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CE **Article #1 (1.5 contact hours)**
Refereed Peer Review

Cataract Evaluation and Treatment in Dogs

KEY FACTS

- Results of a complete ophthalmic examination of a dog with cataracts may reveal subtle abnormalities that may postpone or preclude surgery.
- Proper selection of patients is key to a better prognosis for long-term vision after surgery.
- Any systemic disease should be detected and managed before cataract surgery.

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ABSTRACT: Cataracts are opacities within the lens that may affect a small portion of the lens or the entire lens. Cataracts are a leading cause of blindness in dogs. They may be due to hereditary factors, metabolic diseases, inflammation, or other causes. A thorough ophthalmic and physical examination, complete blood cell count, biochemical evaluation, electroretinography, and ocular ultrasonography should be used to determine whether a patient is a candidate for cataract surgery (phacoemulsification). Several medications are prescribed during the preoperative and postoperative periods. Complications that can occur after phacoemulsification include anterior uveitis, ocular hypertension, glaucoma, corneal ulcers, and retinal detachment.

Cataracts are the most common cause of treatable visual deficits and blindness in dogs. Proper selection of patients is vital to a successful visual outcome after cataract surgery (phacoemulsification). The ideal surgical candidate is a middle-aged patient with an immature cataract and no other ocular or systemic abnormalities. In reality, the perfect situation rarely exists. The purpose of this article is to familiarize the practicing veterinarian with the causes and appearances of cataracts; the ocular abnormalities, revealed by an examination, that may influence the surgical outcome; and the surgical procedure for cataract removal.

LENS ANATOMY AND PHYSIOLOGY

Knowledge of the unique anatomy and physiology of the lens helps one understand cataractogenesis and the treatment of cataracts. In dogs, the lens is a biconvex structure suspended between the iris anteriorly and the vitreous posteriorly by zonules that originate from ciliary body processes. The lens measures 7 mm anterior to posterior and 10 mm in diameter. It consists of two parts: the crystalline lens and the lens capsule. The lens capsule, which envelops the crystalline lens, is a basement membrane secreted by the lens epithelial cells. The anterior capsule thickens throughout life, whereas the posterior capsule remains about 2 to 4 µm thick, approximately twice the thickness of an erythrocyte. The lens epithelial cells, the only metabolically active cells in the lens, occur only subjacent to the anterior lens capsule in the postnatal animal. These epithelial cells undergo differentiation at the lens equator; after the cells lose their nuclei, they become lens fibers. The center of the lens is the nucleus and contains the oldest lens fibers. The tips of the fibers come together to make a Y-shaped pattern in the center of

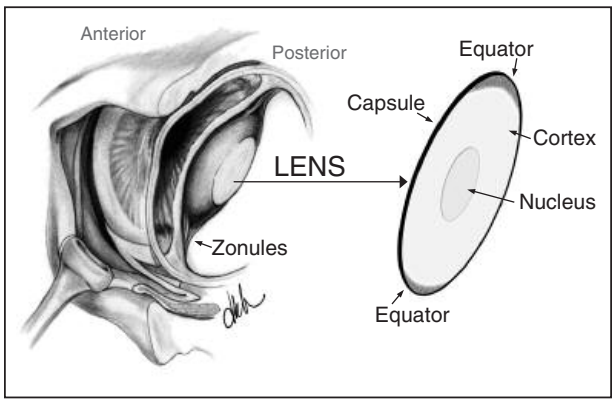

Figure 1—Anatomy of the ocular lens. (©2003 The University of Tennessee College of Veterinary Medicine)

the lens; this pattern is commonly visible on examination. The lens cortex consists of the newer fibers that surround the lens nucleus[1] (Figure 1).

The lens is unique because of its avascularity. Physiologically, all metabolic processes in the lens function to maintain clarity of the lens. Anaerobic glycolysis is the most active metabolic pathway in the normal lens. Glucose from the aqueous humor diffuses across the lens capsule into the lens epithelial cells and is rapidly metabolized to produce ATP. Several other metabolic pathways exist but are used to a minor extent. Most cataracts develop as a result of derangements in metabolic pathways, electrolyte imbalances, or changes in the relative concentrations of the different lens proteins. Some pathologic mechanisms, such as diabetes mellitus, cause osmotic changes in the lens so that excessive fluid enters the lens, with resultant cataracts.[2]

CAUSES

Heredity, metabolic diseases, senile changes, trauma, nutritional deficiencies, toxins, drugs, radiation therapy, and inflammation can all cause cataracts in dogs.[3] Regardless of the inciting or underlying cause, cataracts result from abnormalities in lens metabolism.[4,5] Cataracts may be congenital or may be acquired at any age. The age of the dog at the onset of cataract formation, in addition to a complete history, can help determine the cause.

Inherited or genetic cataracts are the most common kind of cataract in dogs. Inherited mechanisms are suspected in more than 90 breeds of dogs.[3] For example, congenital cataracts are frequently seen in the miniature schnauzer and Labrador retriever. Most cataracts in dogs are recessively inherited, although some breeds, such as the golden retriever and Labrador retriever, have dominantly inherited cataracts.[3] In many breeds, the exact mode of inheritance is still unknown. The age of the dog at the onset of cataracts is also somewhat breed dependent.

Diabetes mellitus is another common cause of cataracts in dogs. Studies have shown that 68% to 80% of dogs with diabetes mellitus develop cataracts.[6,7] Cataract formation may be a more common complication from this disorder in dogs than in humans because of poor glucose regulation or because of species differences in lens metabolism.[7,8] Normally, glucose is metabolized by anaerobic glycolysis in the lens. In the diabetic animal, the enzymes responsible for normal glucose metabolism become saturated; therefore, the sorbitol pathway, in which the enzyme aldose reductase functions, metabolizes glucose.[8] Excessive sorbitol then accumulates in the lens, thereby increasing the osmotic state of the lens and causing subsequent imbibition of water.[3] Diabetic cataracts may develop acutely. Cataracts that occur secondary to diabetes mellitus often imbibe so much water that the lens swells and is referred to as intumescent.

Senile cataracts are common in humans and may also occur in dogs.[3] Distinguishing between inherited cataracts and senile cataracts in old dogs may be impossible. Traumatic cataracts are rare but may result from severe blunt trauma or penetrating injury.[9] If penetrating trauma to the globe causes a very small defect in the lens capsule, the defect may heal uneventfully with only focal cataract formation. If the defect in the lens capsule is large, however, lens proteins leak into the anterior chamber and incite lens-induced uveitis,[3] which is often intractable without phacofragmentation to remove the lens material.

In the past, dogs fed milk replacements as neonates often developed perinuclear cataracts. The cause of these cataracts is unknown but may be related to an arginine deficiency in the milk replacements. These possibly nutrition-related cataracts are rarely seen today because high-quality supplements are available.[3] Hypocalcemia, which can result from renal failure or hypoparathyroidism, causes cataracts with a very characteristic appearance of multiple, punctate cortical opacities.[3] Drugs, such as ketoconazole and dimethyl sulfoxide, can cause cataracts; however, drug-induced cataracts are uncommon.[3] Cataracts can also be seen as a long-term complication of radiation therapy of the head when eyes are included in the radiation field.[10]

Chronic anterior uveitis can lead to cataract formation by altering the aqueous humor, which subsequently affects lens nutrition.[11] The majority of uveitis-induced cataracts are inoperable because of inflammation-induced intraocular tissue changes, such as synechia, secondary glaucoma, and preiridial fibrovascular membranes. Cataracts often develop with

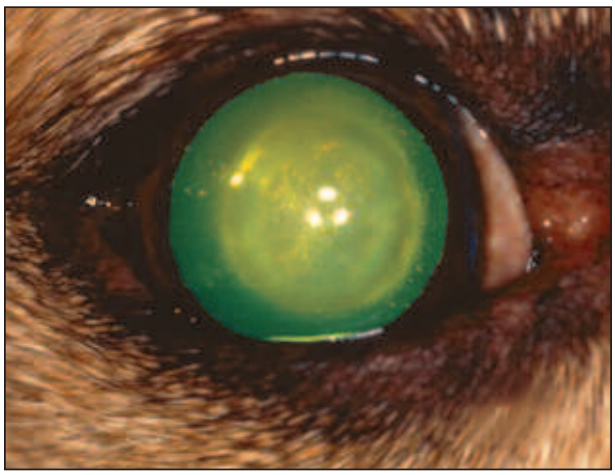

Figure 2—Moderate nuclear sclerosis in a 10-year-old bloodhound.

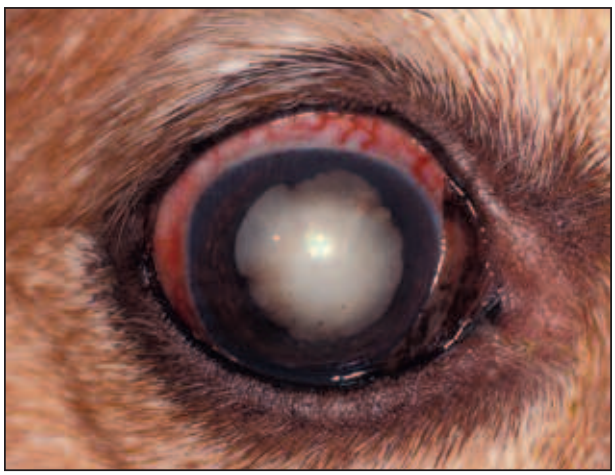

Figure 4—A diabetic cataract and secondary glaucoma in a cocker spaniel. The intraocular pressure was 38 mm Hg. Lesions present include episcleral injection, areas of posterior synechia, pigment dispersion on the anterior lens capsule, and a hypermature cataract. Aqueous flare was visible on slit-lamp biomicroscopy.

other chronic ocular diseases, such as glaucoma, progressive retinal atrophy (PRA), and lens luxation.[12]

The major diagnostic differential for cataracts in dogs is nuclear sclerosis (Figure 2). Nuclear sclerosis is a common aging change seen in dogs older than 7 years of age.[13] Nuclear sclerosis develops because the density of the lens increases throughout life as new lens fibers continue to form. Clinically, nuclear sclerosis appears as a gray, round, homogenous area in the nuclear region of the lens. Nuclear sclerosis does not interfere with examination of the fundus, whereas the fundus may be difficult or impossible to evaluate when a cataract is present. Nuclear sclerosis does not require surgery and

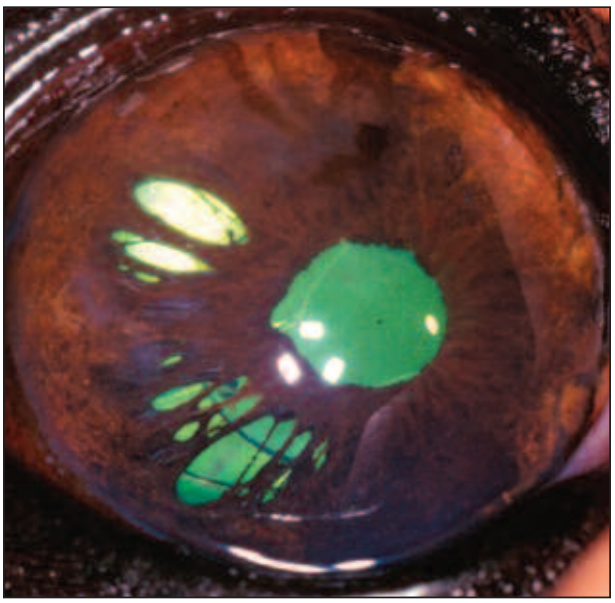

Figure 3—Iris atrophy in a 9-year-old, spayed female Boston terrier. The temporal iris is severely atrophic, and there are full-thickness holes, resulting in a moth-eaten appearance. The pupillary margin is irregular. This patient had cataract surgery, and the edge of the prosthetic lens is visible through the holes in the iris.

usually does not interfere with vision unless it is very advanced, as seen in geriatric dogs (older than 15 years of age).[14] A combination of nuclear sclerosis and cataract is quite common.

OPHTHALMIC EXAMINATION OF CATARACT PATIENTS

Cataracts may be noticed either by an astute owner or by a veterinarian during a routine health examination. Once cataracts are discovered, the veterinarian should do a complete ophthalmic examination. A veterinary ophthalmologist will also perform complete examination when a patient is referred for cataract evaluation. This section reviews the ophthalmic examination and discusses possible findings that may postpone or preclude cataract surgery. A complete ophthalmic examination includes evaluation of pupillary light and menace responses, Schirmer's tear testing, measurement of intraocular pressure, slit-lamp biomicroscopy or light examination of the anterior segment, and indirect or direct ophthalmoscopy after instillation of a mydriatic.

Ocular Reflexes

Pupillary light reflexes should be brisk and complete with any stage of cataract. Although cataracts appear opaque, they do not interfere significantly with light

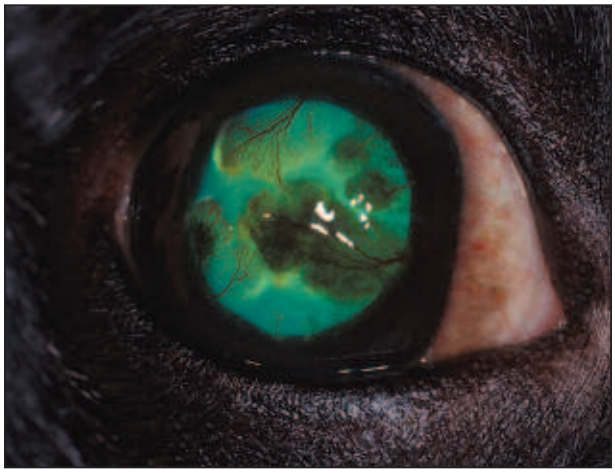

Figure 5—Pigmentary keratitis and neovascularization in a 7-year-old pug. This patient is not a candidate for cataract surgery because of severe corneal changes.

Figure 6—Corneal degeneration in a 5-year-old rottweiler. Corneal neovascularization and mineralization are present.

rays reaching the retina. If the pupils are mydriatic at rest but constrict with light stimulation, early retinal degeneration caused by PRA may be present. If the pupil is mydriatic and nonresponsive to light stimulation, the dog may have end-stage retinal degeneration, iris atrophy, glaucoma, or retinal detachment. A miotic pupil that resists dilation with tropicamide indicates anterior uveitis. Pupils that are fixed or abnormally shaped indicate posterior synechia. Posterior synechia is pathognomonic for either previous or current anterior uveitis. Surgery may not be possible if the synechiae are extensive. Extensive iris atrophy may give the pupil a moth-eaten appearance and cause a sluggish pupillary light response (Figure 3). Iris atrophy does not preclude surgery, however. The menace response, whether present or absent, is not a prognostic indicator in itself.

Ophthalmic Diagnostic Tests

A Schirmer's tear test should always be done before cataract surgery. The normal tear production rate is 15 to 25 mm/min.[15] A rate below 10 mm/min with concurrent conjunctivitis, corneal pigmentation, neovascularization, and scarring indicates the presence of keratoconjunctivitis sicca, a syndrome of decreased tear production. Surgery may still be possible when tear production is low if production can be increased by using topical cyclosporine and if the cornea is not scarred.[15] A borderline tear production rate (12 to 14 mm/min) should be rechecked just before surgery. When borderline tear production exists, the ophthalmologist may elect to use cyclosporine before surgery to increase tear production to a normal level or may elect to treat the dog with artificial tear ointments in the immediate postoperative period because anesthesia may cause a decrease in tear production for the first 24 hours after surgery.[16] Low tear production makes the corneal epithelium unhealthy and prone to ulceration, which can lead to serious complications in the postoperative period. Postoperative corneal ulceration increases morbidity and prevents the use of topical corticosteroids that are needed to reduce surgically induced inflammation.

Abnormal intraocular pressures can indicate subtle disease processes. A low intraocular pressure usually indicates anterior uveitis. High pressures always indicate glaucoma. Although dogs with chronic glaucoma often develop cataracts, owners should be made aware that these dogs are not surgical candidates because of retinal and optic nerve damage caused by elevated intraocular pressure (Figure 4). Dogs may present with bilateral cataracts and unilateral primary glaucoma. We occasionally perform cataract surgery on the nonglaucomatous eye of these dogs. However, we warn owners that the operated eye will most likely develop glaucoma in the future.

Light Examination of the Eye

Adnexal disease is rarely a problem that needs to be addressed in a cataract evaluation. Adnexal disease that requires therapy is usually diagnosed as a separate entity because of the pain or conjunctival hyperemia that it causes. The cornea should be examined for pigmentation, vascularization, and other opacities. Pigmentary keratitis is usually the result of chronic irritation from keratoconjunctivitis sicca, trichiasis, or chronic exposure from macropalpebral fissure or anatomic exophthalmos. Breeds predisposed to pigmentary keratitis include the Boston terrier, Lhasa apso, miniature poodle, Pekingese, pug, and shih tzu[17] (Figure 5). Even rel-

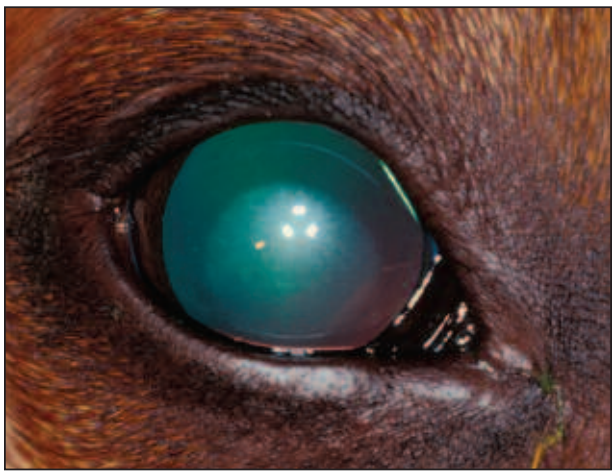

Figure 7—Corneal dystrophy in a dog. A dense, white, circular opacity is present in the axial cornea. This condition was bilaterally symmetric.

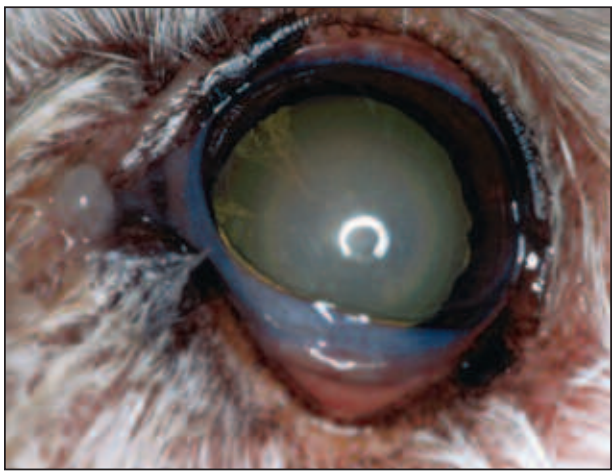

Figure 9—Nuclear sclerosis and an incipient cataract in a 15-year-old mixed-breed dog. The nuclear sclerosis is seen as round, increased lucency in the nuclear region of the lens. Severe nuclear sclerosis is present in the central lens and has the appearance of a pearl. Incipient cataracts can be seen as spokes at the 9 and 11 o'clock positions on the lens.

atively minor corneal pigmentation can prevent visualization of the lens at surgery, which may either result in a poor prognosis after surgery or make the surgery impossible.

Stromal corneal dystrophy or corneal degeneration imparts a white, glittering appearance to the cornea and may limit visualization of the intraocular structures at surgery (Figure 6). The Alaskan malamute, Siberian husky, and bichon frise are a few of the breeds that may develop corneal dystrophy and cataracts (Figure 7). The dachshund, Boston terrier, and Chihuahua develop a specific syndrome, known as corneal endothelial dystrophy, that causes corneal edema[17] (Figure 8). The corneal edema typically originates in the area of the lateral limbus and gradually advances medially to involve the entire cornea. Surgery is generally not recommended for dogs with endothelial dystrophy because the remaining functional endothelium may decompensate with surgery, resulting in an increase in the severity of the corneal edema and corneal ulceration. Any cloudiness or pigmentation of the cornea is greatly intensified under the operating microscope and can impede visualization of the lens at surgery.

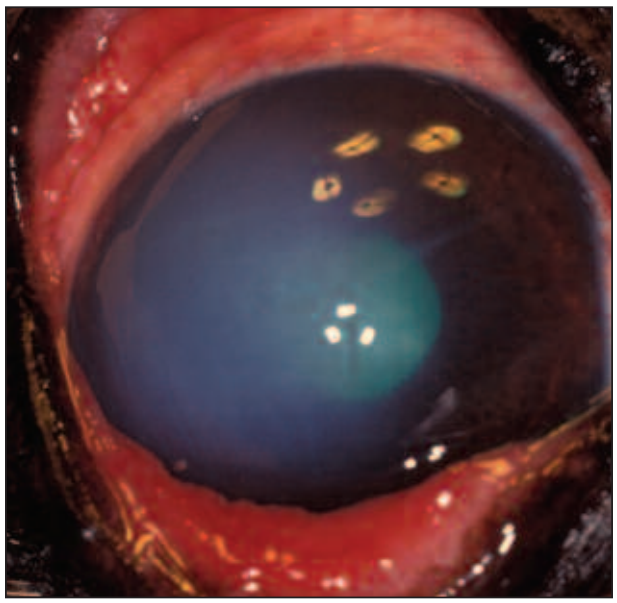

Figure 8—Endothelial dystrophy in a 10-year-old Boston terrier. The temporal aspect of the cornea is diffusely edematous.

The anterior chamber should be evaluated for the presence of aqueous flare. This evaluation can be done by using the small round light or slit beam on the direct ophthalmoscope or a slit-lamp biomicroscope. Lens-induced uveitis is the most likely cause of any aqueous flare. The anterior uveitis must be treated with a topical corticosteroid, such as prednisolone acetate and tropicamide or atropine, so that it is controlled before surgery.

Appearance of Cataracts

Complete evaluation of the lens can be done only after mydriasis is induced with tropicamide or another dilating agent. Staging of the cataract development is necessary to determine the prognosis as well as the necessity for and possibility of doing surgery. The lens cortex, nucleus, and capsule should be evaluated before surgery. Cataract maturity is divided into four categories: incipient, immature, mature, and hypermature.[3] The appearance of a cataract can vary depending on the

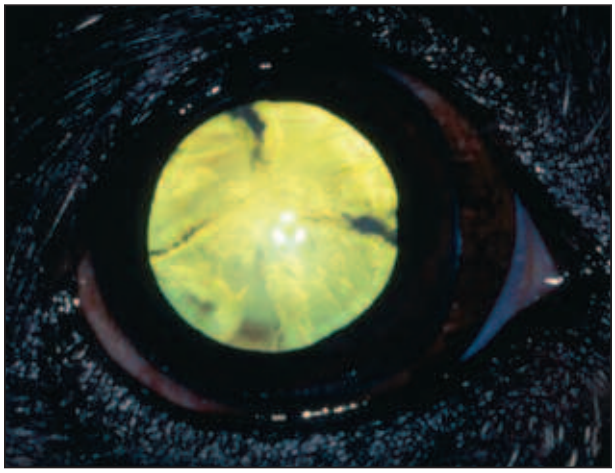

Figure 10—An immature cataract in a Boston terrier. There is only slight attenuation of the tapetal reflex, and several spokes can be visualized within the cataract.

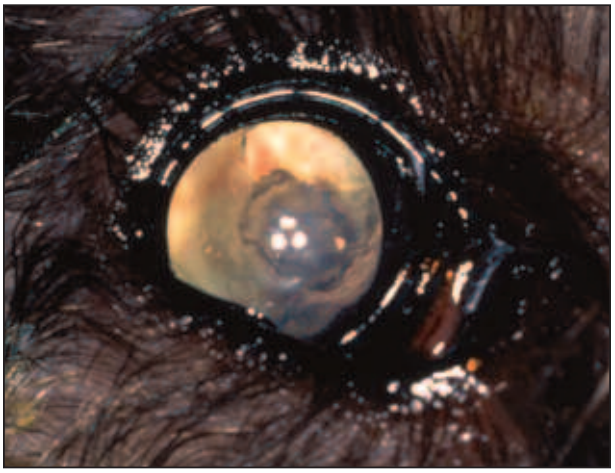

Figure 11—An immature cataract in a 6-month-old miniature schnauzer. A portion of the cataract is denser centrally. This lens had lenticonus, which is a posterior outpouching of the lens. Lenticonus is an inherited condition in miniature schnauzers.

light source and the technique of examination. Compared with a weaker light source (e.g., a penlight), a bright light, such as that from a transilluminator, reveals much more detail and helps with distinguishing a cataract from nuclear sclerosis. A slit-lamp biomicroscope used by ophthalmologists allows acquisition of the greatest amount of detail with regard to maturity and location of the cataract within the lens. With any light source, directly illuminated cataractous areas appear white or gray. If the light goes through the lens and reflects off the tapetum, the cataractous areas may appear dark because they are illuminated from behind (retroillumination).

Incipient cataracts involve only a small portion of the lens and appear as focal white areas in the lens (Figure 9). The tapetal reflection is almost complete, vision is normal, and the entire retina can be visualized via the ophthalmoscope because the cataract is so small that it does not impede examination of the fundus. These cataracts may be static or progressive. Surgery is not indicated at this stage.

Immature cataracts have the greatest degree of variability in appearance. They can range from involving a small portion of the lens to completely preventing fundic evaluation and causing severe vision impairment. Most immature cataracts have a spoke-like appearance, with the density and number of spokes varying (Figures 10 and 11). By definition, a tapetal reflection can always be seen with an immature cataract, but vision may be present or absent. Many immature cataracts progress to vision loss; however, some cataracts, such as the triangular cataracts of golden retrievers and Labrador retrievers, are often nonprogressive[3] (Figure 12). Immature cataracts that are advanced or progressing are at the ideal stage for removal by phacoemulsification.

Mature cataracts involve the entire lens. There is no tapetal reflection, the eye is nonvisual, and the retina cannot be evaluated ophthalmoscopically (Figure 13). The lenses of these dogs have the appearance of white marble. Before the common use of phacoemulsification for cataract removal in dogs, the ideal stage for removal was a mature cataract.[18] With current surgical techniques and ocular medications, however, the prognosis for long-term vision is quite good with earlier removal of cataractous lenses, and the immature stage is now considered ideal.

Hypermature cataracts are cataracts that have begun to liquefy. Lens proteins may leak through the lens capsule and cause anterior uveitis at this stage.[19] The anterior uveitis may be subclinical with no aqueous flare evident, but examination may show that the intraocular pressure is lower than normal and the pupil is resistant to dilation with tropicamide. The appearance of hypermature cataracts varies greatly, according to the degree of liquefaction. These lenses often have a focal or diffuse glittering appearance. The lens capsule may be wrinkled and have the appearance of shrink-wrapping (Figure 14). The anterior chamber may be deeper than normal if significant liquefaction has occurred. In the end-stage hypermature cataract, known as a morgagnian cataract, the cortex has completely liquefied and the lens nucleus has settled ventrally in the lens capsule.

Conflicting reports have appeared as to whether cataract surgery for hypermature cataracts is associated with a poorer prognosis than surgery for immature or

Figure 12—An immature cataract in a Labrador retriever. This triangular cataract is in the posterior aspect of the lens and is characteristic of the inherited cataracts in this breed.

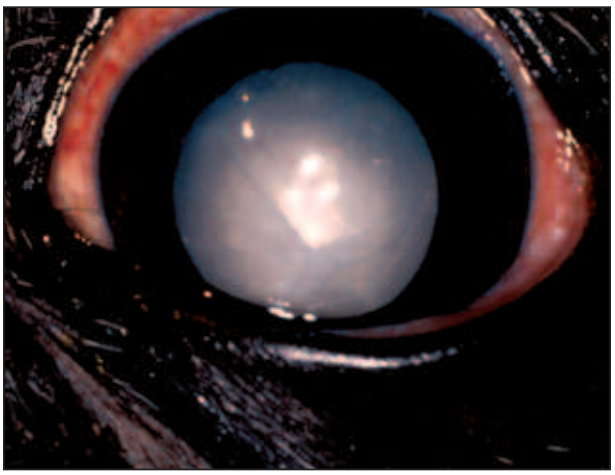

Figure 13—A mature cataract in a shih tzu. The entire lens is opaque, and no tapetal reflection can be visualized through the cataract. The dog was blind.

mature cataracts.[20,21] Certainly, many dogs with hypermature cataracts have lens-induced uveitis, which must be controlled before surgery. Topical antiinflammatory and mydriatic therapy is used to treat and control the uveitis before phacoemulsification.

Chronically luxated lenses may become cataractous[3] (Figure 15). However, it may not be feasible to remove these lenses because the lens may have adhered to the corneal endothelium or because retinal or optic nerve damage from secondary glaucoma may have occurred. The decision to remove luxated lenses must be made on a case-by-case basis. Removal of a cataractous subluxated lens is more difficult than routine cataract surgery and is, therefore, inherently associated with a poorer prognosis than is the uncomplicated cataract, but removal via phacoemulsification or intracapsular procedures may still be attempted if no other abnormalities exist.

Fundic Examination

Fundic examination is quite important in cataract patients. Dogs presented for cataract evaluation frequently have severe vision deficits. The veterinarian or veterinary ophthalmologist should try to determine whether the visual deficits correlate with the kind of cataract present. If the retina can easily be evaluated through a cataract, the dog should be able to see. When visual deficits do not correlate with the degree of cataract development, the veterinary ophthalmologist should suspect retinal degeneration. The most common cause of retinal degeneration in dogs is PRA. Many breeds known to carry the genes for inherited cataracts also carry PRA genes.[22] The earliest signs of retinal degeneration are attenuation of the peripheral retinal vessels and peripheral tapetal hyperreflectivity. Later, generalized tapetal hyperreflectivity, severe vessel attenuation, and optic nerve atrophy occur[23] (Figure 16). Rarely, retinal detachment is visualized in a dog with cataracts (Figure 17). Such eyes are not candidates for cataract surgery. When visual evaluation of the retina is not possible, electroretinography (ERG) and ocular ultrasonography can help determine the function and position of the retina.

SYSTEMIC AND DIAGNOSTIC EVALUATION FOR CATARACT SURGERY

Patients evaluated for cataract surgery should have a complete physical examination. Complete blood cell counts, serum biochemistry profiles, and urinalyses are usually done. Systemic diseases should be evaluated, diagnosed, and treated before surgery. Examination of the urine sediment may reveal occult bacterial cystitis. If so, the patient should be treated appropriately and the cystitis resolved before surgery. In our experience, occult cystitis is more common in diabetic dogs than in nondiabetic dogs. Ideally, dogs with diabetes should have the disorder controlled before cataract surgery, with the blood glucose concentration maintained between 100 and 200 mg/dl.[24] This regulation is necessary because the stress associated with hospitalization and surgery and the perioperative use of topical steroids will likely cause the blood glucose level to rise above 200 mg/dl during the perioperative period. However, dogs with Cushing's disease and diabetes mellitus commonly undergo cataract surgery without complications. In humans, severe dental disease with gingivitis can cause bacteremia, leading to endophthalmitis.[25] We recommend that patients with gingivitis and moderate to severe dental tartar have routine dental prophylaxis per-

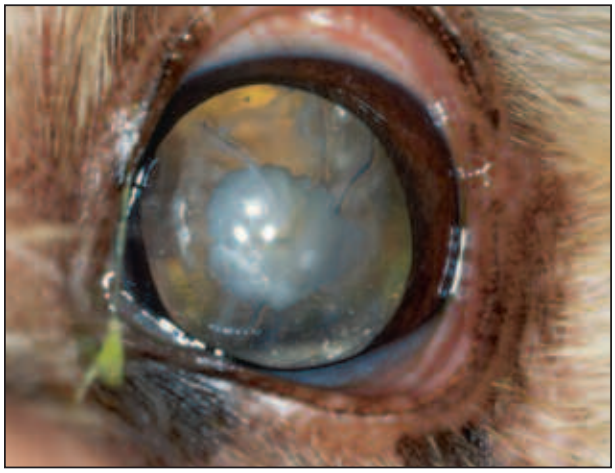

Figure 14—A hypermature cataract in a cocker spaniel. There has been some resorption of the cortex, which allowed a portion of the tapetal reflex peripheral to the nucleus to be observed. Note the glittering appearance of the lens and the wrinkles in the lens capsule.

Figure 15—An anterior luxated cataract in a cocker spaniel. An aphakic crescent can be seen dorsal to the lens. A tapetal reflex accentuates the aphakic crescent. This lens was removed by intracapsular lens extraction. Note the lid masses on the dorsal and ventral eyelids.

formed by the referring veterinarian at least 4 weeks before cataract surgery. Any patient with cardiac disease should be evaluated, and the risk associated with the use of anesthesia should be determined before surgery.

A patient deemed to be a surgical candidate on the basis of the physical examination should undergo ERG and ocular B-scan ultrasonography. ERG allows evaluation of a retina that cannot be visualized. This tool measures electrical activity of the retina and evaluates general retinal function.[26] Patients with PRA will have smaller ERG wave amplitudes than normal dogs. Ocular ultrasonography is used to detect retinal detachments that may not be visible or that are too small or acute to be detected by ERG.[27] We occasionally have patients with acceptable ERG results but with retinal detachments detected ultrasonographically. Patients with retinal detachments or PRA will not benefit from cataract surgery over the long term. Some clients elect cataract surgery for their dog even when there are suboptimal ERG results because they desire the dog to have vision even if it is only temporary.

PREOPERATIVE THERAPY

Once it is determined that surgery will be performed, medical therapy may be initiated. Many preoperative treatment regimens exist, and these vary depending on the personal preferences of the ophthalmologist. The goals of treatment before cataract surgery are to decrease the amount of intraoperative and postoperative intraocular inflammation, decrease the conjunctival bacterial flora, dilate the pupil, and prevent miosis during surgery. Use of topical corticosteroids may be started days to hours before surgery. The duration of corticosteroid treatment depends on the presence of lens-induced uveitis caused by hypermature cataracts and the surgeon's preference. Application of topical NSAIDs is usually initiated 24 hours before surgery. These drugs prevent intraoperative miosis by decreasing prostaglandin formation in the eye.[18] Tropicamide, phenylephrine, and atropine are used in various combinations and time periods before surgery to dilate the pupil so that the lens can be visualized during the operation. Treatment with a broad-spectrum topical antibiotic may be initiated the day before or morning of surgery. IV flunixin meglumine is commonly administered at the time of anesthesia induction, and an antibiotic such as cefazolin is usually given perioperatively.

PHACOEMULSIFICATION

Phacoemulsification (phacofragmentation) is by far the most commonly used method of cataract removal in dogs. This method is the same one used in human cataract surgery today. This technique requires an operating microscope, a phacoemulsification system, and microsurgical instruments. Paralysis of the patient prevents ocular movement and reduces vitreal pressure during the procedure, and it is typically induced with an IV injection of atracurium. This medication causes respiratory paralysis so that mechanical or manual ventilation of the patient is required.[18]

To begin surgery, the cornea, limbus, or sclera is incised. Injection of viscoelastic material, a clear viscous substance, into the anterior chamber maintains the anterior chamber depth and improves visualization of

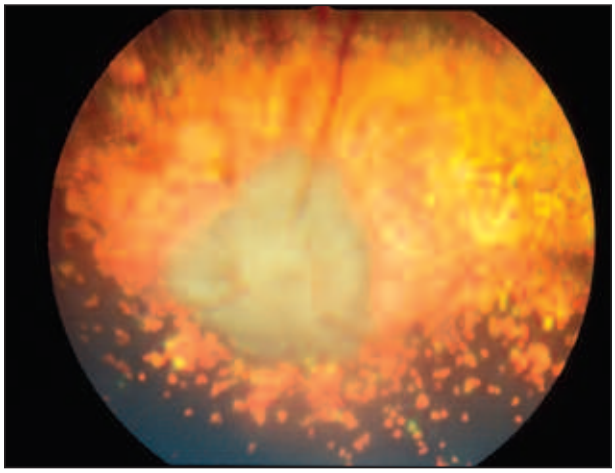

Figure 16—Severe retinal degeneration (progressive retinal atrophy) in an 8-year-old castrated male miniature poodle. There is severe retinal vascular attenuation, optic nerve atrophy, and diffuse tapetal hyperreflectivity. This patient is not a candidate for cataract surgery.

the intraocular contents.[18] A capsulorrhexis is then done to remove a round portion of the anterior lens capsule, which allows access to the cataractous lens material. Ultrasonic energy delivery, irrigation, and aspiration are all achieved through the phacoemulsification handpiece. The ultrasonic energy breaks up the cataract, which is aspirated from the eye.

A prosthetic intraocular lens (IOL) that is placed in the remaining lens capsule after all lens material has been removed helps restore the emmetropic state (no refractive error; Figure 18). Rigid IOLs made of polymethylmethacrylate are currently the most commonly used lenses in veterinary medicine. Foldable IOLs are acrylic and have recently been introduced for use in dogs.[18,28] The primary advantage of foldable lenses is a smaller corneal incision (4 versus 8 mm) and shorter surgical time. The type of IOL used depends on the surgeon's preference. Dogs that do not have an IOL implanted are hypermetropic (farsighted) because they are aphakic (without a lens), and most dogs with IOLs are close to emmetropia.[29] After the IOL is placed in the eye, the incision is sutured.

POSTOPERATIVE THERAPY

An Elizabethan collar is used for 3 weeks postoperatively to prevent trauma to the eyes. Activity is limited, and a harness is used on dogs that are not well leash trained. After surgery, mild anterior uveitis, as indicated by aqueous flare and miosis, is usually present and

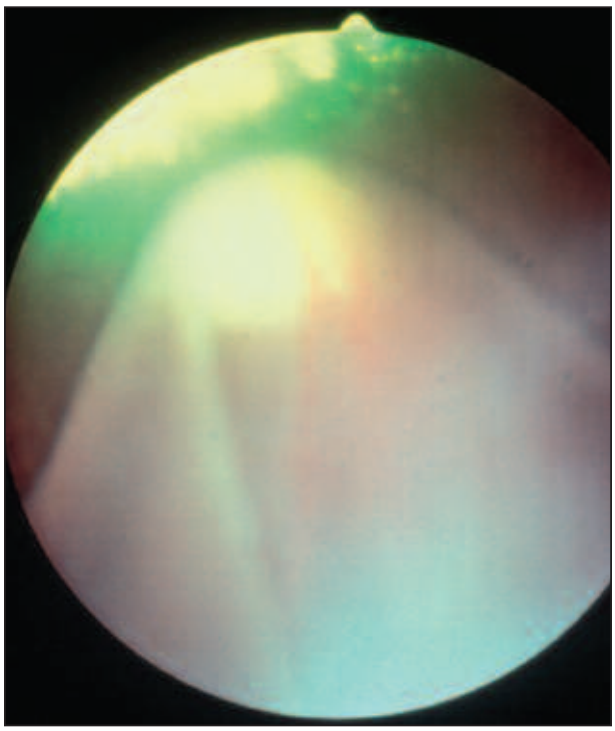

Figure 17—Complete retinal detachment in a dog. The dorsal retina is torn and has fallen ventrally, completely covering the optic nerve. A small amount of hyperreflective tapetum is visible.

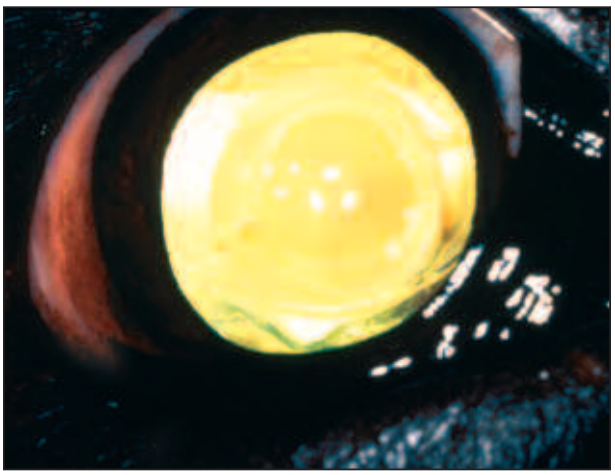

Figure 18—An IOL in a dog. The IOL implant is easily visualized. The central area of the implant is the optic, which is smaller than the normal lens of the dog. The capsulotomy site in the anterior lens capsule can be visualized as the small circle on the surface of the IOL. This patient had an excellent postoperative outcome.

requires treatment with topical corticosteroids or NSAIDs and a mydriatic. Topical and systemic antibiotics are also usually administered after surgery for 2 to 4 months, using a tapering dose, but treatment may be prolonged if the anterior uveitis lingers. This problem appears to be more common in the miniature schnauzer and Boston terrier for unknown reasons. We treat most postoperative cataract patients topically every 6 hours initially and then reduce the frequency of topical medications over the next few weeks as the severity of the surgically induced uveitis subsides. Several visits to check the eyes are commonly scheduled with the ophthalmologist within the first 2 months after surgery. Thereafter, appointments may be scheduled every 6 months to annually.

COMPLICATIONS OF CATARACT SURGERY

Immediately after surgery, some degree of anterior uveitis always occurs. Surgical trauma and exposure of the eye to lens proteins cause anterior uveitis. Treatment with corticosteroids, topical NSAIDs, or mydriatics is dictated by the degree of inflammation. Ocular hypertension, an elevation of intraocular pressure within 6 to 8 hours after surgery, is differentiated from glaucoma by its transient nature and specific cause. The increase in intraocular pressure is due to blockage of the iridocorneal angle by viscoelastic, cellular, and lenticular debris; inflammatory cells; and swelling of the cells in the trabecular meshwork.[30] It is impossible to predict which dogs will develop ocular hypertension postoperatively. Use of a miotic agent can reduce the risk of ocular hypertension by pulling the iris out of the iridocorneal angle, opening the trabecular meshwork, and increasing aqueous humor outflow. Carbachol can be injected intraocularly during surgery to reduce the risk of postoperative hypertension.[31]

Another very frustrating complication that may occur after surgery is corneal ulceration.[32] Ulcers may develop as a result of anesthesia-induced reduction in tear production, reduced healing rates in older patients, and self-trauma. Liberal application of topical lubricants and use of an Elizabethan collar may reduce the risk of postoperative corneal ulceration. These corneal ulcers tend to heal much more slowly than typical traumatic ulcers.

The more common long-term postoperative complications include posterior capsular opacification, persistent uveitis, endothelial degeneration, glaucoma, and retinal detachments.[32] Mild posterior capsular opacification is common in dogs after cataract surgery but rarely causes any noticeable visual deficits. Posterior capsular opacification appears clinically as a haziness or whitening of the posterior lens capsule. This complication begins weeks to months after surgery. The opacification occurs secondary to metaplasia of remaining lens epithelial cells that have migrated posteriorly after surgery and that may produce abnormal lens proteins.

Unfortunately, posterior capsular opacification in dogs is not as amenable to laser surgery as it is in humans; therefore, therapy is generally not attempted unless vision is severely impaired. Use of an IOL reduces the incidence of posterior capsular opacification.[18] Young dogs undergoing cataract surgery are more likely than older dogs to develop this complication. The surgeon may elect to perform a posterior capsulotomy before placing an IOL in young dogs to maintain a clear visual axis and reduce the likelihood that posterior capsular opacification will develop.[18,32]

Some dogs, especially miniature schnauzers and Boston terriers, have persistent uveitis after surgery. This disorder may require the continued use of topical steroids or NSAIDs. Cataract surgery disrupts the blood–aqueous barrier, which in some dogs is not reestablished quickly. Endothelial degeneration most likely occurs secondary to loss or damage of endothelial cells from the trauma of surgery or from inflammation. When endothelial degeneration is severe, it can lead to decreased vision or corneal ulceration.

Glaucoma can develop at any time after cataract surgery; in one study, 28.8% of the dogs that had a follow-up evaluation had glaucoma 12 months postoperatively.[33] The presence of hypermature cataracts, a long phacoemulsification time, and failure to place an IOL were thought to contribute to the development of postoperative glaucoma.[33]

Retinal detachments may occur any time after cataract surgery. The definitive cause of these detachments is unknown; however, they may be due to inflammation or postoperative architectural changes in the eye. Unfortunately, such retinal detachments are generally rhegmatogenous and, therefore, require surgical intervention for repair. Rhegmatogenous detachments occur when a tear develops in the retina. Retinal detachment surgery is in its infancy in veterinary medicine, and most dogs that develop a detachment after cataract surgery will be blind.

PROGNOSIS

The prognosis for a good visual outcome after cataract surgery depends primarily on appropriate selection of patients and compliance with postoperative treatments and follow-up visits by the owner. The short-term success rate for a visual outcome is about 95%.[21,34] Although long-term success rates have not been published, they are probably about 80%.

CONCLUSION

Cataracts are a common cause of blindness in dogs. A successful outcome after cataract surgery depends on the appropriate selection of patients (e.g., by ruling out diseases such as PRA). A significant commitment is required by the owner, especially in the immediate postoperative period, to administer multiple medications to the patient. By becoming familiar with the pre- and postoperative treatment regimen, referring veterinarians can better educate clients who may be considering cataract surgery for their dogs. Also, by performing complete physical and ophthalmic examinations of cataract patients, referring veterinarians may uncover other diseases that need treatment before cataract surgery or may determine that a patient is not a candidate for this surgery. Early referral of patients with an immature cataract gives the best prognosis for a successful surgical outcome.

EDITOR'S NOTE

To find a boarded veterinary ophthalmologist in your area, visit the American College of Veterinary Ophthalmologists Web site at **www.acvo.com.**

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ARTICLE #1 CE TEST

The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. *Choose the best answer* to each of the following questions; then mark your answers on the postage-paid envelope inserted in *Compendium*.

1. The most common cause of cataracts in dogs is
   a. anterior uveitis.
   b. inheritance (genetic).
   c. toxins.
   d. nutritional deficits.
   e. trauma.

2. Congenital cataracts with an inherited basis occur most commonly in which breed?
   a. miniature poodle
   b. miniature schnauzer
   c. cocker spaniel
   d. Boston terrier
   e. Yorkshire terrier

3. Nuclear sclerosis
   a. impairs vision.
   b. eventually requires surgery.
   c. is a normal aging-related change.
   d. blocks visualization of the fundus.
   e. is easy to differentiate from a cataract in a nondilated eye.

4. A complete ophthalmic examination for evaluating cataracts includes
   a. the Schirmer's tear test.
   b. measurement of intraocular pressure.
   c. slit-lamp biomicroscopy.
   d. fundic examination.
   e. all of the above

5. Dogs are not candidates for cataract surgery if they have
   a. glaucoma.
   b. retinal detachment.
   c. chronic uveitis.
   d. pigmentary keratitis.
   e. all of the above

6. _____ cataract has the best prognosis for successful surgery.
   a. A hypermature
   b. An immature
   c. An intumescent
   d. A mature
   e. A subluxated

7. Which characteristic is not an indicator of poor prognosis after cataract surgery?
   a. lack of normal pupillary light responses
   b. lack of a normal menace response
   c. an intraocular pressure of 45 mm Hg

d. a Schirmer's tear test result of 5 mm/min
e. corneal edema

8. Funduscopic abnormalities indicative of PRA do not include
   a. tapetal hyporeflectivity.
   b. tapetal hyperreflectivity.
   c. optic nerve atrophy.
   d. retinal vascular attenuation.
   e. peripheral streaking in the tapetum.

9. Phacoemulsification, the most common technique for cataract removal in dogs, uses which type of energy to break up the lens?
   a. laser
   b. ultrasonic
   c. x-ray
   d. ultraviolet
   e. hydraulic

10. Which type of cataract by definition will not have a tapetal reflection?
    a. incipient
    b. immature
    c. mature
    d. hypermature
    e. morgagnian