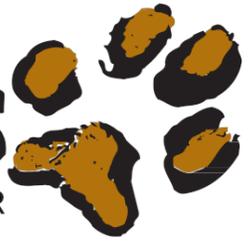




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SouthPaws
 VETERINARY SPECIALISTS & EMERGENCY CENTER



Glaucoma

Glaucoma is one of the leading causes of blindness in animals and people. Glaucoma is defined as increased pressure within the eye (greater than 25 mmHg), beyond that which is compatible with normal ocular function and vision. It is caused by a disturbance in the flow of fluid within and out of the globe. Primary glaucoma occurs when there are no other underlying problems causing the pressure elevation and is usually inherited. Secondary glaucoma occurs when there are other problems within the eye which have contributed to the elevated pressure. Examples of these underlying problems include uveitis (inflammation) and luxation (shifting from the normal position) of the lens.



Glaucoma

The fluid that fills the eye (the aqueous humour) is produced by the ciliary body, a structure which is located behind the iris and circles for 360 degrees around the eye. The fluid produced by the ciliary body flows through the pupil and fills the anterior chamber. Normally, this fluid flows out from the eye through the iridocorneal drainage angle, a structure encircling the eye where the iris and cornea meet. In a normal eye the fluid production and outflow are evenly matched, and this keeps the intraocular pressure steady. Glaucoma occurs when there is an obstruction to the outflow of the fluid which causes the fluid to build up and increase the intraocular pressure. As the pressure increases, several changes can occur within the eye:

- 1) Increased pressure forces fluid into the cornea and disrupts the arrangement of the protein fibers that compose the cornea. This causes the cornea to become edematous, or a cloudy, blue-gray color sometimes referred to as a "steamy" cornea (referring to the appearance of a bathroom mirror after a hot shower).
- 2) Vessels on the sclera (the white of the eye) becomes enlarged and cause the eye to appear red and inflamed.
- 3) With a prolonged pressure increase, the size of the globe will increase because the fibers that make up the sclera are slightly elastic in nature.
- 4) Vision is significantly decreased. This is because the nerve fibers of the optic nerve (responsible for transmitting information from the retina to the brain) and the retina itself are both compromised by increased pressure. The longer the pressure is elevated, the more extensive and permanent the vision loss may be. Recent evidence has shown that increased pressure may cause a chain of event (cellular apoptosis) that causes nerve fiber death within the optic nerve and retina that continues even after the pressure is relieved.

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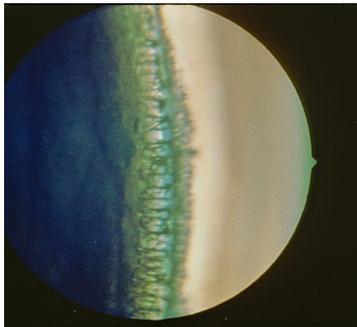
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Glaucoma *continued*

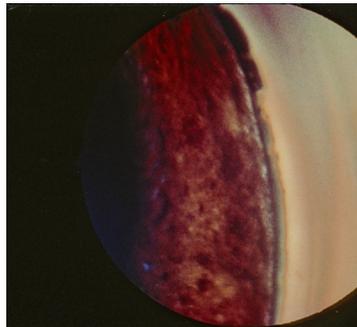
- 5) The lens of the eye is held in place by fibrous strands called zonules. While the sclera is able to stretch a bit due to the increased pressure in chronic glaucoma (leading to increased globe size or buphthalmia), the zonules cannot stretch and instead break. This causes the shifting of the lens from its normal position within the eye.
- 6) Increased pressure and its subsequent changes may cause uveitis (intraocular inflammation) to occur along with the glaucoma.
- 7) Elevations in intraocular pressure may be associated with significant discomfort, especially when such elevations are acute. Humans who experience acute glaucoma compare the discomfort to migraine headaches. Similarly, animals with glaucoma may show discomfort through signs such as keeping the eye closed, pawing or rubbing the eye, lethargy, hiding in dim places, restlessness, decreased appetite, or vomiting. However, because dogs are so stoic, they may not exhibit any of these signs. Occasionally, the decreased activity is contributed to advancing age or arthritis. It is not until the source of the discomfort (elevated intraocular pressure) is alleviated that the animal begins to act normally again.
- 8) Dilation of the pupil combined with absence of response to light stimulation.



Acute glaucoma



Normal angle



Closed Iridocorneal Angle

The accurate diagnosis of glaucoma is based on a thorough ocular examination and measurement of the intraocular pressure with a TONOMETER. GONIOSCOPY is a diagnostic evaluation of the iridocorneal drainage angle and requires a special lens allowing direct visualization of the drainage angle. Gonioscopy may sometimes aid in determining the risk of the

development of glaucoma in the normal eye, especially in breeds predisposed to the development of glaucoma.

PRIMARY GLAUCOMA

Primary glaucoma is usually caused by an inherited abnormality of the drainage angle. Breeds particularly susceptible to inherited primary glaucoma include: several spaniel breeds (including the American Cocker Spaniel and Brittany Spaniels), Basset Hounds, Chow Chows, Chinese Shar Peis, Siberian Huskies, Norwegian Elkhounds, Miniature Poodles, Beagles, and Samoyeds.

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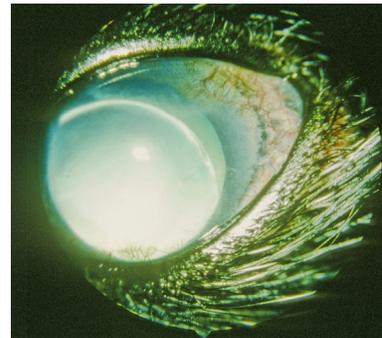
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Glaucoma *continued*

SECONDARY GLAUCOMA

Secondary glaucoma may occur in the following situations:

- 1) Secondary to inherited lens luxation (dislocations): several terrier breeds (including the Jack Russell and Wirehaired Fox) and the Chinese Shar Pei have an inherited zonule loss which leads to a luxation of the lens. If the lens luxates forward through the pupil and into the anterior chamber, it disrupts the flow of the aqueous humour and causes a significant, rapid increase in the intraocular pressure.
- 2) Secondary to uveitis (intraocular inflammation) due to:
 - a) Blockage of the drainage angle by inflammatory cells.
 - b) Closure of the drainage angle due to the inflammatory process (this is common in cats with chronic uveitis and frequently leads to lens luxation).
 - c) Adhesions (synechiations) of the pupil margin to the lens which cause an obstruction of the flow of aqueous humour (iris bombe).
- 3) Secondary to intraocular neoplasia.



anterior luxated lens

GLAUCOMA TREATMENT

A treatment protocol is formulated based upon the classification of glaucoma, the underlying cause of the glaucoma (lens luxation, uveitis, neoplasia), and the prognosis for vision. Treatment of glaucoma is AIMED AT CONTROL, as glaucoma is RARELY CURED! The prognosis for glaucoma is often dependent upon early detection and treatment. However, due to the nature of the disease and what has been recently identified as cellular apoptosis, many animals lose vision in the affected eye despite treatment. In inherited glaucoma, the second, normal eye will become affected within twelve months. It must be stressed that glaucoma is a disease that requires long term vigilant treatment and evaluations.

MEDICAL TREATMENT OF PRIMARY GLAUCOMA

Medical treatments are predominantly aimed at decreasing the production of fluid and increasing its outflow in the hope that this will return the intraocular pressure to equilibrium. These treatments include:

Osmotic Diuretics: Osmotic Diuretics (examples include intravenous Mannitol and oral Glycerin) are used to rapidly decrease the intraocular pressure in acute onset glaucoma.

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Glaucoma *continued*

Beta Blockers: Beta Blockers (examples include Levobunolol, Timolol, Metipranolol, and Carteolol Hydrochloride) work by decreasing the production of fluid by the ciliary body, although there is some evidence that they may also increase the outflow of fluid. The exact mechanism of Beta Blockers action is not fully understood. One possible side effect of Beta Blockers is a decreased heart rate.

Carbonic Anhydrase Inhibitors (CAI): Carbonic Anhydrase Inhibitors (oral examples include methazolamide and dichlorphenamide, topical example include Dorzolamide and Brinzolamide) suppress the production of aqueous humour by the ciliary body. The use of oral carbonic anhydrase may result in panting (due to metabolic acidosis) and decrease the appetite.

Beta Blocker/CAI Combination: A Dorzolamide hydrochloride-Timolol Maleate ophthalmic solution combines the benefits of both drugs into one solution, and their combined effect lowers intraocular pressure more than either component administered alone.

Prostaglandins: Prostaglandins (an example is Latanaprost) decrease intraocular pressure by decreasing the size of the pupil and increasing the uveo-scleral outflow, which is a secondary outflow route for the aqueous humour.

Corticosteroids and Non-Steroidal Anti Inflammatories (NSAIDs): Orally or topically prepared Corticosteroids, as well as oral NSAIDs, may be prescribed to decrease intraocular inflammation associated with glaucoma.

Miotics: Cholinergic agents (examples include Pilocarpine and cholinesterase inhibitors such as Phospholine Iodide) may be used to constrict the pupil. These drugs produce miosis (a contraction of the pupil) through contraction of the sphincter muscles. This causes increased tension on the drainage angle and facilitates aqueous outflow. The use of these drugs, however, can result in ocular irritation and may potentiate uveitis. Miotics may be used in combination with any other glaucoma medications.

Sympathomimetics: Sympathomimetics (examples include Epinephrine or Dipivefrin) have been documented to enhance aqueous outflow while decreasing aqueous production and are also used in the treatment of glaucoma.

SURGICAL INTERVENTIONS

Diode Laser Photocoagulation of the Ciliary Body: In this procedure, pulses of diode laser are delivered via a probe placed on the sclera above the ciliary body. Pulses delivered to 30-60 spots around the eye produce focal destruction of the ciliary body. The production of aqueous humour is reduced by destroying focal points of the ciliary body, hopefully restoring the intraocular

continued

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Glaucoma *continued*

Cylocryotherapy: In this procedure, a glaucoma cryo probe is placed over the ciliary body in 6-10 areas. The subsequent freezing and destruction of the ciliary body reduces aqueous humour production in a manner similar to laser photocoagulation.

GONIOIMPLANT: In this promising alternative for the management of glaucoma, an anterior chamber shunt is placed to facilitate the outflow of fluid onto the sclera and then under a conjunctival pocket. A small tube leads from the device, through a scleral tunnel, and into the anterior chamber. If the intraocular pressure rises above a certain threshold, the excess aqueous humour is siphoned off via this tube to a filtration bleb. The main cause of failure associated with these implants involves the extensive inflammatory response that is present in dogs. This response frequently leads to fibrosis and scarring of the filtration blebe, leading to its failure. Using gonioimplantation in conjunction with laser photocoagulation also shows promise in intraocular pressure control.

TREATMENTS FOR NON-VISUAL EYES

Intraocular Prosthesis: This procedure involves the evisceration of the globe through incisions into the dorsal conjunctiva and sclera. The globe contents are replaced with a solid silicone sphere equivalent to the normal size of the eye and the incisions are sutured. A third eyelid flap may be placed to protect the healing underlying cornea and globe. Inserting an intraocular prosthesis leaves the patients with a comfortable globe that is significantly more cosmetic than enucleation.

Chemical Ablation of the Ciliary Body: This procedure involving the intravitreal injection of 20-25 mg of Gentomycin into the eye often requires mild sedation and centesis of some fluid from the eye prior to the injection. Gentomycin is toxic to the ciliary body and leads to its destructions. The result of the destruction of the ciliary body is that the production of aqueous humour decreases and the intraocular pressure decreases. The then softer-than-normal globe often atrophies (Phthisis bulbi) and can sometimes decrease significantly in size. This procedure can be used as a way to "kill" glaucoma in end-stage, bupthalmic, avidual eyes. Because Gentamycin is also toxic to the retina, it should not be used to treat eyes when there is a potential to save vision. The degeneration of the ciliary body following the gentamycin injection can often cause intraocular inflammation (which may become chronic), with intermittent intraocular hemorrhage.

Enucleation of the Globe: The enucleation of a non-visual, painful eye is also an option. The