Development of glaucoma after phacoemulsification for removal of cataracts in dogs: 22 cases (1987–1997)

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Objective—To determine factors contributing to glaucoma after lens extraction via phacoemulsification in dogs.

Design—Retrospective study.

Animals—22 dogs (29 eyes) with glaucoma and 21 dogs (30 eyes) without glaucoma after phacoemulsification.

Procedure—Medical record review.

Results—Eyes at increased risk for glaucoma included those of Boston Terriers, those with uveal or retinal abnormalities before surgery, and those with intraoperative intraocular hemorrhage. Significant differences between groups were not detected for incidence of preoperative lens-induced uveitis, presence of an intraocular lens, or frequency of an acute postoperative increase in intraocular pressure. Glaucoma developed (mean ± SD) 12.8 ± 14.1 months (median, 10 months; range, 0.25 to 55 months) after surgery. Eighteen of 29 (62%) eyes with potential for vision after onset of glaucoma retained vision for a mean of 16.5 ± 12.8 months (median, 10.8 months; range, 1.5 to 37 months) after glaucoma was diagnosed. Most of these eyes still had vision at the conclusion of the study period.

Conclusions and Clinical Relevance—Risk factors identified by this study will aid in preoperative counseling of clients and refining selection criteria for candidates for phacoemulsification. Careful follow-up for the remainder of the dog's life after surgery may improve long-term success rates by permitting early intervention before intraocular pressure increases substantially and vision is irreversibly lost. Surgery for cataracts may still be worthwhile in dogs with increased risk of glaucoma, especially if elderly, because of the lengthy period to onset of glaucoma after surgery and the beneficial effects of treatment after glaucoma develops. (*J Am Vet Med Assoc* 2001; 218:70–76)

Recent improvements in surgical instrumentation and technique have substantially refined the mechanics of cataract surgery in dogs.[1-5] Although many veterinary ophthalmologists believe these technologic improvements have also increased their success rate at returning vision to cataractous eyes during the long term, the development of glaucoma months to years after extraction of the lens remains a common problem and is one of the major impediments to long-term success in maintaining vision in these patients.[1,4,6-8] This complication is particularly disappointing because, unlike vision impairment attributable to cataracts, blindness attributable to glaucoma is irreversible, and the eye requires additional medical or surgical intervention to control ocular pain caused by the increased **intraocular pressure (IOP)**.[9]

Although it has been suggested that dogs that experience a sudden increase in IOP during the first 24 hours after surgery may be at increased risk of glaucoma in the late postoperative period,[10] it is likely that many other factors are also involved in the pathogenesis of this problem. A better understanding of which dogs will develop glaucoma after extraction of the lens may permit development of a more effective strategy for the prevention of glaucoma in the late postoperative period and may further improve the success rate of surgery for cataracts in dogs. The purpose of the study reported here was to determine features contributing to glaucoma after extraction of the lens via phacoemulsification in dogs, compared with control dogs that did not develop glaucoma.

Criteria for Selection of Cases

A computerized search was performed of all medical records of dogs that underwent extraction of a lens via phacoemulsification from April 1, 1987, to March 1, 1997, at the University of Wisconsin Veterinary Medical Teaching Hospital. Dogs were considered eligible for inclusion in the study if the operated eye never had IOP > 25 mm Hg before surgery, as measured by applanation tonometry,[a] and had no other evidence of glaucoma. A dog was included in the glaucoma group if IOP exceeded 25 mm Hg for longer than 7 days after extraction of the lens via phacoemulsification, and treatment for glaucoma was required. A brief acute increase in IOP within the first 24 hours after surgery was considered to represent ocular hypertension and, alone, was insufficient for the diagnosis of glaucoma. Dogs were included in the control group if they had no evidence of glaucoma, IOP never exceeded 25 mm Hg > 7 days after surgery, they had ≥ 2 years of follow-up examinations performed by an ophthalmologist, and they had not received any antiglaucoma drugs for at least the preceding 12 months. Dogs with cataracts secondary to chronic anterior uveitis were excluded from both groups. Additionally, dogs were excluded if lens-induced uveitis required > 1.5 months of anti-inflammatory treatment prior to surgery to be brought under control, the eye had broad-based poste-

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rior synechiation that extended > 1 clock hour (30°), or preiridal fibrovascular membranes were detected prior to surgery.

Procedures

If the retina was not directly visible prior to surgery, B-scan ocular ultrasonography and bright-flash electroretinography were performed to rule out substantial retinal abnormalities. Lens extraction via phacoemulsification was then performed by an ophthalmologist, by use of a described technique,[2] when there was no evidence of active lens-induced uveitis and any systemic medical abnormalities such as diabetes mellitus were adequately controlled. Implantation of an intraocular lens was in accordance with the owner's wishes and the judgment of the surgeon at the time of surgery. When necessary, dogs in both groups were treated for sudden postoperative increases in IOP during the first 24 hours after surgery, and in a few instances this treatment was continued on a prophylactic basis for several months after surgery. In all dogs in the control group, however, these drugs were ultimately withdrawn, and their discontinuation did not result in an increase in IOP to > 25 mm Hg or any other evidence of glaucoma during at least 20 additional months of follow-up.

Medical records of eligible dogs were reviewed, and the specific preoperative data abstracted included breed, sex, eye affected, age of the cataract (congenital, juvenile, adult, senile), duration of the cataract after detection by the owner or referring veterinarian, stage of cataract (incipient, incomplete, complete, resorbing),[11] cause of the cataract, age at the time of surgery, duration and treatment for lens-induced uveitis if present, and other abnormalities of the eyelids, conjunctiva, cornea, drainage angle,[12] uvea, vitreous, or retina. It was also noted whether IOP increased ≥ 5 mm Hg after dilation of the pupil, compared with values obtained before dilation, or to > 25 mm Hg. The type of medications used on the day of surgery, development and nature of any intraoperative complications, placement of an **intraocular lens (IOL)**, and phacoemulsification time and power were also recorded. Postoperative variables included development of an increase in IOP to > 25 mm Hg during the first 24 hours after surgery, duration of the first episode of uveitis after extraction of the lens, number of episodes of uveitis (until the onset of glaucoma or the last follow-up visit), the nature, route, and duration of administration of drugs used to treat uveitis, and the nature of abnormalities of other ocular tissues. For the purposes of this study, clinically meaningful active anterior uveitis was considered to be absent if aqueous flare was not detectable by use of slit-lamp biomicroscopy and few or no cells were detected in the aqueous humor. The time to the onset of glaucoma, type of antiglaucoma drugs used, and number of months of vision after surgery and onset of glaucoma were also recorded. An eye was determined to be blind if a menace response was not detected or a dazzle reflex to bright light was detected, but other indicators of useful vision were not detected.

Differences in categoric variables between the 2 groups were compared by use of a 2-tailed Fisher exact test. Linear variables (age, duration of cataract, duration of uveitis before and after surgery, phacoemulsification time and power level) were compared by use of a Student *t*-test. All analyses were performed by use of a computerized statistical software package,[b] and significance was set at $P < 0.05$.

Results

One hundred fifty-four dogs underwent phacoemulsification for extraction of a lens in at least 1 eye during the study period; many of these dogs had bilateral surgery. Of these, 22 dogs (29 eyes) and 21 dogs (30 eyes) were eligible for the glaucoma and control groups, respectively. After glaucoma developed, follow-up was maintained until vision loss was complete, the dog was lost to follow-up or died, or the study follow-up period ended (April 1, 1999). Mean (± SD) duration of follow-up for operated eyes in the control group (41.7 ± 12.2 months; range, 26 to 86 months) was significantly ($P < 0.05$) longer than that in the glaucoma group (31.7 ± 22.9 months; range, 2 to 81 months). Mean time to onset of glaucoma was 12.8 ± 14.1 months (median, 10 months; range, 1 week to 55 months).

Boston Terriers were at significantly ($P = 0.042$) greater risk of developing glaucoma after surgery when the data were analyzed by eye (**Table 1**), but not when the data was analyzed by dog ($P = 0.185$).

In general, dogs in both groups received prophylactic treatment for impending glaucoma by the attending clinician when they had IOP > 20 mm Hg and substantial postoperative uveitis or goniodysgenesis before surgery, glaucoma in the opposite eye after surgery, or progressive development of peripheral anterior synechia. Prophylactic treatment was also begun if IOP increased progressively, as judged by results of serial examinations, and postoperative uveitis was absent or static. In a few circumstances, dogs had substantial increases in IOP immediately after surgery, and IOP persisted at higher than expected values instead of returning to baseline within 24 hours, as is typical.[10] For these dogs, the treatment for glaucoma that was initially used after surgery was continued. All dogs in the glaucoma group eventually had increased IOP (> 25 mm Hg) and other signs of overt glaucoma. Treatment typically consisted of topical administration

Table 1—Breed distribution (No. of dogs [No. of affected eyes]) of dogs with glaucoma after extraction of a lens by use of phacoemulsification and control dogs that received the same treatment but did not develop glaucoma

Breed	Glaucoma	Control
Boston Terrier	5 (8)*	1 (2)
Bichon Frise	3 (5)	4 (5)
Cocker Spaniel	3 (3)	1 (2)
Samoyed	1 (1)	1 (2)
Poodle	1 (1)	3 (5)
Siberian Husky	3 (3)	3 (4)
Labrador Retriever	0 (0)	2 (3)
Shetland Sheepdog	0 (0)	2 (2)
Others	6 (8)	4 (5)
Total	**22 (29)**	**21 (30)**

*Significantly ($P = 0.042$) different from No. of operated eyes in control dogs.

of dipivefrin or timolol, alone or in combination. If this was insufficient to keep IOP < 20 mm Hg, topical or oral administration of carbonic anhydrase inhibitors or both was typically added. Some dogs in the glaucoma group also underwent a cyclodestructive procedure with or without a gonioimplant if medical treatment alone was unable to adequately control IOP.

For 19 of 29 (66%) operated eyes in the glaucoma group, glaucoma was anticipated after surgery, and some form of treatment was begun before IOP exceeded 25 mm Hg. In 4 of these 19 eyes, treatment was initiated within 24 hours after surgery because of a large postoperative increase in IOP. This treatment continued for 3 weeks to 6 months after surgery and then was discontinued. Subsequently, glaucoma developed in these eyes 2 to 52 months later and treatment was reinitiated. In another 3 of these 19 eyes, treatment began within 24 hours after surgery, was eventually discontinued, and was later reinstituted prior to IOP exceeding 25 mm Hg, either because of progressively increasing IOP detected at follow-up examinations or because of IOP that was abnormally high in relation to the degree of uveitis that was detected. In another 7 of these 19 eyes, treatment was initiated immediately after surgery (because of large postoperative increase in IOP or IOP that was abnormally high in relation to the degree of uveitis that was detected) and was never discontinued before IOP exceeded 25 mm Hg and the dog met entry criteria for the glaucoma group (in 6 of these 7 eyes, glaucoma developed within 1 to 2 weeks after surgery). In the remaining 5 of 19 eyes, treatment was begun only in the late postoperative period prior to IOP exceeding 25 mm Hg. As for other dogs in the glaucoma group, this treatment was prompted because of progressively increasing IOP detected by follow-up examinations or IOP that was abnormally high given the other changes in the eye. In the remaining 10 of 29 (34%) eyes, glaucoma was not anticipated, and treatment for glaucoma was never given prior to the dog developing overt glaucoma.

For the control group, 5 dogs (8 eyes) received some form of treatment for glaucoma initiated within 24 hours of surgery because of large acute postoperative increases in IOP (6 eyes) or IOP that was abnormally high in relation to the degree of uveitis that was detected (2 eyes). This treatment consisted of administration (topical, parenteral, or both) of antiglaucoma drugs, which was discontinued within 2.5 months after surgery with the exception of 1 dog that received topical administration of antiglaucoma drugs for 8 months after surgery because of IOP that was consistently > 20 mm Hg. Administration of the medication was eventually discontinued in this dog and, during the ensuing 44 months, the eyes remained normotensive and without clinical evidence of glaucoma.

For the control group, 6 of 30 (20%) eyes had lost vision by the end of the follow-up period. Causes of blindness were retinal detachment (2 eyes; at 4 and 51 months after surgery), nonglaucomatous retinal degeneration (3 eyes; 30, 35, and 65 months after surgery), and a malacic corneal ulcer (1 eye; 34 months after surgery). For the 24 eyes that retained vision in the control group, mean follow-up was 39.7 ± 10.1 months (median, 39.5 months; range, 26 to 62 months).

For the glaucoma group, 18 of 29 (62%) had lost vision by the end of the study period. When measured from the time of surgery, extraction of the lens restored vision to these 18 eyes for mean duration of 19.5 ± 13.0 months (median, 14.0 months; range, 1 to 46 months) after surgery. Four of these 18 eyes had lost vision completely because of a variety of other causes before the diagnosis of glaucoma (mean, 22 months after extraction of the lens; mean, 3.5 months before onset of glaucoma), and 7 of these 18 eyes had lost vision completely because of glaucoma by the time the dogs were first noticed to have glaucoma (mean, 14.1 ± 3.1 months after extraction of the lens; median, 14 months; range, 10 to 19 months). Another 7 of these 18 eyes eventually lost vision after the diagnosis of glaucoma (mean, 15.6 ± 12.6 months after the onset of glaucoma; median, 14 months, range, 1.5 to 30 months). Eleven of 29 (38%) eyes maintained vision for 17.0 ± 13.4 months (median, 10 months; range, 4 to 37 months) after the diagnosis of glaucoma and were still being followed at the conclusion of the study. For the eyes that retained potential for vision at the time that glaucoma was first diagnosed (11 eyes that still had vision at last follow-up and 7 eyes that initially had vision but ultimately lost vision), vision was retained for a mean of 16.5 ± 12.8 months (median, 10.8 months; range, 1.5 to 37 months) after diagnosis of glaucoma.

There was a significant (P = 0.006) difference between the 2 groups for stage of cataract, with more resorbing cataracts in the control group than in the glaucoma group (**Table 2**). There were no significant differences between the 2 groups for numbers of incipient, incomplete, or complete cataracts. Significant differences between groups were not detected for age of onset of cataracts (**Table 3**). Preoperative abnormalities affecting the eyelids, conjunctiva, and cornea were

Table 2—Distribution (No. of affected eyes) of stage of cataract development among dogs with glaucoma after extraction of a lens by use of phacoemulsification (22 dogs, 29 eyes) and control dogs that received the same treatment but did not develop glaucoma (21 dogs, 30 eyes)

Stage	Glaucoma	Control
Incipient	3	2
Incomplete	8	5
Complete	17	13
Resorbing	1	10*

*Significantly (P = 0.006) different from No. of affected eyes in dogs with glaucoma.

Table 3—Distribution (No. of dogs) of age at onset of cataract development among dogs with glaucoma after extraction of a lens by use of phacoemulsification (22 dogs, 29 eyes) and control dogs that received the same treatment but did not develop glaucoma (21 dogs, 30 eyes)

Age at onset	Glaucoma	Control
Congenital	1	1
Juvenile	6	4
Adult	14	15
Aged	1	1

Congenital = Birth to 3 months old. Juvenile = 3 months to < 2 years old. Adult = 2 to 10 years old. Aged = > 10 years old.

similar between the 2 groups, but combined abnormalities affecting intraocular structures (uvea and retina) were more common in the glaucoma group ($P = 0.012$, **Table 4**). A significant ($P = 0.02$) difference between the 2 groups was also detected for frequency of intraoperative hyphema. This difference was even more significant ($P = 0.005$) if incidence of intraoperative preretinal and vitreal hemorrhages were added to intraoperative hyphema to form a category that included all forms of intraocular hemorrhage that occurred at the time of surgery (**Table 5**). A significant ($P = 0.03$) difference between the 2 groups was detected for frequency of postoperative development of opacification of the posterior capsule (**Table 6**); opacification developed more commonly in the control than in the glaucoma group. Every dog in the glaucoma group that had opacification of the posterior capsule, except for 1 dog that developed glaucoma 1 week after surgery, had opacification of the posterior capsule before the onset of glaucoma.

Table 4—Distribution (No. of dogs [No. of affected eyes]) of abnormalities involving various ocular structures that were detected before surgery in dogs with glaucoma after extraction of a lens by use of phacoemulsification (22 dogs, 29 eyes) and control dogs that received the same treatment but did not develop glaucoma (21 dogs, 30 eyes)

Structure	Glaucoma	Control
Lid	8 (9)	4 (7)
Conjunctiva	2 (3)	0 (0)
Cornea	4 (6)	1 (1)
Lens*	10 (12)	9 (13)
Uvea	3 (4)	1 (1)
Retina	3 (4)	0 (0)
Intraocular†	6 (8)‡	1 (1)

*Lens-induced uveitis. †Intraocular abnormalities comprised uveal and retinal abnormalities other than cataracts or lens-induced uveitis. ‡Significantly ($P = 0.012$) different from No. of affected eyes in control dogs.

Table 5—Distribution (No. of affected eyes) of intraoperative complications in dogs with glaucoma after extraction of a lens by use of phacoemulsification (22 dogs, 29 eyes) and control dogs that received the same treatment but did not develop glaucoma (21 dogs, 30 eyes)

Complication	Glaucoma	Control
Torn posterior portion of capsule	5	8
Vitreous prolapse	5	4
Intraocular bleeding	7*	0

*Significantly ($P = 0.005$) different from No. of affected eyes in control dogs. Intraocular bleeding comprised hyphema, vitreal bleeding, and preretinal hemorrhage.

Table 6—Distribution (No. of affected eyes) of postoperative complications in dogs with glaucoma after extraction of a lens by use of phacoemulsification and control dogs that received the same treatment but did not develop glaucoma

Complication	Glaucoma	Control
Corneal damage	20	23
Hyphema or hypopyon	3	4
Vitreous in anterior chamber	7	7
Dyscoria	9	10
Opacity of lens capsule	16	25*
Fibrosis around intraocular lens	7	4
Asteroid hyalosis	1	5
Retinal detachment or hemorrhage	6	3
Retinal degeneration	5	2

*Significantly ($P = 0.03$) different from No. of affected eyes in dogs with glaucoma.

Significant differences between the 2 groups were not detected for any other variable, including development of lens-induced uveitis (Table 4) or preoperative duration of treatment for lens-induced uveitis (glaucoma group, 0.9 ± 0.2 months; control group, 1.0 ± 0.3 months). In both groups, preoperative lens-induced uveitis was treated until there was no evidence of active uveitis at the time of surgery, and dogs with chronic lens-induced uveitis were excluded. There were no significant differences between the 2 groups in the nature, route of administration, or duration of administration of anti-inflammatory drugs used after surgery to treat uveitis. For most dogs in both groups, however, low dosages of some form of orally administered (aspirin) or topically administered (nonsteroidal anti-inflammatory drug or corticosteroid) anti-inflammatory medication were administered indefinitely after surgery even if the eye was free of overt signs of anterior uveitis. There also were no differences between groups for the gonioscopic appearance of the drainage angle ($P = 0.08$), although in 10 of 29 eyes in the glaucoma group and 19 of 30 eyes in the control group, the morphologic features of the drainage angle were not recorded. For the glaucoma and control groups, the drainage angle was open in 14 of 29 and 9 of 30 eyes, respectively, and considered narrow or closed in 5 of 29 and 2 of 30 eyes, respectively, as judged by use of a defined grading scheme.[12] Differences were not detected between the 2 groups for development of an acute increase in IOP after surgery (control group, 8/30 eyes; glaucoma group, 11/29 eyes; $P = 0.33$).

Discussion

Multiple variables in the study reported here were associated with increased risk for glaucoma in the late postoperative period, including increased risk in the eyes of Boston Terriers. This breed is reported to be at increased risk of primary open-angle glaucoma and glaucoma secondary to lens luxation,[9] and anecdotal reports among veterinary ophthalmologists also indicate that this breed is generally more prone to complications after extraction of a lens. Precise reasons for this predisposition are unclear, but the facial conformation, active personality, and reduced corneal sensitivity[13] of Boston Terriers undoubtedly increases their risk of inadvertent ocular trauma. Such trauma may ultimately lead to glaucoma in the late postoperative period because eyes that have undergone cataract surgery typically mount a more vigorous inflammatory response than eyes that have not had surgery.[14] Boston Terriers may have been over-represented in our study of glaucoma, compared with other brachycephalic breeds (which presumably also have a similar risk of inadvertent trauma), simply because they were the most common brachycephalic breed to develop cataracts, as in a previous study.[6] It is also possible that breed-related differences in ocular inflammatory responses generated after trauma or surgery, or breed-related differences in susceptibility of the aqueous humor outflow system to obstruction, may increase

risk of glaucoma in the late postoperative period in the eyes of Boston Terriers.

Dogs were also at increased risk of glaucoma if intraocular abnormalities, in addition to cataracts and lens-induced uveitis, were evident prior to surgery. It is understandable how some of these abnormalities (eg, small focal peripheral anterior or posterior synechia) could increase the dog's risk of glaucoma in the late postoperative period. The mechanism is not as clear, however, for other abnormalities in this category, such as uveal cysts, atrophy of the iris, and focal retinal dysplasia. Extremely large uveal cysts that fill the posterior chamber have been described as a cause of glaucoma in Great Danes[15] and Golden Retrievers,[16] but dogs in the study reported here did not have cysts that extensive, so a simple mechanical cause for glaucoma was unlikely. Another hypothesis is that these abnormalities are associated with occult preiridal fibrovascular membranes that may impair outflow of aqueous humor and ultimately lead to secondary glaucoma.[15-18] However, preiridal fibrovascular membranes were not detected before surgery in any dog in our study despite performance of a detailed slit-lamp biomicroscopic examination by at least 1 experienced observer. Nevertheless, histologically detectable preiridal fibrovascular membranes have been reported in dogs with uveal cysts and glaucoma.[15,16] It is also possible, but unlikely, that the dogs classified as having iris atrophy in our study may also have had occult preiridal fibrovascular membranes that resulted in iridal fibrosis, pupillary dilation, and poor pupillary movement. In each eye designated as affected with iris atrophy, however, there was iridal translucency or a frank hole in the iris sphincter region, and overt preiridal fibrovascular membranes were not identified. Therefore, other intraocular abnormalities in this category, which are associated with glaucoma after extraction of the lens, may be serving either as an indicator for the presence of occult preiridal fibrovascular membranes or as some other nonspecific marker of impaired ocular health.

Similarly, the association between intraoperative intraocular hemorrhage and glaucoma in the late postoperative period may also indicate that normal intraocular physiologic function is impaired, because in most of these eyes bleeding occurred from the drainage apparatus immediately upon entry into the anterior chamber and prior to any intraocular manipulations. Although, to some extent, it is typical for blood to spontaneously reflux into the trabecular meshwork when the anterior chamber is decompressed,[10] overt hyphema is rare, and its development may indicate that the blood-aqueous barrier is less stable than usual. Spontaneous intraocular bleeding during surgery may also reflect the presence of occult preiridal fibrovascular membranes that formed as a result of chronic low-grade anterior uveitis that developed prior to surgery.[17,18]

In the study reported here, glaucoma was not associated with preoperative lens-induced uveitis, placement of an IOL, duration of the first episode of uveitis after surgery, or total number of episodes of uveitis that developed after surgery. Overall, 42% of the dogs in our study were treated for lens-induced uveitis prior to surgery, and as expected, every dog in the control and glaucoma group had uveitis immediately after surgery. In each instance, selection criteria required that the mild lens-induced uveitis in these dogs was quickly (typically within a month in each group) and completely controlled prior to surgery. In a previous study in which approximately 16% of dogs had preoperative lens-induced uveitis, a lack of association between preoperative lens-induced uveitis and short-term success rates was reported.[4] However, in an earlier study with a much higher incidence of preoperative lens-induced uveitis (61%) and a follow-up period that was more comparable to, but still shorter than, the one reported here, it was found that preoperative lens-induced uveitis was associated with a substantially poorer long-term surgical outcome.[6] In the latter study,[6] it is possible that dogs with more severe forms of lens-induced uveitis were included in the analysis, whereas they were excluded in the study reported here. Neither study,[4,6] however, attempted to specifically correlate preoperative lens-induced uveitis with glaucoma in the postoperative period.

Despite the lack of association between secondary glaucoma and inflammation in the study reported here, it is likely that inflammation does affect the frequency of postoperative complications such as glaucoma. In our study, dogs with severe lens-induced uveitis or uveitis-induced cataracts were excluded, in part because of the clinical practice policy of the VMTH. This means that only a small subpopulation of dogs with cataracts actually underwent surgery. If dogs with more severe forms of uveitis or all dogs with cataracts, irrespective of other ocular disorders, were included, a more broad range of pre- and postoperative inflammation may have permitted differences between the 2 groups to be detected. Difficulties in describing the nature and intensity of intraocular inflammation also make it difficult to detect differences between groups. For example, it is difficult to compare a dog with surgically induced uveitis that is of short duration but severe intensity with a dog with immune-mediated uveitis of long duration but mild intensity. A much larger prospective study may allow more subtle differences in severity of inflammation to be correlated with development of glaucoma after surgery in the subpopulation of dogs that actually undergo cataract surgery. In any event, cataract surgeons may take comfort in the fact that short-lived readily controlled lens-induced uveitis does not substantially increase the risk of glaucoma in the late postoperative period.

The increased incidence of resorbing cataracts in the control group was surprising and difficult to explain, because it is generally believed that the less advanced the stage of cataract, the greater the probability of a successful outcome.[1,4-7] Dogs in the control group may represent a distinct subpopulation of dogs with milder degrees of cataract resorption than that seen in the general population of dogs with cataracts, because they also had fewer additional intraocular abnormalities before surgery than the glaucoma group. Whether resorption of the lens without other intraocular changes somehow protects against glaucoma in

the late postoperative period is unclear and merits further investigation. This finding indicates, however, that dogs with lens resorption may undergo surgery without substantially increased risk of glaucoma if surgical candidates are judiciously selected.

It is also noteworthy that substantial increase in IOP within the first 24 hours after surgery was not associated with increased risk of glaucoma in the late postoperative period, despite persistence of structural changes in the outflow apparatus detected 24 hours after surgery.[10] Sudden increases in IOP after surgery did not even correlate with development of glaucoma during the first 2 weeks after surgery; only 3 of 8 eyes in this category had acutely increased IOP within the first 24 hours after surgery. Although these periods of ocular hypertension may not herald the development of glaucoma in the long term, such sudden large increases in IOP should not be ignored, because they may pose a substantial threat to vision. Rapid, large changes in IOP may result in substantial deformation of the lamina cribrosa, vascular compromise, and subsequent damage to the optic nerve.[10,19,20] Reperfusion injury may also develop when IOP returns to reference range, and release of neurotoxins by the damaged retina may foster continued degeneration of the optic nerve although IOP returns to baseline values.[21] Recently, intracameral administration of carbachol at the conclusion of surgery has been shown to prevent or substantially reduce the frequency of these acute postoperative increases in IOP.[22]

The secondary glaucoma that develops after extraction of the lens is noticeably different than the typical primary angle-closure glaucoma in dogs, and appears to require a different treatment plan and prognosis.[9] In typical primary angle-closure glaucoma, dogs of certain breeds experience sudden, sometimes intermittent, massive increases in IOP that rapidly result in loss of vision.[9,c] Although prophylactic treatment may substantially delay the onset of this form of glaucoma in these patients, this form of glaucoma usually still has an explosive nature when it becomes overt and surgery becomes the treatment of choice.[9,23,c] Dogs in our study, however, often had less severe, more gradual increase in IOP, and the onset of glaucoma could be anticipated in many dogs, permitting medical intervention prior to substantial increases in IOP. Features that prompted initiation of treatment for glaucoma prior to the onset of overt glaucoma in our study included acute postoperative increases in IOP that resolved slowly; IOP that increased progressively, as judged by results of serial examinations; and IOP that was too high with respect to the intensity of concurrent uveitis or in view of the presence of other high-risk uveal disorders such as goniodysgenesis, peripheral anterior synechia, or glaucoma in the opposite eye. Not every dog with secondary glaucoma in our study developed the disease in this slow, progressive fashion, however, and almost a third of dogs did not have obvious prodromal signs of glaucoma. Additionally, in some dogs, the IOP increased within only a few weeks after surgery, whereas in others the increase in IOP developed only after several years. Whether dogs that develop glaucoma within a few weeks after surgery and those that take years to develop the problem do so by the same or different mechanisms remains to be elucidated.

In our study, the extremely wide range in time to onset of glaucoma (1 week to 55 months) indicates that there is not a point during the postoperative period after which a dog is no longer at risk of developing glaucoma. This large range also suggests that the success rate for cataract surgery continues to decline over time and that dogs that have had cataract surgery need to be monitored for complications for the remainder of their lives. Because median time to onset of glaucoma in our study was 10 months, it seems likely that studies with follow-up periods of approximately < 1 year may miss a substantial number of dogs that ultimately develop complications and overestimate the clinically relevant long-term success rates of cataract surgery. Nevertheless, the risk of glaucoma itself is not a contraindication for attempting surgery, because these dogs are substantially visually impaired and any improvement in vision would likely improve their quality of life substantially. Even if a dog is destined to develop glaucoma and become blind again, the potential for restoration of vision for a median of 14 months indicates that extraction of the lens may still be worthwhile, especially in elderly dogs with limited life expectancy. Additionally, after glaucoma does develop in an eye that still has vision, sight may be maintained by use of aggressive medical or surgical treatment for glaucoma for a median of at least 9 months or, in some patients, for as long as 2.5 years. Again, for older dogs in our study, this period often constituted the duration of their lives. Clearly, careful monitoring for glaucoma after surgery can improve long-term success rates by permitting early intervention before a substantial increase in IOP develops and vision is irreversibly lost.

The sensitivity of our study for detecting subtle risk factors for the development of glaucoma was partially limited by the fact that, although glaucoma is an important complication associated with extraction of the lens, it fortunately developed in only a small percentage of dogs, and thus a small number of dogs were available for study. The number of dogs in the control group was also reduced by the necessity of having a substantial follow-up period with consistent measurements of IOP and examinations to ensure that the probability of these dogs developing glaucoma was extremely low and that mild increases in IOP did not go unrecognized. Longer follow-up for the control group, compared with the glaucoma group, is also the most likely reason why the control group had increased prevalence of opacification of the posterior portion of the capsule.[24,25] The rigorous presurgical screening in our study and previous studies,[3,4,6,7] although applied equally to both groups, undoubtedly excluded many dogs from undergoing surgery and hence may also have masked detection of more blatant predictors of glaucoma in the late postoperative period, such as uncontrollable uveitis, preexisting glaucoma, and extensive synechia. The lack of detailed pre- and postoperative gonioscopic examinations in every dog also may have limited our ability to identify abnormalities of the drainage angle before surgery as a risk fac-

tor for glaucoma after extraction of the lens. Although differences were not significant, numerically there were more dogs with abnormal drainage angles before surgery in the glaucoma group than in the control group; larger sample size may have allowed this difference to reach significance. Despite these limitations, however, results of our study indicate that after the decision has been made to perform extraction of the lens by use of phacoemulsification, select factors may be identified that affect risk of development of secondary glaucoma during the late postoperative period.

[a]Tono-Pen II and XL, Mentor Ophthalmics, Norwell, Mass.
[b]SAS Proprietary Software Release 6.12, SAS Institute Inc, Cary, NC.
[c]Miller PE, Schmidt GM, Vainisi SJ, et al. The efficacy of topical prophylactic therapy in primary closed angle glaucoma in dogs: a multicenter clinical trial (abstr), in *Proceedings*. 29th Ann Meet Am Coll Vet Ophthalmol 1998;15.

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