Anatomy
The canine lens is suspended in the eye by zonular ligaments which arise from the ciliary epithelium and attach to the lens at the equator. The lens capsule is an elastic basement membrane which encloses the underlying anterior epithelium and lens fibers. The size of the lens is approximately 7mm in the anterior/posterior aspect and 10mm as the equatorial diameter. The anterior lens capsule is much thicker (50-70um) than the posterior capsule (2-4 um) and is an important factor when performing cataract extraction. Lens fibers run parallel to each other, running from anterior to posterior. However, the fibers don’t quite reach the anterior and posterior poles or cover the entire circumference and thereby form the upright, anterior ‘Y’ suture and inverted, posterior ‘Y’ suture. The lens is nourished in the embryo by the hyaloid artery arising from the posterior segment and later in life, by the aqueous humor and vitreous. Mittendorf’s dot represents the insertion of the hyaloid artery on to the posterior lens capsule.

Congenital lesions of the lens
Most congenital lesions of the canine lens involve a failure of atrophy of fetal vascular structures. The problem can be as minor as the presence of Mittendorf’s dot, representing the insertion of the hyaloid artery on the posterior lens capsule. An avascular tail leading from Mittendorf’s dot can be seen in the vitreous in many patients. Persistent hyperplastic primary vitreous (PHPV) is the most severe form of failure of atrophy of fetal lenticular vessels and is an inherited disease in Dobermans and Bull Terriers. Congenital lesions of the lens are common in patients with multiple ocular anomalies such as in the Australian Shepherd, Samoyed, Doberman, Saint Bernard, and Labrador Retrievers.

Nuclear (lenticular) sclerosis
Nuclear sclerosis, also referred to as lenticular sclerosis, is a normal age change in all species. Over time the lens becomes less ‘pliable’ which results in a decrease in the focusing ability of the lens. The clinical result is the inability to focus on close-up objects as when we lose the ability to read fine print or a menu in a dimly lit restaurant! In a dog’s world, we usually hear the owner’s concern about the loss of ability for the patient to quickly find a piece of food on the floor. The dog will usually take a few extra seconds to locate the treat. Also, the patient will often times hesitate to climb down stairs, especially if it is dark at the other end of the stairway. However, overall vision remains normal and the owner will often say that the patient can spot a squirrel down the street. Often there is confusion in differentiating nuclear sclerosis from a pathological change we call cataract. The easiest way to differentiate nuclear sclerosis is first, by the clinical signs I mention above and then during the exam with dilation of the pupils. Once the pupils are dilated with tropicamide, nuclear sclerosis will appear as a gray-ish, ice-cube effect ONLY in the center of the lens, the nucleus. You should still be able to examine the fundus through the periphery of the lens through the clear cortex. In fact, sometimes to help a very elderly patient out with some improved peripheral vision if we elect not to pursue surgical removal of a very dense sclerotic lens, we can use intermittent dilation therapy at home to help the patient navigate more confidently.

Cataracts
Cataracts form when the water and electrolyte pumping systems fail to perform properly. Most cataracts in dogs are inherited and therefore, are seen in young patients. Other causes of cataract include: metabolic such as Diabetes mellitus, trauma, uveitis. Cataracts are classified based on age of onset, location, degree of maturation, and cause. Clinically, the most useful categories are location and degree of maturation. Locations of a cataract include: capsular, subcapsular, cortical, nuclear, and equatorial. The location of a cataract provides implications of cause and prediction of whether the lesion will progress. For example, a triangular posterior subcapsular cataract in each eye of a middle aged Golden Retriever probably won’t progress, whereas, diffuse cortical and equatorial cataract formation in a 2 year old Cocker Spaniel will probably progress quickly and require surgery. Stages of maturation include: incipient, immature, mature, and hypermature. Incipient cataracts are early focal lesions. Immature cataracts are the next stage and allow some viewing of the fundus. Mature cataracts are opaque and the fundus can’t be viewed. As the lens becomes hypermature, liquefaction of protein occurs and the capsule becomes wrinkled. Leakage of lens protein often results in lens-induced uveitis.

*Treatment of cataracts
Various medical treatments have been investigated for dissolving cataracts but none of these has been effective. Surgical removal remains the only method of treatment. Prior to surgery the patient must be evaluated for any concurrent ocular problems that would preclude surgery such as glaucoma, severe corneal disease, retinal diseases, etc. If the ophthalmic exam is normal, an electroretinogram is performed to evaluate retinal function and many surgeons (including myself) perform ultrasonography to evaluate for retinal detachment, vitreal diseases, or neoplasia.
There are three basic types of removal. Intracapsular extraction involves rupturing the zonules and removing the entire lens as a single piece through a large corneal incision. This method was the first method historically to remove cataracts. Extracapsular extraction, where the lens cortex and nucleus are removed but the posterior capsule and some of the anterior capsule are left in the eye as a “dividing wall” between the anterior and posterior segments, has been used for most of this century until the early 1970’s. In 1971 (or thereabouts), Dr. Charles Kellman devised a method to shatter the cataract into several pieces through a small corneal incision by a method called phacofragmentation and then modified to phacoemulsification. Phacoemulsification remains the preferred method for cataract removal by ultrasonic shattering of the lens. A 3.2 mm incision is created in the superior limbus as a 2-plane, valve-like approach. The anterior chamber is filled with viscoelastic material to maintain a full working depth and protect the fragile corneal endothelium. Next, a capsulotomy in the anterior lens capsule is performed to provide access to the lens material. The phacoemulsification process is a combination of irrigation of fluid into the eye, emulsification of the cataract by ultrasonic vibration of the tip, and aspiration of particles until all that remains is the “capsular bag”, a combination of posterior and equatorial capsule and the anterior capsule with the round capsulotomy. Depending on surgeon preference and specific patient criteria, an intraocular lens (IOL) may be placed. There are many different styles of IOL’s but most are made of PMMA with a central optic and supporting haptics and have a dioptic power of 40-42D to obtain emmetropia in canine eyes. The incision is closed with 8-0 to 10-0 suture material. Postoperatively, anti-inflammatory medications, usually steroids, are used to reduce anterior uveitis. Follow-up rechecks are required over about an eight week period to monitor resolution of uveitis, intraocular pressure, and incisional healing.
So, we veterinary ophthalmologists have some pretty cool toys to do our work from slit lamp biomicroscopes to tonometers to high resolution ultrasound machines, electoretinography, phacoemulsification and more! We are able to combine various disciplines to manage our patients from anatomy to physiology to medicine to surgery to pharmacology and as you have certainly heard, “The eye is the window to the soul!” In reality, as my crazy neurologist friend likes to point out, we’re still dealing with a 2 cm organ in the patient’s head. Dry eye, dry eye, corneal ulcer, glaucoma, dry eye, corneal ulcer, eyelid tumor….there really aren’t THAT many diseases, in reality, that we deal with during our busy days so, what do we do as ophthalmologists to make our days a bit more interesting? Well, I think about some things that nobody has ever published or maybe even presented at a conference, but chats with other eyeball colleagues on the phone or in the hallways of meetings reveals that they think about some of these things too!

1. **Contralateral Dry eye disease**: Often times when a patient presents with a very severe eye disease in one eye, such as a deep melting corneal ulcer or intense glaucoma, we find that the tear production in the contralateral eye is diminished. Then, once we resolve the problem in the diseased eye the Schirmer tear test improves without any lacrimogenic drugs being used on the fellow eye. What’s up with that?!

2. **Westie KCS “Syndrome”**: While we’re on the topic of dry eye, I have noted over the years that West Highland White Terriers have what I refer to as the ‘mountain response’ to tear stimulating drugs when they are being treated for dry eye disease. At one point the tear production is ‘reasonable’ (never great) and then without changing any treatment or reduction in client compliance, the next visit will show a decreased tear test. Then wait another 6 months and the tear production may improve again. This continues forever!

3. **Dry Eye Corneal Pathology**: Another KCS related interesting phenomenon is that of differences in corneal pathology in certain types of dry eye patients. Many chronic cases of KCS will result in severe corneal pathology of vascularization, pigmentation, edema and these cases may have only mild to moderate suppression of their Schirmer tear tests. Then, in certain cases, namely the German Shepherd dog or congenital KCS, the corneas will remain clear of any pathology forever, despite total lack of lacrimation with Schirmer results of 0 mm/min.

4. **Extended wear use of Bandage contact lenses**: Certain cases of age-related crystalline corneal degeneration can be difficult to manage with simple lubricants as the patient often remains very uncomfortable with intense blepharospasm. We have found that soft bandage contact lenses can be tolerated for long term use in these patients to greatly improve comfort.

5. **Cherry Eye “Dilemma”**: Controversy continues regarding a patient who has had a prolapsed gland of the third eyelid for an extended period time and has developed KCS in that eye. Dr. Rhea Morgan confirmed in JAAHA in 1991 that if a prolapsed gland is left in the prolapsed position, that patient is at much higher risk of developing KCS. However, the question is: Should we repair that chronically prolapsed gland or simply remove it? In other words, would it benefit the patient, other than cosmetic effects, to replace the gland in the hopes of re-initiating tear production from that gland? The controversy continues.

6. **“He Doesn’t Seem Painful”**: Often times a patient presents with a severely diseased eye that we know is extremely painful in humans such as a buphthalmic, chronic glaucomatous eye. Unfortunately, there is no reasonable option in these cases other than removal of the eye but when I explain that the eye is painful (as evidenced by increased activity after such an eye is removed), the owner often states that the patient doesn’t seem painful. How do we convince the client that the eye IS painful?

7. **“Blinder than Blind”**: Now, in comes a patient with bilateral severe glaucoma with no hope of vision; the cornea is completely opacified and edematous, there are bilateral chronic cataracts with synechiae, and pressure values in the 60’s. No way does this patient see but the owner elects bilateral ciliary body chemical ablation to eliminate pain. The owner returns for the two week follow-up convinced that since the procedure the patient has “lost vision”. Hmmmm?

8. **Cataract surgery results in blue eyed dogs**: I love doing phacoemulsification in blue-eyed dogs as they always look absolutely beautiful after surgery as if we never even did surgery. I’m talking even at the 1 day or 1 week rechecks it’s difficult to tell that the eye had major intraocular surgery- little to no aqueous flare, no redness, etc. Bring on those Huskies with cataracts!

9. **Latanoprost for acute glaucoma**: a drop of latanoprost in an acute glaucomatous eye often (maybe 75%) results in rapid return of vision, reduction in pressure, and a nice clear cornea. This drug is not designed to do this in people and the drug does not seem to have much of an effect in our chronic canine cases but in a TRULY acute case, it can be miraculous!
10. Ocular melanomas- despite the aggressive nature of this tumor elsewhere on the dog and cat body such as mouth or skin, the eye seems to be a privileged site in protection against metastasis. It’s rare to see metastasis of epibulbar or iris melanomas in dogs and cats. Similarly, diagnosis of an adnexal carcinoma (third eyelid) usually brings a good prognosis after surgical excision and even a diagnosis of hemangiosarcoma of the conjunctiva has a very low (non-existent) metastatic rate.
Often times the client presents a patient for ‘sudden blindness’ they noticed only recently. However, many times the patient in fact began to have problems long before the onset of the apparent ‘sudden blindness’ and it is the job of the practitioner to play detective to uncover the true story! Careful examination of both eyes of the patient will reveal the complete ophthalmic situation, regardless of some possible mis-leading information from the client’s history.

Complete evaluation of the patient involves a systematic approach to the examination, starting from the front of the eye with examination of the adnexa and progresses to the posterior aspect of the eye, including the fundus, retro bulbar tissue, and cortex of the brain. Although ophthalmologists seem to have ‘cool toys’ in the exam room, a thorough eye exam requires only minimal equipment and an accurate interpretation of clinical findings. Necessary exam equipment includes: Schirmer tear test strips, fluorescein stain strips, a good focal light source such as a Finoff transilluminator, slit beam aperture on a direct ophthalmoscope head, tonometer, and indirect or direct ophthalmoscope.

First, we will cover causes of pseud-sudden blindness where the client suspects recent onset of blindness, but in fact, the disease has been developing over time. These cases occur far more frequently than you might suspect and it can often be difficult to convince the owner that the problem has existed for some time.

**Pseudo-sudden blindness**

**Keratoconjunctivitis sicca (dry eye disease)**

You would not believe the numbers of cases I’ve seen for emergency exams because the owner panics that the dog has sudden lost vision, only to find out that the patient actually has had chronic dry eye. How can this happen?- Simply, the dog has been developing pigmented corneas for several months or years and nobody ever cared much about the chronic mucus and redness. As the dog seemed to behave normally, a gradual loss of vision was also never noted UNTIL recently when that last bit of clear cornea became pigmented and now the owner suspects sudden vision loss. Examination of this patient reveals mucus, conjunctival hyperemia, completely pigmented corneas, and a low Shirmer tear test value. Often times the Schirmer is ‘0’ in these patients but any value less than 15mm/min is considered abnormally low. Treatment is aimed at improving tear production and decreasing the pigmented corneas with combination of topical cyclosporine (Optimmune) and topical steroid ointment. Reversal of the pigment is quite successful and the patient regains vision over several weeks to months.

**Pigmentary keratitis**

Although pigmentary keratitis is part of the KCS discussion above, it also occurs as a separate problem in such breeds as the Pug, Lhasa, Pekingese, Shih Tzu, and other Brach cephalic breeds. The diagnosis is made by the presence of pigmented corneas in an otherwise, healthy eye. There is no indication of dry tear film or decreased Schirmer tear tests, lids blink properly, and there is no entropion. Again, owners suspect recent onset of blindness but the pigmented corneas were just not noted until the dog lost that one last area of clear cornea. Treatment consists of topical cyclosporine and/or topical steroids.

**“Pannus” (chronic superficial keratitis)**

Similar to the patient with dry eye or pigmentary keratitis, the dog that presents for sudden blindness that actually has chronic pannus, has been developing the characteristic granulation type corneal reaction for months or years prior to going blind. Again, the patient was able to cope with gradually decreasing vision until the last bit of clear cornea became involved with the fleshy infiltrate. The disease is most commonly associated with German Shepherd dogs but is also seen in Greyhounds and miniature Dachshunds. In the German Shepherd dog, the most common age group is 5-7 years old but there is a younger group of 2-3 year old dogs. Interestingly, the younger age group tends to have a more aggressive form of the disease and therefore, requires more intense treatment. Treatment consists of LIFELONG topical steroids and/or cyclosporine. The disease is very rewarding to treat in most cases as the vision returns over a few months. Medication can NEVER be discontinued but the dose can be decreased once the corneas have cleared.

**Facial paralysis- absent menace response**

One of my favorite stories involves a diabetic patient who I had performed cataract surgery on a couple of years prior to emergency re-referral for blindness in one eye. As I began my exam of this patient I noted absolutely quiet, clear eyes with excellent clarity to the intraocular lens implants. As I performed a menace reflex test in this patient, I noted that the patient did not blink as he was threatened by my motioning hand; upon further examination of his neuro-ophthalmic reflexes, the situation became clear when his Pupilebral reflex on that same eye as negative. This patient could see fine but had developed facial nerve paralysis and THAT’S why he didn’t blink with the menace reflex!
**First eye chronically blind, 2nd eye new problem**

Another quite common situation that makes the client suspect recent onset of blindness is a patient who has had an undetected blind eye but good vision out of the fellow eye. Then something occurs in the fellow eye to cause a sudden onset of blindness in that one eye but the patient is now blinded since the first eye was already blind. My favorite case here was a high strung German Shepherd who presented for ‘sudden blindness’ after the dog ran through a glass door during a thunderstorm. Examination revealed a quiet right eye but a very squinty left eye. This patient had undetected hypoplasia in the right eye and actually NEVER had vision out of that eye thereby demonstrating that many/most dogs can show normal vision behavior with only one functioning eye. The left eye had developed severe corneal edema and laceration from a piece of glass and had temporarily lost vision from this eye. Another example of this situation include vitreal degeneration of Shih Tzus where there has been complete retinal detachment in the first eye for some time (possibly since birth) and some time later the second eye becomes detached. In summary, any dog who has been blind in the first eye for some time and suddenly develops a problem in the remaining eye may seem to have sudden vision loss from BOTH eyes.

**Chronic glaucoma**

Glaucoma is usually acute in dogs causing sudden vision loss one eye at a time but in some cases such as in the Shar Pei, glaucoma causes a more gradual vision loss. In these dogs, the glaucoma has been ‘chipping away’ at the optic nerves for some time but only recently they lost those last few functioning nerve cells to appear suddenly blind. Chronic glaucoma is recognized by: 1. Buphthalmic globe 2. Haab’s striae which are stretch marks in the corneal endothelium revealing long straight opacities noted in the cornea 3. Luxated or subluxated lens and 4. Optic nerve cupping and atrophy where the nerve is sunken below the level of the sclera (‘red numbers’ on the direct ophthalmoscope). It amazes me how many times owners suspect sudden blindness in chronic glaucoma cases.

**Chronic retinal atrophy**

Here’s another good one for pseudo-sudden blindness- A dog has retinal atrophy/degeneration such as the patient with inherited Progressive Retinal Atrophy (PRA). This dog has been losing vision for months to years but has been able to mask any significant vision deficits until the last healthy part of the retinas degenerate. Often times we are looking at a 5-7 year old Labrador Retriever with PRA who has a canine companion in the home who has served as a ‘guide dog’ for this patient. The visually impaired patient follows the tail of the normal vision dog and thereby hides any vision problems to the owners. PRA is an inherited disease in many breeds, at first causing decreased vision in dim light and eventually complete loss of vision with no known treatment.

**Non-responsive pupil(s)**

Pupillary light responses evaluate both the afferent and efferent parts of the visual pathway. This reflex relies on a light stimulus to evaluate the afferent aspects (retina, optic nerve, pupillary light center in the brain), and the efferent oculomotor nerve to the iris sphincter muscle. In the case of an unresponsive pupil, we must be sure that the iris is not dilated from some other problem besides a true vision problem. Non-blinding causes of a non-responsive pupil include iris atrophy and dilating medications. Iris atrophy is very common in one or both eyes of aging patients such as miniature poodles and other toy breeds. The diagnosis is made by evaluation of a ‘ragged edge’ to the pupil and often times there are leftover strands of iris crossing the pupil. In a patient with a dilated pupil without iris atrophy, we should question the owner carefully about any recent use of dilating agents such as atropine. Atropine can last up to a couple of weeks in a non-inflamed eye. Hint: Manufacturers color code the cap of any dilating agent to be RED (atropine, Tropicamide, Phenylephrine, cyclopentolate).

**Sudden blindness**

**Sudden acquired retinal degeneration syndrome**

Sudden Acquired Retinal Degeneration Syndrome is a sudden blindness syndrome in dogs that is characterized by acute loss of vision with extinguished electroretinogram. The syndrome is found in pure and mixed breeds but is over-represented in the miniature schnauzer and dachshund and many dogs exhibit concurrent clinical signs including weight gain, polydipsia/polyuria, polyphagia around the time of blindness. The concurrent signs hint at the possibility of Cushing’s disease but the relationship between the blindness and the endocrine problem has not been confirmed. The ophthalmic examination is usually unremarkable; some patients demonstrate retinal vascular “box-carring” or strictures of the vessels and over several months the tapetum becomes hyperreflective. The cause of SARDS is unknown. Various factors have been discussed but to date no specific conclusions have been made. Theses patients seem to have an unusual amount of excitement or panic when they become suddenly blind, bumping into walls without any slowing or sensing of objects in front of them.

**Optic neuritis**

Another cause of true sudden blindness occurs in patients with inflamed optic nerves. Most of the time the exam is unremarkable except for dilated, non-responsive pupils. This disease is the major differential diagnosis with SARDS and the differences with the exam are subtle, mostly based on the difference of pupillary light response. Sometimes we will see papilledema if the nerve head is swollen on the intraocular portion but be careful not to confuse papilledema with optic nerve myelination in normal dogs, especially larger breeds like the retrievers. Optic nerve swelling of the intraocular portion is often accompanied by small retinal hemorrhages around the nerve head. However, most cases of optic neuritis do NOT show any changes with the nerve since the inflammation is presumed to be of the long, extra-ocular portion of the nerve. The patient with optic neuritis may show neurological signs, compatible
with associated glaucomatous meningoenephalitis (GME) and may require further neurological workup with cerebrospinal fluid analysis. The cause of optic neuritis is often idiopathic and maybe immune-mediated in many cases; these patients usually respond to systemic immuno-suppressive drugs such as corticosteroids, azathioprine, or cyclosporine and vision will return if treated within about 2 weeks of onset of blindness. Recurrences can happen even when the medications are gradually tapered over months.

**Acute bilateral glaucoma**

Two words…Bassett Hounds and Cocker Spaniels! Although glaucoma usually occurs one eye at a time in breeds that are predisposed to the blinding condition, sometimes a patient, especially these breeds, will be stricken by the disease in both eyes at the same time. As opposed to the patient who has pseudo-sudden blindness with chronic glaucoma, these patients do not have the clinical signs noted above which are indicative of a more chronic onset. Acute glaucoma includes: corneal edema, elevated intraocular pressure, blindness WITHOUT optic nerve atrophy, buphthalmos, or other chronic corneal changes such as vascularization. A VERY common scenario goes something like this: A dog has had blindness from glaucoma in the first eye and then suddenly develops acute glaucoma in the second eye, thereby completely blinding the patient. Again, predisposed breeds such as the Bassett and Cocker will usually acquire glaucoma one eye at a time with an average time span between eyes of about 6 months. However, I have seen many cases where we just about get the first eye dealt with when the second eye becomes involved. Treatment for glaucoma depends on personal preference, chronicity of the problem, elevation of intraocular pressure, and client preferences. Treatment options for acute glaucoma include: medical management with systemic and topical drugs, diode laser cyclophotocoagulation, and anterior chamber shunts make up the majority of treatments. Treatment for blind, chronic glaucoma is aimed at reduction of long-term pain including: enucleation, evisceration with intraocular prosthesis, diode laser cyclophotocoagulation, and chemical cycloablation.

**Diabetic cataracts**

Diabetic cataracts occur when the blood sugar level is very high, NOT the length of time that the patient has been diabetic. Therefore, it is most common to find that our diabetic dogs acquire cataracts within a few months of diagnosis since their blood glucose values are highest around the time of diagnosis of the diabetes. These cataracts usually develop very quickly once they start to form and can cause lens-induced uveitis at the same time. Treatment for the diabetic cataracts is the surgical removal by phacoemulsification after the patient has been well regulated on insulin and all uveitis is under control.

**Retinal detachment**

There are many causes of retinal detachment in the dog including infectious diseases, neoplastic, metabolic, and idiopathic/immune-mediated. The patient presents for sudden blindness and examination often reveals complete retinal detachment as in a ‘morning glory’ type detachment. The patient should be thoroughly worked up within reason to search for a cause of the detachment. If no cause is found, then the patient may have ‘immune-mediated, steroid responsive’ type of detachment which responds dramatically to systemic corticosteroids. Essential workup prior to ‘shot-gunning’ with steroids should include: complete blood count, chemistry profile, blood pressure measurement, and any specific tests based on findings such as lymph node aspirate, chest radiographs, urinalysis, or infectious serology.

**Hepatic encephalopathy and cortical blindness**

As I tell clients, the eye is just the antenna that sends the light signals to the brain for interpretation, ie vision. In some cases the ‘antenna’ may be working just fine but the signal is not being read by the brain. This can happen for any number of brain lesions but it can also occur when there is liver disease resulting in a buildup of waste products that cause a metabolic neurological disease. Puppies can have liver shunts resulting in intermittent blindness and other neurological disease such as ‘head pressing’ in corners; this often happens to the patient around the time of feeding when waste products such as ammonia will be high. Tumors of the visual cortex will also cause blindness in a patient with an otherwise normal eye exam.
The cornea is a transparent structure that comprises the front fifth of the globe. The cornea functions to support the contents of the eye; it also refracts and supports light. The cornea is nourished by the tear film and the aqueous humor since it is avascular. The cornea is elliptical with a wider horizontal diameter than vertical, although in domestic animals the difference is minimal so the eye appears round. Corneal thickness may vary both based on the species and the area of the cornea measured; an average width for most species in 0.5mm. The cornea is highly innervated with the nerves originating from the long ciliary nerves, a branch of the ophthalmic division of the trigeminal nerve. Pain receptors are located in the superficial cornea and pressure receptors in the stromal area.

The transparency of the cornea results from its avascular anatomy, nonkeratinized surface epithelium maintained by a precellular tear film, absence of pigment, size and organization of the stromal collagen fibrils. The physiologic state of the cornea also affects transparency, for instance lack of a functioning precorneal tear film will lead to vascularization, pigmentation, and scarring. The anatomic layers of the cornea are 1. The epithelium, 2. Bowman’s layer (rarely present) 3. the stroma, 4. Descemet’s membrane, and 5. the endothelium. Different types of collagen and glycosaminoglycans form a very precise and ordered arrangement creating corneal transparency. The stroma of the cornea occupies 90% of the cornea. The cornea is 75-85% water, but is relatively dehydrated compared to the rest of the body. The state of deturgescence is derived from active movement of water out of the cornea by endothelial cells. Energy dependent Na+/K+ adenosine triphosphate pumps, as well as carbonic anhydrase, are active in moving the water. The presence of specific glycosaminoglycans allows the pumps to function effectively. The epithelium acts as a physical barrier to movement of water into the cornea. Damage to the endothelium or epithelium, changes in the glycosaminoglycans, or even pressure on the cornea may change the spatial orientation of the precise collagen organization leading to corneal opacity.

Based on published studies removal of the corneal epithelium will allow absorption of water into the cornea; over 24 hours the cornea will increase in thickness by 200%. Removal of the corneal endothelium results in a much greater change leading to a 500% increase in thickness as the permeability of the cornea increases by sixfold. Both are important in maintaining deturgescence, but the endothelium is far more critical. Descemet’s membrane is the basement membrane of the endothelium and is a very thin acellular structure. It is comprised of acellular collagen and this may be why stromal and epithelial cells do not migrate over Descemet’s membrane very well thus creating a very unstable corneal ulcer. The last layer of the cornea is the endothelium. It is a single cell layer thick with little to no regenerative ability. As the cornea ages and the endothelial cells die or stop functioning the ability of the cornea to maintain deturgescence is affected. In the young dog endothelial cell density is 3000cells/mm²; when the cell density decreases to 500-800cells/mm² the cornea decompensates and corneal edema develops. This may be due to age-related degeneration, inherited abnormalities, from trauma, or other metabolic events.

Ulcers are a commonly treated corneal issue. The majority of ulcers will heal in 4-5 days with a first level antibiotic such as neomycin-bacitracin-polymixin or gentocin treatment. If an ulcer does not heal within 5 days, then the underlying cause needs to be determined to facilitate treatment and resolution. Complicated corneal ulcers need upper tier antibiotics, oral antibiotics and additional medications such as serum to achieve healing. Common causes of complicated corneal ulcers are keratoconjunctivitis sicca, epithelial and/or endothelial degeneration affecting healing, infectious agents, and physical irritants such as ectopic cilia. Even superficial corneal ulcers such as indolent ulcers may be complicated ulcers.

Ulcers that appear malacic or infected may need treatment with multiple antibiotics. Cytology and a culture and sensitivity are recommended to identify the best antibiotic treatment. Additional therapy for these ulcers may include serum, topical tear replacement, oral antibiotics, oral anti-inflammatories, and analgesics. Two oral antibiotics may be indicated, one based on the culture and sensitivity result and the second, doxycycline for anti-protease activity against matrix metalloproteases that can create further malacia. Higher tier topical antibiotics include medications such as late-generation fluoroquinolones, e.g., Vigamox; empiric combinations such as an aminoglycoside with a cephalosporin, e.g., cefazolin-fortified tears are also good for more complicated corneal ulcers until the culture and sensitivity results are received. If the ulcer progresses or has a prolonged course surgery is indicated.

Indolent ulcers are superficial corneal ulcers with a prolonged, frustrating course. The attachment of the epithelium to the underlying stroma may become dysfunctional resulting in a nonhealing superficial ulcer. A hyalinized zone develops between the epithelium and the anterior stroma. Stromal fibroplasia develops underneath the hyalinized zone. Previous studies have shown that topical tetracycline ointment may facilitate healing. Sodium chloride ointment may decrease the corneal edema that usually develops with this ulcer. Topical Adequan, diluted 1:1 in artificial tears solution and applied q8hours may stimulate healing. Overall topical medical treatment has less than a 50% success rate; the majority of these ulcers require a procedure such as a keratotomy to accomplish healing.
Viral keratitis and ulceration occurs most commonly in cats, however viral ulcers have been documented in dogs, especially immunosuppressed patients. Viral ulcers in cats may present as a classic dendritic ulcer or as a geographic corneal ulcer. Dogs may present with dendritic ulcers or develop non-ulcerative keratitis in the peripheral cornea. For both species topical treatment with 0.1% idoxuridine (4-8 times daily application), 0.5%-1% trifluridine (4-6 times daily application), or 0.5% cidofovir (twice daily application) is recommended. Cats may also benefit from oral famcyclovir (62.5-250 mg PO bid). At this time the efficacy of oral lysine is under investigation so the author does not routinely prescribe this medication. Topical antibiotics as well as oral antibiotics may also be indicated for ulcerative corneal disease.

Fungal infection is also a cause of corneal ulceration. Cytology, as well as culture and sensitivity are important in identifying the active organism. Topical miconazole, 1 or 2% may be empirically started until results are back. Prescription topical antifungal medications are costly. Antibiotic treatment should be used concurrently as co-infection is common.

Cats that present with corneal ulcers may progress to develop a sequestrum f the ulcer does not heal in a timely manner. FHV-1, as well as chronic irritation, e.g., from trichiasis, may lead to corneal ulceration and sequestrum formation. Generally, once a sequestrum forms surgery is needed to resolve it.

Non-ulcerative corneal disease may occur for many reasons. Immune-mediated keratopathies are common in both dogs and cats. Chronic superficial keratitis or pannus affects many German Shepherds and Greyhounds; however, individuals of other breeds or mix’s may be affected. Treatment consists of an immunosuppressant medication to address the afferent arm of the immune system and block the immune response. Additional short- or long-term treatment with an anti-inflammatory to address the clinical inflammation is also needed. Tacrolimus or cyclosporine in conjunction with a topical steroid or nonsteroidal anti-inflammatory (NSAID) are used. Corneal epithelial degeneration may occur primarily or as a progression of inherited or acquired corneal dystrophy. Exposure or poor tear film quality may be a precursor to this abnormality. Previous corneal trauma or scleral inflammation may also result in corneal degeneration. Lipid deposition or lipid keratopathy presents in a similar manner. Treatment with a topical tear supplement and a nonsteroidal anti-inflammatory may decrease the density of the corneal deposits and degeneration. If tear film quality issues are also present a lacrimostimulant such as cyclosporine may also improve the health of the cornea and the epithelium overlying the degeneration.

Corneal dystrophy may develop from an inherited etiology, or as an acquired disease. Metabolic abnormalities such as poor thyroid function, Cushing’s disease, diabetes mellitus, etc. Tear film abnormalities, both quality and quantity may also be causative. Treatment as for corneal degeneration is recommended.

Pigmentary keratitis is another form of non-ulcerative keratitis that evolves from chronic corneal irritation. It often has a multifactorial etiology and occurs when peri-limbal melanocytes migrate into the cornea and deposit melanin granules within corneal epithelial cells. Treatment involves diagnosing the etiology, correcting it, and preventing progression. Tear production should be monitored over time in these patients as keratoconjunctivitis sicca is often a contributing factor. Surgical removal of pigment has been performed but often pigment recurs. Treatment of accompanying keratitis with a topical anti-inflammatory and treatment with a lacrimostimulant is recommended.

Corneal endothelial degeneration resulting in corneal edema may occur as an inherited defect. The Boston terrier, dachshund, and Chihuahua are predisposed breeds. Age-related changes, trauma including corneal ulcers, and uveitis may also be an etiology. Corneal endothelial cells form the single cell layer endothelium; these cells have little to no regenerative capability. Hypertonic saline, either drops or ointment, applied q 8-24 hours will help to dehydrate the corneal stroma and control the disease. Treatment with a topical anti-inflammatory will address the associated corneal inflammation. Progressive or nonresponsive corneal edema may result in so much fluid in the corneal stroma that bullae develop. Bullous keratopathy may be the end stage of corneal endothelial degeneration. At this point surgery is needed, such as thermo- or laser keratoplasty. Gunderson flap placement may also improve and control corneal edema.

Corneal epithelial inclusion cysts and dermoids are additional corneal abnormalities. Cyst formation may be treated medically with nonsteroidal anti-inflammatories. If significantly large surgical removal may be indicated, however they are benign. Dermoids are congenital abnormalities and are usually located adjacent to the limbus. If small they may not need to be removed. If the dermoid is large or has associated trichiasis then surgical removal is indicated.

Corneal masses need to be biopsied and additional treatment based on the definitive diagnosis.

References
Glaucoma is a group of diseases that result in an elevated intraocular pressure (IOP) and blindness. Aqueous humor (AH) dynamics play a key role in the development of glaucoma. AH is produced as a filtrate of blood by the cells of the ciliary body. AH provides nutrition, oxygen, and removes waste from the non-vascular tissues of the eye, such as the lens. AH is continuously produced and flow is not auto-regulated due to the reliance for survival of the tissues of the eye on it. If the AH cannot “drain” out of the eye normally and becomes backed up then the IOP will increase. Visually it is a matter of a constantly flowing faucet with a partially to completely clogged drain.

Closed angle glaucoma is the most common form of glaucoma in dogs. Gonioscopy is used to evaluate the width of the iridocorneal angle (ICA) as well as the anatomy of the pectinate ligaments. The pectinate ligaments should be well differentiated with drainage space in between them. If the ICA width is narrow to closed AH outflow will be compromised. If the pectinate ligaments are not well differentiated compromise will also occur. The worst scenario occurs when the ICA width, as well as the pectinate ligament structure is abnormal; this would be termed goniodysgenesis with a closed or narrow angle and pectinate ligament dysplasia. The Burmese cat has inherited narrow/closed angle glaucoma and the Siamese cat breed may have pectinate ligament dysplasia.

The majority of canine patients develop glaucoma in middle age with females the more frequently affected gender. Potential hypotheses for the development of glaucoma in middle age include decreased metabolism of an already compromised iridocorneal angle, subclinical uveitis leading to clogging of a compromised angle, microscopic increases in the dimension of the lens as it becomes sclerotic pushing the iris forward and impacting an already compromised angle, reverse pupillary block, etc. Reverse pupillary block occurs when the tone of the iris decreases during sleep and the iris flops back and comes in contact with the lens. A temporary adhesion occurs and AH is blocked and cannot circulate through the pupil to the anterior chamber and then drain out. There are many other possible hypotheses, but these are some of the current ones. Recent research suggests that a cascade of events begins leading to the predisposition of further glaucoma episodes following the initial event; one glaucoma episode may affect the long and short posterior ciliary arteries compromising blood flow, including oxygen and nutrients to the eye and ciliary body. In an already metabolically or anatomically compromised eye this is a dangerous development.

Damage to the retinal ganglion cells and the optic nerve (ON), made of the axons of the ganglion cells results through several different modalities. Traditionally we think of primary glaucoma with a closed iridocorneal angle, compromise to aqueous humor outflow, an increase in IOP, damage to the retina and ON leading to blindness. Primary glaucoma may occur in other ways as well. Normotensive and open angle glaucoma are other primary glaucoma categories.

As more research is completed on glaucoma some of the dynamics of open angle glaucoma have been described. Changes occur in the trabecular meshwork of the ICA leading to blockage there even though the angle appears open on gonioscopy. There are other changes on a molecular level within the optic nerve also.

Several smaller spikes that resolve by themselves or are misdiagnosed as conjunctivitis by the owner or primary care veterinarian precede many major glaucoma spikes. Every red eye should have an IOP measurement! Several tonometers are currently available and all are accurate within the normal range of IOP. As the IOP increases most tonometers slightly underestimate IOP; as IOP decreases the tonometers overestimate IOP. For dogs the normal IOP range is approximately 8mm Hg to 25mm Hg, although generally an IOP over 20mm Hg requires further diagnostics and treatment. Cats have a little more range with the end of normal in the upper 20’s.

The signs of glaucoma in dogs are hyperemic conjunctiva and injected sclera. Over an IOP of 40mm Hg the corneal endothelial pumps fail and the cornea becomes edematous. The pupil usually dilates unless pupillary block or uveitic glaucoma is present. The third eyelid may be elevated secondary to pain. Blepharospasm, tearing, and rubbing may also occur. Cats have more subtle signs of glaucoma; often the only sign is mydriasis. Anterior chamber depth is also an indicator in cats; too shallow or too deep are both indicators of AH issues. Feline glaucoma tends to have a slow, insidious onset so presentation is generally later in the disease process.

If a glaucoma spike occurs medical treatment is usually the initial treatment. Hyperosmotics include mannitol and glycerin. Mannitol (1-1.5 gm/kg iv) is a larger molecule so has more osmotic draw and is the more effective medication. Glycerin (0.75 of body weight in lbs = mls PO) may be administered and can be effective, especially if the glaucoma is not uveitic. Mannitol starts working within 30 minutes and lasts for approximately 4 hours. Glycerin has a slightly shorter duration of action. During that time food and water should be withheld. After 4 hours small amounts of water can be re-introduced. After the patient is consuming water normally food can be offered.

Other medical glaucoma treatment includes 1) medications to decrease the production of aqueous humor, 2) medications to increase outflow through the ICA or conventional route, 3) medications to increase outflow through the uveoscleral or unconventional route.
Prostaglandin analogues are also important in early emergency therapy. Xalatan (latanoprost) and Travatan are the major topical medications used on an emergency basis to treat elevated IOP. They work by maximizing outflow through the uveoscleral route, the alternative outflow tract. These two medications produce miosis as a side effect. Neither is that effective in cats; they do not have the appropriate receptor to bind Xalatan or Travatan and only 3% of AH exits the eye by the uveoscleral route in cats. Generally, either medication should create an IOP decrease in 30 minutes in dogs. Usually PM dosing or bid dosing is used. The other long-term effect is on the trabecular meshwork. With consistent use remodeling of the meshwork occurs that allows greater AH outflow.

Carbonic anhydrase inhibitors are another mainstay of therapy. Dorzolomide or Trusopt and brinzolomide or Azopt are topical medications. Methazolamide and the older acetazolamide are oral medications. These medications work by decreasing the production of aqueous humor. Timolol also decreases the production of aqueous humor, however the exact mechanism is not known. Timolol and dorzolomide may be combined into one drop, Cosopt. Topical medications containing timolol should be used with caution in small patients or patients with cardiac and pulmonary disease as it is a beta-2 blocker. Betaxolol may be used instead as it is more specific in targeting beta-1 receptors.

Other medications include demecarium bromide, both 0.125% and 0.25%, and pilocarpine. These are both miotics and constrict the pupil pulling iris tissue away from the ICA with the goal of allowing a larger exit space for aqueous humor. Pilocarpine in particular is irritating with a short half-life. Epinephrine or Dipivefrin is also an older glaucoma medication that slightly decreases aqueous humor production and increases outflow.

Many patients are acutely refractory to medical therapy or slowly become non-responsive as the disease progresses leading to elevated IOP’s. Visual patients will need a procedure to decrease the production of aqueous humor; some also need increased aqueous outflow capability as well. The state of the art therapy is endolaser cyclophotocoagulation and glaucoma valve placement. Internal lasering (80-90% plus success rate) is more effective than external or transcleral laser (50-60% success rate) therapy. All therapies to decrease aqueous humor formation lead to post-operative uveitis and the potential complication of cataract formation.

Phacoemulsification is performed with endolaser treatment so the complication of cataract development is removed. All surgeries also have the complication of secondary glaucoma associated with post-operative inflammation. Generally, once the ciliary body has healed and the uveitis resolved the risk of immediate post-operative glaucoma is fairly low. Placement of a glaucoma valve may be performed first to address the possible post-operative IOP spikes. Over the last 1-2 years more ophthalmic surgeons are performing goniouvalve placement first with increased success rates since the immediate post-operative IOP spikes are controlled.

Comfort surgeries include enucleation, evisceration with intrascleral prosthesis placement, ciliary body ablation, and cyclocryotherapy. The first two have a 99% success rate and the latter two 80-85% success rate.

The newest frontier in glaucoma treatment is trying to prevent retinal and optic nerve damage. As retinal cells die calcium channel function is affected. Calcium channel blockers such as amlodipine may help to prevent neural damage when IOP’s are elevated. Gabapentin also decreases voltage-gated calcium channel current. Namenda (memantine) is used in Alzheimer’s disease treatment. Its neuroprotective properties have been shown to prevent damage to retinal cells during IOP spikes. Tramadol is a NMDA antagonist and may block destructive biochemical interactions in retinal cells. Many other medications have promise in glaucoma therapy; in general, these drugs target molecular interactions in the cell.

Other forms of glaucoma such as secondary, pigment dispersion, and aqueous misdirection glaucoma utilize many of the drugs and surgeries mentioned above. Secondary glaucoma often occurs from uveitis; treatment involves symptomatic therapy and addressing the cause of the uveitis, often an ocular manifestation of systemic disease. Anti-inflammatory therapy and antibiotics may play a major role in therapy.

Cats develop secondary glaucoma most commonly. Cats also develop aqueous misdirection or malignant glaucoma. The AH is misdirected posteriorly into the vitreal cavity through small breaks in the hyaloid face near the vitreous base; increased vitreal pressure leads to anterior displacement of lens and iris. Carbonic anhydrase inhibitors are the favored therapy. If medical therapy doesn’t work phacoemulsification with anterior and posterior capsulotomies and vitrectomy has a favorable outcome.

Pigmentary glaucoma may develop in Golden retrievers and other breeds. It has an inherited basis in Golden retrievers. Pigment dispersion and uveal cyst formation are factors in pathology and progression; the role of uveitis in the etiology is still in question. The uveitis may follow the dispersion of pigment. Cairn terriers also develop pigmentary glaucoma that has a poor prognosis; current surgical therapies are not effective. Cats also may develop glaucoma secondary to the pigment accumulation associated with diffuse iris melanoma. This tumor has a poor prognosis if glaucoma develops. The globe should be enucleated and submitted for histopathology. Additionally, further work-up to assess metastasis should be performed.

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**Corneal Surgery**  
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Surgery techniques continue to evolve with the goal of maintaining function and clarity of the cornea. Most surgeries are performed under the operating microscope for magnification and visualization. Microsurgical instrumentation is used with 8-0 or 9-0 suture material. Culture and sensitivity, as well as cytology should be performed on infected corneas to initiate the appropriate post-surgical treatment. A corneal ulcer that is 50% or greater depth in the cornea is considered a surgical ulcer. Descemetoceles and corneal perforations require surgery and are more emergent than a deep corneal ulcer. Other abnormalities that usually require surgery are corneal sequestrum formation, corneal lesions compatible with neoplasia, and developmental or acquired lesions with resultant visual impairment.

Classic surgeries utilizing conjunctival grafting or flaps are still common, although typically more scarring is seen with this procedure. A modified conjunctival; third eyelid flap may be performed with head loupe magnification and has acceptable results in respect to healing although overall corneal clarity is decreased. This procedure seems to be more successful in cats, but many dogs have been successfully treated as well.

Pedicle grafts, island grafts, hood grafts, and bridge grafts may be used for tectonic support, availability of a vascular supply, and to maintain corneal integrity. The grafted conjunctival tissue will heal and develop inflammation. Once healing is complete anti-inflammatory will facilitate graft contraction and improve clarity. Usually the harvested conjunctiva is autogenous tissue.

Corneal grafting techniques include corneocutaneous transpositions, corneoscleral transpositions, and corneal grafts. The first two are autogenous, however corneal grafts may also be from non-autogenous donor tissue. Transpositions are advantageous both because they are autogenous tissue and final visual acuity is better than using conjunctival tissue. The cornea adjacent to the lesion is advanced into the defect and the attached conjunctiva is also moved forward so the cornea has a blood supply. The clear cornea is now in the lesion area and the attached conjunctiva remains peripheral adjacent to the limbus so healing is facilitated and cosmesis and vision are better.

Conical grafts may be autogenous, allogenic, or transgenic. The abnormal cornea is removed using a penetrating keratoplasty and a donor button, typically 1mm larger than the defect is positioned in the defect. After suturing the graft into place post-operative medications should include an immunosuppressant to decrease the risk of rejection.

Ammonium is being used as a grafting material. Theoretically, this is stem cell-like tissue being used to treat the corneal abnormality. It is antifibrinogenic, anti-angiogenic, anti-proteolytic, avascular, and contains growth factors. Amnion acts as a substrate for epithelial growth and reinforces adhesion of basal epithelial cells. It promotes epithelial differentiation, prevents apoptosis, precludes white blood cell infiltration, prevents fibroblast activation into myofibroblasts. This technique has a good success rate and results in good vision and cosmesis. Corneal ruptures and infected ulcers do not respond as well to ammonium grafting. The ammonium needs to be prepared and stored appropriately to ensure viability. Once the amnion is thawed it must be used and cannot be re-frozen. Pericardial tissue has also been used for corneal repair.

Biomaterials and scaffolds may also be used as a grafting material to encourage healing. They are readily available and aid the body in its own healing response by providing a matrix for cell attachment. Rapid cellular infiltration and vascularization of the corneal wound occurs with regeneration and proliferation of tissue. Scar formation is decreased. The biomaterial may be sutured into the cornea and used with another material such as conjunctiva to treat penetrating keratoplasties or for less severe corneal defects may be placed on the cornea and positioned using a third eyelid flap. The author has used biomaterial to address corneal malacia; the proteases and healing factors in the material have similar properties to serum in arresting corneal melting.

Corneal laceration repair can be fairly straightforward if a simple partial or full thickness laceration is present. For irregular lacerations or lacerations with iris prolapse or lens rupture anterior chamber irrigation and aspiration and/or cataract surgery may need to be performed. If an infection is present and the cornea needs significant debridement a concurrent corneal grafting technique may be needed to replace resected tissue. Plant material if embedded in the cornea often leads to corneal malacia, most likely from enzymes in the plant material. Both bacterial and fungal cultures may be warranted to identify appropriate medical therapy. Other foreign bodies may require extensive reconstruction as the foreign body should not be pulled out of the cornea, rather removed from underneath or irrigated out of the cornea. An incision may be needed adjacent to the foreign body to lift it from underneath and remove it from the cornea without leaving microparticulate material in the cornea that could lead to abscess formation. If the resulting lesion is deep enough or corneal malacia has developed around the foreign body a conjunctival graft or other material to restore normal corneal integrity may be needed. Culture and sensitivity, as well as cytology should be performed prior to placing material over the foreign body site. Copious irrigation should be performed prior to the grafting procedure.

Corneal abscess formation occurs when infectious agents get trapped in the cornea due to epithelialization isolating the infectious material. Appropriate medical treatment may be adequate to address the issue, but the abscess may develop and encompass the whole
Corneal stroma leading to a potential descemetocele or corneal rupture. If a deep ulcer develops secondary to the abscess the area needs to opened, debrided and lavaged. Culture and sensitivity, as well as cytology should be submitted. Then a graft may be used to restore the cornea and to preserve the eye. All of the above techniques work well for this type of corneal repair as long as an adequate vascular supply is provided for the repair.

Corneal adhesive therapy is another method of treating corneal wounds. It may decrease stromal melting by preventing leukocyte migration, however it is generally not used for deeper melting ulcers. The exothermic reaction after application may lead to perforation during polymerization. Adhesives are more commonly used to treat anterior stromal ulcers and refractory ulcers in dogs.

Keratectomy, superficial or deep is used to remove corneal lesions such as feline sequestrum, tumor, or in the treatment of refractory corneal ulcers. If the lesion is full thickness conversion to a penetrating keratoplasty may be necessary with adjunctive grafting techniques. Keratectomy may also be utilized to remove lesions from the cornea. Often once the keratectomy is performed an adjunctive technique such as a conjunctival graft may be utilized to improve tectonic properties. A keratectomy may be used to remove excess conjunctiva after symblepharon develops; in this case immunosuppressants and anti-inflammatories may be prescribed immediately post-operatively to decrease scarring during healing.

Other corneal surgery techniques involve treatment of corneal endothelial degeneration that is leading to bullous keratopathy. Felines may also develop bullous keratopathy from chronic oral steroid treatment. A thin conjunctival graft called a Gunderson flap may improve corneal integrity, as well as prevent recurrent ulceration and malacia. The flap is placed over an area of bulla, even though there is not a corneal stromal defect. The vascular properties of the graft provide an egress for the fluid leading to corneal edema from the corneal endothelial degeneration.

Other surgical treatment for bullous keratopathy in canines is a thermokeratoplasty or laser keratoplasty. These techniques utilize energy to dehydrate the cornea and create a very thin translucent layer of scar tissue to prevent the fluid associated with the corneal edema from creating bullae. This in turn will decrease the corneal edema by removing this access route for fluid entering the cornea when the corneal epithelium has healed and is sealed. The thickness of the cornea increases by 200% when the corneal epithelium is not intact. This treatment is generally reserved for advanced cases as it does create scar tissue and it can be painful post-operatively until healed. A bandage contact lens is often placed to aid in comfort and healing.

Artificial corneas may be embedded into the axial cornea to address severe corneal edema and bullous keratopathy. This is used for humans with severe corneal endothelial degeneration or Fuch’s corneal dystrophy, but is not as successful in animals.

Lastly, superficial chronic corneal epithelial defects can be a challenge to treat. Debridement is an initial technique to stimulate healing and the production of new micro-adhesion complexes between the corneal epithelium and anterior stroma. Other techniques include linear grid keratotomy, anterior stromal micropuncture, burr keratotomy, corneal adhesive treatment, and in the worst case scenario a superficial keratectomy.

References
Eyelid disease: Medical and surgical therapy

Where would a dog’s eyeballs be without eyelids to protect them and nourish them? A dog’s eyelids form a protective barrier over the globe. The lining conjunctiva provide a smooth surface for the cornea during the blink process. The associated mucous producing goblet cells create the building blocks for the tear film. The fatty glandular secretions of the meibomian glands prevent tear evaporation and protect the tear film. The wave-like motion of the eyelid closure helps to entrap and remove foreign material from the conjunctival sac, as well as smooth and evenly distribute the tear film and move it toward the lacrimal sac. eyelids are such small structures that work in concert to protect the eye and improve vision. Eyelid diseases are common in dogs and are a significant source of discomfort in many cases.

Eyelid diseases can be divided into the usual “DAMNIT V” categories. These diseases are challenging from a diagnostic standpoint for the general practitioner due to the lack of magnification. Knowing and understanding the anatomy of the eyelids will help determine the correct medical and/or surgical therapy. The eyelids surround the palpebral fissure, or eyelid opening. There is the upper eyelid, also known as superior or dorsal eyelid, and the lower eyelid, also known as the ventral or inferior eyelid. The outside edges, the medial and lateral canthus, anchor the eyelids and provide stability and shape. The medial canthal ligament firmly fixes the medial canthus and subcutaneous tissues and peristium. The lateral canthal region is more mobile and can be poorly developed. The retracto anguli oculi lateralis muscle aids the lateral canthal ligament. In many breeds, the lateral canthus is poorly developed to the point that it contributes to entropion. Muscles that open the eyelid are the Muller muscle, also known as the levator anguli ocularis medialis, (sympathetic fibers) the levator palpebral superioris muscle (CN III), the frontalis muscle ( upper eyelid, CN VII) the malaris and the pars palpebralis of the sphincter colli profundus muscles ( lower eyelid, CN VII). Closing the eyelid involves the circular orbicularis oculi muscle (CN VII), and the retracto anguli oculi lateralis which pulls the eyelid opening laterally during closure. The skin of the eyelids are very thin. Dogs have 2-4 rows that are irregularly spaced, of eyelashes or ciliae. Within the hair follicles, there are two types of glands, the gland of Moll, a modified sweat ( apocrine gland)and the gland of Zeis a modified sebaceous gland. These glands can become inflamed or abscessed, causing an external hordeolum or stye. Inside of the eyelid, the tarsal, or meibomian glands are sebaceous glands (holocrine type) that produce the very important outer lipid or oily fraction of the precorneal tear film. These glands range from 20-40 glands along each eyelid and their orifices, open onto the eyelid margin. In the corner of the eye, the lacrimal caruncle contains sebaceous glands. The eyelids are lined by conjunctiva and that conjunctiva is also covered by the oily fraction of the precorneal tear film to facilitate smooth eyelid movements against the globe. There are goblet cells in the conjunctiva that create mucin. Within the eyelids, the tarsal plate is a thin, flexible tarsus that provides a surface for muscle attachment. Eyelids are innervated by several branches of the trigeminal nerve for sensation, and the facial nerve for movement. The blood supply is primarily from the medial and lateral palpebral arteries but is also from deeper orbital blood vessels.

Blepharitis is an inflammation of the eyelids and it is often a multifactorial disease. The inflammation can be focal, or diffusely involve the entire eyelid. Self-induced trauma due to the severe discomfort of this disease, often causes secondary complications that can make the original cause difficult to diagnose. Chronic inflammation causes scarring and malfunction of the glands of the eyelids, which can lead to tear film abnormalities. A bacterial blepharitis can occur due to a juvenile pyoderma, or “puppy strangles” and is of staphylococci spp, but both staphylococci and streptococci spp will cause a diffuse eyelid inflammation. The eyelids will be edematous, hyperemic and may or may not contain pustules, or pyogranulomas. Treatment involves topical and oral antibiotic therapy. If this occurs for several days to week, the eyelids can become fibroed and eyelid function and muscle activity is altered. Entropion or ectropion can occur after several months that may require surgical intervention. Blepharitis can also be mycotic, parasitic, or immune-mediated. There are several autoimmune or immune-mediated diseases that can affect the eyelids. They can occur alone, or in association with other systemic conditions. Pemphigus type immune mediated conditions that affect the mucocutaneous junction can affect the eyelids, and a subset affects the medial canthus. Medial canthal ulcerative blepharitis affects just the medial canthus in the german shepherd, Dachshund and Kerry Blue Terrier. Blepharitis can also be caused as a drug reaction to aminoglycosides (neomycin and gentamicin are the most common) and dorzolamide. Changing antibiotic or glaucoma therapy and the addition of a topical steroid is often necessary and will resolve the condition. Treatment involves corticosteroids and occasionally additional immune suppression through cyclophosphamide or azathioprine. Meibomianitis is a chronic, common, often over-looked inflammation of the meibomian glands that can cause increased tear evaporation and then dry eye. It also causes a qualitative tear film deficiency. This can occur in allergic blepharitis as well. Treatment includes hot compresses or “soaks” with warm wet compresses to help improve expression of the meibomian glands. Topical corticosteroids in the short term and longer applications of topical antihistamines such as Naphcon A and Zaditor are often effective in treatment. However, meibomianitis is often a “managed” disease.
and not a completely curable condition. LipiFlow Heating system uses heat and pressure on the eyelids to treat meibomian gland
dysfunction and cystic eye diseases in humans.

Eyelid masses and benign neoplasia is very common in the dog. Inflammatory masses such as histiocytosis and nodular fascitis are
uncommon. Meibomian gland adenomas and adenocarcinomas and other epithelial tumors are the most common. These tumors are
benign, often crumbly tumors that will back up the gland and cause a secondary impaction or chalazion. Excision is usually curative.
Other eyelid tumors include melanomas, fibromas and papillomas. Melanomas are usually benign and excision is curative. Sometimes
cryosurgical alone or in combination with excision is also effective. Surgical removal of eyelid tumors depend on the size of the mass,
the extent of the eyelid involvement, and the site. Typically 25% of the eyelid length can be removed without losing function of the
eyelid. Closure is by 1 or 2 layers. Four points of contact at the eyelid margin with a “figure 8” suture or horizontal mattress suture to
achieve perfect apposition of the eyelid margin is recommended.
In the past hour, the anatomy and physiology of the canine eyelids was discussed along with the surgical procedures for minor eyelid neoplasms. This hour will focus on the more complicated reconstructive techniques for removal of larger masses and larger areas of the eyelid and forehead. These are techniques that require the removal of 1/3 or more of the length of the eyelid. We are going to focus on types of sliding skin grafts, the semicircular skin graft, and the more complicated mucocutaneous subnormal plexus flap and subdermal plexus graft. These blepharoplastic techniques can be modified as needed for the individual patients and are limited by the skill and imagination of the surgeon.

Lateral canthotomy
In this procedure, a modified lateral canthotomy can be used to lengthen the eyelid by a little bit. If the mass is right at the 25-35% involvement of the eyelid, and just a small lengthening of the palpebral fissure is necessary, then a modified lateral canthotomy can be performed. A triangle of skin is removed from the lateral canthus to permit a shift of lateral canthal skin into either the upper or lower eyelid. This skin must be lined by the adjacent conjunctiva, which can be undermined with Steven’s tenotomy scissors and used to line the canthal skin. The edge of the skin should be trimmed on an angle to help prevent trichiasis upon contracture during healing. The conjunctiva is tacked with 6-0 or 7-0 vicryl in a simple interrupted pattern. The knots can be buried or placed on the palpebral surface. The skin is closed with 4-0 to 6-0 simple interrupted, non absorbable, monofilament suture.

Semicircular skin graft
If the mass requires more than a small portion of the lateral canthus, a semicircular skin graft can be used to repair larger eyelid defects. These defects allow for restoration of the eyelid in defects that affect 40-60% of the eyelid. The eyelid mass is excised full thickness, using the 4-sided or “house” method. The skin excision at the lateral canthus is extended in a curvilinear fashion. A triangle of skin, matching the length of the eyelid that is removed along with the mass, is removed and the flap is slid medially into the defect. This “Burow’s triangle” of skin that is removed at the lateral end of the semi circular graft prevents the puckering of skin at this site. The lateral canthus needs to be undermined and the conjunctiva sutured to the new skin. The defect is closed as indicated above for the modified lateral canthotomy.

For these next procedures, it is important to remember the basic principles of reconstructive surgery. The base of the graft needs to widen more than the tip to assist in the perfusion of the most distal aspect of the graft. Ischemia is not your friend! The base of the graft needs to have the proper blood supply. Tissue grafts will contract, so the graft needs to be at least 0.5-1 cm wider and larger. The graft needs to be free of tension and should “sit” within the defect on its own accord, without tension. In the subcuticular space, dead space should be eliminated and sutures are placed to reduce the tension on the graft and to approximate the skin margins. The donor skin should be slightly everted but in perfect apposition with the surrounding skin. Simple interrupted sutures are placed using 4-0 to 6-0 non-absorbable monofilament.

Post operative oral antibiotic therapy is important and an E collar for two weeks is a must! Warm compresses that apply gentle pressure and some movement to reduce swelling and promote circulation to the grafts are recommended for at least one week.

Mucocutaneous subdermal plexus flap
- This procedure is also called the “Lip to Lid” and
- The flap is lifted as apedle and rotated medially and sutured into the eyelid defect
- The conjunctiva is spared to line the new eyelid
- Lid is made slightly larger and then “made to fit” into the defect.
- No clinical consequence to the shortening of the upper lip
- Lip movement can be reflected in the movement of the new eyelid

Axial pattern flaps
These grafts use the blood supply at the base of the ear. The caudal and rostral auricular arteries encircle the ear and come off of the carotid. The superficial temporal artery is another source. These grafts can be used to replace portions of the upper eyelid and medial canthus, as well as to close large resultant defects during orbital surgeries.

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The dilated pupil: what are the causes?

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The iris is part of the vascular tunic, and is an extension of the ciliary body that covers the anterior portion of the lens. It is a diaphragm that is derived from the neural crest during development and divides the anterior ocular compartment into the anterior and posterior chambers. The anterior portion of the iris is composed of a central pupillary zone and a peripheral ciliary zone. These two zones are separated by a demarcation called the iris collarette. The function of the iris is to control the amount of light that enters the eye, and it is used for accommodation, or focusing, in some species. During periods of reduced light, the pupil will dilate to allow more light to enter the retina and maximally stimulate the rod photoreceptor cells. The iris is divided into the anterior border layer, the stroma and sphincter muscle and the posterior epithelial layers. The iris dilator muscle is peripherally located in the posterior iris stroma. The iris is innervated by both sympathetic and parasympathetic myelinated and nonmyelinated fibers. The pupil size is determined by the balance of these two muscle groups and their respective innervation.

The iris sphincter muscle, a constrictor, is the strongest of the two, and is controlled by the Oculomotor Nerve, or CN III. The Oculomotor Nerve provides primarily parasympathetic control of the iris. The dilator muscles are innervated by sympathetic fibers. There are different alpha and beta receptors in the iris that cause different ocular effects in different species. Therefore, medications can act differently across multiple species.

The pupillary light reflex is made of several parts and represents 20% of the optic tract axons that bypass the lateral geniculate nucleus and visual cortex. When light hits the photoreceptors, the rods and cones, in the peripheral retina, that light is transmitted into electrical energy and the affarent pathway of that action potential travels through the retina to the optic nerve. The affarent pathway continues to the optic tract, and 20% peels off and dives deep into the pretectal area of the brain. The efferent pathway begins when the pretectal area extends to the anteromedian nuclei in the brain that is rostral to the Edinger-Westphal nucleus. These parasympathetic fibers travel to the ciliary ganglion via CN III and have their effect on the iris sphincter muscle. This causes the resultant constriction and miosis.

Causes for pupillary dilation (in no particular order)

1. Retinal or optic nerve disease
   a. It takes a significant lesion to cause this loss and often the PLR is the last to disappear, after vision, in disease of the retina that cause vision loss. In these diseases, the pupil may respond to light, but be slightly dilated in ambient room light. In cases of optic neuritis, there is often no movement of the iris. In dogs that did not develop an optic nerve normally (optic nerve hypoplasia) the PLR is absent. Cats with hypertension will have dilated pupils in ambient light that may or may not respond to brighter light, depending on the damage to the retina. Dogs with Sudden Acquired Retinal Degeneration have varying degrees of pupil constriction.

2. Oculomotor nerve paralysis- this can occur due to a space occupying lesion such as neoplasia or mass, or from orbital disease causing pressure on the nerve. A retrobulbar abscess, or orbital meningioma can have varying effects on the pupil size.

3. Sympathetic “override”- stress and epinephrine can cause the sympathetic nervous system to take over and actively dilate the pupil. Other stimuli for pupil dilatation are sudden body pain, asphyxia and other causes of anxiety.

4. Pharmacologic- suppose this dog saw another doctor previously and was treated with atropine for a corneal ulcer? Atropine will cause the pupil to stay dilated for 96-120 hours. Repeated administration can make it last even longer. Datura inoxia, or Jimsonweed will cause mydriasis with direct contact as well Nightshade and Angel’s trumpet.

5. Glaucoma- increased intraocular pressure will mechanically blow the pupil open. When the pressure exceeds the systolic ophthalmic artery pressure, an afferent pupillary defect develops due to the transient retinal ischemia. Iris sphincter function is impaired with pressurization and can even be noted at intraocular pressures below the diastolic ophthalmic artery pressure. Further sphincter impairment occurs with higher intraocular pressures. The iris sphincter muscle itself, and not the neuromuscular junction, appears to be effected by the elevated pressure. The iris dilator function seems to be relatively resistant to the effects of increased intraocular pressure.

6. Lens instability- if the lens is pushed forward, it will dilate the pupil and push it open as there is increased lens to iris contact.

7. Iris atrophy- as the iris sphincter muscle atrophies with age, the stroma will progressively thin and the pupillary margin will develop a scalloped, and sometimes “moth-eaten” appearance.
8. Synechiae- in cases of uveitis, or sometimes in cases of lens instability. Inflammatory cells, fibrin and fibroblasts will cause adhesions of the iris to the lens or peripheral cornea.

9. Iris hypoplasia or iris coloboma- Australian Shepherds and dogs with the merle gene can have developmental defects in the iris that will cause a dyscoria (abnormally shaped pupil) or lack of movement and adequate dilation. When it is necessary to determine if the pupil is dilated due to a neurologic problem, vs a mechanical problem, a parasympathomimetic, pilocarpine, can be given topically. Response to this medication rules out a primary iris sphincter problem such as iris atrophy, or synechia.