Uveitis is one of the most common and clinically important ophthalmic disorders in domestic cats. Uveitis in cats has been associated with trauma, primary or metastatic neoplasia, abnormalities of the lens, infectious agents, and idiopathic causes. In a retrospective histopathologic study of 158 eviscerated or enucleated feline globes, idiopathic lymphocytic-plasmacytic uveitis was the most common cause of uveitis. In a study of cats with uveitis without obvious systemic disease, 48% of affected cats had bilateral ocular involvement, 72% were sexually intact or castrated males, and the mean age of affected cats was 9.6 years. Sequelae to uveitis include secondary glaucoma, synchiae, lens luxation, cataract formation, phthisis bulbi, and blindness.

The pathogenesis of uveitis involves breakdown of the BAB, which is comprised of the vascular endothelial cells of the iris, tight junctions between the nonpigmented ciliary body epithelial cells, and the posterior pigmented epithelium of the iris. Increased permeability of the BAB results in increased aqueous humor protein concentration. The severity of the BAB disruption is directly proportional to the protein concentration of the aqueous humor and intensity of aqueous humor flare. Mediators of BAB breakdown include prostaglandins, leukotrienes, platelet-activating factor, neuropeptides such as calcitonin gene-related peptide and substance P, interleukins, nitrous oxide, and bradykinin. Endogenous prostaglandins play an important role in the initiation and maintenance of intraocular inflammation.

**Objective**—To compare inhibitory effects of topically applied 1% prednisolone acetate suspension, 0.03% flurbiprofen solution, 0.1% dexamethasone suspension, and 0.1% diclofenac solution on paracentesis-induced blood-aqueous barrier breakdown in cats.

**Animals**—9 healthy cats.

**Procedures**—Paracentesis of the anterior chamber was performed in both eyes of each cat. One eye of each cat was treated with a topically administered anti-inflammatory medication (1% prednisolone [n = 7 cats], 0.03% flurbiprofen [7], 0.1% dexamethasone [9], or 0.1% diclofenac [8]) immediately following paracentesis and at 6, 10, and 24 hours after paracentesis. The contralateral untreated eye served as the control eye. Each cat had a 6-day washout period between experimental drugs. Breakdown of the blood-aqueous barrier was quantified by use of laser flaremetry.

**Results**—Topical administration of 1% prednisolone significantly reduced aqueous humor flare at 4, 8, and 26 hours after paracentesis. Topical administration of 0.1% diclofenac significantly reduced aqueous humor flare at 8 and 26 hours after paracentesis. Topical administration of 0.1% dexamethasone and 0.03% flurbiprofen did not significantly decrease flare at any time point. There were significant differences in intraocular pressures between NSAID-treated eyes and untreated contralateral eyes.

**Conclusions and Clinical Relevance**—Topical administration of 1% prednisolone and 0.1% diclofenac significantly reduced intraocular inflammation in cats with paracentesis-induced uveitis. Topical administration of 1% prednisolone or 0.1% diclofenac may be appropriate choices when treating cats with anterior uveitis. Topical administration of diclofenac and flurbiprofen should be used with caution in cats with a history of ocular hypertension.
ocular inflammation. Prostaglandins also cause miosis, hypotony, and increased permeability of the anterior uveal vasculature.\textsuperscript{27,28,35,36} Prostaglandins can be produced by almost all ocular tissues, including the iris and ciliary body.\textsuperscript{37,38} In feline eyes, EP1 prostaglandin receptors have been detected in the iris sphincter and ciliary muscles, whereas FP prostaglandin receptors have been detected only in the iris sphincter muscles.\textsuperscript{39}

Experimental paracentesis-induced disruption of the BAB has been detected in several species, including cats.\textsuperscript{28,29,40–42} The ciliary body epithelium in the anterior pars plicata is most likely the site of disruption of the BAB after paracentesis of the anterior chamber.\textsuperscript{26,43,44} Enlargement of intercellular spaces and poorly defined fenestrae were found in the anterior pars plicata ciliary body epithelium by use of electron microscopy following paracentesis in cynomolgus monkeys.\textsuperscript{41} Paracentesis-induced breakdown of the BAB is thought to be mediated primarily by prostaglandins.\textsuperscript{28,45–49}

Several methods can be used to evaluate aqueous humor flare or aqueous humor protein concentration, including slit-lamp examination, fluorophotometry, microprotein assays, and laser flaremetry. Laser flaremetry is a rapid, noninvasive, reproducible, and quantitative method to evaluate inflammation in the anterior segment of the eye.\textsuperscript{50,51} Laser flaremetry is a useful method to detect increases in aqueous flare and hence disruptions of the BAB in cats with ocular inflammation.\textsuperscript{9}

Because uveitis is a common clinical entity in cats and most cases are idiopathic, treatment of uveitis is aimed at decreasing intraocular inflammation, preventing the complications associated with intraocular inflammation, and relieving signs of pain and discomfort. Treatment of uveitis in veterinary medicine commonly includes the use of anti-inflammatory medications in addition to a specific antimicrobial agent when an infectious etiology has been identified. Anti-inflammatory medications can be administered topically, systemically, or subconjunctivally to treat intraocular inflammation. The route of administration is determined by the severity of the inflammation and the location of the inflammation within the eye. In general, topical anti-inflammatory treatment is recommended to treat anterior uveitis.

Anti-inflammatory treatment includes the use of corticosteroids and NSAIDs. Corticosteroids prevent biosynthesis of arachidonic acid and subsequent formation of prostacyclin, thromboxane A\textsubscript{2}, prostaglandins, and leukotrienes.\textsuperscript{52–54} Glucocorticoids also decrease prostaglandin synthesis at the level of the cyclooxygenase pathway,\textsuperscript{55} and they may also induce local expression of somatostatin, which has anti-inflammatory properties.\textsuperscript{56} Nonsteroidal anti-inflammatory drugs bind to and inhibit cyclooxygenase, which is the enzyme that converts arachidonic acid to prostaglandins (prostaglandin E\textsubscript{2}, prostaglandin D\textsubscript{2}, prostaglandin F\textsubscript{2}, and prostaglandin I\textsubscript{2}) and thromboxane A\textsubscript{2}.\textsuperscript{54,57} Cats do not tolerate systemic NSAIDs as well as other species do because of their lesser ability for glucuronide conjugation; therefore, topical application of these drugs may be more appropriate for this species. Presently available topical ophthalmic NSAIDs in the United States include 0.09% bromfenac, 0.1% diclofenac, 0.03% flurbiprofen, 0.4% and 0.5% ketorolac, and 0.1% nepafenac. Topical formulations of indomethacin are not commercially available in the United States at this time.

Although there have been several ophthalmic studies\textsuperscript{56,31,49,67} evaluating the efficacy of systemic and topically applied anti-inflammatory agents in dogs, there has only been 1 study reported in the literature in cats.\textsuperscript{58} Because of species differences in stability of the BAB\textsuperscript{24,69} and differences in the metabolism of drugs, it is necessary to evaluate the efficacy of anti-inflammatory medications in individual species.

The purpose of the study reported here was to compare the inhibitory effects of 4 commercially available topical anti-inflammatory drugs (1% prednisolone acetate suspension, 0.03% flurbiprofen solution, 0.1% dexamethasone suspension, and 0.1% diclofenac solution) on paracentesis-induced intraocular inflammation in cats by use of laser flaremetry.

Materials and Methods

Animals—Nine young adult random-source domestic shorthair cats (7 neutered males and 2 sexually intact females) were used in this study. Cats were tested for FeLV, FIV, and heartworm infection and had complete physical examinations. An ophthalmic examination including slit-lamp biomicroscopy, fluorescein staining, applanation tonometry, and indirect ophthalmoscopy was performed prior to the study. This study was approved by the Purdue University Animal Care and Use Committee.

Flaremetry—Aqueous humor protein concentration was estimated by measuring aqueous humor flare by use of a laser flare-cell meter\textsuperscript{70} and the procedure described for the use of this meter in cats.\textsuperscript{32} To allow proper positioning, cats were sedated with ketamine hydrochloride\textsuperscript{5} (5 mg/kg, IM) and xylazine\textsuperscript{4} (0.25 mg/kg, IM) prior to laser flaremetry. Flare measurements were reported in photon counts per millisecond, and the mean of 10 measurements was recorded as the flare value for each eye.

Disruption of the BAB by use of anterior chamber paracentesis—Paracentesis of the anterior chamber was performed in both eyes of each cat. One eye of each cat was treated with 1 drop of a topically administered anti-inflammatory medication (1% prednisolone acetate suspension\textsuperscript{5} [n = 7 cats], 0.03% flurbiprofen solution\textsuperscript{7} [7], 0.1% dexamethasone suspension\textsuperscript{9}, or 0.1% diclofenac solution\textsuperscript{8}) immediately following paracentesis and at 6, 10, and 24 hours after paracentesis. The number of cats in each treatment group varied because a small number of cats developed mild to moderate transient conjunctivitis that resolved during the study. Cats with conjunctivitis at the beginning of the drug-testing portion of the study were excluded. The contralateral untreated eye served as the control eye. Each cat was allowed a 6-day washout period before treatment with the next experimental drug.\textsuperscript{57} The order of the anti-inflammatory medication experiments and the eye treated were as follows: 1% prednisolone acetate administered to the right eye, 0.03% flurbiprofen administered to the left eye, 0.1% dexamethasone...
administered to the right eye, and 0.1% diclofenac administered to the left eye. Paracentesis of the anterior chamber was performed by use of the technique described for cats. A 30-gauge needle attached to a 3-mL syringe was introduced into the cornea of each eye 1 to 2 mm anterior to the limbus. The needle was advanced into the center of the anterior chamber, and aqueous humor was slowly removed over 3 to 5 seconds. Aqueous humor was removed until the anterior chamber was almost completely collapsed. The volume of aqueous humor removed ranged from 0.7 to 1.1 mL. Laser flare measurements were taken at 4 hours (after 1 treatment), 8 hours (after 2 treatments), and 26 hours (after 4 treatments) after paracentesis. Laser flaremetry, slit-lamp biomicroscopy, and measurement of IOP with an applanation tonometer were performed before each new experiment to ensure that these variables were not abnormal at the beginning of each experiment.

**Statistical analysis**—Data are expressed as mean ± SEM values for flare and IOP. Protein concentrations were calculated from flaremetry results by use of the mean value from each time point and the following equation: \( y = 0.96x + 0.60 \), where \( y \) is the logarithm of the protein concentration and \( x \) is the logarithm of flare (photon counts/ms).\(^{29}\)

Percentage inhibition of flare induced by the anti-inflammatory drugs was calculated for each drug. The mean flare concentration at each time point was corrected by subtracting the pretreatment measurement of that group from the time point measurements at 4, 8, and 26 hours. Then, by use of the corrected data, the following equation was used: percentage inhibition of flare = ((corrected mean for control eye group – corrected mean for treated eye group)/corrected mean for control eye group) \( \times 100\).\(^{29}\) A 1-sided paired student t test was performed. Values of \( P \leq 0.05 \) were considered significant.

### Results

**Clinical evaluation**—All cats had negative results for FeLV, FIV, and heartworm tests. The ophthalmic examinations were within reference limits in all cats. The amount of aqueous humor flare at each time point was subjectively evaluated by 1 investigator (AJR) by use of a slit-lamp biomicroscope. According to the scale developed by Hogan et al,\(^{21}\) there was no visible flare in any of the eyes prior to paracentesis at the beginning of each experiment. The amount of flare was graded to be between 0 and 2+ in all eyes at 4 and 8 hours after paracentesis. At 26 hours after paracentesis, most of the cats had no visible flare and only 1 cat in the prednisolone group and 5 cats in the dexamethasone group had trace flare. The anterior chamber appeared to be completely reformed in each cat by 4 hours after paracentesis.

**Drug inhibition of flare increase**—Mean pretreatment flare values in all eyes ranged from 7.5 to 11.1 photon counts/ms. Pretreatment flare values for the left and right eyes were not significantly different. Mean ± SEM flare values and the calculated aqueous humor protein values for the control eye and treated eye for the 4 anti-inflammatory drugs were summarized (Table 1). The flare values were considered important if the treated eye had less flare, compared with the control eye.

For the 1% prednisolone acetate suspension, there were significant differences between the control and treated eye at 4, 8, and 26 hours after paracentesis (\( P = 0.03, 0.001, \) and 0.001, respectively). For the 0.03% flurbiprofen solution, there were no significant differences between the control eye and the treated eye at any of the time points. For the 0.1% dexamethasone suspension, there was a significant difference between the control eye and the treated eye at 4 hours. At this time point, the flare value was higher in the treated

<table>
<thead>
<tr>
<th>Time and treatment</th>
<th>Control eye</th>
<th>Treated eye</th>
<th>Protein (mg/dL)</th>
<th>Flare (photon counts/ms)</th>
<th>Protein (mg/dL)</th>
<th>Flare (photon counts/ms)</th>
<th>( P ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 hour</td>
<td></td>
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</tr>
<tr>
<td>1% prednisolone</td>
<td>7.5 ± 0.8</td>
<td>7.4 ± 0.9</td>
<td>27.5</td>
<td>27.2</td>
<td>0.45</td>
<td></td>
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</tr>
<tr>
<td>0.03% flurbiprofen</td>
<td>9.7 ± 1.3</td>
<td>9.3 ± 0.6</td>
<td>35.3</td>
<td>33.9</td>
<td>0.37</td>
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</tr>
<tr>
<td>0.1% dexamethasone</td>
<td>8.0 ± 0.7</td>
<td>8.5 ± 0.3</td>
<td>29.3</td>
<td>31.1</td>
<td>0.27</td>
<td></td>
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</tr>
<tr>
<td>0.1% diclofenac</td>
<td>11.1 ± 1.4</td>
<td>9.2 ± 0.7</td>
<td>40.1</td>
<td>33.5</td>
<td>0.06</td>
<td></td>
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<tr>
<td>4 hours</td>
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</tr>
<tr>
<td>1% prednisolone</td>
<td>162.9 ± 34.6</td>
<td>104.0 ± 17.4</td>
<td>528.9</td>
<td>343.8</td>
<td>0.03</td>
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<tr>
<td>0.03% flurbiprofen</td>
<td>115.1 ± 22.9</td>
<td>92.0 ± 14.2</td>
<td>379.0</td>
<td>305.7</td>
<td>0.12</td>
<td></td>
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<tr>
<td>0.1% dexamethasone</td>
<td>127.0 ± 26.9</td>
<td>146.0 ± 28.0</td>
<td>416.5</td>
<td>476.2</td>
<td>0.02</td>
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<tr>
<td>0.1% diclofenac</td>
<td>157.7 ± 16.5</td>
<td>104.7 ± 10.8</td>
<td>412.4</td>
<td>346.1</td>
<td>0.15</td>
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<tr>
<td>8 hours</td>
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</tr>
<tr>
<td>1% prednisolone</td>
<td>75.0 ± 9.0</td>
<td>40.9 ± 4.0</td>
<td>251.2</td>
<td>140.4</td>
<td>0.001</td>
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<tr>
<td>0.03% flurbiprofen</td>
<td>70.6 ± 21.2</td>
<td>33.6 ± 4.0</td>
<td>237.1</td>
<td>116.2</td>
<td>0.06</td>
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<tr>
<td>0.1% dexamethasone</td>
<td>59.5 ± 12.7</td>
<td>52.6 ± 12.7</td>
<td>201.2</td>
<td>176.7</td>
<td>0.19</td>
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<tr>
<td>0.1% diclofenac</td>
<td>75.0 ± 10.5</td>
<td>43.6 ± 5.6</td>
<td>264.1</td>
<td>149.2</td>
<td>&lt; 0.001</td>
<td></td>
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</tr>
<tr>
<td>26 hours</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>1% prednisolone</td>
<td>24.3 ± 3.4</td>
<td>13.9 ± 1.9</td>
<td>85.1</td>
<td>49.8</td>
<td>0.001</td>
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</tr>
<tr>
<td>0.03% flurbiprofen</td>
<td>16.8 ± 2.5</td>
<td>12.8 ± 1.8</td>
<td>59.7</td>
<td>46.0</td>
<td>0.11</td>
<td></td>
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</tr>
<tr>
<td>0.1% dexamethasone</td>
<td>22.4 ± 2.9</td>
<td>16.1 ± 4.0</td>
<td>78.7</td>
<td>57.4</td>
<td>0.07</td>
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<tr>
<td>0.1% diclofenac</td>
<td>29.8 ± 3.8</td>
<td>16.0 ± 2.3</td>
<td>102.9</td>
<td>57.0</td>
<td>0.005</td>
<td></td>
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</tr>
</tbody>
</table>

\( P \) values indicate comparison between the aqueous humor flare values in contralateral untreated eyes versus treated eyes.

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AJVR, Vol 72, No. 6, June 2011

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Discussion

In this study, we evaluated the ability of 4 commercially available topical anti-inflammatory medications to inhibit paracentesis-induced BAB disruption in cats by use of laser flareometry. Several reports have indicated the efficacy of topically administered prostaglandin-inhibiting drugs to decrease the disruption of the BAB in dogs. Various concentrations and frequency of use of the drugs used, methods of disrupting the BAB, and methods of assessing breakdown of the BAB have been used. These discrepancies and the variation in BAB stability among species highlight the necessity of evaluating the efficacy of anti-inflammatory medications in different species.

In the present study, 1% prednisolone acetate was the only drug that significantly decreased the flare values at all 3 time points. In a study in which topically administered steroidal drugs and NSAIDs were used to compare effectiveness at preventing paracentesis-induced BAB disruption by use of fluorophotometry in dogs, pretreatment with topically administered 1% prednisolone acetate and 0.03% flurbiprofen was more effective than pretreatment with 0.1% dexamethasone sodium phosphate. Several factors limit the therapeutic effect of a topically administered corticosteroid ophthalmic preparation, including corneal permeability, degree of steroid receptor binding, extent of ocular metabolism of the drug, and effect of the drug on the target cell. The greatest barrier to intraocular penetration of topically administered corticosteroids is the corneal epithelium, which is lipophilic. Lipophilic acetate and alcohol corticosteroid suspensions penetrate the cornea more readily than do sodium salts of the steroid phosphate.

Although previous studies have indicated the BAB-stabilizing effects of topically administered flurbiprofen in dogs, in the present study, flurbiprofen did not significantly decrease the aqueous flare values at any of the time points. Measured by use of fluorophotometry, topical pretreatment for 48 hours with 0.03% flurbiprofen more effectively decreased fluorescein leakage into the anterior chamber than did pretreatment with 1% prednisolone when an Nd:YAG laser was used to create an anterior capsulotomy in dogs. In that study, flurbiprofen also was much more effective at maintaining mydriasis than was prednisolone acetate and there were no significant differences in IOP between the groups. In another study performed by the same authors in which Nd:YAG laser anterior capsulotomy-induced inflammation was used, topically administered flurbiprofen was as effective as flunixin meglumine administered IV at maintaining mydriasis, compared with untreated controls. Possible factors that affect the bioavailability of topically administered NSAIDs in the eye include corneal penetrability, corneal stromal protein binding, and corneal stromal metabolism. It is also possible that in the present study, there was inhibition of inflammation in the contralateral eye, which would decrease the difference between the treated eye and the contralateral control eye. There also may be important species differences between dogs and cats that may explain the different results between those studies and the present study.

Dexamethasone 0.1% suspension did not have a significant effect on decreasing flare in the present study. Dexamethasone sodium phosphate was found in another study to be the least effective at stabilizing the BAB in dogs, compared with 0.03% flurbiprofen and 1% prednisolone acetate. However, it is known that dexamethasone sodium phosphate has poor corneal penetration, and this could account for its poor efficacy in that study. Experimental studies indicate that dexamethasone suspension is superior to dexamethasone sodium phosphate in anti-inflammatory activity. In a model of endotoxin-induced uveitis evaluated in cats, topically administered dexamethasone beloxil (AL-2512) was more effective in reducing inflammatory cell influx into the anterior chamber than was topically administered 1% prednisolone acetate.88 In

Table 2—Mean ± SEM IOP in the same eyes as in Table 1.

<table>
<thead>
<tr>
<th>Treatment and time</th>
<th>Control eye IOP (mm Hg)</th>
<th>Treated eye IOP (mm Hg)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretreatment</td>
<td></td>
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</tr>
<tr>
<td>1% prednisolone</td>
<td>15.7 ± 0.9</td>
<td>17.1 ± 0.9</td>
<td>0.01</td>
</tr>
<tr>
<td>0.003% flurbiprofen</td>
<td>18.5 ± 1.7</td>
<td>17.4 ± 1.3</td>
<td>0.12</td>
</tr>
<tr>
<td>0.1% dexamethasone</td>
<td>14.7 ± 0.8</td>
<td>15.2 ± 0.2</td>
<td>0.22</td>
</tr>
<tr>
<td>0.1% diclofenac</td>
<td>15.1 ± 1.0</td>
<td>14.8 ± 0.8</td>
<td>0.30</td>
</tr>
<tr>
<td>4 hours</td>
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</tr>
<tr>
<td>1% prednisolone</td>
<td>8.6 ± 0.8</td>
<td>8.6 ± 0.8</td>
<td>0.5</td>
</tr>
<tr>
<td>0.003% flurbiprofen</td>
<td>9.8 ± 0.6</td>
<td>12.3 ± 1.6</td>
<td>0.05</td>
</tr>
<tr>
<td>0.1% dexamethasone</td>
<td>8.4 ± 0.6</td>
<td>8.7 ± 0.6</td>
<td>0.28</td>
</tr>
<tr>
<td>0.1% diclofenac</td>
<td>8.0 ± 0.7</td>
<td>9.4 ± 0.8</td>
<td>0.09</td>
</tr>
<tr>
<td>8 hours</td>
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<td></td>
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<tr>
<td>1% prednisolone</td>
<td>7.9 ± 1.5</td>
<td>8.9 ± 1.6</td>
<td>0.23</td>
</tr>
<tr>
<td>0.003% flurbiprofen</td>
<td>8.9 ± 1.0</td>
<td>12.6 ± 0.7</td>
<td>0.002</td>
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<tr>
<td>0.1% dexamethasone</td>
<td>8.8 ± 1.0</td>
<td>8.7 ± 1.2</td>
<td>0.42</td>
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<td>0.1% diclofenac</td>
<td>8.1 ± 0.6</td>
<td>9.8 ± 0.5</td>
<td>0.002</td>
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<tr>
<td>26 hours</td>
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<tr>
<td>1% prednisolone</td>
<td>15.7 ± 2.1</td>
<td>15.7 ± 1.8</td>
<td>0.5</td>
</tr>
<tr>
<td>0.003% flurbiprofen</td>
<td>15.9 ± 0.8</td>
<td>16.7 ± 1.4</td>
<td>0.18</td>
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</tr>
<tr>
<td>0.1% diclofenac</td>
<td>11.0 ± 1.0</td>
<td>13.1 ± 1.2</td>
<td>0.03</td>
</tr>
</tbody>
</table>
that study,86 0.1% dexamethasone beloxil and 1% prednisolone acetate failed to significantly reduce aqueous humor protein concentrations. In the present study, it is possible that if the cats had been pretreated with topically administered dexamethasone or treated more frequently, there may have been more of an anti-inflammatory effect.

In the present study, 0.1% diclofenac significantly decreased the flare values at 8 and 26 hours. In a study39 comparing the efficacies of topically administered NSAIDs at preventing paracentesis-induced BAB breakdown in dogs, 1% diclofenac suspension was superior to 1% suspensions of flurbiprofen, suprofen, and tolmetin. In that study,94 flurbiprofen and suprofen were also effective at preventing BAB disruption. However, it is difficult to extrapolate this information into a clinical setting because these drugs are not presently commercially available in 1% suspensions. Higher drug concentrations may permit greater intraocular concentrations.77

In the present study, 1% prednisolone acetate inhibited the flare by 38% at 4 hours, which was greater than the other 3 drugs. At 8 hours after paracentesis, 1% prednisolone acetate, 0.03% flurbiprofen, and 0.1% diclofenac inhibited the flare increase by approximately 50% or more, which should be beneficial clinically. At 26 hours, all 4 drugs inhibited the increase in flare by approximately 50% or more. Prednisolone acetate– and diclofenac-treated eyes had significant decreases in flare, compared with the contralateral control eyes.

A significant difference in IOP between the flurbiprofen-treated eyes and the control eyes at 4 and 8 hours after paracentesis was found in the present study. There were also significant differences between the diclofenac-treated eyes and the control eyes at 8 and 26 hours. There were no significant differences between the corticosteroid-treated eyes and the control eyes at 4, 8, or 26 hours after paracentesis. In another study,63 flurbiprofen and prednisolone acetate applied topically 4 times/d had no effect on the IOP in dogs that had inflammation induced with an anterior capsulotomy by use of an Nd:YAG laser. It is important to note that in that study,63 all 6 time points were either at or prior to 60 minutes after the initiation of inflammation, which may not be sufficient time to evaluate the effect of these drugs on IOP. Results of several other studies indicate that flurbiprofen increases the IOP in experimental BAB disruption65,67 and decreases aqueous humor outflow78 in dogs. In a study67 in which pilocarpine was applied topically to dogs, topical administration of 1% prednisolone acetate, 0.03% flurbiprofen, and 1% suprofen significantly inhibited the decrease in IOP, compared with the values for the control group. In that study,67 topically administered NSAIDs increased the IOP in the contralateral eyes and treated eyes.

The present study had several limitations. It is possible that the anti-inflammatory medication that was applied to the treated eye in each cat may have had an effect on the contralateral control eye in the same cat, which would have decreased the difference in flare between the 2 eyes. It is also possible that the drugs may have had a longer effect than the allotted 6-day washout period, although none of the cats had visible flare on slit-lamp biomicroscopic examination or increased flare counts via laser flaremetry examination prior to each experiment. Other limiting factors of the present study were the small number of cats used and the variability in flare values, most notably at 4 hours after paracentesis (the SEM ranged from 10.8 to 34.6). This variability was likely caused by interanimal differences in stability of the BAB. In a previous study29 evaluating the effects of topically applied 2% pilocarpine and paracentesis of the anterior chamber in cats, evaluated by use of laser flaremetry, paracentesis of the anterior chamber induced a greater breakdown of the BAB and also resulted in larger SDs, compared with topically applied pilocarpine. The effect on the BAB of repeated paracentesis in feline eyes has not been previously evaluated, and repeated paracentesis may also have had an effect on the results.

Results of this study provide a basis for selecting an appropriate topically administered anti-inflammatory medication in treating cats with anterior uveitis. On the basis of the results of this study, 1% prednisolone acetate is recommended for treatment of anterior segment inflammation in cats. In cases in which a corticosteroid is contraindicated, diclofenac would be a good choice. It is possible that more frequent applications of topically administered anti-inflammatory medications or pretreatment with topically administered anti-inflammatory medications prior to disruption of the BAB (ie, surgery) may be more effective at decreasing flare, although this was not evaluated in the present study. Results also suggested that topically administered NSAIDs should be avoided or used with caution in feline patients with a history of ocular hypertension.

References


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