METROPOLITAN VETERINARY ASSOCIATES
ESTABLISHED 1986

newsletter

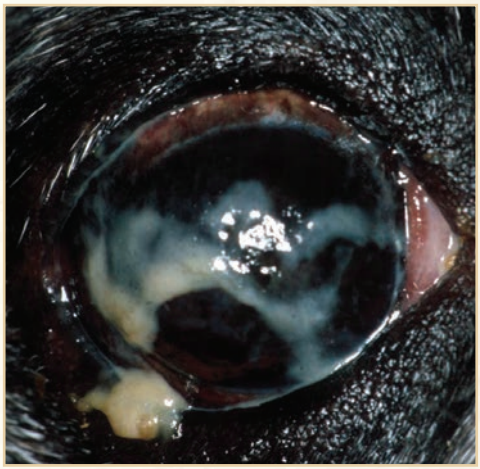
ISSUE: VOLUME 4 · NUMBER 1 SPRING – APRIL 2015

CANINE DRY EYE

CANINE KERATOCONJUNCTIVITIS SICCA (KCS)

Tear abnormalities are among, if not the, leading cause of canine ocular surface disease. Canine keratoconjunctivitis sicca (dry eye) can be an extremely frustrating disease to treat and manage. The response to therapy varies and patients may 'relapse', at times acutely, after years of successful management. This is a disease that requires life-long therapy, good client education and life-long monitoring.

Tear abnormalities can be grouped into quantitative and qualitative disorders. Quantitative tear deficiency can most often be diagnosed by performing the Schirmer tear test (STT). The STT I, most commonly used in clinical practice, is done without topical anesthesia and thus both the basal and reflex tears are measured. (The STT II uses topical anesthesia so only the basal tear level is measured.) The Schirmer tear test should be performed on any patient that presents with an ocular problem and must be performed prior to application of any eye drops (i.e. fluorescein stain or topical anesthetic). Although many 'dry eyes' are blepharospastic with conjunctival hyperemia and mucoid discharge, this is not always the case. Qualitative tear deficiencies are not as straightforward to diagnose. The tear film break-up time is a test that can be used to measure tear quality, however, it is not often done in clinical practice. The health of the precorneal tear film can be evaluated by the veterinary ophthalmologist using a slit lamp biomicrosope.



Conjunctival hyperemia, mucoid discharge, and diffuse corneal pigment causing vision loss in a dog with chronic KCS.

What is the precorneal tear film and why is it so important?

The precorneal tear film (PTF) is crucial for preserving ocular surface health. It provides the avascular cornea with nutrition (oxygen, glucose, electrolytes), lubricates the lids and ocular surface, provides protective antimicrobial proteins, immunoglobulins and growth factors, and removes debris and exfoliated cells.

The PTF is made up of three primary components: lipid, mucin and aqueous fluid. These components are intricately intermingled over the corneal and conjunctival surfaces. The lipid layer is secreted by the meibomian glands which line the upper and lower eyelid margins. Its main job is to prevent evaporation of tears and provide the stable and even dispersion of tears. Normal blinking and compression of the eyelids is important for release of lipid from the meibomian gland opening. The mucin layer is secreted by goblet cells within the conjunctiva, corneal and conjunctival epithelial cells, and the lacrimal gland. It decreases bacterial adherence, anchors the tear film to the corneal epithelium, lubricates and facilitates normal refraction. The aqueous layer is secreted by the orbital lacrimal glands and gland of the third eyelid. The aqueous portion of the PTF provides the cornea with nutrition, lubricates, and removes waste (bacteria, carbon dioxide, lactic acid, debris). Lacrimal gland secretion is controlled by the parasympathetic and sympathetic nerve system, hormones and various proteins (i.e. epidermal growth factor). Neurologic and hormonal control of these secretions is not well understood in dogs.

Ocular surface health requires more than normal tears. The eyelids work with the PTF to preserve the normal refractive quality of the cornea (i.e. vision) and protect the globe from injury. Many common ocular problems in some breeds, such as the pug, can be blamed in part for their physiologic exophthalmos preventing normal

SPECIALTY SPOTLIGHT

OPHTHALMOLOGY

Dr. Corr joined Metropolitan Veterinary Associates in September 2010 following completion of her residency in ophthalmology at the University of Pennsylvania School of Veterinary Medicine. She spent her summers during veterinary school at the University of Pennsylvania working as a veterinary assistant for MVA and it was always her dream to return as a specialist. Amanda truly enjoys all aspects of veterinary ophthalmology and working with companion animals. Her specific interests include eyelid surgery, cataract extraction and inherited ocular disease. Dr. Corr and Dr. Gross are at MVA 5-6 days a week.

Amanda Corr, VMD, DACVO

METROPOLITAN VETERINARY ASSOCIATES®
ESTABLISHED 1986

Providing Specialized Veterinary Services
& 24 HOUR EMERGENCY CARE

CARDIOLOGY
Michael Miller, MS, VMD, ABVP
Risa Roland, DVM, DACVIM (Cardiology)

DERMATOLOGY
Karen B. Farver, DVM, DACVD

EMERGENCY SERVICES
James Buckman, VMD, PhD
Jason Chamberlin, VMD
Robert Gaunt, VMD
Jennifer McGough, VMD
Meghan Romano, DVM
Marisa Suvannavejh, VMD
Dana Yard, VMD

INTERNAL MEDICINE
John V. DeBiasio, DVM, DACVIM
James F. Dougherty, MS, VMD
Leslie A. Kuczynski, VMD, DACVIM

NEUROLOGY
Jerry W. Northington, DVM

ONCOLOGY
Suzanne Rau, DVM, DACVIM (Oncology)

OPHTHALMOLOGY
Amanda Corr, VMD, DACVO
Stephen L. Gross, VMD, DACVO

RADIOLOGY
Robert C. McLear, VMD, DACVR
Lisa Suslak, VMD, DACVR

SURGERY
Lori W. Cabell, DVM, DACVS
A. Jon Nannos, DVM
Jacqui Niles, BVETMed, SAS, DACVS
Catherine Popovitch, DVM, DACVS, DECVS
Timothy M. Schwab, VMD, DACVS

HOSPITAL ADMINISTRATOR
Stacey Connell

HOSPITAL MANAGER
Suzie Weaver

AAHA ACCREDITED The Standard of Veterinary Excellence
AAHA ACCREDITED REFERRAL The Standard of Veterinary Excellence

blinking excursions. A common condition such as entropion becomes more complicated when one considers not only the consistent irritation from rolling in of the lids, but also the resultant abnormal dispersion of the tear film, and lack of normal lid compression. In any dog, maintaining homeostasis of the ocular surface also requires normal function of the very complex mucosal and ocular immune system.

What are the causes of KCS?

Certain breeds are more likely to develop KCS where a genetic component is suspected. This includes the American Cocker Spaniel, Cavalier King Charles Spaniel, West Highland White Terrier, English Bulldog, Pekingese, Lhasa Apso, Shih Tzu, Pug, and Yorkshire Terrier.

MOST COMMON UNDERLYING CAUSES OF KCS

Immune-Mediated: The definitive cause of KCS is not identified in most cases and may be classified as 'idiopathic KCS'. The overwhelming majority of cases are believed to be due to a tissue-specific (lacrimal gland) immune-mediated disorder. Lymphocytic-plasmocytic inflammation of the lacrimal tissue leads to glandular atrophy and reduced aqueous production.

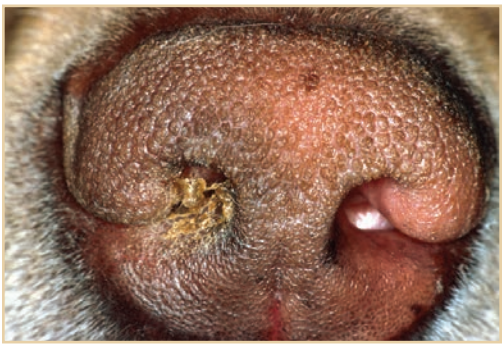
Congenital: Cases of congenital lacrimal gland hypoplasia and aplasia have been described most often in Yorkshire Terriers. These dogs present early in life most commonly with unilateral signs. The Schirmer tear test in these dogs is typically severely reduced (less than 5 mm/min).

Drug-Induced: The medications most commonly implicated in drug-induced KCS are sulfa drugs. This is an idiopathic reaction. In some dogs the reaction is transient and tear production improves once the medication is discontinued. In other dogs the damage is permanent. Dogs weighing less than 12 kg may be at significantly greater risk for developing KCS if sulfa drugs are used.

General and Topical Anesthesia: Preanesthetic and anesthetic drugs reduce tear secretion for at least 24 hours following their use. This highlights the importance of using post-operative, in addition to, intraoperative topical lubrication especially in breeds predisposed to KCS. Topical anticholinergics, Atropine being the most commonly used, are well known to reduce tear production.

Iatrogenic: The gland of the third eyelid can produce up to 30% of the aqueous layer which is why excision of a prolapsed gland is contraindicated. Removal of the third eyelid gland in the dog can lead to KCS, especially in breeds predisposed to the disease. Failure to surgically correct a prolapsed third eyelid gland can also lead to glandular scarring, atrophy and KCS.

Neurogenic: A primary loss of parasympathetic innervation to the lacrimal glands (CN VII) and/or loss of sensory innervation to the cornea (CN V) can also lead to KCS. These cases may have a history of orbital traumatic or inflammatory disease. Because the parasympathetic branch of the facial nerve innervates the lateral nasal glands, dogs with neurogenic KCS may also present with dryness and crusting of the ipsilateral nares.



Dry right nares in a dog with neurogenic KCS of the right eye.

Systemic and Metabolic Disease: Dogs with diabetes mellitus, hyperadrenocorticism, and hypothyroidism are more likely to develop KCS. Diabetic dogs have been shown to not only have reduced STT values but also alterations in the mucin component of tears and reduced corneal sensitivity. This can especially be a problem in diabetic dogs post-cataract extraction that are also challenged by tear film alterations secondary to general anesthesia. In one study, within two weeks after cataract surgery, diabetics were twice as likely to develop KCS compared to non-diabetics.

What are the consequences and clinical signs of poor tear health?

The ultimate outcome of tear film disease is ocular surface inflammation – keratitis and conjunctivitis – leading to cell damage, necrosis and a cycle of perpetual deterioration. Initially, the corneal and conjunctival epithelium become dehydrated and hypertonic. Eventually the corneal epithelium, subepithelium and underlying stroma become hypoxic. Irritation occurs due to lack of lubrication and resultant friction by the eyelids and third eyelid. Toxic metabolites (i.e. lactic acid) accumulate and microorganisms colonize. Clinically, within 12-24 hours, a normal eye with normal tear and adnexal function may develop acute keratoconjunctivitis and a deep melting ulcer or perforation.

Clinical signs will vary based upon the duration KCS has been present and the extent of dryness. In the early stages, the eye may be mildly hyperemic with no, little or intermittent mucoid discharge. These cases are often misdiagnosed as bacterial conjunctivitis and treated with topical antibiotics. Ointments may lead to some improvement due to the lubrication. However, as KCS progresses the clinical signs are ongoing and may become more severe. Corneal ulcers in these cases also tend to be extremely slow to heal or non-healing without treatment of the underlying primary problem of KCS.

With progressive disease, consequences may be severe. Chronic keratitis can lead to permanent corneal fibrosis/scarring and pigmentation which can lead to vision loss. Acute or chronic KCS may lead to corneal ulceration. Corneal ulcers

are more likely to become infected in the face of tear deficiency. Accumulation of mucoid discharge can lead to blepharitis and periocular dermatitis.

How is KCS diagnosed and how are the Schirmer Tear Test results evaluated?

Any patient presenting with an ocular complaint should have their tear production measured with a Schirmer tear test. The following is a general guideline for interpreting the STT:

>/= 15 mm/min = normal
11-14 mm/min = early or subclinical KCS
6-10 mm/min = moderate to mild KCS
</= 5 mm/min = severe KCS

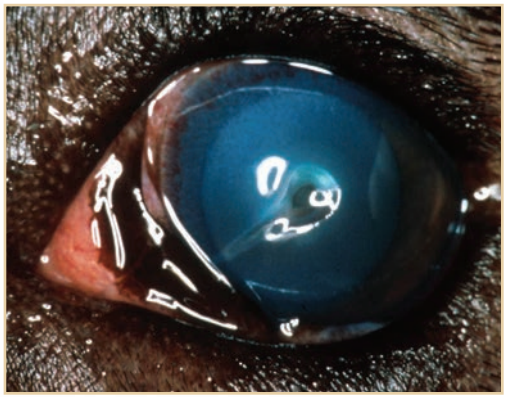
The STT needs to be evaluated in light of the clinical findings. For example, if a blepharospastic eye has a Schirmer tear test of 15 mm/min and stains positive for corneal ulceration, this would be considered abnormal. Although the STT value is within the normal range, one would expect epiphora, or excessive tearing (STT > 20mm/min), in an eye with corneal ulceration. Without treatment of the tear deficiency, healing is expected to be prolonged and recurrence of corneal ulceration is likely.

The use of Atropine in a patient with KCS creates a dilemma due to the fact that it is an anticholinergic drug and can reduce tear production. In eyes with KCS in addition to corneal ulceration or uveitis, atropine should be used judiciously or possibly not at all. This again highlights the importance of measuring tear production in any patient presenting with an ocular problem.

Tear production and Schirmer tear test values vary daily and weekly in canines. Therefore, in a patient with low normal tear production (i.e. STT of 15-16 mm/min), one must consider that tear production is likely to dip below normal at times. These patients may initially improve with tear replacement therapy (lubrication), however, specific treatment of tear deficiency is often necessary.

What is the therapy for KCS?

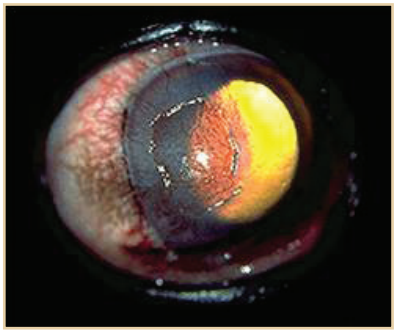
Treatment of KCS is specific to the individual patient and varies significantly on the underlying cause of tear deficiency, severity, ability to treat the patient, and client compliance. In general, most cases of KCS are treated with lacrostimulants, lacromimetics and anti-inflammatories.



Deep infected central corneal ulcer in a dog with acute severe onset of KCS.

Lacrostimulants increase the aqueous portion of the tear film. They can be divided in to cholinergic agents or, more commonly used, immunomodulating drugs. Pilocarpine is the cholinergic agent that can be used in cases of neurogenic KCS to stimulate tear production through the parasympathetic pathway.

Immunomodulating agents are commonly used and can be extremely effective in treating and controlling KCS. Cyclosporine A inhibits T-helper and T-cytotoxic cells. This allows T-suppressor cells, which sustain normal lacrimal function, to predominate. Cyclosporine A is frequently used to treat dry eye in people and its immunomodulating and tear stimulating properties have been well documented in dogs. Substantial increases in STT are most commonly observed within 3 weeks. Measuring STT three hours after administration of topical cyclosporine will give the most accurate assessment of response. Dogs with pre-therapy STT values of 2 mm/min or more have an 80% chance of improvement. Those with pre-therapy STT values of 0-1 mm/min have a 50% chance of responding. Systemic absorption of long-term topically applied cyclosporine (at appropriate concentrations) has not been shown to have a clinically evident alteration of peripheral cellular immunity.



Indolent superficial corneal ulcer with corneal neovascularization in a dog with untreated KCS.

Tacrolimus is an immunomodulating drug with a similar mechanism of action to cyclosporine. In vitro, it is 10-100 times more potent than cyclosporine. In dogs that do not have the desired response to cyclosporine, tacrolimus may provide superior tear stimulation.

Lacromimetics are most often used in conjunction with lacrostimulants. Lacromimetics can be used to replace deficiencies in all three tear components (aqueous, mucin, lipid). There is an overwhelming number of tear substitutes commercially available. The majority of these products are polymer combinations. Other products may be polyvinyl alcohol solutions, cellulose-based solutions, or viscoelastic containing solutions. Ointment formulations enhance ocular surface hydration and prolong contact time, however, these products may be challenging to apply. There are an increasing number of gel formulations which are preferable in the sense that they are easier to administer and contact time is improved over a drop. One of the most important things to consider when choosing a topical lubricant is that preservatives used in many of these products, such as benzalkonium chloride and chlorobutanol, cause epithelial toxicity and should not be used more than 6 times daily.

With today's arsenal of immune modulating drugs and lacromomimetics, surgery for KCS is infrequently necessary. Parotid duct transposition (PDT) remains the most common surgical therapy for KCS. In this procedure, the oral papilla of the parotid duct is transposed and attached to the lower conjunctival fornix. Unfortunately, saliva can be extremely irritating to the ocular surface leading to mineral deposition, periocular dermatitis and persistent irritation. Topical medication is needed to control these side effects and at times the duct requires partial or complete ligation (i.e. reversal of the PDT). Another possible complication is ductal occlusion. A less complicated surgical therapy that may be beneficial for dogs with KCS, especially brachycephalic breeds, is partial permanent tarsorrhaphy. The goal is to provide greater protection of the ocular surface and conserve existing tears.

METROPOLITAN VETERINARY ASSOCIATES & EMERGENCY SERVICES
2626 Van Buren Avenue
Norristown, PA 19403
ph: 610.666.1050 – fax: 610.666.1199

FRIDAY
4.10.15
by Denise Wyse, CVT and Nicole Thomas, BLS certified

Registration at 6:00PM

"INTRODUCTION TO THE RECOVER INITIATIVE: New CPR Guidelines for Veterinary Technicians"

2 PVMA credits for veterinary technicians

WEDNESDAY
5.13.15
By Amanda Corr, VMD, DACVO

Registration at 6:00PM

"Tear Disorders and Ocular Surface Disease in Dogs and Cats"

PVMA District 7 Meeting

For questions or to R.S.V.P. for any of these events please contact Sarah Spurgeon at events@metro-vet.com or 610.666.1050

METROPOLITAN VETERINARY ASSOCIATES®
ESTABLISHED 1986

2626 Van Buren Avenue
Norristown, PA 19403
610.666.1050 – fax: 610.666.1199
Metro-Vet.com

Paws of Promise Foundation

A 501(c)3 charitable organization

Join us on Thursday, May 21st for a happy hour benefitting the Paws of Promise Foundation!

DATE: Thursday, May 21st

TIME: 5:00pm – 7:00pm

LOCATION: Great American Pub, 123 Fayette Street, Conshohocken, PA 19428

ENTRY: $20 entry, includes two drink tickets and light appetizers

FOR MORE INFORMATION: https://www.facebook.com/events/726946444070417/

The Paws of Promise Foundation is a 501(c)3 charitable organization focused on helping good owners afford emergency and critical care for their pets in times of financial need.

Some of the pet's we've been able to help:

To learn more about Metropolitan Veterinary Associates' Paws of Promise Foundation, visit: Metro-Vet.com/PawsofPromise

Welcome Dr. Robert Gaunt

Metropolitan is happy to welcome Dr. Robert Gaunt to our Emergency Services team!

Dr. Gaunt attended University of Pittsburgh where he received his Bachelor of Science degree in Biology in 2009. After Pittsburgh he proceeded on to the University of Pennsylvania receiving his veterinary degree in 2013. Dr. Gaunt was accepted into a rotating medicine and surgical internship from Red Bank Veterinary Hospital in NJ. Upon completion in July 2014, he worked in a general practice until February 2015, when he joined Metropolitan Veterinary Associates. His special interests are Emergency Medicine.

In his spare time he enjoys Philadelphia sports, running, hiking, traveling, soccer and drawing. He resides at home with his four year old shepherd Skye, whom he rescued from North Philadelphia.