inoculating the bacterial organism. Also unknown is the role of residual lens cortical epithelium in the development of a localized infection.

Clinically, patients with localized endophthalmitis correlate well with the experimental studies in nonhuman primate eyes of Beyer et al, who reported the protective barrier effect of the posterior lens capsule in exogenous bacterial endophthalmitis. Also unknown is the role of residual lens cortical epithelium in the development of a localized infection.

Localized endophthalmitis may create the clinical appearance of posterior capsular opacification (PCO). There are at least four cases reported of inflammation precipitated in otherwise quiet eyes by Nd:YAG laser capsulotomy. Tetz et al reported a case of P. acnes endophthalmitis precipitated by Nd:YAG laser posterior capsulotomy for PCO performed 10 months after the primary surgery. There was no evidence of inflammation before the laser procedure, and their case was eventually treated with removal of the IOL and capsule. Visual acuity returned to 20/50, limited by CME. Meisler et al reported a case of P. acnes endophthalmitis developing within a month after Nd:YAG laser capsulotomy in a patient with PCO without intraocular inflammation. The laser procedure was performed 3 months after the primary surgery, and their patient required removal of the IOL and capsule despite aggressive antimicrobial treatment. Visual acuity returned to 20/60, limited by CME. Carlson and Koch described a patient who developed P. acnes endophthalmitis after Nd:YAG laser posterior capsulotomy performed 4 months after cataract surgery for PCO in an eye that was otherwise without evidence of inflammation. Their case demonstrated the potential for successful treatment without removal of the IOL or capsule. Their patient returned to 20/25 visual acuity despite mild CME and an epimacular membrane. Carlson et al reported another patient who developed Staphylococcus aureus endophthalmitis after Nd:YAG laser capsulotomy for PCO that developed in an otherwise quiet eye. The capsulotomy was performed 2 years 8 months after the original cataract surgery. The isolation of S. aureus was surprising because this species is generally more pathogenic than P. acnes. The organism apparently remained viable within the cap-
sule for an unusually long time without causing intraocular inflammation. This patient responded to antimicrobial and anti-inflammatory treatment without IOL removal and returned to a visual acuity of 20/25. In addition to the above four cases, four additional cases (three *P. acnes*, one *S. epidermidis*) have been reported with inflammation prior to capsulotomy that progressed and were possibly aggravated by the laser procedure.1,164,181,197

Piest et al reported a patient who underwent IOL exchange for presumed toxic lens syndrome.197 The lens capsular sac was retained and the inflammation persisted, although it improved with intense corticosteroid treatment. The patient eventually underwent Nd:YAG laser capsulotomy for PCO and developed recurrent hypopyon. The patient improved after complete removal of the capsular sac and all cortical remnants combined with anterior vitrectomy. Abrahams reported worsening of *P. acnes* endophthalmitis after Nd:YAG laser capsulotomy for PCO in a patient who appeared to have had a localized infectious plaque on the corneal endothelium without clear documentation of PCO resulting from *P. acnes*.1 The patient underwent sector iridectomy, removal of the retained inflammatory material, and removal of the IOL and the capsular bag along with anterior- and midvitrectomy. Histopathologic and cytologic studies of the capsular bag were unable to identify evidence of a local infection.

**Laboratory Diagnosis**

Aqueous and vitreous cultures may be negative when the infection is localized within the capsular bag. If a localized process suspicious for an infection is clearly visible within the capsular bag, laboratory evaluation should include material from the sequestrum. The material should undergo Gram stain and direct inoculation onto bacterial and fungal media. To improve sensitivity and specificity, media for anaerobic bacteria should include at least one broth (e.g., Thiol, Thioglycolate, or chopped meat enriched broth) and one agar plate (e.g., prereduced Brucella blood agar, supplemented Columbia base agar, or CDC anaerobic blood agar). The laboratory should be specifically instructed to incubate the specimen for at least 14 days before discarding it as negative and to consider organisms such as *P. acnes* as pathogens and not laboratory contaminants. Organisms obtained in low number or under the influence of previous antibiotic therapy may take longer to grow than the standard 7-day incubation period recommended by the American Society for Microbiology. Isolating the organisms on multiple media decreases the likelihood that a contaminant or false-positive culture has occurred.

**Indications for Intraocular Lens Removal**

Based on the literature regarding the medical and surgical management of patients with localized endophthalmitis, it is difficult to predict which patients will respond to treatment without removal of the IOL and capsular bag. Problems in interpreting the literature include the small number of well-documented cases and the variability of case definition and spectrum of case severity and treatments. Selection bias favors reporting cases that undergo IOL removal, as many of the early series were not diagnosed until the specimen was evaluated by histopathology. Isolated cases that are successfully managed without IOL removal are probably less likely to be reported than cases that involve IOL removal or exchange.

Removal of the IOL and capsular bag should be considered in patients who do not respond adequately to limited capsulectomy, removal of the sequestrum, and injection of antibiotic within the capsular bag. We recommend intraoperative injection of 1 mg of vancomycin in patients suspected with *P. acnes*, and we may repeat this postoperatively at the slit-lamp. The techniques for capsular biopsy and IOL-capsular bag removal are described elsewhere.35

**Optical Indications**

INCORRECT INTRAOCULAR LENS POWER

Incorrect IOL power selection results in an unsatisfactory refractive error. Extreme postoperative refractive surprises have been reduced by improved understanding of the limitations of the older, empirically derived IOL calculation formulas and improved accuracy in the preoperative measurement of axial length and corneal curvature.

Potential sources of error in determining the power of an IOL include 1) incorrect axial length measurement; 2) incorrect corneal power determination, particularly in patients with previous refractive surgery; 3) error associated with incorrect use or selection of IOL calculation formulas, including the reduced accuracy of empirically derived formulas with unusually long or short eyes; 4) alteration of the effective IOL power by placing the optic in a position different than expected by the preoperative calculation, such as sulcus placement instead of capsular bag placement; 5) insertion of the wrong IOL during surgery; 6) incorrect power labeling or incorrect A-constant or “surgeon factor” of the IOL; or 7) multiple sources of error that can produce an unanticipated refractive result. The majority of surprise errors are explained by remeasuring the preoperative parameters.

Sources of error can be reduced by 1) measuring both eyes preoperatively for comparison; 2) repeating any measurement that is statistically unusual by
itself or in comparison with the other eye; 3) comparatively cross-checking keratometry with computerized corneal analysis; 4) educating the technical staff to improve consistency, minimize distraction during testing, and alert the surgeon to any difficulty encountered during measurement; 5) determining the accuracy postoperatively in preparation for IOL selection in the second eye and looking for consistent errors that might require modification of the A-constant or “surgeon factor;” and 6) anticipating tolerance of error and adjusting surgery accordingly—e.g.: targeting for $0.5$ sphere in a patient who desires emmetropia, as an error in the hyperopic direction is tolerated less well in the nonaccommodating pseudophakic eye than an error in the myopic direction.

MALPOSITION OF THE INTRAOCULAR LENS

Malposition of the IOL may be the result of the original surgical placement of the lens, or it may develop postoperatively because of external or internal forces. External forces include trauma or eye rubbing (Fig. 15). Internal forces include a size disparity between the IOL and the site of fixation, scarring, peripheral anterior synechiae, and capsular contraction. Decentration of tilting of an AC-IOL may produce optical problems from aberration or edge glare.

Fig. 15. A: Open-loop, Kelman-style AC-IOL placed horizontally to avoid peripheral iridectomy superiorly. B: The same eye, 2 $\frac{1}{2}$ years later with IOL rotation and peaking of the pupil. This process may develop in association with eye rubbing and/or a size disparity between the IOL and the dimensions of the anterior chamber angle.

Fig. 16. Sunrise syndrome—Asymmetric IOL loop fixation in combination with postoperative capsular contraction results in unopposed forces on the inferior IOL loop with resulting migration of the IOL superiorly.

Fig. 17. Pea pod effect—Postoperative capsular contraction with one or more tears in the anterior capsule produces forces capable of extruding one or both loops out of the capsule, resulting in IOL decentration.
Optical complications caused by wrong IOL power or malposition comprise the most common indication for removal or exchange of a modern PC-IOL. Although malposition of a PC-IOL may reduce the quality of vision, it is less likely than malposition of an AC-IOL to cause mechanical injury or inflammatory sequelae.

The pathogenesis of malposition may be related to a variety of locations of loop fixation, to the forces of capsular contraction, or to a combined mechanism. Before the development of capsulorhexis, it was common for the surgeon to place the inferior IOL loop within the capsule while releasing the superior loop into the ciliary sulcus, thus producing asymmetric loop fixation. Because significant decentration was anticipated, PC-IOLs had large optics (7 mm) and long total lengths (13–14 mm). Subsequent healing from capsular fusion and contraction potentially caused the inferior loop to exert forces on the optic unopposed by forces from the superior loop within the sulcus. Migration of the optic superiority produces the “sunrise syndrome,” which may cause symptomatic edge glare or aberration (Fig. 16).

A tear in the anterior capsule may allow one or both IOL loops to migrate out of the capsular bag under the forces of capsular contraction, producing “pea podding” (Fig. 17).

Capsulorhexis is a major surgical advance that contributes to long-term IOL centration and reduces the likelihood of needing IOL removal or exchange (Table 2). Despite an intact capsulorhexis, decentration may still occur, as seen in the capsular

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**TABLE 2**

*Advantages of Capsulorhexis*

| 1. Assures capsular bag fixation to improve short- and long-term IOL centration. |
| 2. Allows use of newer IOLs with smaller optic and shorter overall length, as well as use of foldable IOL designs that tend to decenter without capsulorhexis. |
| 3. Improves the safety of surgical techniques involving nuclear cracking or chopping. |
| 4. Improves the efficacy of surgical techniques such as cortical-cleaving hydrodissection and facilitates cortical removal by expanding the capsular bag and decreasing aspiration of the capsular flaps. |
| 5. Provides fixation that avoids uveal contact. |
| 6. Improves IOL power calculations by increasing predictability of IOL location. |
| 7. Produces less zonular stress than is created during puncture techniques. |
| 8. Provides better support for sulcus-fixated IOLs in cases developing significant intraoperative disruption of the posterior capsule. |
| 9. Potentially reduces posterior capsular opacification by facilitating more complete cortical removal in addition to enhanced contact inhibition from apposition between the optic and the capsule. |
| 10. Provides an additional barrier against scleral collapse during penetrating keratoplasty when performing a triple procedure. |

IOL = intraocular lens.

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Fig. 18. Capsular contraction syndrome producing appositional fibrosis and contraction of the anterior capsular opening, evidence of capsular tension with stria formation and mild decentration of the IOL from capsular forces.

Fig. 19. Capsular contraction with dense fibrosis of the anterior capsule and complete closure of the anterior capsular opening.
contraction syndrome. This has been described by several authors, including Shepherd, Hansen, Davison, and Masket. The emphasis of the original reports was the development of closure of the anterior capsular opening. Many of these cases develop capsular striae, a sign demonstrating that capsular forces can produce IOL decentration (Figs. 18 and 19). This complication can be prevented by a large capsulorhexis.

In a separate but related mechanism, an eccentric capsulorhexis may allow capsular fusion peripheral to the optic with progressive adherence. B and C: This fusion produces IOL decentration from the edge of the capsulorhexis.

The silicone-plate–design IOL is particularly susceptible to the forces of capsular contraction and may decenter, rotate, tilt, or buckle because of several properties specific to the plate design (Fig. 21). With forces from capsular compression, the plate lens is unable to compensate as well as a loop-design lens, which essentially adjusts for disparity between the size of the IOL and capsular bag. Plate-design lenses have a smaller arc of contact with the capsular fornix, reducing anchoring forces that normally reduce the potential for rotation and decentration. Sil-
icone does not adhere well to the capsule, and compression produces folding or buckling out of the plane of the IOL rather than compression within the plane, as with a loop-design lens.

An additional concern is the potential for dislocation into the vitreous after Nd:YAG laser capsulotomy (Fig. 22). Carlson et al reported 10 cases of silicone-plate IOL dislocation and attempted to identify specific risk factors for this complication. Eight of 10 cases occurred despite waiting more than 3 months after cataract removal before performing the laser surgery. This complication was not specific to an individual surgeon nor was there anything unusual about the IOL powers or axial lengths. Evidence of capsular tension was present in at least 6 of 10 patients who developed a capsular split during the first few laser shots. In all cases, there was no immediate dislocation of the IOL, but rather spontaneous dislocation occurring several days to weeks after the procedure. Most patients retained excellent vision, although 6 underwent IOL removal and 1 developed a retinal detachment resulting in vision at the level of counting fingers. Recent plate lens modifications, including enlarged holes within the plate or the presence of a loop extending from the plate, may provide additional anchoring to resist the forces associated with capsular contraction.

Newer IOL designs, improved surgical techniques, and a greater understanding of IOL fixation and postoperative capsular contraction should reduce the incidence of IOL malposition. Presently, management options for IOL malposition include 1) observation, 2) pharmacologic miosis, 3) repositioning (Fig. 23), 4) repositioning combined with McCannel suture fixation (Fig. 24), 5) removal (Fig. 25), or 6) exchange. The decision to retain and reposition the IOL with or without a McCannel suture requires that the IOL be of the appropriate power and design, and be capable of adequate fixation with acceptable risk and with long-term safety and stability. In the absence of these characteristics, IOL removal or exchange should be considered. Selection of treatment is based on the patient’s symptoms, visual needs and expectations, and an assessment of which option is likely to provide the best long-term benefit with the least risk.

**Patient Counseling**

Patients faced with removal or exchange of an IOL will have various reactions depending on their level of understanding, expectation, and emotional stability. Emotions may range from relief that their problem may be solved to anger toward their original surgeon. In a discussion of IOL removal or exchange, the content of the message should offer...
adequate information for the patient to make an informed choice regarding additional surgery. The delivery should offer a cushion of hope around the delivery of “bad news.” The emotional environment should be maximally supportive. Helpful techniques include in-person rather than over-the-phone discussion and avoidance of interruptions or a rushed appearance during the consultation. The presence of close family members and any technical staff that the patient may be close to can provide support to the patient.

Verbal and nonverbal feedback during counseling will help assess how the patient is comprehending and receiving the news. The staff may alert the ophthalmologist to patients’ comments about recent bad news regarding health or family stress, indicating that it may not be the most appropriate time to discuss IOL removal. Family support during the patient counseling may be particularly helpful for patients who may have trouble remembering everything that was discussed.

Most patients, when confronted with the possibility of IOL removal or exchange, are already aware of problems surrounding their previous surgery and exhibit relief and optimism when additional treatment is offered to improve their present situation or minimize future complications.

**Conclusion**

There are few decisions in ophthalmic surgery that are as complex and problematic as the decision to remove or exchange an IOL. While some decisions appear clear-cut, e.g., removing a closed-loop AC-IOL during penetrating keratoplasty, other cases are less certain, e.g., when the patient has excellent vision that requires chronic topical corticosteroids to control inflammatory complications or when a clear cornea has IOL-induced accelerated corneal endothelial cell loss.

Issues that help the surgeon determine whether or not the risk-to-benefit ratio favors IOL exchange over continued observation include the following: 1) severity, duration, and chronology of the problem; 2) response to nonsurgical treatments; 3) natural history of a specific IOL (e.g., closed-loop design AC-IOL); 4) likelihood that surgical removal would provide substantial relief or benefit; 5) ease of surgical removal and potential for aggravating or creating additional complications; 6) status of the other eye; 7) patient and family expectations and visual needs; and 8) life expectancy and overall health of the patient. The surgeon must balance these issues and determine the potential for visual rehabilitation and the likelihood that surgical intervention will improve the patient’s quality of life and ability to function. The surgeon should seek consultation if the prob-

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**Fig. 24.** A: Visually significant PC-IOL dislocation resulting from inadequate capsular support. B: Successfully managed with a McCannel suture supporting the IOL loop to the peripheral iris. A single suture is usually adequate in patients that have residual capsule supporting at least one loop. Absence of capsular support may require an additional suture to avoid a “windshield wiper” effect.

**Fig. 25.** Intraocular lens removal is the best option in a patient with an anterior chamber IOL and recurrent granulomatous uveitis unresponsive to medical management.
REMOVAL OR EXCHANGE OF INTRAOCULAR LENSES

It is incompletely defined or if the surgeon lacks experience with the more technically challenging IOL exchange procedures. The patient must fully understand the treatment options, including the potential risks associated with both surgical and nonsurgical treatments. Preoperative counseling allows the patient to develop reasonable expectations and make an informed choice regarding care.

The frequency and spectrum of IOL-related complications is changing as a result of improved surgical techniques, instrumentation, and improved quality of IOLs. Patients continue to develop higher expectations and are less tolerant of surgical failure. Patients are also living longer and the trend has clearly developed toward the use of IOLs in younger patients. While IOL-related problems are less frequent and less severe overall, they may become more apparent in younger patients with longer follow up.

Methods of Literature Search

Search terms included various combinations of the terms listed in the “Key words” section of this article. A Medline search was performed from 1985 to present through the National Library of Medicine. Articles obtained from the reference list of other articles were reviewed and included when considered appropriate. Criteria for inclusion included the present clinical value or the original importance of the article to a particular subject.

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