OPTOMETRIC CLINICAL PRACTICE GUIDELINE

Care of the Adult Patient with

Cataract

American Optometric Association

OPTOMETRY: THE PRIMARY EYE CARE PROFESSION

Doctors of optometry are independent primary health care providers who examine, diagnose, treat, and manage diseases and disorders of the visual system, the eye, and associated structures as well as diagnose related systemic conditions.

Optometrists provide more than two-thirds of the primary eye care services in the United States. They are more widely distributed geographically than other eye care providers and are readily accessible for the delivery of eye and vision care services. There are approximately 32,000 full-time equivalent doctors of optometry currently in practice in the United States. Optometrists practice in more than 7,000 communities across the United States, serving as the sole primary eye care provider in more than 4,300 communities.

The mission of the profession of optometry is to fulfill the vision and eye care needs of the public through clinical care, research, and education, all of which enhance the quality of life.

Healthy People 2010 Vision

American Optometric Association

OPTOMETRIC CLINICAL PRACTICE GUIDELINE
CARE OF THE ADULT PATIENT WITH CATARACT

Reference Guide for Clinicians

Prepared by the American Optometric Association Consensus Panel on Care of the Adult Patient with Cataract:

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NOTE: Clinicians should not rely on the Clinical Guideline alone for patient care and management. Refer to the listed references and other sources for a more detailed analysis and discussion of research and patient care information. The information in the Guideline is current as of the date of publication. It will be reviewed periodically and revised as needed.

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INTRODUCTION

Optometrists, through their clinical education, training, experience, and broad geographic distribution, have the means to provide effective primary eye and vision care for a significant portion of the American public and are often the first health care practitioners to examine and diagnose patients with cataracts.

This Optometric Clinical Practice Guideline for the Care of the Adult Patient with Cataract describes appropriate examination and treatment procedures to reduce the risk of visual disability from cataract. It contains recommendations for timely diagnosis, treatment, and, when necessary, referral for consultation with or treatment by another health care provider. This Guideline will assist optometrists in achieving the following goals:

- Identify patients at risk of developing cataracts
- Accurately diagnose cataracts
- Improve the quality of care rendered to patients with cataracts
- Effectively manage patients with cataracts
- Identify and manage postoperative complications
- Inform and educate patients and other health care practitioners about the visual complications and functional disability from cataracts and the availability of treatment.

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I. STATEMENT OF THE PROBLEM

Cataract is the major cause of blindness in the world and the most prevalent ocular disease.[1] In the United States, cataracts are the most frequently cited self-reported cause of visual impairment and the third leading cause of preventable blindness.[2,3] Visual disability from cataracts accounts for more than 8 million physician office visits per year.[4]

When the disability from cataract affects or alters an individual's activities of daily living, surgical lens removal with intraocular lens implantation is generally the preferred means of treating the functional limitations. In the United States, more than 1.35 million cataract surgical procedures were paid for by Medicare in both 1990 and 1991, making it the most common surgery for Americans over the age of 65.[5-7] Between 1987 and 1988, 97 percent of cataract surgery patients received intraocular lens implants,[8] and by 1991, the annual cost of cataract surgery and associated care in the United States was approximately $3.4 billion.[9] Cataract surgery compares favorably to other health care interventions (e.g., hip replacement or aortic valve replacement) in terms of approximate cost per quality-adjusted life year (defined as the financial cost of surgery and aftercare balanced against the improved quality of life).[10]

The initial diagnosis of cataract may be made by any of a number of providers, such as a primary care physician, optometrist, or ophthalmologist. The patient's decision to proceed with cataract surgery to decrease disability involves consultation with an optometrist and/or ophthalmologist. Often the patient has a long-term relationship with the optometrist who is the patient's primary eye care provider before a cataract develops. The optometrist is often the first to detect, diagnose, and counsel the patient concerning the presence of cataracts and other eye diseases. The optometrist serves not only as counselor but also as an advocate for quality surgery and postsurgical care. If surgical intervention is undertaken, the optometrist is likely to be involved in providing postoperative and continuing care for cataract patients.[11,12]

Optometrists and ophthalmologists may work together as a team to provide complete preoperative, intraoperative, and postoperative care to meet the patient's needs. This cooperative care provided by doctors of optometry and ophthalmic surgeons for patients with eye disease or requiring eye surgery has come to be known as "comanagement."[13] It is commonly used in the treatment of patients with cataract.

A. Description and Classification of Cataract

A cataract is any opacity of the lens, whether it is a small local opacity or a diffuse general loss of transparency. To be clinically significant, however, the cataract must cause a significant reduction in visual acuity or a functional impairment.[14] For purposes of this Guideline, the definition of a cataract is an opacification of the lens that leads to measurably decreased visual acuity and/or some functional disability as perceived by the patient.

Cataracts may occur as a result of aging or secondary to hereditary factors, trauma, inflammation, metabolic or nutritional disorders, or radiation.[15,16] Age-related cataracts are the most common. The three common types of cataract are nuclear, cortical, and posterior subcapsular (See Appendix Figure 3 for the ICD-9-CM classification of cataracts). A cataract-free lens is one in which the nucleus, cortex, and subcapsular areas are free of opacities; the subcapsular and cortical zones are free of dots, flecks, vacuoles, and water clefts; and the nucleus is transparent, although the embryonal nucleus may be visible.

Cataracts may be graded by visual inspection and assignment of numerical values to indicate severity. Alternative grading systems advocated for use in epidemiological studies of cataract are the Oxford Clinical Cataract Classification and Grading System,[17] the Johns Hopkins system,[18] and the Lens Opacity Classification System (LOCS, LOCS II, and LOCS III).[19-22] Photographs of slit lamp cross-sections of the lens are used as references for grading nuclear opalescence and nuclear color, and photographs of the lens seen by retroillumination are used as references for grading cortical and posterior subcapsular cataract.

Most systems use a sequence of four photographs for each of the cataract characteristics to be evaluated. The recently introduced LOCS III system uses six photographic references for nuclear color and nuclear opalescence and a series of five photographic references for cortical and posterior subcapsular opacities.[22] In these systems, a numerical grade of severity is assigned to each reference photograph, and to interpolate the appearance of cataracts that fall between the reference photographs clinicians can use decimals to grade the cataracts in finer incremental steps.[23]

In most clinical settings, reference photographs are not available. Therefore, a less-sensitive four-point grading system modified from LOCS II[21] is commonly used. Despite its limitations, this simple 1, 2, 3, 4 grading scale can be used to record the extent of nuclear, cortical, and posterior subcapsular lenticular opacity changes from one visit to the next. A practical guide for this clinical form of cataract grading is shown in Table 1:

- Nuclear sclerosis (NS) may be graded by evaluating the average color and opalescence of the nucleus as a continuum from grade 1 (mild or early) to grade 4+ (severe advanced milky or brunescent NS).
- Cortical cataract (CC) and subcapsular opacities should be visualized as "aggregate" and quantified on the basis of the percentage of intrapupillary space obscured.
- Posterior subcapsular cataract (PSC) is graded on the basis of percentage of the area of the posterior capsule obscured. A PSC in the line of sight may be much more debilitating and the description of grading should reflect this (e.g., grade 2+ PSC in line of sight).

Table 1

Grading the Three Common Types of Cataracts*

Cataract Type	Grade 1	Grade 2	Grade 3	Grade 4
Nuclear Yellowing and sclerosis of the lens nucleus	Mild	Moderate	Pronounced	Severe
Cortical Measured as aggregate percentage of the intrapupillary space occupied by the opacity	Obscures 10% of intra-pupillary space	Obscures 10%-50% of intra-pupillary space	Obscures 50%-90% of intra-pupillary space	Obscures more than 90% of intra-pupillary space
Posterior subcapsular Measured as aggregate percentage of the posterior capsular area occupied by the opacity	Obscures 3% of the area of the posterior capsule	Obscures 30% of the area of the posterior capsule	Obscures 50% of the area of the posterior capsule	Obscures more than 50% of the area of the posterior capsule

* Designation of cataract severity that falls between grade levels can be made by addition of a + sign (e.g., 1+, 2+). Grading of cataracts is usually done when the pupil is dilated.

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B. Epidemiology of Cataract

1. Prevalence and Incidence

Studies on the prevalence of cataract have focused on different sample populations:

- The National Health and Nutritional Examination Survey (NHANES) studied both genders and all races, sampled from a broad range of communities.[24]
- The Watermen Eye Study included men only from a selected region.[25]
- The Framingham Eye Study included both genders in a small community.[26]
- The Beaver Dam Eye Study included both genders in a rural community.[27]

The NHANES study showed a progressive increase in lens opacities with age. Approximately 12 percent of participants of ages 45-54, 27 percent of those ages 55-64, and 58 percent of those ages 65-74 had lens opacities. Of the 65-74 year age group, 28.5 percent had lens opacities with associated vision decrease.[24]

The Watermen Eye Study examined lens opacities for fishermen in age ranges from 30 to 94 years and found a progressive increase in lens opacities with age. Cataract was present in approximately 1.8 percent of men under the age of 35 years. Lens opacities causing vision loss were found in approximately 5 percent of the age 55-64 group, 25 percent of the age 65-74 group, and 59 percent of the 75-84 age group.[25]

The Framingham Eye Study showed the prevalence of cataracts without vision loss ranged from 41.7 percent in persons ages 55-64 to 91.1 percent in persons ages 75-84. The prevalence of lens opacity with decreased vision was 4.5 and 45.9 percent, respectively, for the same age groups.[26]

The Beaver Dam Eye Study evaluated the prevalence of cataract in adults between the ages of 43 and 84 years. Overall, 17.3 percent had

NS more severe than level 3 in a five-step scale of severity. The investigators found cortical opacities in 16.3 percent of this population, PSC in 6 percent. Women were more commonly affected than men.[27]

Estimates of the incidence of cataract can be inferred from prevalence data. In the Framingham population, 10-30 percent of persons ages 55-75, respectively, developed lens opacities, but only 15 percent went on to have impaired vision worse than 20/30 by age 75.[28] The incidence of cortical and nuclear cataracts tabulated for the Watermen Eye Study are shown in Table 2.[29]

Table 2

Estimated Incidence of Cortical and Nuclear Cataracts: Watermen Eye Study*

Age	Cortical Cataract	Nuclear Cataract
30-39	1%	1%
40-49	3%	2%
50-59	8%	12%
60-69	17%	32%
70-79	32%	51%
80+	32%	55%

In addition, some ongoing surveys are accumulating data on the incidence and progression of cataracts due to age. The National Health Interview Survey, an ongoing nationwide study using self-reports, found that in 1986, 141 per 1,000 persons age 65 reported having cataracts.[30]

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The incidence increased to 233 per 1,000 reporting cataract at age 75 and older.

2. Risk Factors

In addition to age, risk factors for the development of cataract include:

- **Diabetes mellitus**. Persons with diabetes mellitus are at higher risk for cataracts, and persons with diabetes who have cataracts have a higher morbidity than those without cataracts.[31]
- **Drugs**. Certain medications have been found to be associated with cataractogenesis and vision loss. There is an association between corticosteroids and posterior subcapsular cataracts.[32] Drugs such as phenothiazine or other thiazines and chlorpromazine have been associated with the induction of cataract formation. Antihypertensive agents have not shown a high association with onset of cataract.[33]
- **Ultraviolet radiation**. Studies have shown that there is an increased chance of cataract formation with unprotected exposure to ultraviolet (UV) radiation. These studies find that patients living in environments with high UV-B radiation levels have a higher incidence of cataract.[25,34] Also, if not protected, persons with higher occupational exposure to UV light are at greater risk for cataract than those with lower occupational exposure rates.[35]
- **Smoking**. An association between smoking and increased nuclear opacities has been reported.[36-38]
- **Alcohol**. Several studies have shown increased cataract formation in patients with higher alcohol consumption compared with patients who have lower or no alcohol consumption.[39,40]
- **Nutrition**. Although the results are inconclusive, studies have suggested an association between cataract formation and low levels of antioxidants (e.g., vitamin C, vitamin E, carotenoids). Further study may show that antioxidants have a significant effect on decreasing the incidence of cataract.[41,42]

C. Clinical Background of Cataract

1. Natural History

Although cataracts may be categorized by a variety of methods, this Guideline classifies adult-onset cataracts on the basis of their location within the three zones of the lens: capsule, cortex, or nucleus. The capsule is the "bag" that encloses the lens with the epithelium layer attached anteriorly. The nucleus and cortex form the central and more external contents, respectively.

The mechanism of cataract formation is multifactorial and, therefore, difficult to study. Oxidation of membrane lipids, structural or enzymatic proteins, or DNA by peroxides or free radicals induced by UV light may be early initiating events that lead to loss of transparency in both the nuclear and cortical lens tissue.[15,16] In cortical cataract, electrolyte imbalance leads to overhydration of the lens, causing liquefaction of the lens fibers.[15] Clinically, cortical cataract formation is manifested by the formation of vacuoles, clefts, wedges, or lamellar separations that can be seen with the slit lamp.

Nuclear cataracts usually occur secondary to deamidation of the lens proteins by oxidation, proteolysis, and glycation. The proteins aggregate into high-molecular-weight (HMW) particles that scatter light. Colored products formed from amino acid residues in this process (urochrome) may be present. The increasing optical density of the nucleus may cause index myopia that results in myopic shift of the refractive error. In addition, the central region of the lens acquires a murky, yellowish to brunescent appearance that is visible in optic section with the slit lamp.[15,43]

Age-related PSCs are created by loss of lens fiber nuclei and replacement epithelial cells that aberrantly migrate toward the posterior pole. These epithelial cells cluster, form balloon cells, and interdigitate with adjacent lens fibers and the deeper cortical fibers, breaking them down. The result is the lacy, granular, iridescent appearance of PSCs.[44]

2. Common Signs, Symptoms, and Complications

The hallmark symptoms of cataract are decreased vision and increased problems with glare. Changes in refractive error may also occur. Mild cataracts that do not significantly affect vision (e.g., cortical changes or smaller PSCs) may be identified clinically. In such cases, patients may be advised to watch for vision changes, such as reduced visual acuity or contrast sensitivity or seeing multiple images, which may be constant or occur only under certain conditions. The patient should be advised that the presence of a lens opacity does not necessarily warrant surgical intervention. When vision loss affects the ability to perform activities of daily life, consideration should be given to cataract extraction.

3. Early Detection and Prevention

The biological processes of cataract formation are becoming more clearly understood, but there is still no clinically established treatment to prevent or slow the progression of cataract. Research on the prevention of cataract has centered on risk factors and the control of diseases, such as diabetes, which are associated with some types of cataracts.[45]

The results of research on the effect of medical treatment on the prevalence of cataract have been inconsistent. In three clinical studies, no association was found between the use of aspirin and the risk of developing cataracts.[46-48] Therefore, current findings do not support the use of aspirin as preventive or protective against cataract development.

Cataract is a multifactorial disease; different factors are associated with the development of different types of opacities. Nevertheless, a simple, low-cost and low-risk preventive strategy is to reduce exposure to sunlight, decrease or discontinue smoking, and possibly increase antioxidant vitamin intake.

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II. CARE PROCESS

This Guideline describes the optometric care provided a patient with cataract. The components of patient care described are not intended to be all inclusive because professional judgment and individual patient symptoms and findings may have a significant impact on the nature, extent, and course of the services provided. Some components of care may be delegated.

A. Diagnosis of Cataract

Many patients with undiagnosed cataract first present for examination when they experience symptoms of reduced vision that affects their daily activities. Such patients should undergo a comprehensive eye and vision examination* with particular attention given to inspection of the lens of the eye. The essential elements of this evaluation include:

1. Patient History

Demographic data (e.g., patient's age, gender, race) should be collected prior to the initiation of the patient history. The patient history should reveal whether the vision loss was of acute or gradual onset. It is unusual for a cataract to cause an acute onset of vision loss; however, sometimes a cataract may have been present for years but discovered only when the vision in the better eye was compromised. Patients should be asked about vision problems under special conditions (e.g., low contrast, glare).

The patient history should include a review of refractive history, previous ocular disease, amblyopia, eye surgery, and trauma. The patient should be asked about difficulties performing various visual tasks, which may include, but not necessarily be limited to:

- Ambulation
- Driving

* Refer to the Optometric Clinical Practice Guideline on Comprehensive Adult Eye and Vision Examination.

- Reading under both dim and high-illumination conditions
- Reading medicine labels
- Performing occupational and avocational activities.

A general health history should include information about any health or related problems that may have a bearing on the etiology or prognosis of the patient's cataract and suitability for surgery. So that the patient can be properly counseled, a thorough review of the patient's current medication(s) should determine whether a patient is taking any medication that might influence surgical decisions or procedures. The patient's allergies to antibiotics or anesthetics and any special needs relevant to surgery should be explored.

2. Ocular Examination

Examination of the eye involves identifying the nature and severity of the cataract and assessing any other diseases that might contribute to symptoms or limit the potential for good vision following cataract surgery. Elements of the ocular examination may include, but are not limited to, the following:

- Measurement of visual acuity under both low and high illumination
- Biomicroscopy with pupillary dilation, with special attention to the three clinical zones of the lens and the classification and quantification of the cataract
- Stereoscopic fundus examination with pupillary dilation
- Assessment of ocular motility and binocularity
- Visual fields screening by confrontation, and if a defect is noted, further investigation by formal perimetry
- Evaluation of pupillary responses to rule out afferent pupillary defects
- Refraction to rule out refractive shift as a cause for the decreased vision
- Measurement of intraocular pressure (IOP).

3. Supplemental Testing

Additional testing may be necessary to assess and document the extent of the functional disability and to determine whether other diseases (e.g., corneal disease, optic nerve disease, or retinal disease) may limit preoperative vision or may prove to limit postoperative vision.[49,50] Contrast sensitivity, glare testing, potential acuity testing, threshold visual fields or Amsler grid testing, fluorescein angiography, corneal pachymetry/endothelial cell count, specialized color vision testing, B-scan ultrasonography, tonography, and electrophysiology testing are not required as a part of the preoperative workup; however, individual circumstances, as documented in the patient record, may justify their use (Table 3).

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Table 3

Supplemental Testing to Document the Presence of Coexisting Eye Disease, the Extent of Functional Disability, and Potential for Improvement

Visual Fields

- Suspicion or evidence of retinal detachment, eye tumor, glaucoma, retinitis pigmentosa, coloboma, optic nerve disease, or tumor

Amsler Grid

- Suspicion of macular disease

Corneal Pachymetry/Endothelial Cell Count

- History of previous corneal transplant or corneal surgery
- Suspicion of corneal thickening or endothelial dystrophy
- Planned phacoemulsification

B-Scan Ultrasonography

- Poor or limited view of the fundus
- History or suspicion of intraocular mass or tumor

Tonography

- Suspicion of glaucoma or diagnosed open angle glaucoma

Specialized Color Vision Testing

- Suspicion of optic nerve or macular disease

Electrophysiology

- Suspicion of optic nerve disease
- Family history of retinal disease
- Assessment of visual function in a patient with a mature cataract

Contrast Sensitivity and/or Glare Testing

- Patients' symptoms of functional disability far worse than suggested by visual acuity measurements

Potential Acuity Testing

- Prediction of postoperative acuity

[redacted]

B. Management of Cataract

Care of the patient with cataract may require referral for consultation with or treatment by another optometrist or an ophthalmologist experienced in the treatment of cataract, for services outside the optometrist's scope of practice. The optometrist may participate in the comanagement of the patient, including both preoperative and postoperative care. The extent to which an optometrist can provide postoperative treatment for patients who have undergone cataract surgery may vary, depending on the state's scope of practice laws and regulations and the individual optometrist's certification. Appendix Figure 1 presents a flowchart for the optometric management of the adult patient with cataract.

l. Basis for Treatment

The treatment decision for the patient with cataract depends on the extent of his or her visual disability.

a. Nonsurgical Patient

Most people over the age of 60 years have some degree of cataract formation. However, some persons do not experience a decrease in visual acuity or have symptoms that interfere with their activities of daily living. If the patient has few functional limitations as a result of the cataract and surgery is not indicated, it may be appropriate to follow the patient at 4 to 12-month intervals to evaluate eye health and vision and to determine whether functional disability develops.

It is important for patients to have a basic understanding of cataract formation, the ocular signs and symptoms associated with cataract progression, and the risks and benefits of surgical and nonsurgical treatments. Patients should be encouraged to report all ocular symptoms such as blurred vision, decreased vision in glare or low-contrast conditions, diplopia, decreased color perception, flashes, or floaters. Because most cataracts progress over time, it is important that patients understand that timely followup examinations and management are

important for proper decision making and intervention to prevent further vision loss.

b. Surgical Patient

In most circumstances, there is no alternative to cataract surgery for correcting visual impairment and/or increasing functional ability. The patient should be provided information about the findings of the eye examination, the option of surgical intervention, and any factors that could adversely affect postoperative visual acuity or ocular health. Potential benefits and possible complications should be discussed. In addition, the patient should be advised that cataract surgery is an elective procedure in most cases that should be performed only if his or her visual acuity and functional ability are compromised. This information should be provided before the patient decides whether or not to proceed with cataract surgery.

If the patient has made the decision to proceed with cataract surgery, the optometrist should assist the patient in selecting the ophthalmic surgeon and making the necessary arrangements for the procedure. The optometrist should provide the surgeon with the results of the diagnostic and presurgical examination.

2. Available Treatment Options

a. Nonsurgical Treatment

Incipient cataracts may cause refractive error shift, blur, reduced contrast, and glare problems for the patient. The initial treatment for symptomatic cataracts may include changing a spectacle or contact lens prescription to improve vision, incorporating filters into the spectacles to decrease glare disability, advising the patient to wear brimmed hats and sunglasses to decrease glare, and dilating the pupil to allow for viewing with more peripheral areas of the lens.

Changing the lens prescription to compensate for any changes in refractive error will often significantly enhance the patient's vision. However, as a result of spectacle correction of an unequal or unilateral

refractive change, an image size difference may occur. Prescribing lenses with equal base curves and center thicknesses may help to reduce this problem. The patient developing a cataract in one eye may have difficulty with tasks requiring good binocular vision and may be a candidate for contact lenses or a spectacle-contact lens combination. Contact lenses usually help to minimize image size differences.[51] Similarly, unequal or unilateral refractive changes may induce a vertical deviation that results in visual discomfort or diplopia when near tasks are performed. This problem can often be managed by decentration of the spectacle lenses, changing bifocal position, or prescribing dissimilar segment styles, prism power, or contact lenses.

Impairment of vision due to loss of lens transparency is largely related to the presence of short wavelength (blue and near ultraviolet) light in the environment. Tinted or filter spectacle lenses, such as pink #2 or #3, or #1 grey or green, can decrease the amount of light entering the eye. Lenses that selectively filter short wavelengths may improve image contrast by reducing light scatter and lens fluorescence.[52] Other special lenses, such as Corning CPF filters, may be helpful.[53,54] Improved visual acuity and contrast sensitivity may be obtained by using a filter that blocks light with wavelengths shorter than 480 nm.[55]

Counseling the patient regarding illumination control can also improve patient comfort and functional visual ability. The use of a visor indoors or a wide-brimmed hat and sunglasses outdoors may help to reduce problems with glare. The appropriate positioning and use of light sources, such as reading lights, can be beneficial.

Under certain circumstances, if the cataract is centrally located, improved vision might be obtained by the instillation of mydriatic drops, such as phenylephrine (2.5%) or tropicamide (0.5%),* provided there are no contraindications.[56] The benefits of this pharmacological treatment must be weighed against the side effects of loss of accommodation and

* Every effort has been made to ensure the drug dosage recommendations are accurate at the time of publication of the Guideline. However, as treatment recommendations change due to continuing research and clinical experience, clinicians should verify drug dosage schedules with information found on product information sheets. This is especially true in cases of new or infrequently used drugs.

photosensitivity due to pupillary dilation. IOP should be evaluated periodically during therapy.

The patient should be advised of how the cataract might affect performance of visual tasks and visually guided activities. For example, an individual who has 20/50 Snellen visual acuity in each eye, but elects to defer cataract surgery, should be advised of the possible risks due to impaired ability to perform tasks such as driving a car or operating machinery. When the cataract formation is in one eye, the loss of stereopsis should be discussed. If the cataract continues to progress and vision deteriorates, but surgery is not a viable option or is declined by the patient, a low vision evaluation may be appropriate.

b. Indications for Surgery

Surgery is indicated when cataract formation has reduced visual acuity to the level that it interferes with the patient's lifestyle and everyday activities, and when satisfactory functional vision cannot be obtained with spectacles, contact lenses, or other optical aids. The vision needs of the patient, as they relate to his or her lifestyle, occupation, and hobbies, should be considered.

Indications for surgery are usually based on the level of Snellen visual acuity and have been divided into two groups of patients: those with visual acuity of 20/40 or better and those with 20/50 or worse.

In patients with 20/40 visual acuity or better, special attention should be paid to patient complaints of decreased vision during specific tasks, monocular diplopia or polyopia, or large refractive difference between the eyes. Debilitation due to any of these factors may indicate a need for surgical intervention. There should be measurement and documentation of contrast sensitivity and visual function under glare conditions.[50]

In patients who have worse than 20/40 visual acuity, surgical intervention is more likely to be appropriate because of reduced ability or inability to perform daily tasks. The patient should be informed about cataract and advised if his or her current level of vision does not meet state law requirements for driving. The patient should be advised of the

probability of improvement in his or her visual ability following surgery, the risks of surgery, and the chances of a further decrease in vision if surgery is not undertaken. The alternatives to surgery (i.e., refractive and/or low vision correction) should be discussed. The patient should be advised that, although cataract extraction with intraocular lens (IOL) implantation is the standard of care today, cataract extraction and vision correction with aphakic spectacles or contact lenses is an alternative to lens implantation.

Other special indications for surgery are lens-induced disease such as uveitis, phacomorphic or phacolytic glaucoma, or existing concomitant ocular disease such as diabetic retinopathy, in which a clear view of the retina is necessary to monitor retinal changes and perform ocular treatments such as photocoagulation.

When a cataract is present in the fellow eye, surgery need not be considered if visual improvement of the first operated eye allows the patient good visual function and comfort or if correction with glasses or contact lenses adequately resolves the patient's complaints and meets his or her lifestyle and occupational goals. Surgery on the second eye, when deemed necessary, may be performed after vision in the first operated eye has stabilized so that the patient can function adequately during the postoperative period for the second eye. The examination process and counseling related to surgery for the second eye should be the same as for the first eye.

c. Special Surgical Considerations

Coexisting conditions in which cataract extraction with IOL implantation are usually contraindicated include:[57,58]

- Active proliferative diabetic retinopathy (unless cataract removal is necessary to allow visualization of the retina)
- Rubeosis iridis and/or neovascular glaucoma
- Microphthalmos
- Buphthalmos.

Various preoperative conditions may affect some aspects of cataract surgery, as discussed in the following paragraphs.

- **Anterior uveitis**. It is common for a patient with recurrent anterior uveitis* to develop cataract due to the intraocular inflammation and/or steroid therapy. In general, surgery is only considered when the anterior uveitis has been quiet and stable for a period of time.[59] It may be beneficial to treat the eye with topical steroids for a week before cataract surgery and to provide more aggressive treatment with topical anti-inflammatory medications following the surgery.

- **Corneal guttata**. This condition places the patient at risk for postoperative corneal edema, a risk that should be discussed with the patient before surgery. This is evaluated pre-operatively by the endothelial cell count and pachymetry. The surgeon may use a viscoelastic substance to protect the endothelium and to reduce the risk for postoperative corneal edema.[60] If the patient develops chronic corneal edema following cataract extraction, penetrating keratoplasty may be needed.

- **Diabetes mellitus**. Patients with diabetes mellitus** are at greater risk of developing cystoid macular edema (CME) and should be counseled appropriately. When there is any evidence of clinically significant macular edema, rubeosis iridis, and/or proliferative diabetic retinopathy, the condition should be treated and stabilized prior to cataract surgery, if possible. When laser photocoagulation is not performed prior to cataract surgery, the patient should receive careful retinal examination in the early postoperative period because diabetic retinopathy can accelerate following cataract surgery.[61-63]

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* Refer to the Optometric Clinical Practice Guideline for Care of the Patient with Anterior Uveitis.

** Refer to the Optometric Clinical Practice Guideline for Care of the Patient with Diabetes Mellitus.

- **Glaucoma**. The glaucoma* patient who also has a cataract provides a special challenge.[64] A thorough clinical evaluation should be performed to ensure that glaucoma is stable prior to cataract surgery and to determine whether the cataract contributes significantly to reduction of visual acuity. When the IOP is not well controlled or there are moderate to advanced optic nerve and visual field changes, a combined cataract extraction and trabeculectomy may be performed.

- **Retinal detachment**. A history of retinal detachment** in the cataractous eye or the fellow eye or the existence of lattice degeneration places the patient at greater risk for detachment. However, recent studies have reported that retinal detachment rates and the results of repair are not influenced by IOL implantation, although repair may be more difficult.[58, 65]

d. Other Contraindications for Surgery

In addition to the previously described contraindications, surgery should not be performed when the patient is unfit for surgery due to underlying systemic disease or coexisting medical conditions, when the patient is not mentally competent to consent to surgery, or when no guardian or other responsible person can consent to surgery for the patient. Moreover, even if the risks and benefits have been explained to a patient with profound vision loss but he or she does not desire surgery, it should not be done. Cataract surgery is contraindicated when it will not improve visual function due to the presence of coexisting ocular disease. However, cataract surgery may be necessary to improve visualization of the posterior segment for treatment of coexisting disease.

* Refer to the Optometric Clinical Practice Guidelines for Care of the Patient with Primary Angle Closure Glaucoma and Care of the Patient with Open Angle Glaucoma.

** Refer to the Optometric Clinical Practice Guideline for Care of the Patient with Retinal Detachment and Related Peripheral Vitreoretinal Disease.

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e. Surgical Procedures

Under most circumstances, the standard of care in cataract surgery is removal of the cataract by extracapsular cataract extraction (ECCE), using either phacoemulsification (PE) or nuclear expression.[65,66] ECCE has replaced intracapsular cataract extraction (ICCE) as the standard of care for primary cataract extraction although ICCE is still used under certain special circumstances.[65] The following brief descriptions show the nature as well as special indications and risks of each surgical procedure.

- **Extracapsular cataract extraction by phacoemulsification**. After the opening incision and anterior capsulotomy, an ultrasonic probe emulsifies the hard nucleus, enabling the surgeon to remove the lens material using a suction device. This procedure maintains the normal depth of the anterior chamber. The wound is then enlarged to allow insertion of a posterior chamber IOL into the capsular bag. Depending on the configuration of the wound, the incision may be closed with a single suture or without sutures.

- **Extracapsular cataract extraction by nuclear expression**. Following the opening incision and anterior capsulotomy, the nucleus is expressed from the capsular bag and removed in one piece through the incision. The residual cortex is removed by irrigation and aspiration. This procedure requires a larger incision, usually necessitating several sutures to close the wound.

- **Intracapsular cataract extraction**. Following the opening incision, the entire lens is extracted in one piece, with the nucleus and cortex still enclosed in the lens capsule. Because this procedure requires a very large incision and carries a much higher risk of loss of vitreous and postoperative complications, it is seldom performed. However, it may be preferable to remove the cataract by this procedure in special circumstances (e.g., damaged zonules secondary to trauma).

Over the past 15 years cataract surgery has made a significant transition from inpatient to outpatient surgery. This change has resulted in a

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decrease in the cost of patient care without an adverse effect on surgical outcomes.[67]

3. Patient Education

Surgical candidates should be informed of the risks involved with cataract surgery. Those risks include serious complications (e.g., endophthalmitis) which may result in vision worse than that prior to surgery or in total vision loss.[68] Other complications that may also occur (e.g., CME) may require additional medications or prolonged followup, but do not necessarily result in long-term vision loss.[69]

Patients should be advised of the advantages and disadvantages of the available cataract extraction techniques and intraocular lenses and the postoperative care available to them. The qualifications of the surgeon(s) and the setting for delivery of care should be discussed. Patient counseling may include a discussion of the following aspects of the surgery:

- **Anesthesia**. Local anesthesia by peribulbar or retrobulbar injection is most commonly used; facial block is less common. Topical anesthesia has recently become more widely used. The choice of anesthesia is determined by the surgeon. Based on both safety and efficacy, it should allow completion of surgery with the least pain and lowest risk to the patient.

- **Location and type of incision**. Superiorly located incisions (3-10 mm, depending on surgical method) are made posterior to the limbus and termed "scleral pocket," "scleral tunnel," or "posterior" incisions. Single stitch or self-sealing wounds are generally preferred. The clear corneal procedure has gained widespread acceptance and is now frequently used. This type of incision appears to be astigmatism neutral in most cases.

- **Intraocular lens options**. Although anterior chamber lenses may be necessary in the case of secondary IOL implantation or capsular rupture during cataract extraction, posterior chamber lenses are the choice for nearly all patients today.[70] A posterior chamber IOL

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placed in the capsular bag is associated with less risk of decentration, inflammation, and posterior capsular opacification, and with fewer complications associated with neodymium-yttrium aluminum garnet Nd:YAG laser capsulotomy, should it become necessary.[71] The IOL may be of rigid or soft plastic, with single-vision or multifocal optics. There are many alternative designs for the haptic portion of the lens, which positions it within the capsule.

- **Medications**. A combination of topical and oral antiglaucoma, antibiotic, and anti-inflammatory medications may be administered to the patient before, during, and after the operation.

- **Disposition**. Following surgery, the patient may receive an eye patch or eye shield, depending on the type of anesthesia and incision. The patient can usually be discharged the same day. Under most circumstances, the patient will be seen the next day for the initial postoperative evaluation.

- **Continuing postoperative care**. The expected schedule for postoperative visits and the need for an ophthalmic lens prescription should be explained to the patient.

4. Prognosis and Followup

Cataract extraction with IOL implant leads to improved vision in the majority of patients.[13] It also leads to better ability to perform daily activities and improved quality of life and mental status.[72]

The decision on the delivery of postoperative care shall be made by the patient, the optometrist, and the operating surgeon. Many optometrists provide postoperative care following cataract surgery. When the optometrist assumes care of the patient, the surgeon should provide a written statement regarding the transfer of care. The surgeon should inform the comanaging optometrist of both the surgical procedure used and any unusual events or complications during the surgery. If postsurgical complications arise (Table 4), the comanaging optometrist will inform or consult with the operating surgeon.

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Table 4
Postoperative Complications
That May Arise Following Cataract Surgery

Early Emergent Complications	Early Less-Emergent Complications	Intermediate to Late Complications
• Ocular hypertension	• Ptosis	• Ptosis
• Malignant glaucoma	• Diplopia	• Diplopia
• Wound leak with shallow or flat anterior chamber	• Wound leak with well-formed anterior chamber	• Ocular hypertension or glaucoma
• Endophthalmitis	• Acute corneal edema	• Epithelial downgrowth
• Iris prolapse or vitreous in the wound	• Hyphema	• Chronia corneal edema/corneal decompensation
• Intraocular lens dislocation	• Intraocular lens decentration/papillary capture	• Chronic anterior uveitis
	• Choroidal detachment	• Posterior capsular opacity
	• Anterior ischemic optic neuropathy	• Psuedophakic cystoid macular edema

[redacted]

a. Postoperative Care in the Absence of Complications

General guidelines for postoperative followup (See Appendix Figure 2) are as follows:

- **First visit** (24-36 hours postoperative). At this visit, the patient's visual acuity, both unaided and with pinhole, is measured. Keratometry and retinoscopy may be performed. The IOP is tested and the anterior segment is examined to ensure IOL centration and intactness of the structures. The following structures should be evaluated with the slit lamp: conjunctiva, cornea, anterior chamber, IOL, capsule, and wound. Funduscopic examination is performed when there are symptoms of very poor vision or retinal disease.

  At the conclusion of the first postoperative visit, the patient is counseled regarding followup care (e.g., instructed in how to use the antibiotic and/or steroid drops, advised concerning the level of physical activity permitted, and warned of symptoms that require emergency care) and the second postoperative visit is scheduled.

- **Second visit** (7-14 days postoperative). Visual acuity, both unaided and with pinhole, should be measured . Tonometry and slit lamp examination should be performed as outlined for the first visit. Dilated fundus examination should be performed when indicated by signs or symptoms of retinal disease.

  At the conclusion of the second visit, the patient should be informed of his or her progress and instructed to continue or taper the antibiotic and/or steroid drops. If only an antibiotic drop was prescribed, it may be discontinued at this point. In the case of a steroid-only drop or an antibiotic/steroid combination drop, tapering may begin if the eye is quiet. This is generally accomplished by decreasing the dosage frequency by 1 less daily drop each week.[73] The patient should again be advised regarding the level of physical activity permitted and warned of symptoms requiring emergency care. An appointment should be scheduled for the third visit.

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- **Third visit** (3-4 weeks postoperative). The examination and instructions to the patient are the same as for the second visit. A refraction may be performed, and spectacles may be prescribed if the eye appears quiet and stable. An appointment should be scheduled for the fourth visit, if needed.

- **Fourth visit** (6-8 weeks postoperative). Examination and instructions to the patient are the same as for the second visit. A dilated fundus examination is recommended at the final postoperative visit if one has not been done earlier. If the patient's eye is quiet, any topical medications still in use can be discontinued. A refraction should be performed, and the patient may be given the final postoperative prescription, if it was not given at the previous visit.

Spectacle or contact lens correction is usually needed after cataract surgery with IOL implantation to correct any residual refractive error or pseudophakic presbyopia. Residual refractive error may be due to planned or unexpected undercorrection or overcorrection by the IOL power and/or due to pre-existing corneal astigmatism or induced corneal astigmatism caused by suturing of the incision. A near vision prescription may also be needed to compensate for the loss of focusing power of the eye due to removal of the lens. Some ophthalmic surgeons prefer to set a goal of low myopia (less than 1 diopter [D]) rather than emmetropia for postoperative refractive error. This may allow the patient to function at many intermediate and near visual tasks without a lens prescription.

The "optimal" postoperative refraction for a particular patient may be based on the refractive status of the other eye, whether cataract surgery is planned or anticipated for both eyes, and the specific visual status and needs of the patient. There are several special considerations for prescribing glasses for patients who have undergone cataract extraction with IOL implants. For patients who have had surgery in one eye only, more than 1.50 D of anisometropia may result in asthenopia related to correction with different spectacle lens powers. The option of the use of a contact lens on the fellow eye should be discussed with the patient. In

patients with presbyopia undergoing cataract extraction in one eye only, the fellow eye typically has a near add power of approximately 2.50 D, which allows a normal correction for the operated eye of approximately 2.50 D, bringing the two eyes into balance at near. However, for younger patients who have some residual accommodative function, a monocular add for the operated eye is required to bring it into focus with the fellow eye when reading.

Ultraviolet protection is provided in most implanted IOLs. This protection is needed in the replacement lens because the natural lens normally absorbs UV radiation and shields the retina from exposure. For patients who have IOLs without UV absorption, the benefits of UV protection (e.g., reducing the risk of age-related macular degeneration) should be discussed. UV protection can also be provided in spectacles and contact lenses.

For patients who have anisometropia of 3 D or less, vision can usually be satisfactorily corrected with spectacle lenses. In patients with greater differences between lens powers, and even in some patients whose lens powers differ by as little as 1 D, symptoms of diplopia and asthenopia (particularly at near), headache, or photophobia may occur. Aniseikonia also needs to be considered in the spectacle management of these patients. If spectacle management is not satisfactory, a contact lens, IOL exchange, or refractive surgery may be considered to balance the refractive correction between the two eyes.

Postoperative astigmatism may be influenced by size or location of the incision, suturing method, or suture tension. When astigmatism is caused by tight suturing, sutures may be cut after approximately 4-6 weeks. Often this can reduce astigmatism, which typically has the steepest corneal meridian directly aligned with the orientation of the tightest suture. When induced corneal steepening in the vertical meridian is associated with tight sutures, suture-cutting should be considered before glasses are prescribed to reduce the astigmatism in the spectacle correction.

In most patients with pseudophakia, the appropriate refractive correction is usually determined 4-8 weeks after the surgery. By that time, sutures may have been cut, and the eye has healed enough that the refraction is stable. However, some patients may show fluctuating refractive error several weeks after surgery. Such patients may have to wait longer until the refraction has stabilized. Alternatively, a temporary pair of glasses may be offered to improve vision, even though the glasses may have to be modified in a few months when the refraction has stabilized.

- **Subsequent followup visits**. A 3-month postoperative examination is optional. Most patients should be seen at about 6 months after the surgery and then as indicated thereafter.

b. Postoperative Care of Early Emergent Complications

- **Ocular hypertension**. During the early postoperative period (1-14 days), the retention of lens particles and inflammatory debris, viscoelastic material, red blood cells, vitreous, or pigment cells may obstruct the trabecular meshwork and cause an elevation of the IOP.[74] Fortunately, this elevation is usually transient and, under most circumstances, IOP will return to baseline or lower.

The clinical signs of ocular hypertension may depend on the degree of elevation of the pressure. The conjunctiva may be injected. Corneal edema may be present, due to associated trauma or to the pushing of aqueous into the cornea by high IOP. Postoperative microcystic edema may indicate increased IOP. The anterior chamber angle should be assessed to rule out closed angle as a cause of the elevated pressure. The IOP should be tested by applanation tonometry.

When increased IOP is due to an obstruction of an open angle,* it should be treated with aqueous suppressants. Beta blockers, alpha-agonists, carbonic anhydrase inhibitors (CAIs), and/or oral

* Refer to the Optometric Clinical Practice Guidelines on Care of the Patient with Open Angle Glaucoma and Primary Angle Closure Glaucoma.

hyperosmotic agents are used to lower the IOP postoperatively, provided there are no contraindications.[75] The use of topical corticosteroids to suppress associated inflammation may also improve aqueous outflow by decreasing the swelling of the trabecular meshwork.[76]

Glaucoma with closed-angle mechanism* can also occur if a portion of the implant obstructs the pupil, creating pupillary block. This obstruction is possible with any type of IOL but is more common with anterior chamber IOLs.[77] Pupillary block has also been reported with posterior chamber IOLs, especially sulcus-fixated IOLs.[78] In this instance, clinical inspection reveals iris bombé around the edge of the implant. Generally, there is no iridectomy, or the implant haptic may be occluding the iridectomy. It is also possible for the anterior face of the vitreous to occlude the iridectomy. The IOP is usually elevated; however, persistent elevation of the IOP may lead to ciliary body shutdown, in which case, a hypotonous IOP may occur.[79]

Other conditions that may cause a shallow anterior chamber include malignant glaucoma, choroidal detachment, and wound leak with hypotony.

The goal of therapy for closed angle glaucoma** is to medically break the attack, followed shortly by laser or surgical intervention. In some circumstances, when the patient cannot be immediately treated by laser iridotomy, a mydriatic drop can be instilled to dilate the pupil, relieving the pupillary block as the edge of the pupil expands past the edge of the implant.[80] It is prudent not to use a long-acting mydriatic because aggressive miotic therapy would be required to reverse the mydriasis before a laser iridotomy.

** Refer to the Optometric Clinical Practice Guideline for Care of the Patient with Primary Angle Closure Glaucoma.

- **Malignant glaucoma**. When the clinician observes an elevated IOP and a uniformly shallow anterior chamber, malignant glaucoma should be suspected. This rare condition occurs when aqueous is misdirected behind the vitreous face so that the vitreous and iris lens diaphragm are pushed forward.[81]

  This condition often requires surgical and/or laser treatment. Aqueous suppressants, cycloplegics, and hyperosmotics may be used to lower the IOP. Ultimately, many patients require a vitrectomy to break the causative vitreociliary block.[80,81]

- **Wound leak with shallow or flat anterior chamber**. If the incision is not properly closed or if it reopens postsurgically, aqueous can percolate through the wound. The patient with this condition may present with or without pain, and the IOP is usually low (typically below 8 mm Hg). Clinical signs include corneal edema and/or irregularity, and the wound may gape. The instillation of fluorescein, using a sterile fluorescein strip, facilitates the observation of a stream of aqueous with hyperfluorescent borders, cascading from the area of leakage (positive Seidel sign). Choroidal detachments are usually associated with the lowered IOP.

  Any other condition resulting in a shallow anterior chamber should be ruled out in the differential diagnosis (e.g., pupillary block glaucoma and malignant glaucoma in which the IOP is elevated). Choroidal detachment without a wound leak may cause a shallow anterior chamber. Therefore, the integrity of the wound should be carefully tested.

  The management of wound leak requires consultation with the surgeon. In some cases, the surgeon may recommend administering a strong cycloplegic, followed by pressure-patching for a few days to seal the wound. It may be necessary to repair the wound by suturing the area of dehiscence.[82]

- **Endophthalmitis**. This rare complication following cataract surgery occurs in approximately 0.02-0.50 percent of cataract patients as a result of direct microbial invasion of the anterior chamber at the time of surgery.[68,83,84] The more virulent bacteria causing endophthalmitis include Streptococcus and Pseudomonas species; moderately virulent bacteria include Staphylococcus aureus; less virulent varieties, Staphylococcus epidermidis and Propionibacterium acnes. Many other microbes are less frequently the cause of endophthalmitis. In addition, in some climates fungal endophthalmitis may present in a delayed but aggressive fashion.[85]

  The clinical presentation of endophthalmitis depends on the virulence of the offending microorganism (Table 5). In those microorganisms which are extremely virulent, inoculation usually occurs during surgery or with an infected, leaking wound and clinical signs and symptoms generally occur within 72 hours. The patient presents with severe pain, loss of vision, lid and corneal edema, and conjunctival injection. A severe anterior chamber reaction may occur, possibly with hypopyon formation, and the pupillary area may be bridged by a fibrinous membrane. Upon dilation, the pupil may show a capsular infiltrate. The vitreous should be inspected thoroughly for the presence of cells because many microorganisms, particularly the Staphylococcus bacteria, show a predilection for growth in the vitreous humor. There may be retinal hemorrhage or congested optic nerve as well. The retinoscopy reflex is generally dim, due to the accumulation of cells and protein in the aqueous and vitreous.

Table 5

Endophthalmitis: Causative Organisms

Organism (In Order of Frequency)	Onset	Presentation of Signs and Symptoms	Vision Prognosis
Staphylococcal epidermidis	Days to weeks	Mild to moderate	Good to poor
Staphylococcal aureus	Days to weeks	Mild to moderate	Good to poor
Gram-negative species	1-4 days	Severe	Poor
Streptococcus species	1-4 days	Severe	Poor
Propionibacterium acnes	Weeks to months	Indolent	Good to poor

Endophthalmitis should be suspected in any eye with pronounced inflammation. Although the onset of endophthalmitis generally occurs within the first week following surgery, it may occur as early as 1 day or as late as many months after surgery.[86] Sometimes microorganisms become trapped behind the IOL and they can be released by a Nd:YAG capsulotomy.[87] The differential diagnosis of infectious endophthalmitis encompasses all forms of severe sterile endophthalmitis.

When endophthalmitis is suspected, the optometrist must inform the cataract surgeon immediately. The patient should be promptly referred to a surgeon who specializes in managing intraocular infections (e.g., a vitreoretinal subspecialist) as an anterior chamber and vitreous tap may be required.[88] Because the intraocular infection can progress rapidly, endophthalmitis represents a true ocular emergency. If the intraocular

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inflammation is believed to be sterile and corticosteroids are prescribed, then the patient should be asked to return within 6 hours for re-examination. If the condition has progressed, prompt referral is needed to rule out infectious endophthalmitis.

- **Iris prolapse or vitreous in the wound**. The iris or vitreous may become incarcerated in the wound as a result of either poor wound closure, trauma, or increased IOP.[89] A dark mound of iris tissue may be observed in the wound or a strand of vitreous may be seen stretching through the pupil to the wound. The wound may be agape and the pupil may be distorted with a peak toward the site of incarceration.

  Iris prolapse or vitreous in the wound places the patient at risk for CME, endophthalmitis, and retinal detachment.[68,90-92] The surgeon should be contacted immediately. In most cases, surgical or laser correction will be required.

- **Intraocular lens dislocation**. With current surgical procedures, the dislocation of either a posterior chamber or an anterior chamber IOL is quite rare. An IOL dislocation occurs when the IOL moves from its proper position because it has lost most of its tissue support. A posterior chamber IOL can dislocate because of excessive trauma in removing the cataract, causing the capsule to be partially torn or the zonules broken. If capsular bag support is lost, the IOL placed in the bag may dislocate. Similarly, if the zonular attachments to the capsular bag have been broken (by surgical or prior trauma) and the IOL is implanted in the ciliary sulcus, the IOL may dislocate with time. Anterior chamber lens dislocation is rare unless the IOL has been placed in an eye with a large-sector iridectomy.

  Patients with IOL dislocations usually experience symptoms of sudden vision loss and/or diplopia. Slit lamp examination confirms that the IOL is out of position. The most common direction of displacement for the posterior chamber IOL is inferior, a condition often referred to as the "sunset syndrome." Less frequently the IOL dislocates in other directions. A

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dislocated IOL requires the prompt attention of the operating surgeon. The patient should be instructed to limit physical activity and, if possible, to maintain a head position that will make further dislocation less likely.

- **Retinal break and detachment**. Cataract surgery is a risk factor for retinal break or detachment. Modern surgical techniques have lowered the risk of retinal detachment. Current epidemiology statistics show approximately a 1.0 percent risk for retinal detachment following planned extracapsular or phacoemulsification cataract surgery.[93-95] The incidence increases with intraoperative capsular bag rupture, creating vitreous prolapse and loss, or with ICCE.[96-98] Any iatrogenic retinal detachment usually occurs within 6 months of surgery.

  The patient may present with symptoms of vitreoretinal traction, including flashing lights and floaters. When a detachment occurs, the patient may report a dark curtain or shade appearing in the peripheral visual field; however, some patients remain asymptomatic.

  The suspicion of retinal break or detachment indicates the need for a complete ocular examination. In such cases the IOP is most often low; however, it may be unchanged or elevated. In chronic retinal detachment, there may be an anterior chamber reaction. Funduscopic examination reveals typical signs of retinal breaks or detachments. Pigment and/or red blood cells may be observed in the vitreous humor. When any retinal elevation is noted postoperatively, the differential diagnosis should include choroidal detachment and other conditions (e.g., choroidal tumors) that may cause nonrhegmatogenous retinal detachments.

  A postsurgical retinal detachment is an ocular emergency, requiring prompt referral to the operating surgeon or a vitreoretinal subspecialist. In this situation, the optometrist should instruct the patient to avoid eating or drinking liquids because surgery under general anesthesia may be necessary.

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c. Postoperative Care of Early Less-Emergent Complications

- **Ptosis**. Commonly observed within a few weeks of cataract surgery, ptosis may result from prolonged akinesia, patching, trauma from the lid speculum, superior rectus levator aponeurosis trauma, and myotoxicity.[99] The patient generally complains of a lid droop. Patients who have a ptosis prior to surgery are more likely to have an exacerbation of the condition.

  In the early postoperative period (1-14 days), the patient should be reassured that most cases of early postoperative ptosis resolve in days to months. Occasionally, however, the ptosis does not improve and surgical correction may be needed.

- **Diplopia**. Double vision following cataract surgery can result from prolonged akinesia of the extraocular muscles, especially following retrobulbar anesthesia.[100] The patient reports symptoms of diplopia and there will be a noncomitant strabismus.

  Differential diagnosis of diplopia includes any pre-existing strabismus that could have been masked by a very dense cataract. If good postoperative visual acuity is attained in an eye that previously had poor vision, diplopia may occur. Furthermore, the effects of thyroid orbitopathy may be exacerbated by ocular surgery.[101]

  Patients should be reassured that most diplopia will improve within the first few days of surgery. If symptoms do not improve spontaneously, the patient should be evaluated and managed as recommended in the section on Postoperative Care of Intermediate to Late Complications of Diplopia (page 41).

- **Wound leak with well-formed anterior chamber**. Inadequate wound closure or postsurgical trauma that opens the wound may cause the aqueous to create a fistula which lowers the IOP, creating hypotony. At examination, the wound may appear to be agape and the IOP may be 5 mm Hg or lower. A diagnostic test in which sterile fluorescein solution is instilled and the wound is observed

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with blue light can be used.[102] If a cascade of aqueous with brightly stained borders is observed coursing down the cornea below the wound, the test is positive (Seidel sign) and a wound leak is confirmed.

There may be other causes for hypotony, however. Occasionally following cataract surgery, the ciliary body may stop producing aqueous. In addition, patients with anterior uveitis or retinal detachment may have low IOPs.

If the anterior chamber has a normal configuration, conservative treatment of the wound leak is generally indicated. This usually includes consulting with the surgeon and administering topical antibiotics and cycloplegics. A pressure patch may be applied to the eye and the patient observed on a daily basis. If there is no response after several days, surgical repair may be indicated.

- **Acute corneal edema**. Corneal epithelial and stromal swelling can occur following cataract surgery as a result of trauma, inflammation, or an acute rise in the IOP. The patient complains of blurred vision, foreign body sensation, and sometimes pain. Slit lamp examination may show epithelial microcysts or bullae, stromal edema and thickening, and folds in Descemet's membrane.

If IOP is over 30 mm Hg, it should be treated with aqueous suppressants, provided there are no contraindications. Excessive inflammation should be treated by frequent instillation of steroid drops. Topical hyperosmotics may be used to relieve the discomfort from bullae or microcysts.[103] However, these agents probably have no effect in resolving the stromal edema. Bandage contact lenses may be used in painful bullous keratopathy of acute nature; however, the risk of infection should be weighed against the benefits of this method of relieving pain.

Provided the corneal endothelium was healthy preoperatively, the edema should resolve within 1 or 2 weeks. If there are pre-existing endothelial abnormalities (e.g., corneal guttata or Fuchs' dystrophy), a slower recovery may be expected and permanent corneal edema becomes a possibility.

- **Hyphema**. Blood cells may accumulate in the anterior chamber following cataract surgery.[104-106] The incidence of postoperative hyphema has been estimated at 1.5-5.0 percent of cases following cataract surgery.[107] Risk factors for developing hyphema include the position of the incision, the concomitant use of anticoagulants, the degree of iris manipulation and trauma, and pre-existing iris neovascularization or Fuchs' heterochromic iridocyclitis.[107] Measures that help prevent hyphema include a more anterior incision to avoid the scleral vessels, the application of thermal cautery to the conjunctival and episcleral vessels, and discontinuing anticoagulants prior to surgery.

Although it has been shown to reduce the incidence of postoperative hyphema, discontinuing anticoagulants prior to surgery is controversial. There is speculation that if the anticoagulants are discontinued, the patient is at a greater risk for cardiovascular complications.[108] Studies have shown that patients who do not discontinue anticoagulants prior to surgery have equally successful visual outcomes as patients who discontinue anticoagulants,[109,110] despite the fact that the risk for hyphema increases when anticoagulants are continued.

In hyphema, the usual complaint is a precipitous, painless vision loss. Red blood cells in the anterior chamber may be coagulated, diffuse, layered, or form a dense fibrin ball ("eight-ball" hyphema), and there is an approximately 10 percent risk that the IOP will be elevated, although this IOP rise is usually transient.[111] If red blood cells become trapped between the IOL and the posterior capsule (endocapsular hematoma), visual acuity may be markedly decreased. In fact, the usual complaint is a precipitous loss of vision, depending on the extent of the hyphema. If there has been a tear in the posterior capsule and a loss of vitreous, vitreous hemorrhage may occur.

The patient can be reassured that his or her vision should improve. IOP over 30 mg Hg should be treated with aqueous suppressants unless their use is contraindicated, and as the hyphema clears, the patient should be evaluated for complications. Because the red blood cells are sequestered, an endocapsular hematoma will not resolve and Nd:YAG capsulotomy is necessary to disperse the red blood cells and restore clear vision.[112] Vitreous hemorrhage clears much more slowly and there is a risk for ghost cell glaucoma following the resolution of the red blood cells in the vitreous.[111] Therefore, patients with vitreous hemorrhage should have their IOP followed for several months.

- **Anterior uveitis.**[*] Modern surgical procedures have greatly reduced the incidence of intraocular inflammation. However, inflammatory reactions still may occur as a result of a variety of factors, including intraoperative trauma, hypersensitivity to irrigating solutions, response to viscoelastic agents, physiologic lens anaphylaxis, infection, and mechanical irritation by the implant.[113]

  Anterior uveitis usually causes pain and photophobia. Both the conjunctiva and episclera may be injected. There are inflammatory signs in the anterior chamber, and there may be fibrin on the corneal endothelium or surface of the IOL. In severe inflammatory disease, a pupillary membrane may form and, less commonly, there may be hypopyon.[114] The clinician should look for any signs of lens fragments retained in the anterior and posterior chamber.

  When a sterile inflammation is suspected, the patient should be treated with topical corticosteroids every 2-4 hours. Cycloplegic drugs may be used to dilate the pupil. Rarely, hourly steroid drops, sub-Tenons steroid injection, or oral steroids will be needed. Any time there is a disproportionate inflammation, endophthalmitis should be suspected. The treatment of

[*] Refer to the Optometric Clinical Practice Guideline for Care of the Patient with Anterior Uveitis.

endophthalmitis is discussed in the section on Postoperative Care of Early Emergent Complications (page 32).

- **Intraocular lens decentration/pupillary capture**. Decentration of the IOL, in either the posterior or the anterior chamber, occurs when the IOL is not symmetrically located within the pupil yet maintains most of its tissue support. A posterior chamber IOL most often becomes decentered when one haptic is located in the bag and the other haptic in the ciliary sulcus. The IOL is decentered toward the haptic that is in the sulcus. Decentration of an anterior chamber IOL can occur when the haptic rotates through an iridectomy, tilting the opposite haptic forward. If it contacts the cornea, the IOL may cause corneal edema.

  Patients with decentered IOLs may present with a variety of symptoms (e.g., blurry vision, diplopia, or an arc-like shadow corresponding to the edge of the implant); however, many patients remain asymptomatic.

  To ascertain that an IOL is in its proper position, it should be carefully examined with and without pupillary dilation (with the exception of an iris fixated IOL where dilation is contraindicated). For an anterior chamber IOL, gonioscopy should be performed to determine whether the haptic has rotated through the iridectomy. Because the haptics of posterior chamber IOLs may also rotate through the iris, the goniolens should be used to observe the structures anterior to the iris near the base of the angle.

  The treatment of an IOL decentration depends on the severity of symptoms and associated inflammatory complications. In a patient who has no symptoms and a quiet eye, observation without treatment may be appropriate. If visual acuity is diminished as a result of the decentration or as a result of inflammatory complications (e.g., corneal edema, anterior uveitis, or CME), surgical repositioning or IOL exchange may be considered. When the IOL dislocation causes significant symptoms, the surgeon should be consulted regarding the possibility of surgical correction.

Pupillary capture occurs when the pupillary margin becomes partially or totally anterior to an anterior chamber IOL or posterior to a posterior chamber IOL. Patients complain of symptoms similar to those from IOL decentrations, and they are at risk for chronic inflammation and CME. If pupillary capture occurs early in the postoperative period, dilating, then constricting the pupil may break the capture. The patient should be referred to the operating surgeon for further evaluation.

Surgical correction of longstanding pupillary capture may be necessary if visual acuity is compromised due to corneal edema, anterior uveitis, or CME. When visual acuity is not compromised, the condition may be monitored.

- **Choroidal detachment**. When serous fluid accumulates in the suprachoroidal space choroidal detachment can occur. The shunting of fluid into this space is generally precipitated by low IOP, causing hydrostatic pressure to decrease in the anterior uveal veins. The plasma proteins add an osmotic force that draws more fluid into the space, increasing the detachment.[115] Low IOP can result from a wound leak or ciliary body shutdown.

The patient with choroidal detachment may have decreased visual acuity or mild discomfort, and the IOP is usually below 8 mm Hg. The characteristic clinical signs are smooth elevations of the peripheral choroid and retina. These appear very solid but vary in extent. The differential diagnoses include retinal detachment and retinal mass.

Under most circumstances, a choroidal detachment is managed conservatively. If the anterior chamber is well formed and if the choroidal detachments are small to moderate in size, it is recommended that the patient be maintained on a strong cycloplegic agent, such as atropine (1%), 3-4 times per day, along with a topical steroid 4-6 times daily.[116] If the cause of the hypotony is from a wound leak, that should be treated as recommended in the section on Postoperative Care of Early Emergent Complications (page 30).

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If the choroidal detachment causes a shallow anterior chamber, the optometrist should consult the surgeon because the patient with this condition may need to have the choroidal detachment drained and the anterior chamber reformed. Furthermore, if the choroidal detachment is quite large, choroidal tissues from both sides of the ora may touch each other. This condition, called a "kissing" choroidal detachment, increases the risk of retinal detachment and requires immediate consultation with the operating surgeon because surgical correction may be needed.

- **Anterior ischemic optic neuropathy**. Anterior ischemic optic neuropathy (AION) is a very rare complication of cataract surgery[117,118] in which there is typically acute vision loss, altitudinal visual field defect, and, sometimes, afferent pupillary defect. Any AION in the postoperative period should be evaluated to differentiate between arteritic and nonarteritic ischemic optic neuropathy.

d. Postoperative Care of Intermediate to Late Complications

- **Ptosis**. Late ptosis occurs in as many as 5.5-13 percent of patients undergoing cataract surgery.[119,120] Causes of ptosis include superior rectus trauma, levator aponeurosis disinsertion, eyelid edema, lid speculum trauma, prolonged patching, and myotoxicity.[99,100,121-123]

Postoperative ptosis may be an exacerbation of pre-existing ptosis. Postoperative ptosis usually improves with time; therefore, regular observation is recommended. If the patient's lid does not return to the normal position after 6 months, lid surgery should be considered.[99]

- **Diplopia**. Postoperative diplopia can occur because of monocular factors (e.g., astigmatism, media opacity, corneal irregularity, IOL decentration, IOL aberrations, or macular disease).[124] Binocular diplopia can occur as a result of a strabismus. A recent epidemiologic study showed that 8.0 percent of diplopia in

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individuals over 60 years of age is the result of strabismus following cataract extraction.[125]

Strabismus can occur for a variety of reasons. Surgical trauma may exacerbate a pre-existing disease such as Graves' orbitopathy.[101,126] In addition, postoperative diplopia can result when a dense cataract is removed from a patient with pre-existing strabismus. With the improved postoperative visual acuity, the patient may complain of new diplopia.

The treatment of the diplopia depends on whether it is monocular or binocular. Careful correction of refractive error can sometimes eliminate monocular diplopia. If IOL decentration is the cause of the monocular diplopia, it should be addressed. Binocular diplopia should be monitored for up to 6 months before surgical correction is considered. In the interim, the optometrist can prescribe Fresnel prism lenses to help the patient achieve single vision in the primary field of gaze.[127] In the presence of a large angle of strabismus, or in the event spectacle correction is impractical, surgery should be considered after 6 months.

- **Ocular hypertension or glaucoma**. Ocular hypertension or glaucoma may be induced by steroids, IOL irritation, or synechial angle closure. Any patient who has increased IOP in the late postoperative period and who is taking topical steroids should be suspected of having a steroid response. Steroid responses are more likely to be found in individuals with glaucoma or a family history of glaucoma. The degree of response is influenced by which topical steroid is used and by its dosage frequency and length of use.

  It may take several weeks for the response to occur. If the eye is quiet and the IOP is elevated, the steroid can be discontinued or swiftly tapered. It may take up to a month for the IOP to normalize. If the anterior chamber is inflamed, then an alternative steroid such as fluorometholone, which has a lower potential for causing a steroid response or a topical nonsteroidal anti-inflammatory (NSAID), may be considered.[128]

Ocular hypertension or glaucoma can be induced by irritation from the IOL if the optic or haptic of an IOL is in contact with vascularized tissue. This occurs commonly with the iris-fixated and anterior chamber IOLs;[129,130] however, it can also occur with posterior chamber IOLs that are fixated in the ciliary sulcus.

A classic presentation of IOL irritation is the uveitis-glaucoma-hyphema (UGH) syndrome, which is generally associated with iris-fixated or anterior chamber IOLs.[131] In this condition, hyphema and anterior chamber inflammation result in IOP elevation from obstruction of the trabecular meshwork with inflammatory and hemorrhagic debris. Milder variants of UGH also exist.

The iris should be carefully inspected for signs of transillumination defects. If any portion of the IOL touches vascularized tissue, chafing can cause inflammation or pigment dispersion.[132] Sulcus-fixated IOLs, particularly IOLs with multiple piece construction, can abrade the posterior pigment epithelium of the iris and cause some of the signs of pigmentary glaucoma. Moreover, lenses fixated in the ciliary sulcus can sometimes cause pronounced postoperative iritis. Although modern anterior chamber IOLs, with their flexible haptics and point fixation, are less likely to induce irritative effects, they still are more likely to cause irritation than posterior chamber IOLs.

The treatment for IOL-induced irritation depends on the severity of associated changes. Initially, the inflammation may be managed medically with topical corticosteroids, and the IOP elevation may be treated with aqueous suppressants provided there are no contraindications. If the irritative effects continue, the optometrist should consult with the operating surgeon regarding either IOL exchange or repositioning.

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Synechial angle closure-induced glaucoma can occur if part of the IOL touches vascularized tissue, resulting in inflammation and the formation of peripheral anterior synechiae (PAS). Anterior chamber IOLs pose the greatest risk, particularly the older-design implants that have broad points of contact in the trabecular sulcus.[81,133] Synechial angle closure occasionally occurs in patients with sulcus-fixated posterior chamber IOLs.[81,134,135]

In this type of glaucoma, the IOP may be elevated and the pupil distorted toward the site of the PAS. Gonioscopic evaluation may reveal PAS covering the haptics of an anterior chamber IOL (cocooning). When a sulcus-fixated posterior chamber IOL is associated with synechial angle closure, gonioscopy may reveal synechiae overlying that portion of the iris closest to the haptic fixation.

Treatment involves controlling the IOP with aqueous suppressants, if not contraindicated, and controlling the inflammation with topical steroids. The operating surgeon should be consulted because IOL removal may be required.

- **Epithelial downgrowth**. Generally occurring due to poor wound closure in which conjunctival epithelium grows over the posterior surface of the cornea, across the structures of the angle and onto the iris, epithelial downgrowth decreases visual acuity.[81] Slit lamp examination shows a translucent membrane extending from the wound area onto the corneal endothelium with a line observed at the leading edge. If it extends onto the iris, this membrane can create tractional distortion of the pupil.

  Epithelial downgrowth is a severe complication. When it is suspected, the optometrist should immediately refer the patient to the operating surgeon. Aggressive surgery is indicated, with the ultimate goal of preserving the structure of the globe because in this complication corneal transparency is almost invariably lost.

- **Chronic corneal edema/corneal decompensation**. This may be the result of corneal edema that occurs immediately after surgery

and does not clear, or it may occur later in a clear cornea that suffers a gradual loss in endothelial cell function. Patients with corneal edema generally complain of irritation and they may have pain, depending on whether epithelial bullae form. Stromal edema causes thickening of the stroma and folds in Descemet's membrane. Epithelial microcysts and bullae can result from severe stromal edema. Causes of the corneal edema (e.g., a haptic touching the cornea, causing localized corneal decompensation or elevated IOP) should be identified and treated.

The treatment of corneal edema is directed toward keeping the patient comfortable and ultimately restoring corneal clarity. When the IOP is elevated, lowering it with aqueous suppressants, if not contraindicated, may help resolve the edema. Inflammation should be treated with topical anti-inflammatory medication. If pain results from the epithelial bullae and microcysts, topical hyperosmotics may alleviate the discomfort.[103] In cases of severe pain, a bandage contact lens may be indicated. When an IOL is irritating the corneal endothelium, surgical correction is indicated.

When the corneal edema is persistent, the patient should be followed for 4 months. However, when the patient has a clear cornea following cataract surgery and subsequently develops corneal edema, resolution of the edema is unlikely. The optometrist should refer the patient with unresolved or late-developing edema to a cornea subspecialist for consideration of penetrating keratoplasty.

- **Late hyphema**. Due to irritative effects of the IOL, small vessels in the ciliary body, iris, or trabecular sulcus can be severed and cause a late postoperative hyphema. This condition occurs most often with iris-fixated IOLs but can also occur with anterior chamber and sulcus-fixated posterior chamber IOLs.[132]

  Patients often complain of episodes of intermittent visual "white-outs" caused by red blood cells being released into the anterior chamber (white-out syndrome). These white-outs may last from hours to days. Between episodes, the patient may have good visual

acuity and only a few red blood cells circulating in the anterior chamber. The anterior chamber may become devoid of cells as a result of the eye's ability to absorb red blood cells rapidly. However, the IOP may become elevated. When white-out symptoms are encountered in a postoperative patient, the clinician should evaluate the condition to rule out amaurosis fugax as a cause.

The treatment of recurrent late postoperative hyphema may include prescribing a low-concentration miotic drop to stabilize the pupillary motion in an attempt to eliminate the recurrent hyphema. However, miotics can increase the inflammatory response; therefore, the eye should be carefully monitored. Surgical consultation is often required, and IOL exchange may be considered.

- **Chronic anterior uveitis**. IOL irritation, retained lens fragments, infection, or the use of miotics may cause anterior uveitis. These complications have been discussed in the section on Postoperative Care of Early Less-Emergent Complications of Anterior Uveitis (page 38). Typical signs and symptoms of anterior uveitis include pain, photophobia, and decreased vision. Slit lamp examination reveals conjunctival and episcleral injection. There may be keratic precipitates and anterior chamber reaction, along with precipitates on the intraocular lens. IOP may be elevated, and PAS may be present.

In an eye with chronic anterior uveitis, the clinician should suspect infectious endophthalmitis. It is now recognized that the onset of endophthalmitis may be delayed until months or years after cataract surgery because the microorganisms become sequestered behind the IOL.[136]

The goals of treatment for chronic anterior uveitis include identifying and eliminating its cause and suppressing the inflammation. When possible, the cause of the inflammation should be eliminated first. For example, if an IOL is causing tissue damage, IOL exchange or repositioning may be considered. Any

lens fragments may be removed surgically.[137] To control the inflammation, prednisolone acetate (1%) may be used every 2-4 hours, depending on the degree of inflammation. Topical cycloplegic drugs may be used as well.[73,138]

- **Posterior capsular opacity**. One of the most common sequelae of cataract surgery is the development of a posterior capsular opacity, which occurs in up to 50 percent of eyes within 5 years of surgery.[139,140] The onset of opacification is at anytime from months to years following surgery.[139] Younger patients are at greater risk. The opacification of the posterior capsule occurs as the result of cell proliferation, which extends from the equator of the lens capsule to the posterior surface.[141,142]

The patient complains of a gradual and painless loss of vision. Slit lamp examination reveals opacification of the posterior capsule, which may take the form of capsular pearls or tissue fibrosis. Posterior capsular opacity is treated by Nd:YAG capsulotomy. The indications for treatment of an opacified posterior capsule are similar to those of cataract. When a patient has functional vision loss, laser treatment should be considered. A patient without functional difficulties should be monitored.

- **Pseudophakic cystoid macular edema**. CME results when there is an increased permeability of parafoveal retinal capillaries and serous leakage occurs in the intraretinal layers.[143-147] Possible etiologies for this phenomena include inflammation, trauma, and vitreous traction. CME can be classified into angiographic CME, which is only observable by fluorescein angiography (FA), and clinical CME, which causes significant loss of vision and cysts in the macula. Angiographic CME is more common than clinical CME. Clinical CME occurs in 3-5 percent of cataract surgery patients.[148]

The reduction in visual acuity due to CME occurs from 1 to 4 months after surgery. The clinician should evaluate the anterior segment for any signs of anterior chamber inflammation and look for any vitreous that may be incarcerated in the wound. IOL

position should be checked. The funduscopic examination should concentrate on the macular area. Because CME may be difficult to detect, the examination should be done at high magnification with a bright illumination source. Translucent petaloid cysts emanating from a central yellow spot may be observed. Sometimes there is a mild swelling of the optic nerve and an occasional dot hemorrhage in the macula. When CME is not observed by funduscopy or when macular drusen are present, an FA should be performed to rule out other etiologies, such as subretinal neovascularization.

The management of CME depends on its clinical presentation. The incarceration of any vitreous in the wound should be treated by Nd:YAG vitreolysis.[149,150] IOL displacement that creates an irritation might require surgical treatment. When there is anterior chamber inflammation, topical steroidal and nonsteroidal anti-inflammatory agents should be applied to the eye for up to a month. If there is no improvement after a month, the clinician may consider oral nonsteroidal anti-inflammatory drugs or subconjunctival steroid injections.[151] Orally administered CAIs are sometimes used, but their efficacy is debatable.

If there is no apparent cause for the CME, the clinician may monitor the patient over time or begin treating the eye with topical nonsteroidal or steroidal anti-inflammatory agents or acetazolamide. Clinical studies have shown that in a significant number of untreated eyes the CME will resolve and there will be a good return of visual acuity.[148] There have been no controlled studies showing that any treatment for CME is of benefit.

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CONCLUSION

Cataract is a common problem in an aging population. Reduced vision due to cataract can greatly affect the patient's ability to perform day-to-day activities. Proper care through both nonsurgical and surgical intervention can lead to improved productivity, reduction of personal suffering, and substantial cost savings for the affected individuals, their families, and the health care system as a whole.

Every patient with cataract should be informed of the presence of the condition. The optometrist should discuss with the patient the natural course of the cataract and the treatment options, as well as the importance of routine examinations. Cataract patients whose vision loss is correctable with spectacles should be informed that the lens opacities may progress and require other spectacle lens changes or surgery. Patients who cannot otherwise achieve adequate vision for their activities of daily living should be informed that only surgery can help rehabilitate their vision (i.e., that further spectacle changes would be of limited value). A candidate for cataract surgery must be informed of all of the risks and benefits of surgery. The patient should be provided complete information on the pros and cons of the various surgical techniques, the skills of the surgeons in the area, and the expected outcome and schedule for postoperative care. The patient who has had cataract surgery should receive proper and timely postoperative care and proper monitoring of both overall ocular health and vision status.

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IV. APPENDIX

Figure 1
Optometric Management of the Adult Patient With Cataract: A Brief Flowchart

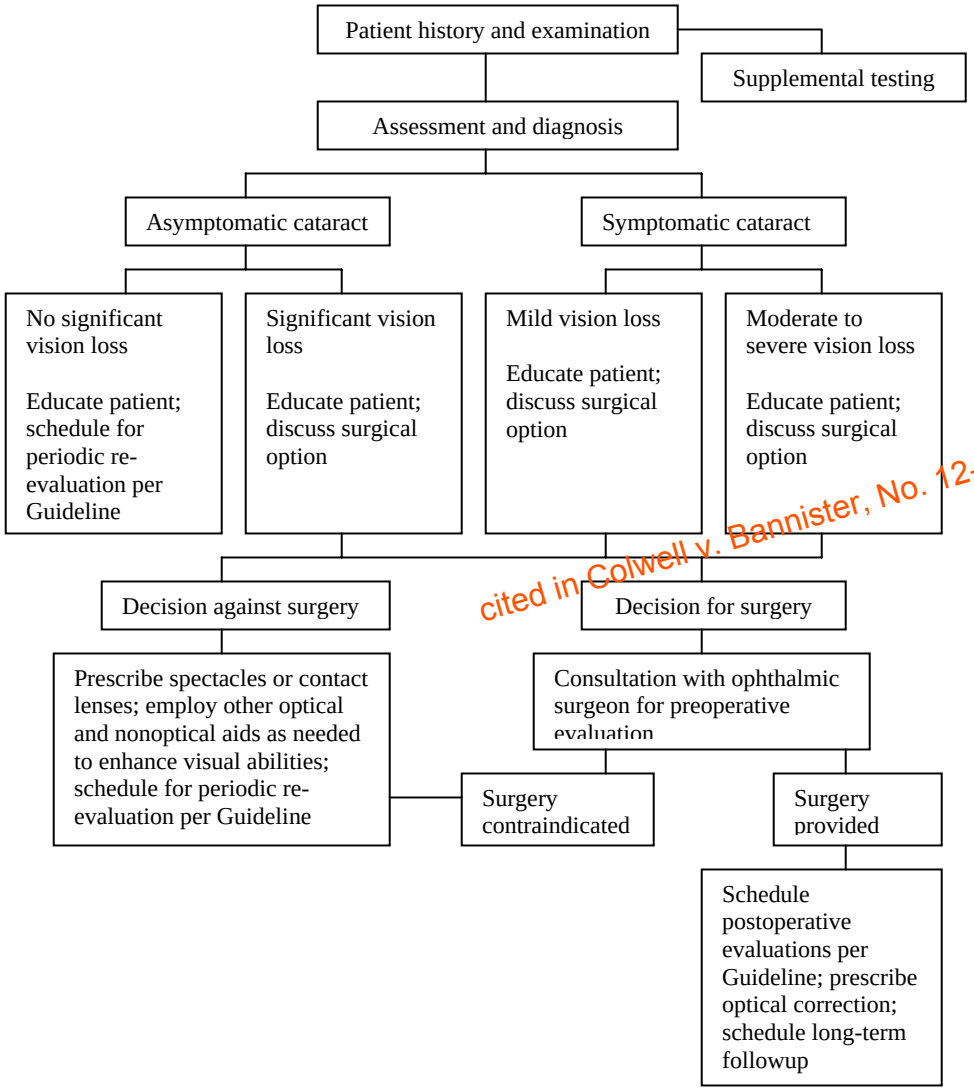

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Figure 2
Frequency and Composition of Evaluation and Management Visits for an Uncomplicated Clinical Course Following Cataract Surgery✱

Postoperative Visits	History	Visual Acuity Unaided and With Pinhole*	External and Slit Lamp Exam
# 1 One day	Yes	Yes	Yes
# 2 7-14 days Usually 1 week	Yes	Yes	Yes
# 3 3-4 weeks	Yes	Yes	Yes
# 4+ 6-8 weeks	Yes	Yes	Yes++
# 5* Subsequent visits 3-6 months	Yes	Aided visual acuity with pinhole	Yes

* Pinhole VA: assess if visual acuity worse than 20/30 unaided.
\+ Optional visit: Some clinicians elect to schedule three postoperative visits, others four prior to determining a final spectacle prescription
++ Consider need to cut sutures if high astigmatism is present.
✱ Figure 2 extends horizontally on page 63.

[redacted]

Figure 2

Refraction	Tonometry	Dilated Fundus Exam**	Management Plan
	Yes	If indicated by symptoms of very poor vision or retinal disease	Administer topical antibiotic/steriod; Counsel patient
	Yes	If indicated by signs or symptoms of retinal disease	Continue and/or taper medications; counsel patient
Yes	Yes	Yes+++	Continue and/or taper medications; counsel patient Prescribe refractive correction
Yes	Yes	Yes+++	Discontinue medications if exam is normal; counsel patient Prescribe/modify refractive correction
If vision is reduced	Yes	If indicated based on findings and symptoms+++	Reschedule for yearly evaluation or as needed

** Dilated fundus exam: provided at least once during the postoperative period.
+++ Check clarity of posterior capsule.

[redacted]

Figure 3
ICD-9-CM Classification of Cataract

Cataract **366**
Excludes: congenital cataract (743.30-743.34)

Infantile, juvenile, and presenile cataract **366.0**

Nonsenile cataract, unspecified **366.00**

Anterior subcapsular polar cataract **366.01**

Posterior subcapsular polar cataract **366.02**

Cortical, lamellar, or zonular cataract **366.03**

Nuclear cataract **366.04**

Other and combined forms of nonsenile cataract **366.09**

Senile cataract **366.1**

Senile cataract, unspecified **366.10**

Pseudoexfoliation of lens capsule **366.11**

Incipient cataract **366.12**
Cataract: Water clefts
coronary
immature NOS
punctuate

Anterior subcapsular polar senile cataract **366.13**

Posterior subcapsular polar senile cataract **366.14**

Cortical senile cataract **366.15**

[redacted]

Nuclear sclerosis **366.16**
Cataracta brunescens
Nuclear cataract

Total or mature cataract **366.17**

Hypermature cataract **366.18**
Morgagni cataract

Other and combined forms of senile cataract **366.19**

Traumatic cataract **366.2**

Traumatic cataract, unspecified **366.20**

Localized traumatic opacities **366.21**
Vossius' ring

Total traumatic cataract **366.22**

Partially resolved traumatic cataract **366.23**

Cataract secondary to ocular disorders **366.3**

Cataracta complicata, unspecified **366.30**

Glaucomatous flecks (subcapsular) **366.31**
Code first underlying glaucoma (365.0-365.9)

Cataract in inflammatory disorders **366.32**
Code first underlying condition, as:
chronic choroiditis (363.0-363.2)

Cataract with neovascularization **366.33**
Code first underlying condition, as:
chronic iridocyclitis (364.10)

[redacted]

Cataract in degenerative disorders **366.34**
Sunflower cataract
Code first underlying condition, as:
chalcosis (360.24)
degenerative myopia (360.21)
pigmentary retinal dystrophy (362.74)

Cataract associated with other disorders **366.4**

Diabetic cataract **366.41**
Code first diabetes (250.5)

Tetanic cataract **366.42**
Code first underlying disease, as:
calcinosis (275.4)
hypoparathyroidism (252.1)

Myotonic cataract **366.43**
Code first underlying disorder (359.2)

Cataract associated with other syndromes **366.44**
Code first underlying condition, as:
craniofacial dysostosis (756.0)
galactosemia (271.1)

Toxic cataract **366.45**
Drug-induced cataract
Use additional E code, if desired, to identify drug or other toxic substance*

Cataract associated with radiation and other physical influences **366.46**
Use additional E code, if desired, to identify cause*

After-cataract **366.5**

After-cataract, unspecified **366.50**
Secondary cataract NOS

Soemmering's ring **366.51**

[redacted]

Other after-cataract, not obscuring vision	**366.52**
After-cataract, obscuring vision	**366.53**
Other cataract Calcification of lens	**366.8**
Unspecified cataract	**366.9**
Congenital cataract and lens anomalies	**743.3**
Excludes: infantile cataract (366.00-366.09)	
Congenital cataract, unspecified	**743.30**
Capsular and subcapsular cataract	**743.31**
Cortical and zonular cataract	**743.32**
Nuclear cataract	**743.33**
Total and subtotal cataract, congenital	**743.34**
Congenital aphakia Congenital absence of lens	**743.35**
Anomalies of lens shape Microphakia Spherophakia	**743.36**
Congenital ectopic lens	**743.37**
Other	**743.39**

* *To determine E code, please see SUPPLEMENTARY CLASSIFICATION OF EXTERNAL CAUSES OF INJURY AND POISONING (E800-E999).*

[redacted]

Abbreviations of Commonly Used Terms

AION	-	Anterior ischemic optic neuropathy
CAI	-	Carbonic anhydrase inhibitor
CC	-	Cortical cataract
CME	-	Cystoid macular edema
D	-	Diopter
DNA	-	Deoxyribonucleic acid
ECCE	-	Extracapsular cataract extraction
FA	-	Fluorescein angiography
HMW	-	High molecular weight
ICCE	-	Intracapsular cataract extraction
IOL	-	Intraocular lens
IOP	-	Intraocular pressure
Nd:YAG	-	Neodymium-yttrium aluminum garnet
NHANES	-	National Health and Nutritional Examination Survey
nm	-	Nanometer
NS	-	Nuclear sclerosis
NSAID	-	Nonsteroidal anti-inflammatory drug
PAS	-	Peripheral anterior synechia
PE	-	Phacoemulsification
PSC	-	Posterior subcapsular cataract
UGH	-	Uveitis-glaucoma-hyphema
UV	-	Ultraviolet

[redacted]

Glossary

Anterior chamber The space in the eye, filled with aqueous humor, that is bordered anteriorly by the cornea and a small portion of the sclera and posteriorly by a small portion of the ciliary body, the iris, and that portion of the lens which presents through the pupil.

Biomicroscopy Examination of ocular tissue using a bright focal source of light with a slit of variable width and height and a binocular microscope with variable magnification.

Capsulotomy Disruption of the lens capsule by surgical incision or laser.

Cataract An opacity of the crystalline lens or its capsule.

Choroidal detachment Separation of the choroid from the sclera, usually caused by traction from within or by accumulation of serous fluid in the perichoroidal space.

Corneal guttata Dystrophy of the endothelial cells of the cornea appearing as wart-like excrescences of Descemet's membrane projecting toward the anterior chamber.

Cortical cataract A cataract in which the opacity lies in the cortex of the crystalline lens and the opaque areas are usually oriented radially.

Crystalline lens A biconvex, normally transparent and resilient lenticular body, which serves as the focusing component of the eye and allows for accommodation. It is directly behind the pupil of the eye and nested in the patellar fossa of the vitreous body.

Cystoid macular edema (CME) A swelling of the retina in the macula region caused by serous fluid accumulating in the retinal tissue, usually assuming a petaloid configuration.

Diplopia A condition in which a single object is perceived as two rather than one.

[redacted]

Endophthalmitis An ocular emergency of severe intraocular inflammation due to infection, but possibly occurring as an allergic reaction following a cataract operation.

Epithelial downgrowth Growth of conjunctival epithelium into the eye, usually due to poor wound closure or trauma.

Extracapsular cataract extraction (ECCE) Surgical removal of the cataractous crystalline lens by incising the anterior capsule of the lens and expressing the lens substance, leaving the posterior capsule intact.

Fluorescein angiography (FA, FANG) A diagnostic procedure whereby sodium fluorescein dye is injected intravenously and its passage is observed as it transits the retina and choroid.

Glaucoma A group of ocular diseases with various causes that ultimately are associated with a progressive optic neuropathy leading to loss of vision function. Glaucoma is often associated with abnormally increased intraocular pressure.

Gonioscopy A diagnostic procedure to examine the angle of the anterior chamber in which a specialized corneal contact lens and a biomicroscope are used.

Hyphema Blood in the anterior chamber.

Hypotony, ocular Abnormally low intraocular pressure.

Intracapsular cataract extraction (ICCE) Surgical removal of a cataractous crystalline lens, together with its capsule.

Intraocular lens (IOL) A plastic lens that is surgically implanted to replace the crystalline lens of the eye.

Intraocular pressure (IOP) The pressure within the eye relative to the constant formation and drainage of the aqueous humor.

Iridectomy Surgical removal of part of the iris.

[redacted]

Myotoxicity Toxic reaction of the muscle fibers.

Nuclear cataract An opacity of the central nucleus of the crystalline lens.

Nuclear expression A procedure during extracapsular cataract extraction in which the nucleus of the lens is removed in one piece.

Peripheral anterior synechia (PAS) An adhesion between the peripheral iris and the anterior chamber angle or peripheral cornea.

Phacoemulsification (PE) A surgical procedure during extracapsular cataract extraction in which the nucleus of the lens is emulsified by ultrasound and removed by aspiration.

Posterior capsular opacification A clouding of the posterior capsule that occasionally occurs following extracapsular cataract extraction.

Posterior chamber The space in the eye delimited by the posterior surface of the iris, the ciliary processes and the valleys between them, the zonule of Zinn, and the anterior surface of the crystalline lens. It includes the canal of Hanover, the canal of Petit, and the retrolental space of Berger.

Posterior subcapsular cataract (PSC) A lens opacity involving the posterior region of the lens, especially beneath the posterior lens capsule.

Ptosis Drooping of the upper eyelid below its normal position.

Pupillary block Blockage of the normal flow of aqueous humor from the posterior chamber into the anterior chamber through the pupil.

Pupillary capture Occurs when the pupillary margin is located partially or totally anterior to an anterior chamber IOL or posterior to a posterior chamber IOL.

Refraction Clinically, the determination of the refractive error of the eye or eyes (e.g., myopia, hyperopia, astigmatism, anisometropia).

Seidel sign A cascading stream of hyperfluorescence, observed with a slit lamp following instillation of sodium fluorescein, indicating a leak of aqueous humor through a wound. Also, a sickle-shaped scotoma appearing as an upward or downward prolongation of the physical blind spot, indicating glaucoma, is referred as *Seidel's sign*.

Sunset syndrome Sudden diplopia and/or vision loss that occurs when a posterior chamber IOL is decentered or dislocated inferiorly due to poor zonular support or haptic-sulcus fixation.

Tonometry A procedure for measurement of the pressure within the eye. Clinically, tonometry measures the intraocular tension.

UGH syndrome Uveitis, glaucoma, and hyphema that may occur in a pseudophakic eye, due to abnormal contact of the IOL with vascularized tissues.

Visual acuity The clearness of vision that depends upon the sharpness of focus of the retinal image and the integrity of the retina and visual pathway.
It is expressed as the angle subtended at the anterior focal point of the eye by the detail of the letter or symbol recognized.

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