

# Animal Eye Center of NJ

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## **Glaucoma:** When the Pressure Gets To Be Too Much...

Glaucoma is a very common cause of blindness in dogs, resulting from either an acute or chronic increase in intraocular pressure. It is less common in cats, and features of the disease differ between the two species. Normal intraocular pressure in dogs and cats ranges from 15 to 25 mmHg when measured with an applanation tonometer (such as a TonoPen). Increased intraocular pressure produces irreversible damage to the optic nerve and retina causing vision impairment. If pressure remains uncontrolled, the damage will become permanent and blinding, as the retina and optic nerve will lose their function entirely. For this reason, swift treatment of high intraocular pressure is imperative for preserving vision and long-term comfort.

Intraocular pressure is normally achieved through a balance of both aqueous humor production and aqueous humor outflow. Aqueous humor is produced by the folds of the ciliary body behind the iris. This fluid then flows forward through the pupil and into the anterior chamber. From here, the fluid can exit the anterior chamber by one of two "routes".

The **conventional outflow** pathway allows the aqueous humor to exit via the iridocorneal angle, through the trabecular meshwork, and back into the systemic circulation. This comprises approximately 85% of outflow in dogs and 97% in cats.

The **unconventional outflow** pathway involves absorption of aqueous humor through the base of the iris and the ciliary body, and eventual drainage into the venous circulatory system.

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Like many other ocular diseases, glaucoma in dogs often presents as a "red eye". Therefore, a careful ocular examination and diagnostics are imperative for determining a course of therapy. Clinical signs consistent with glaucoma may differ depending on the duration of disease.

### Clinical signs of **acute glaucoma** may include:

Pain (blepharospasm, epiphora)	Mydriasis
Episcleral congestion	Blindness
Corneal edema	

### Clinical signs of **chronic glaucoma** may include:

Pain (more variable than in acute cases)	Corneal striae (Haab's striae)
Episcleral congestion	Buphthalmos
Corneal edema	Blindness
Mydriasis (may be more variable than acute cases)	

Clinical signs of glaucoma in cats are generally more subtle. The most consistent clinical sign is a fixed and dilated pupil. Other signs such as overt pain, episcleral congestion, and corneal edema are seen less consistently than in dogs.

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The pathophysiology of glaucoma is complicated, but in general cases can be categorized into either **primary** or **secondary glaucoma**.

**Primary glaucoma** is breed-related or inherited. A large number of breeds can be affected including Cocker spaniels, Siberian huskies, Chow chows, Shar peis, Beagles, Basset hounds, Briards, and Boston terriers. Primary glaucoma develops due to structural and functional impairment of the iridocorneal angle. Presenting age may be variable, but most dogs are middle-aged or older at time of onset.

**Secondary glaucoma** is glaucoma that develops due to pre-existing ocular disease. The most common causes are chronic anterior uveitis and/or lens disorders such as chronic cataracts or lens luxation/subluxation. This can affect any breed of dog. This is also the most common cause of glaucoma in cats, being 19 times more common than primary glaucoma.

Making the distinction between primary and secondary glaucoma is a crucial part of ocular assessment, as presence of other ocular diseases may have implications for prognosis and treatment.

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**The primary goals of glaucoma treatment are to decrease intraocular pressure and subsequently relieve pressure on the optic nerve and retina.** This can be achieved using a number of different systemic and topical drugs. Choice of drugs should be dictated by the nature of the glaucoma.

**Osmotic agents** can be used in the treatment of **acute** glaucoma. These agents dehydrate the vitreous, widen the iridocorneal angle, and therefore improve aqueous outflow. They are available in intravenous and oral formulations.

**Mannitol: 1-2 g/kg, IV bolus, over 20 minutes**  
**Glycerin: 1-1.5 g/kg PO, mixed with food**

Contraindications for mannitol or glycerin include generalized dehydration, renal insufficiency, or cardiac disease. While mannitol can be used safely in diabetic patients, glycerin cannot as it is metabolized to glucose and can exacerbate hyperglycemia. Due to absorption variability, glycerin's hypotensive effect is less predictable. Oral administration of glycerin also commonly induces nausea and/or vomiting.

**Carbonic anhydrase inhibitors (CAI)** are agents that decrease aqueous humor production by inhibiting enzyme activity at the ciliary body. These drugs are indicated in all cases of glaucoma, whether primary or secondary. They are available in either topical or oral formulations as listed below.

**Brinzolamide (Azopt): 1 drop to the affected eye, TID-QID**  
**Dorzolamide (Cosopt): 1 drop to the affected eye, TID-QID**  
**Methazolamide: 2-10 mg/kg PO BID to TID**

Oral methazolamide, especially at higher doses, can be associated with metabolic acidosis that leads to panting, lethargy, and/or vomiting and diarrhea. Therefore, this drug should be used carefully in patients that have pre-existing metabolic disease. Cats are exceptionally sensitive to the side effects of methazolamide, so oral administration should be avoided. Brinzolamide has been shown to be less effective than dorzolamide in cats.

**Beta blockers** decrease aqueous humor production at the level of the ciliary body. They may also have some minor effects on aqueous outflow. In general, these agents are weak and best used in combination with other glaucoma medications. They are available in topical formulations, either alone or in combination with carbonic anhydrase inhibitors.

**Timolol maleate (0.25%, 0.5%): 1 drop to the affected eye BID**  
**Timolol/dorzolamide (Cosopt): 1 drop to the affected eye BID**

Side effects are uncommon with topical beta blockers, but the active agents can be absorbed into the systemic circulation. Therefore, these agents are contraindicated in patients with pre-existing cardiac disease or lower airway disease such as asthma or obstructive pulmonary disease. Beta blockers should also be avoided when lens luxation is suspected, as they can induce miosis of the pupil and can exacerbate glaucoma by further obstructing aqueous flow

**Prostaglandin analogs** are powerful agents that encourage aqueous outflow through the unconventional pathway. They are well-tolerated and available in a number of topical formulations.

**Latanoprost (Xalatan): 1 drop to the affected eye SID-BID**  
**Bimatoprost (Lumigan): 1 drop to the affected eye SID-BID**  
**Travoprost (Travatan): 1 drop to the affected eye SID-BID**

Systemic side effects are not reported. PAs, however, should not be used in cases where lens luxation is suspected. These agents cause intense miosis of the pupil and can exacerbate glaucoma by further obstructing aqueous flow. They should also be used with caution in patients with overt uveitis, as their prostaglandin analog activity can exacerbate existing inflammation. **Also noteworthy is that PAs are not effective in cats.**

The following algorithms can help the clinician determine a course of therapy for glaucoma. Again, treatment for each patient may vary according to their species, duration and nature of disease, prognosis for vision, and systemic health status.

# Canine Glaucoma

Corneal edema  
Episcleral congestion  
PLR deficit/mydriasis  
IOP > 25 mmHg



Lens in anterior chamber?

Hypopyon, Hyphema,  
Tumor, Trauma

No additional findings

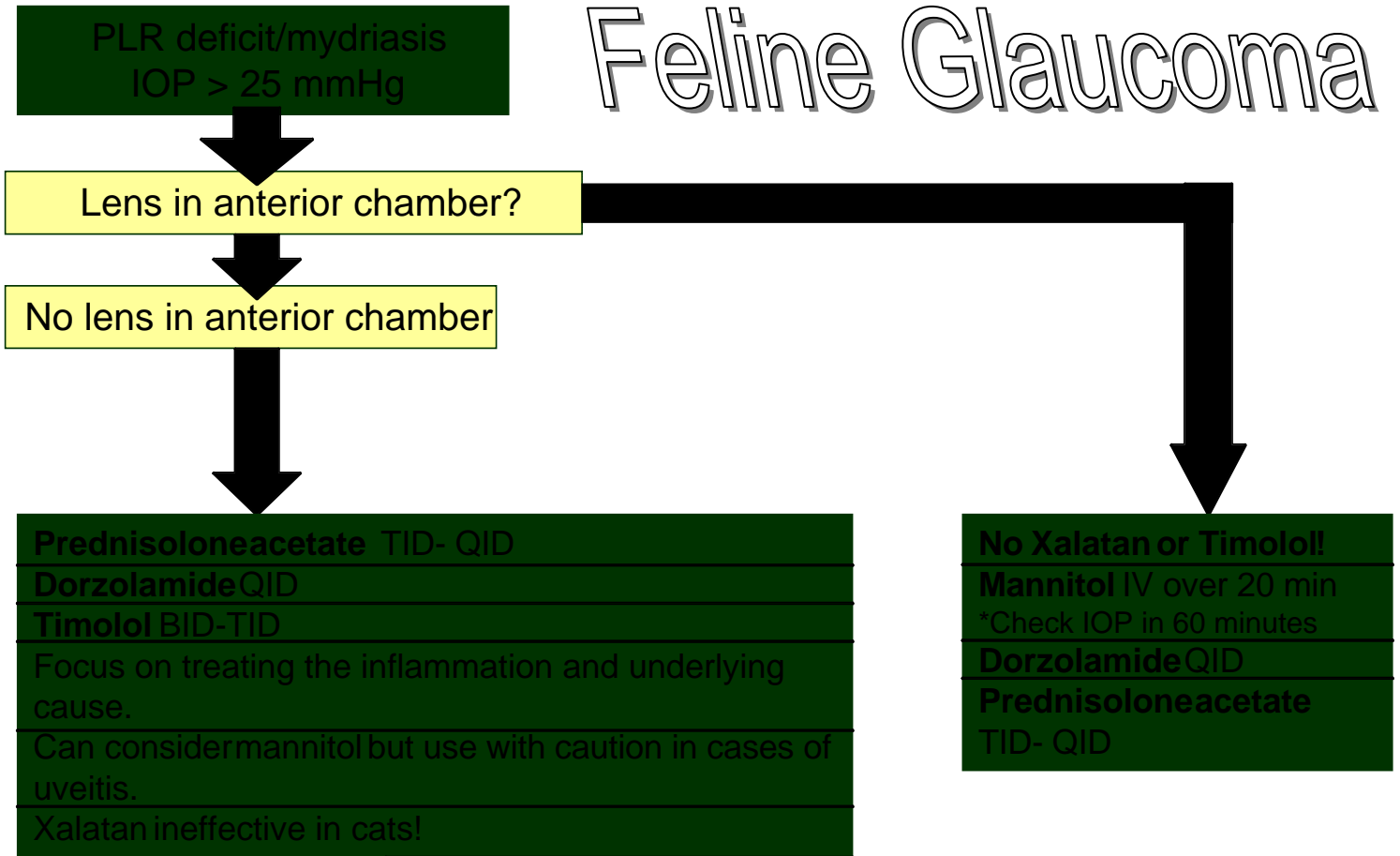


1 drop **Xalatan**  
\*Check IOP in 30 minutes  
If no response, IV bolus  
**mannitol** over 20 minutes  
\*Check IOP in 60 minutes  
**Dorzolamide** QID and/or  
**methazolamide** PO BID  
**Xalatan** BID  
**Timolol** BID-TID  
**Prednisolone acetate** TID- QID

**Prednisolone acetate**  
TID- QID  
**Dorzolamide** QID and/or  
**methazolamide** PO BID  
**Timolol** BID-TID  
Focus on treating the  
inflammation and underlying  
cause.  
Can consider mannitol and/or  
Xalatan but use with caution in  
cases of uveitis.

**No Xalatan or Timolol!**  
**Mannitol** IV over 20 min  
\*Check IOP in 60 minutes  
**Dorzolamide** QID  
and/or **methazolamide**  
PO BID  
**Prednisolone acetate**  
TID- QID

# Feline Glaucoma



## **Some additional considerations...**

**\*If an eye is diagnosed with primary glaucoma, the contralateral eye is at high risk (50% likelihood) of developing glaucoma within 8 months of diagnosis.**

Prophylactic treatment for the eye and evaluation by an ophthalmologist may be recommended.

**\*Long-term response to medical glaucoma therapy is variable in dogs and cats, regardless of underlying cause. In cases where vision can be preserved, surgical treatment of glaucoma may be pursued.** This can be performed by a veterinary ophthalmologist and may include:

Laser cyclophotocoagulation: laser treatment of the ciliary body to decrease aqueous humor production; can be performed transsclerally or endoscopically  
Glaucoma shunt: subconjunctival valved shunt placed to improve aqueous outflow

Laser treatment alone (transscleral), is reported to decrease IOP by 58% over a one year postoperative period. Maintenance of vision, however, at 6 months postoperatively ranged from 37-50% in two separate studies. When combined, these procedures are reported to maintain IOP control in 75% of eyes. At one year postoperatively, 41% of operated eyes were visual.

**\*Newer considerations in glaucoma therapy are neuroprotective agents.** While evidence-based indications for their use have not been determined, some ophthalmologists recommend use of NMDA inhibitors such as memantine. Oral amlodipine has also been suggested to minimize retinal/optic nerve ischemia.

**\*Eyes with chronic and/or refractory glaucoma carry a poor prognosis for long-term return to vision as well as a guarded prognosis for long-term control of IOP and comfort.** Therefore, these dogs and cats may be considered better candidates for procedures such as enucleation, evisceration with intrascleral prosthesis, or chemical ablation of the ciliary body (gentamicin/dexamethasone intravitreal injection).