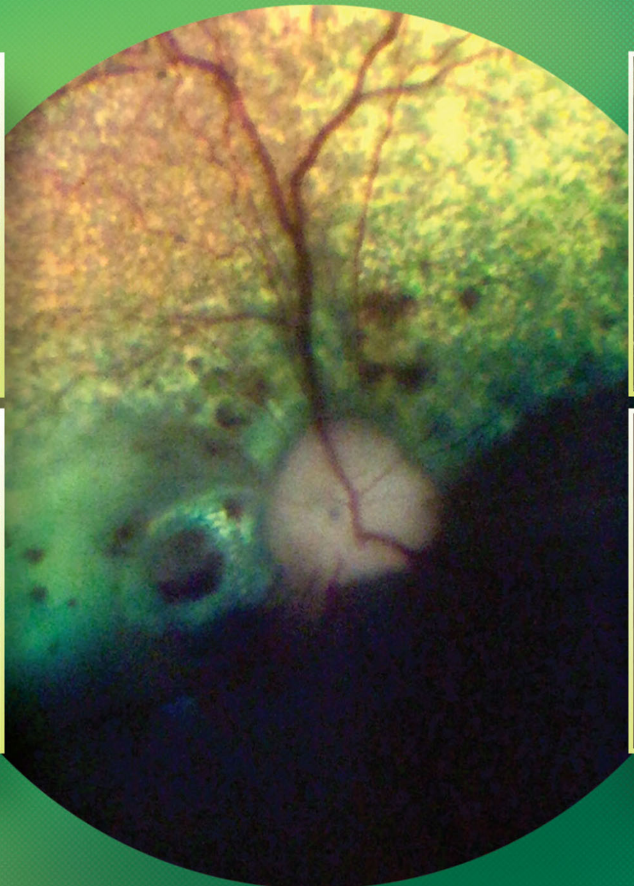
Second Edition

Clinical Atlas of Canine and Feline Ophthalmic Disease

Edited by

Douglas Esson • Sara Calvarese





WILEY Blackwell

CLINICAL ATLAS OF
CANINE AND FELINE
OPHTHALMIC DISEASE

CLINICAL ATLAS OF CANINE AND FELINE OPHTHALMIC DISEASE

SECOND EDITION

EDITED BY

DOUGLAS W. ESSON
Eye Care for Animals
Tustin, CA, USA

and

SARA CALVARESE
Tustin, CA, USA

WILEY Blackwell

This edition first published 2022

Edition History
John Wiley & Sons, Inc (1e, 2015)

Registered Office(s)
John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA
John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial Office
John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA

For details of our global editorial offices, customer services, and more information about Wiley products visit us at www.wiley.com.

Wiley also publishes its books in a variety of electronic formats and by print-on-demand. Some content that appears in standard print versions of this book may not be available in other formats.

Library of Congress Cataloging-in-Publication Data applied for

HARDBACK ISBN: 9781119665847

Cover image: Courtesy of Douglas Esson
Cover design by Wiley

Set in 10/12pt Minion Pro by Straive, Chennai, India
10 9 8 7 6 5 4 3 2 1

CONTENTS

PREFACE

It is some years since we made the conscious decision to further our post-graduate knowledge of the complex and fascinating world of veterinary ophthalmology. With the passing of the intervening years, we have had the immense good fortune to interact with a number of exceptional clinician-scientists, all of whom have generously shared their time, experience, and knowledge with us. In particular, we are indebted in this regard to Drs. Peter Bedford, Randy Scagliotti, Kirk Gelatt, David Wilkie, Paul Miller, Phil Pickett, Ivan Schwab, Dick Dubielzig, Bill Dawson, and Mark Sherwood.

We are frequently asked to give lectures about various ophthalmic topics to audiences ranging from students to experienced ophthalmologists, and it is most commonly through photographic images, that we are able to share our thoughts and experience.

The field of veterinary ophthalmology, detailed ophthalmic texts, and the peer-reviewed ophthalmic literature represent a sometimes-challenging and potentially confusing arena and as a consequence, we have sought here to provide the busy general practitioner with a clear, systematic, repeatable clinical picture of the most frequently encountered ophthalmic conditions in small animal practice. Images are intentionally presented in the same way as cases would be encountered.

In order to expand this project beyond that of simply an image atlas, we have also tried to provide clear, concise, updated, and clinically relevant information as it pertains to each of these conditions. This information has been supported with small number of relevant references for those to wish to read further.

ACKNOWLEDGMENTS

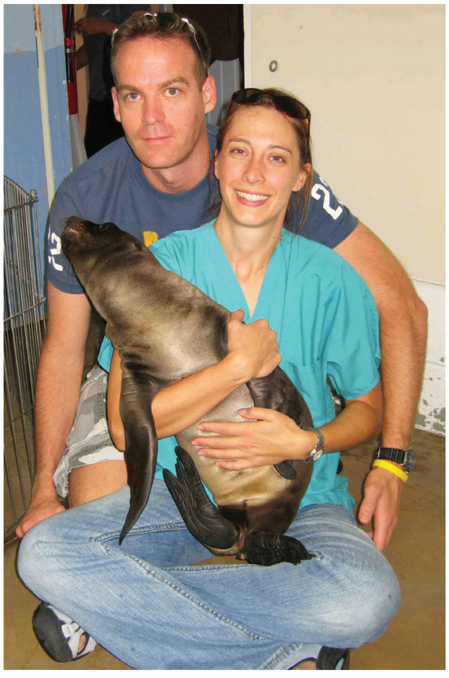
Many individuals have contributed to the development of this book:

The project, from its inception, has been shaped by our editors and publishers at Wiley, with special thanks due to Erica Judisch and Merryl Le Roux.

The images within this volume have predominantly been sourced from own collection; however, we have relied on friends and colleagues to discuss clinical presentations, help source cases (and in some instances provide images) in order to achieve completeness. In this regard we would like to acknowledge Drs. Anne-Michelle Armour, Randy Scagliotti, Al MacMillan, David Wilkie, Matthew Fife, Anastasia Komenou, Peter Bedford, David Williams, David Donaldson, Emily Moeller, Joanna Norman, Keith Collins, Nick Millichamp, Mark Haskins, Gwen Lynch, Amy Knollinger, and Kristina Narfstrom.

We are additionally grateful to a number of the affiliated specialists with whom we work, for their willingness to source and discuss cases commonly presented to the services of internal medicine, dermatology, oncology, and surgery. In this regard, we would particularly like to acknowledge Drs. Wayne Rosenkrantz, Colleen Mendelsohn, David Bommarito, and Tony Cambridge.

For Justin & Emily
With all our love
Doug Esson & Sara Calvarese
California, 2021



Section 1

Anatomy and Physiology

CHAPTER 1
NORMAL OCULAR ANATOMY

The orbit – comprising bones, connective tissue, lacrimal and salivary glandular tissue, adispose tissue, blood vessels, and nerves.

The eyelids (comprising skin, orbicularis oculi muscles, deep tarsal and superficial conjunctival tissue). These tissues also contain mucous-producing goblet cells, lipid-producing meibomian glands, and the openings of the nasolachrymal drainage system.

The third eyelid, comprising a T-shaped cartilaginous structure, which surrounds the lachrymal gland of the third eyelid and is covered with conjunctival tissue.

The external shell of the eye, comprising the scleral tissues posteriorly and cornea anteriorly. The cornea is composed of an outer epithelium (with its basement membrane), central stroma, and underlying endothelial layer (with its basement or "Decemet's" membrane).

The uveal tract, composed of the anterior iris and ciliary body and the posterior choroid, provides vascular supply to the retina.

The lens, which is suspended from the ciliary body by zonular ligaments and surrounded by the lens capsule.

The neuroretina which comprises the photoreceptors as well as multiple layers of supporting neuroglia and the retinal ganglion cells which coalesce to form the optic nerve which exits the globe posteriorly, through the porous lamina cribrosa.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

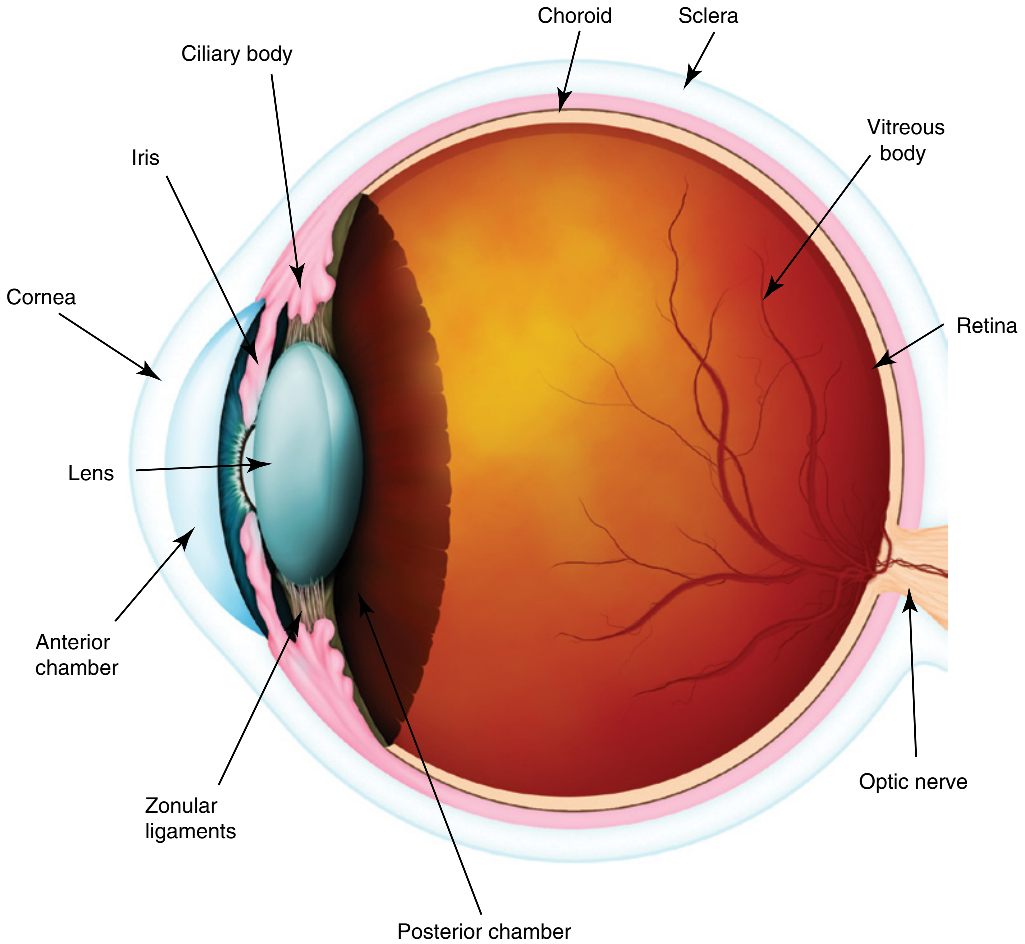


Figure 1.1 Normal Ocular Anatomy.

CHAPTER 2
ORBIT

The orbit comprises the incompletely enclosed space in which the globe and associated adnexal tissues are located.

The external boundaries of the canine and feline orbit are defined by the frontal, lacrimal, zygomatic, temporal, presphenoid, palatine and maxillary bones. The orbit is completed externally by the zygomatic arch (arising from the zygomatic and temporal bones) and the orbital ligament.

The orbital contents are enclosed in periorbital connective tissue which is reflected over the extraocular muscles, nerves, vessels, and globe as Tenons capsule. This connective tissue is contiguous with the orbital bony periosteum cranially and the cranial dura mater posteriorly at the level of the optic canal and orbital fissure.

As well as the globe, the orbit contains the connective tissues, a variable fat pad, nerves blood vessels, and extraocular muscles.

The extraocular muscles comprise the four rectus (dorsal, ventral, medial, and lateral), two oblique (dorsal and ventral), and single retractor bulbi (which inserts around the optic nerve at the posterior pole).

The dorsolateral orbital space also contains the orbital lacrimal gland.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

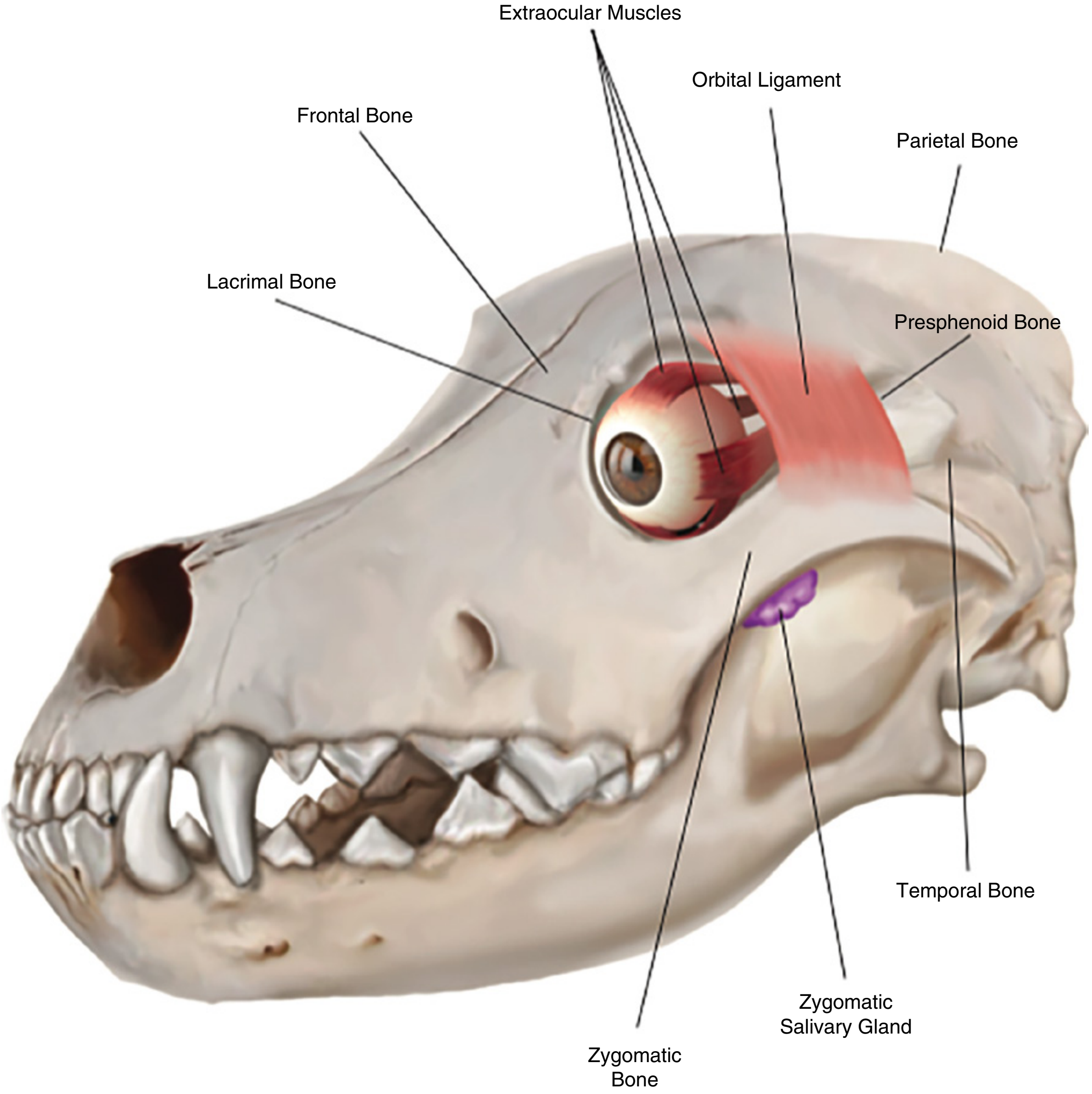


Figure 2.1 Anatomy of the Orbit.

CHAPTER 3
EYELIDS

The palpebral fissure is defined by the upper and lower eyelids (palpebrae).

The medial and lateral canthi are supported by the medial and lateral palpebral ligaments, which attach to the frontal and zygomatic bones, respectively.

External skin transitions to palpebral conjunctiva at the level of the eyelid margins (which also contain the tarsal/Meibomian glands), with bulbar conjunctival tissues continued and reflected across the globe to the level of the corneoscleral limbus. The upper lids bear sensory cilia (eyelashes).

The medial canthus contains a thickened caruncle of tissue, typically covered with finely haired skin.

Beneath the eyelid skin and connective tissues, the palpebral fissure is surrounded by the orbiculatris oculi muscle which is attached medially to the medial palpebral ligament and laterally to the retractor anguli oculi muscle. The orbicularis oculi serves to close the palpebral fissure.

In addition, the levator palpebre superioris muscle extends from the deep orbital tissues to the upper eyelid and serves to elevate the upper lid.

Finally, the medial canthus contains the nictating membrane (third eyelid), comprising a fold of palpebral conjunctiva supported by a T-shaped cartilaginous frame. This structure contains the lacrimal gland of the third eyelid and serves to maintain and protect the precorneal tear film and corneal surface.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

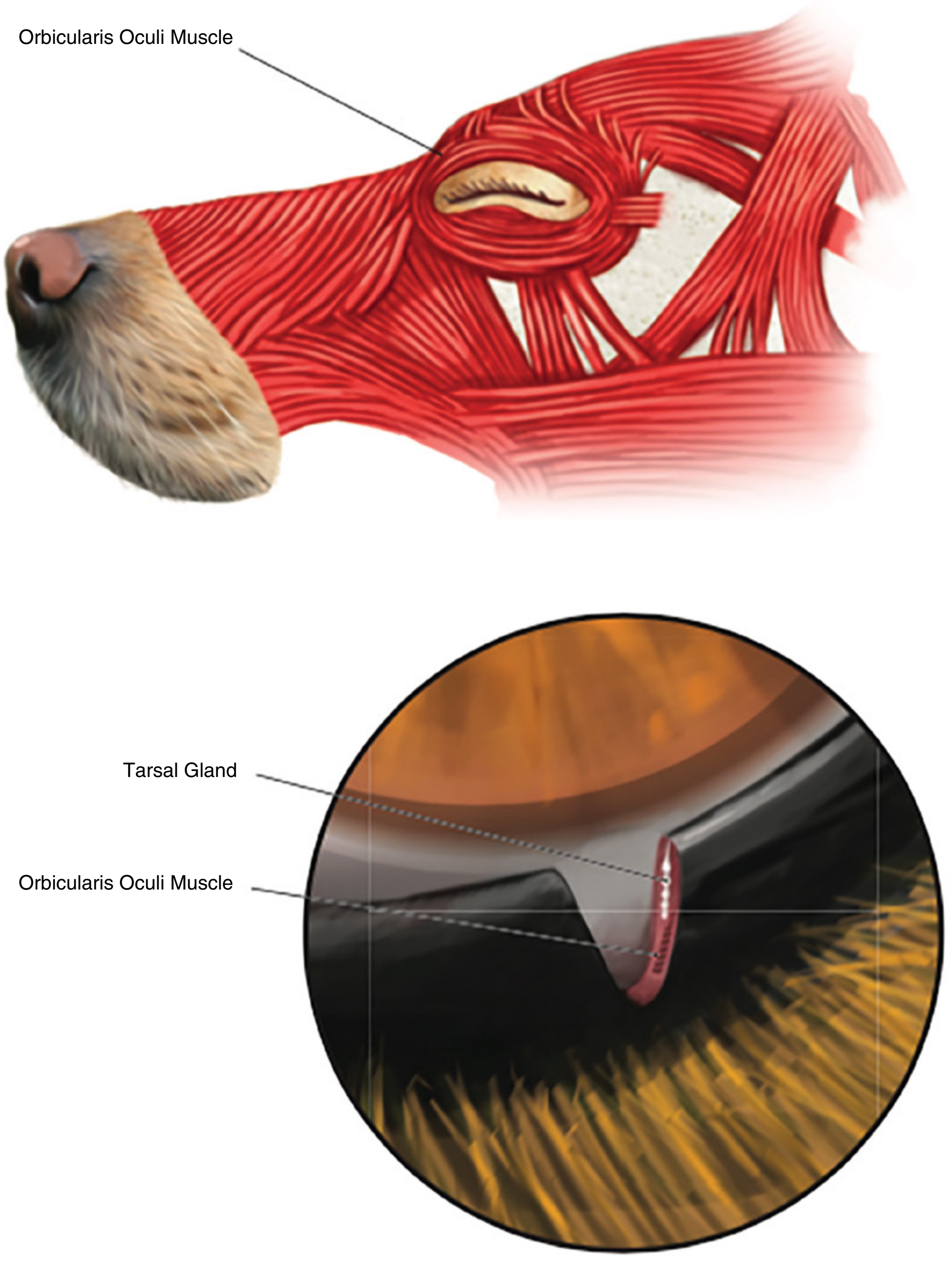


Figure 3.1 Anatomy of the Eyelids.

CHAPTER 4
LACRIMAL SYSTEM

The precorneal film (PCTF) lubricates and protects the ocular surface, removing irritants and supporting the ocular surface immune response via the activity of lysozymes and antibodies. This function is aided by eyelid blinking.

The PCTF comprises three basic constituents.

- Mucin which is secreted from goblet cells within the conjunctival tissues and serves to interface/adhere the PCTF to the cornea as well as assisting with immune function by distributing IgA and IgG across the ocular surface.
- Lipids which are secreted from the eyelid tarsal or Meibomian glands (modified holocrine sebaceous glands) and which serve to stabilize the PCTF.
- Aqueous, which is secreted by the orbital and third eyelid-associated lacrimal glands, consisting primarily of water, electrolytes, glucose, and proteins including enzymes (proteases and inhibitors of proteases.). Aqueous fluid lubricates the cornea, carries nutrition/waste to/from surface tissues, and also removes biological debris.

The orbital lacrimal gland is located ventral to the zygomatic process of the orbital bone, with ductular openings within the dorsolateral fornix. The lacrimal gland of the third eyelid is associated the cartilaginous support of this structure with ductular openings on the bulbar surface of the third eyelid.

Innervation to the lacrimal glands is overwhelmingly autonomic (parasympathetic) although a sensory input based on trigeminal mediated corneal sensitivity contributes. Fibers originate within the nucleus of the facial nerve (the rostral salivary nucleus), in the medulla oblongata. Preganglionic nerves (which together form the major petrosal nerves) pass through the petrous temporal bones before joining with the deep petrosal nerves to form the nerves of the pterygoid canal. The parasympathetic aspect of these nerves then synapses within the pterygopalatine ganglia located in the floor of each orbit. Postganglionic neurons follow the zygomatic nerves (the first branch of the maxillary division of the trigeminal nerves) and synapse with the acini of the lacrimal glands to stimulate lacrimation.

The PCTF is drained via a single superior and single inferior nasolacrimal punctum, via the lacrimal sac and nasolacrimal duct to the external nostrils.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

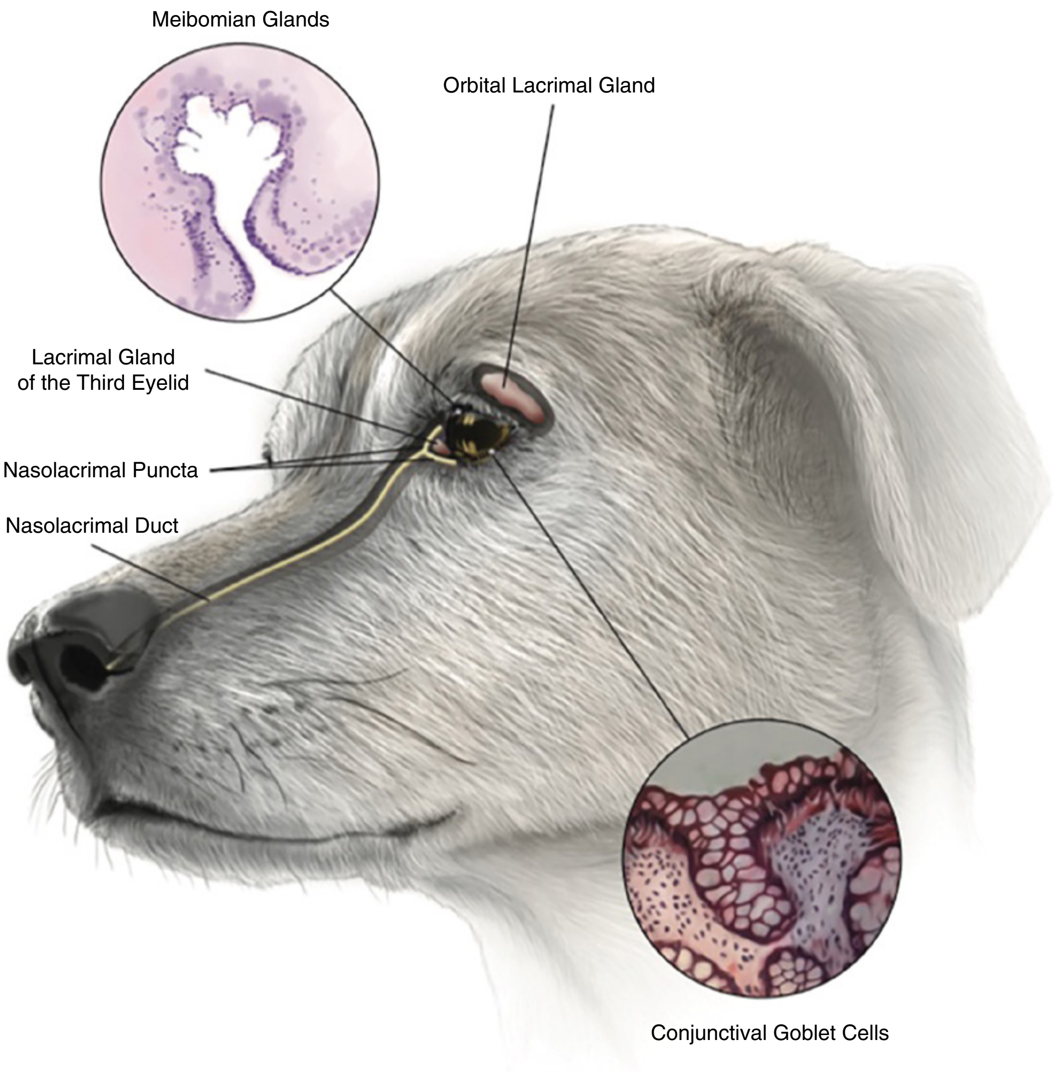


Figure 4.1 Anatomy of the Lacrimal System.

CHAPTER 5
THE CORNEOSCLERAL TUNIC

The corneoscleral tunic (comprising the anterior transparent corneal and posterior opaque scleral tissues) surrounds and protects the ocular contents.

The cornea is composed of three major layers:

- Outer epithelium, made up of non-keratinized stratified squamous cells which are in a constant state of turnover, and which present a lipophilic transparent surface barrier.
- Central stroma composed of regularly arranged collagen fibrils and which represents approximately 90% of the corneal thickness.
- Inner endothelium made up of a single layer of endothelial cells and their underlying basement ("Descemet's") membrane. Endothelial cells are richly supplied with Na+-, K+-ATPase pumps and serve to maintain stromal deturgescence.

The cornea receives significant sensory innervation from the trigeminal nerve as well as some autonomic innervation via the superior cervical ganglion. Corneal sensory and sympathetic nerves exert important neuromodulatory effects on cellular physiology, including proliferation, differentiation, adhesion, and migration.

The scleral shell is composed predominantly of collagen and serves to provide structural support for the ocular contents and a site for the insertion of extraocular muscles as well as allowing the optic nerve to exit the globe posteriorly via the porous lamina cribrosa.

Protective conjunctival tissues cover the corneoscleral shell and are contiguous with the external eyelid margins, reflecting across the surface of the globe and terminating at the corneoscleral interface.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

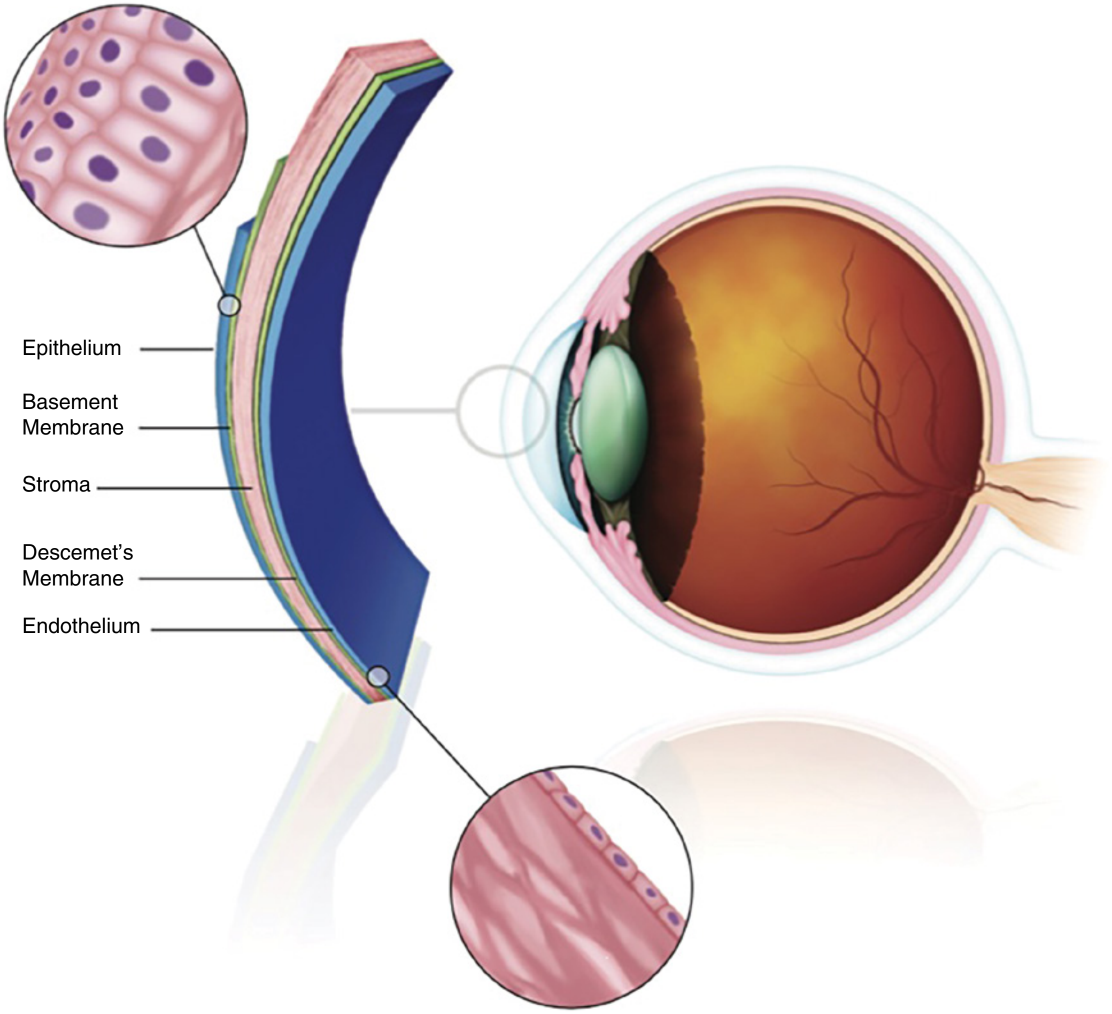


Figure 5.1 Anatomy of the Cornea.

CHAPTER 6
LENS

The biological lens is a transparent, biconvex, and avascular intraocular structure which together with the cornea, serves to focus light onto the retina.

The lens is surrounded by a capsule comprised of an elastic transparent basement membrane.

Beneath the capsule, the anterior lens surface contains a single layer of cuboidal epithelial cells, which are responsible for lenticular homeostasis including nutrition and hydration.

Epithelial cells are also the source of the lens fibers which comprise the majority of the lens volume. New lens fibers are continually layed down throughout life in a concentric pattern. With increasing age, densely compressed lens fibers give the central (nuclear) zone a grayish relucency, frequently described as "nuclear sclerosis." Proteins within the lens, chiefly comprising α-, β-, and γ-crystallins maintain optical density.

The lens is suspended in position by circumferentially arranged zonular fibers which extend around 360°, from the equatorial lens capsule to the uveal ciliary processes. Variations in muscular tension on the ciliary/zonular attachments facilitate subtle alterations in lens shape and focal length, facilitating focus through a process known as accommodation.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

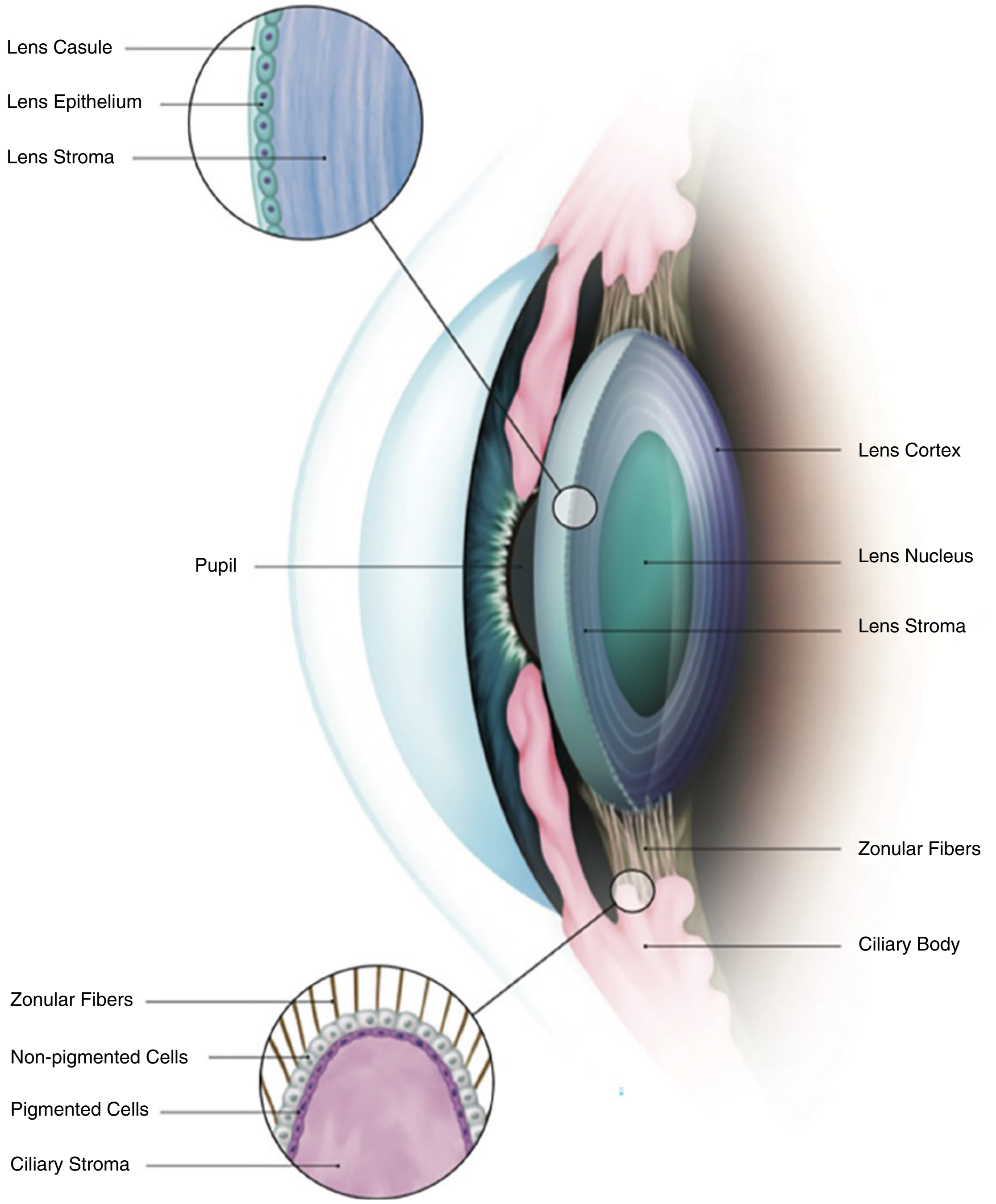


Figure 6.1 Anatomy of the Lens.

CHAPTER 7
UVEA

The uveal tract is the heavily pigmented and richly vascular ocular tunic which encompasses the iris and ciliary body anteriorly and the choroid posteriorly, providing mechanical support, nutrition, and immune-integrity.

The **iris** comprises an annular muscular structure, containing both constrictor and dilator muscles, which controls the amount of light entering the eye as well as the tension on supporting lens zonules and resultant lens position. Variable melanin pigment within iridal tissues contribute to coloration.

The posterior iris base comprises the stroma and folds of the **ciliary body**, inserting against sclera and transitioning to the neuroretinal tissues around 360° at the "ora serrata."

Nonpigmented ciliary tissues are the source of aqueous humor (AH) production through a combination of active secretion and passive ultrafiltration. AH is responsible for oxygen and nutrient delivery as well as metabolic waste removal for the avascular tissues of the lens and cornea. AH is drained via dual pathways.

AH is predominantly drained through the pectinate ligament structure into the trabecular meshwork within the iridocorneal angle and returns to circulation via the vessels of the angular vascular plexus. This is termed the "conventional" drainage pathway. A smaller, variable, and dynamic degree of AH drainage also takes place through the connective tissues of anterior iris and ciliary musculature, exiting the eye through the suprachoroidal space and scleral vasculature. This is termed "unconventional" drainage pathway.

The balanced production and drainage of aqueous maintains a stable intraocular pressure.

The **choroid**, comprises the vascular bed overlying the posterior sclera which supports the fragile neuroretinal tissues. Choroidal vasculature is responsible for oxygen and nutrient delivery as well as metabolic waste removal for the outer layers of the retina.

Finally, the uvea is responsible for maintaining the vascular and cellular integrity of ocular tissues via the blood ocular barriers. These barriers are maintained via tight junctions between the cellular and capillary endothelial tissues at the sites of the anterior "blood-aqueous" and posterior "blood-retinal" barriers.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

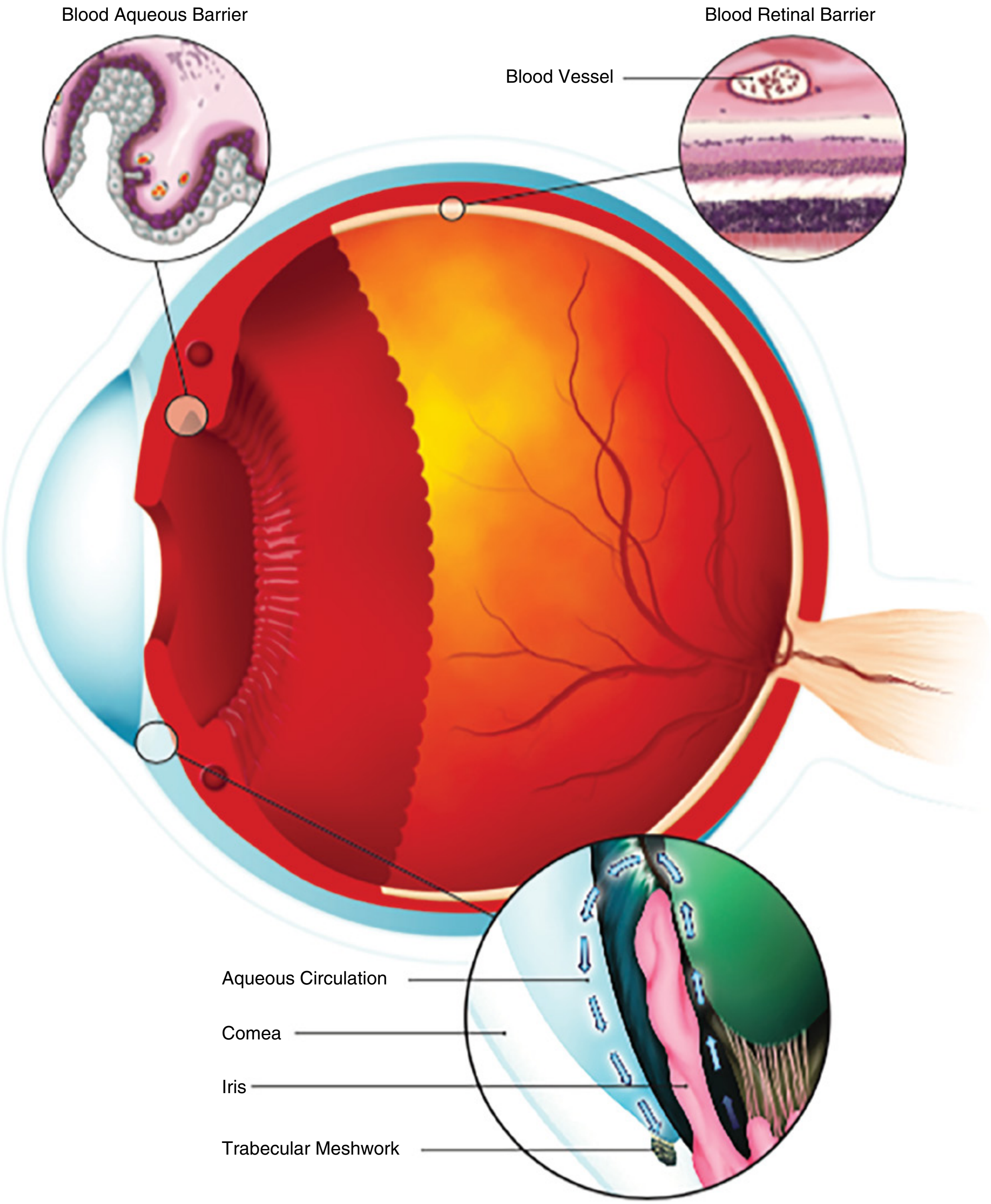


Figure 7.1 Anatomy of the Uvea.

VetBooks.ir

CHAPTER 8
VITREO RETINAL

The neuroretina is the innermost, light-sensitive tissue of the eye.

The optics of the eye create a focused two-dimensional image of the visual world on the retina, which translates that image into electrical neural impulses to the brain to create visual perception.

The neuroretina consists of several layers of neurons and supporting cells, interconnected by synapses and is supported by an outer layer of pigmented epithelial cells. The primary light-sensing cells in the retina are the photoreceptors (rods and cones). Rods function mainly in dim light and provide black-and-white vision. Cones function best in well-lit conditions and are responsible for the perception of color and high-acuity vision.

Photosensitive ganglion cells contribute to managing circadian rhythms as well as the pupillary light reflex.

Light striking the retina initiates a cascade of chemical and electrical events that ultimately trigger nerve impulses to the visual centers of the brain via the optic nerves.

The neuroretina itself comprises ten distinct layers, comprising (from closest to the vitreous):

1. The Inner Limiting Membrane is the basement membrane generated by Müller cells.
2. The Nerve Fiber Layer which is composed retinal ganglion cell axons
3. The Ganglion Cell Body layer
4. The Inner Plexiform Layer which contains synapses between bipolar, amacrine, and ganglion cells
5. The Inner Nuclear Layer which contains the nuclei and surrounding cell bodies of amacrine and bipolar cells
6. The Outer Plexiform Layer which contains synapses between horizontal and bipolar cells and photoreceptors
7. The Outer Nuclear Layer which contains nuclei and cell bodies of photoreceptors (rods and cones)
8. The Outer Limiting Membrane which facilitates connectivity with photoreceptors via junctional complexes and also acts as a junctional barrier to macromolecules
9. Photoreceptor Layer which contains the inner and outer rod and cone photoreactor segments
10. The Retinal Pigment Epithelium is a single layer of cuboidal epithelial cells which adjoins the underlying choroid and provides nourishment and supportive functions to the neuroretina

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

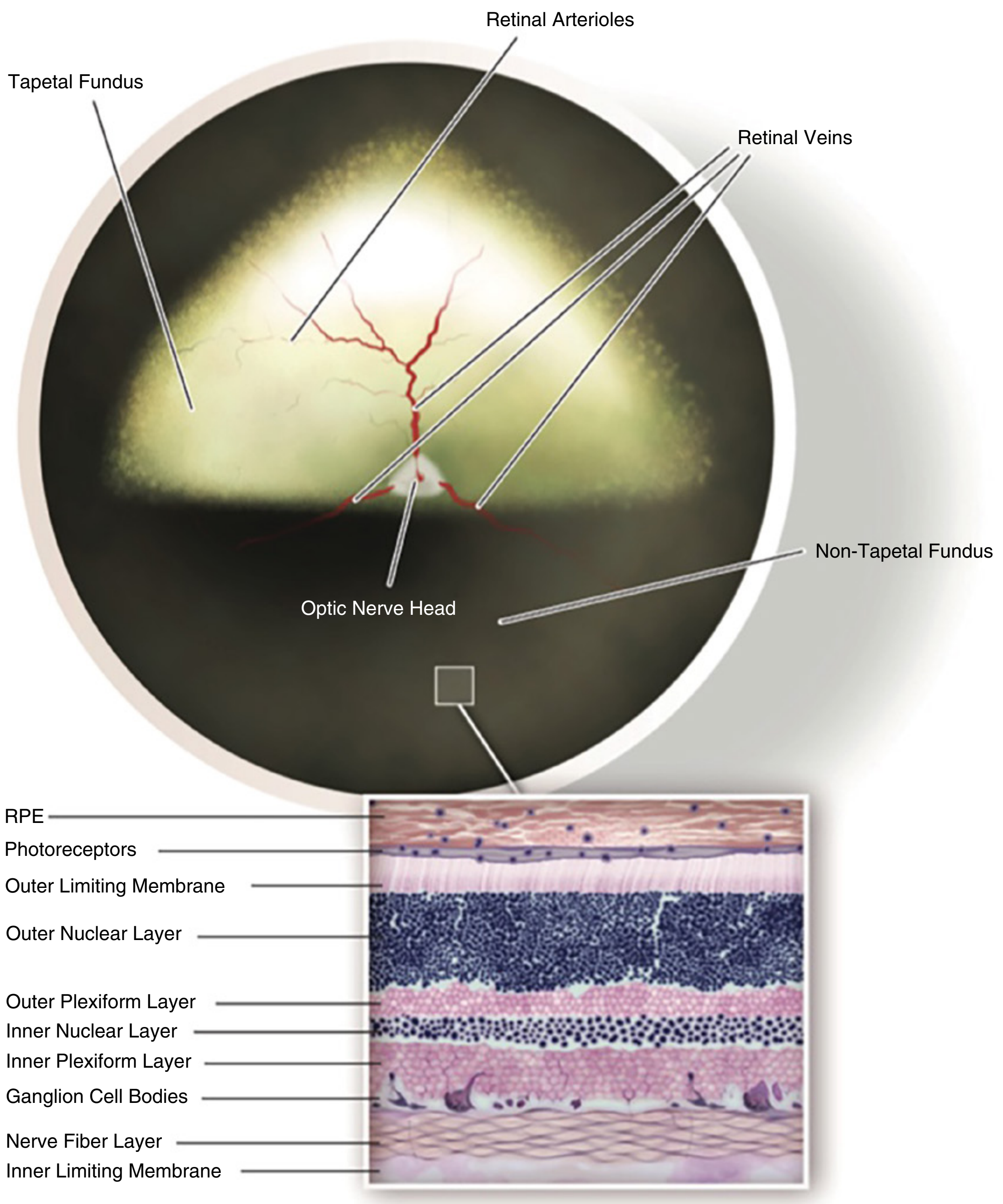


Figure 8.1 Anatomy of the Retina.

CHAPTER 9
NEUROPHTHALMOLOGY

As well as generalized sensory and motor innervation to the tissues and structures of the eye, three major neurophthalmic pathways are recognized.

THE VISUAL PATHWAY

Upon reaching the retina, light photons interact with (rod and cone) photoceptor opsins, resulting in signal generation via polarization. Photoreceptors synapse via bipolar cells onto retinal ganglion cells (RCGs) which coalesce to form the optic nerves (ONs). After exiting the globes via the porous lamina cribrosa, the ONs form a chiasm at which the nerve fibers decussate and branch, to form the left and right optic tracts. The optic tracts terminate in the lateral geniculate nuclei (LGN) in the thalamus. The optic radiations (one on each side of the brain) carry information from the lateral geniculate nuclei to the visual cortices which are responsible for processing and interpreting visual images.

SYMPATHETIC PATHWAY

Sympathetic innervation to the eye mediates the action of the iris dilator and sphincter muscles, the smooth muscle of the orbit and the Muller's muscles of the upper and lower eyelids. These pathways originate in the hypothalamus, exit the spinal chord with the ipsilateral ventral thoracic roots, and synapse in the cranial cervical ganglia, adjacent to the tympanic bullae. Post-ganglionic fibers follow the ophthalmic divisions of the trigeminal nerve before entering the eyes via the long ciliary nerves.

PARASYMPATHETIC PATHWAY

CN III provides somatic efferent innervation to the dorsal, medial, and ventral rectus and ventral oblique muscles of the eye, as well as the levator palpebral muscles. This nerve also contains the parasympathetic fibers, which synapse at the level of the ciliary ganglia and innervate the pupillary muscles (through "double reciprocal innervation") as well as the lacrimal tissues of the eye.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

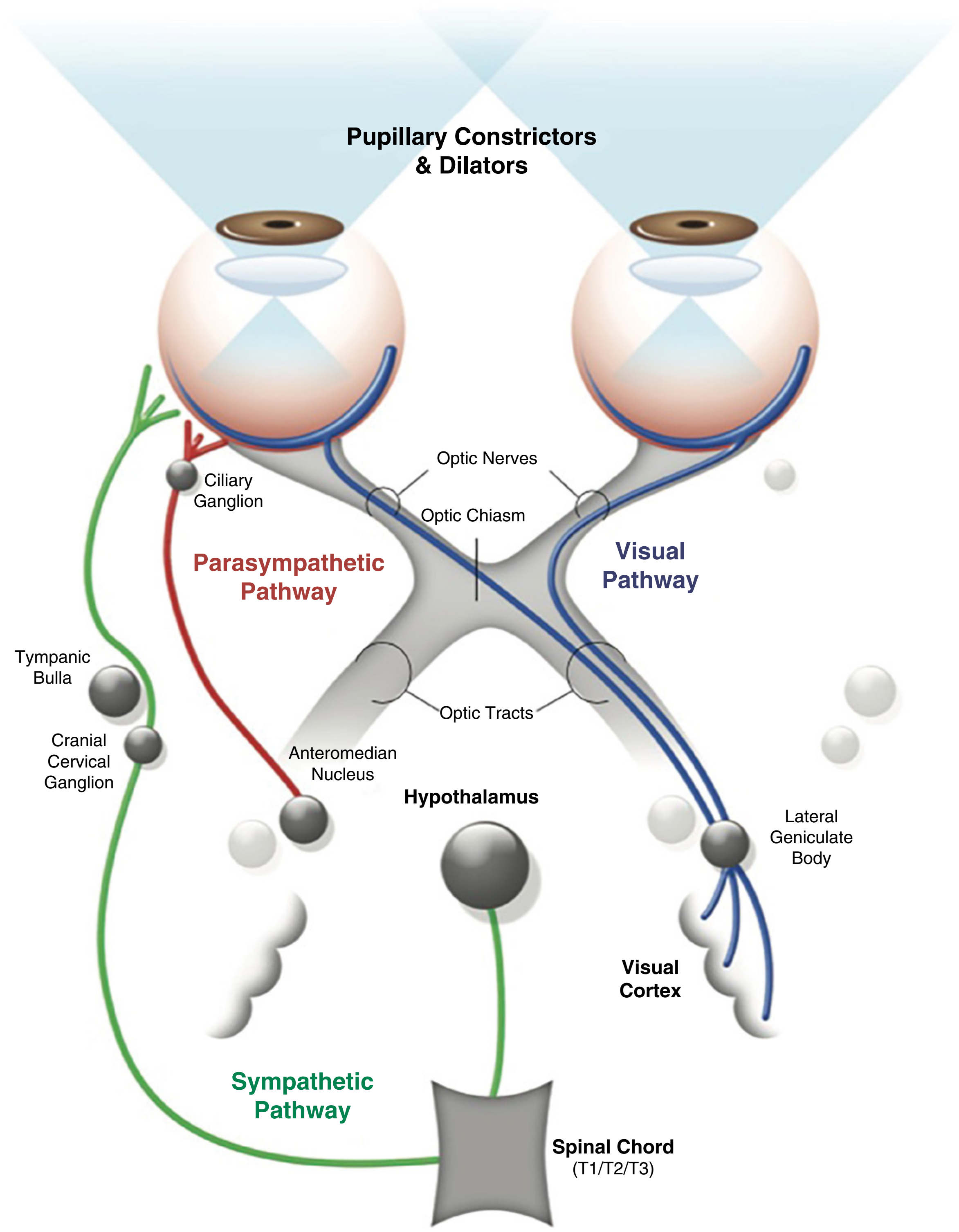


Figure 9.1 Neurophthalmic Pathways.

Section 2

Normal Variations

CHAPTER 10
NORMAL PIGMENTARY VARIATIONS

Both canine and feline irides may demonstrate a range of pigmentary variations.

True ocular albinism (the complete lack of pigment) is rare. The term subalbinism describes pigment dilution resulting in variably gray to bluish colored iridal tissue, a finding common in animals with light hair coat colors. The term heterchromia iridis describes variable combinations of pigment within either one or both irides.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 10.1 Normal variation in pigmentation between left and right eyes.

Figure 10.2 Normal heterochromic variation in pigmentation within a light colored iris.

Figure 10.3 Normal heterochromic variation in pigmentation within a dark colored iris.

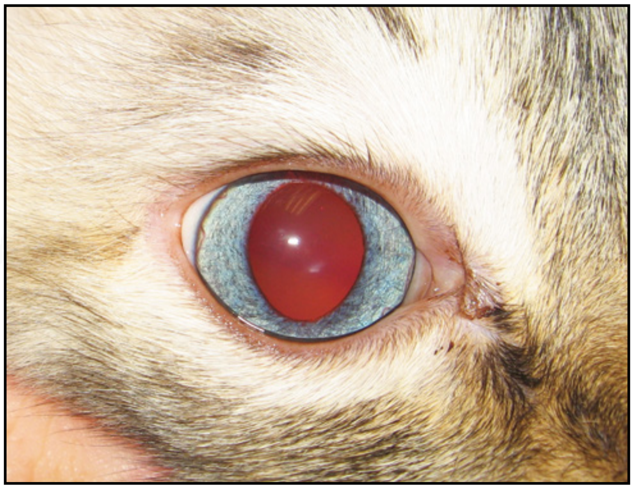


Figure 10.4 Normal blue iris (the "red reflex" of the subalbinotic fundus being visible through the pupil).

CHAPTER 11
THE NORMAL CANINE FUNDUS

The canine fundus exhibits a wide variation in normal appearance, comprising a tapetal as well as a non-tapetal region, the optic nerve head, associated vasculature and multi-layered neuroretina, all of which overly the chorioidal vascular bed. The juvenile canine fundus typically appears bluish in color until maturation within the first 3–4 months of life.

The specialized cells of the tapetal region contain reflective material comprising zinc/cysteine as well as a poorly to non-pigmented retinal pigment epithelial layer (RPE), which facilitates low-light vision. This region is typically bright yellow to green in coloration.

The non-tapetal fundus is usually dark in color due to the presence of pigment within the RPE.

The optic nerve ("optic disc," "optic papilla") appears as a variably shaped and variably myelinated white to pink structure within the fundus, representing the accumulation of ganglion cells and displaying an incomplete vascular circle surrounding a central physiologic pit.

Radiating from the ONH are 3–4 large veins and 15–20 smaller arterioles.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

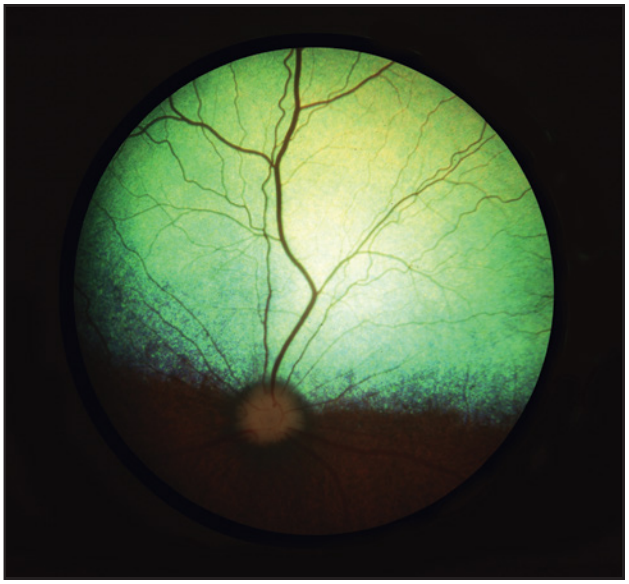


Figure 11.1 Normal pigmented canine fundus (predominantly green).

Figure 11.2 Normal pigmented canine fundus (predominantly yellow).

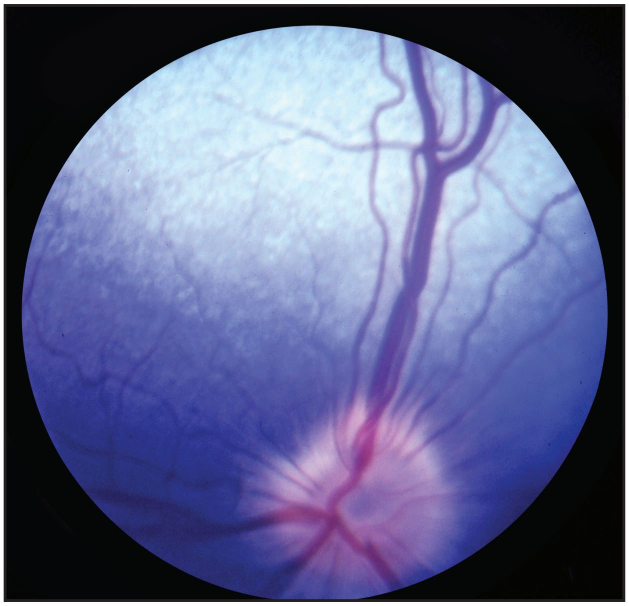


Figure 11.3 Normal pigmented canine fundus The bluish color indicates immaturity.

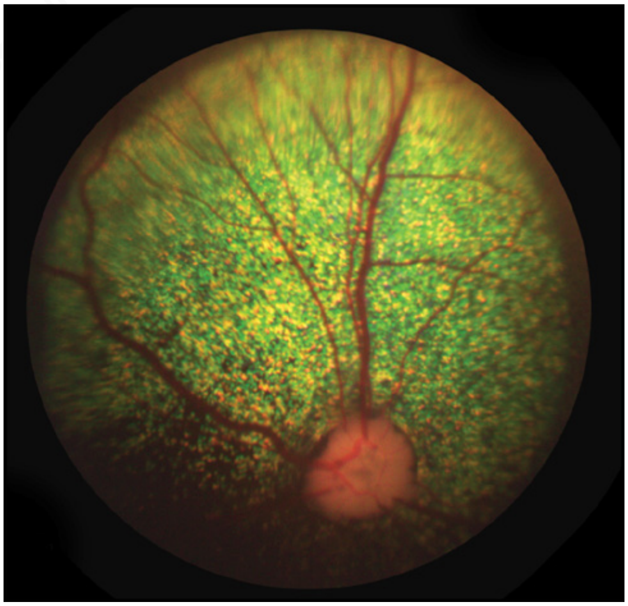


Figure 11.4 Normal pigmented canine fundus (speckled).

CHAPTER 12
THE NORMAL FELINE FUNDUS

The feline fundus exhibits a wide variation in normal appearance, comprising a relatively large tapetal as well as non-tapetal region, the optic nerve head (ONH), associated vasculature and multi-layered neuroretina, all of which overly the chorioidal vascular bed.

The specialized cells of the tapetal region contain reflective material comprising zinc/riboflavin as well as a poorly to nonpigmented retinal pigment epithelial layer (RPE), which facilitates low-light vision. This region is typically bright yellow to green in coloration.

The non-tapetal fundus is usually dark in color due to the presence of pigment within the RPE.

The optic nerve ("optic disc," "optic papilla") appears as a small, circular, and unmyelinated white to gray structure within the fundus, representing the accumulation of ganglion cells.

Three major pairs of arterioles as well as larger venules radiate from the ONH.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 12.1 Normal pigmented feline fundus (predominantly green), note the poorly myelinated optic nerve head.

Figure 12.2 Normal pigmented feline fundus (predominantly yellow), note the poorly myelinated optic nerve head.

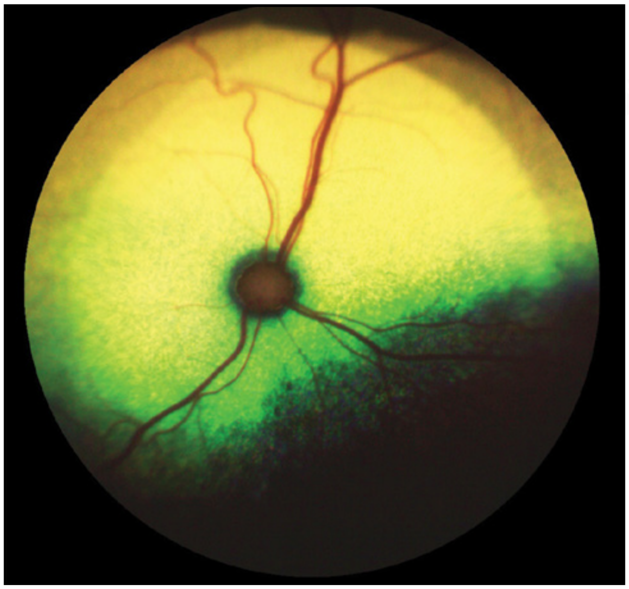


Figure 12.3 Normal pigmented feline fundus (predominantly green), note the poorly myelinated optic nerve head.

Figure 12.4 Normal pigmented feline fundus (predominantly yellow), note the poorly myelinated optic nerve head.

CHAPTER 13
THE NORMAL SUBALBINOTIC FUNDUS

Dogs or cats displaying blue irides, heterochromic irides, and/or merled coat coloration, typically display "subalbinotic" fundi. In these animals, the tapteal region may be variably reduced to absent in association with a variable to complete lack of pigment within the non-tapetal fundus. As a result, underlying choroidal vasculature is visible against the white scleral background. The subalbinotic fundus represents a normal variation in coloration.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 13.1 Normal (canine) subalbinotic fundus. Choroidal vessels are clearly visible against the white scleral background.

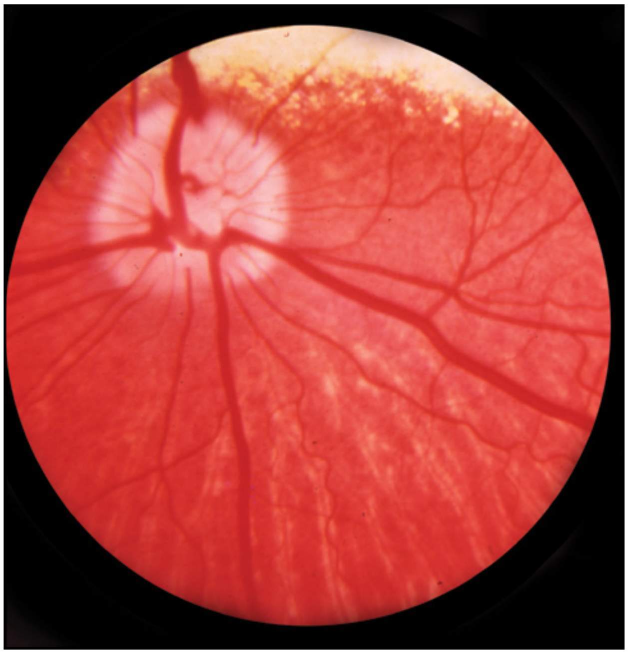


Figure 13.2 Normal (canine) subalbinotic fundus. Choroidal vessels are clearly visible against the white scleral background.

Figure 13.3 Normal (canine) subalbinotic fundus. Choroidal vessels are clearly visible against the white scleral background.

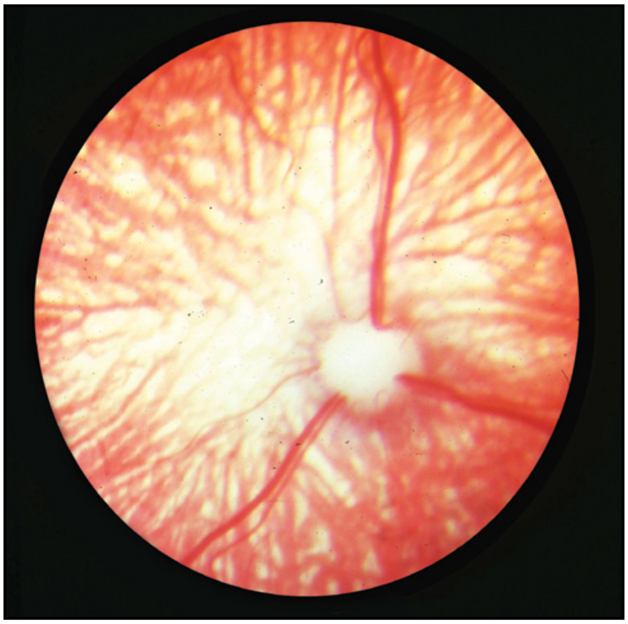


Figure 13.4 Normal (feline) subalbinotic fundus. Choroidal vessels are clearly visible against the white scleral background.

CHAPTER 14
NORMAL MYELIN VARIATIONS

The optic nerve head (ONH) comprises coalescing ganglion cells as they converge prior to exiting the globe caudally through the porous lamina cribrosa. This region is variably myelinated; poorly in the cat and variably in the dog. Variations in the amount of myelin present may result in range of normal appearances when this region is visualized.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

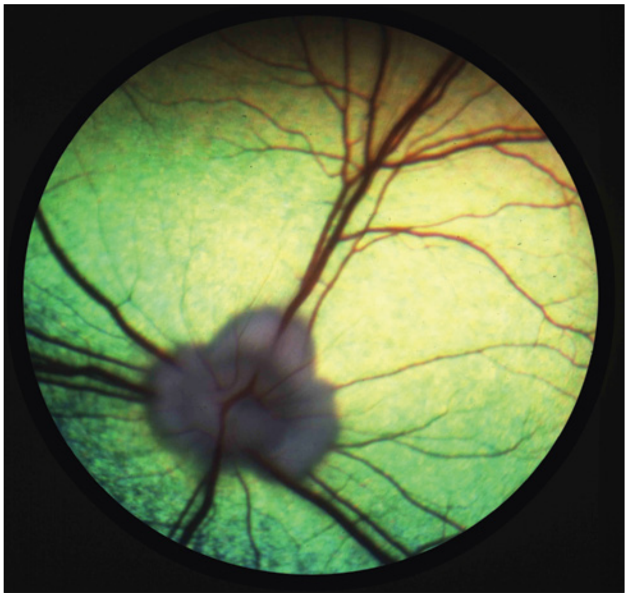


Figure 14.1 Normal moderately to heavily myelinated canine optic nerve head.

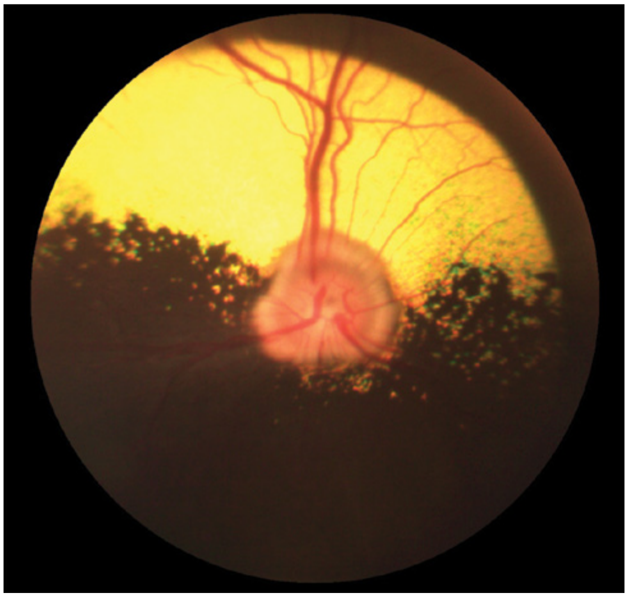


Figure 14.2 Normal moderately to heavily myelinated canine optic nerve head.

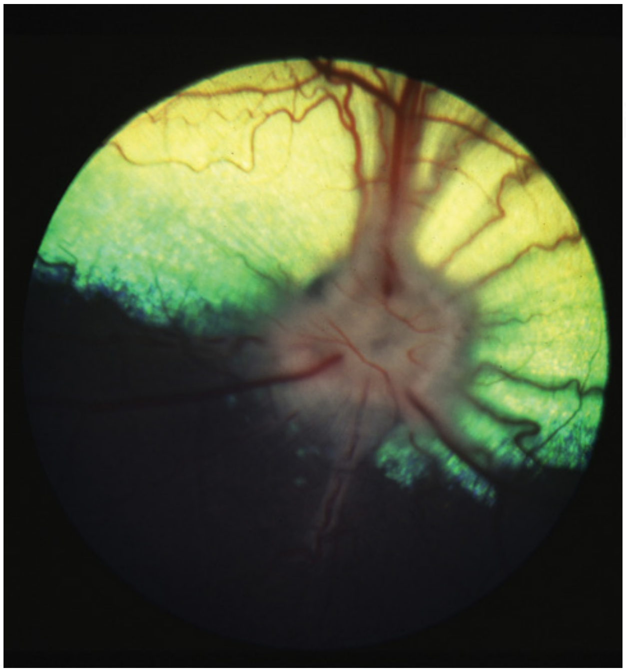


Figure 14.3 Normal moderately to heavily myelinated canine optic nerve head.

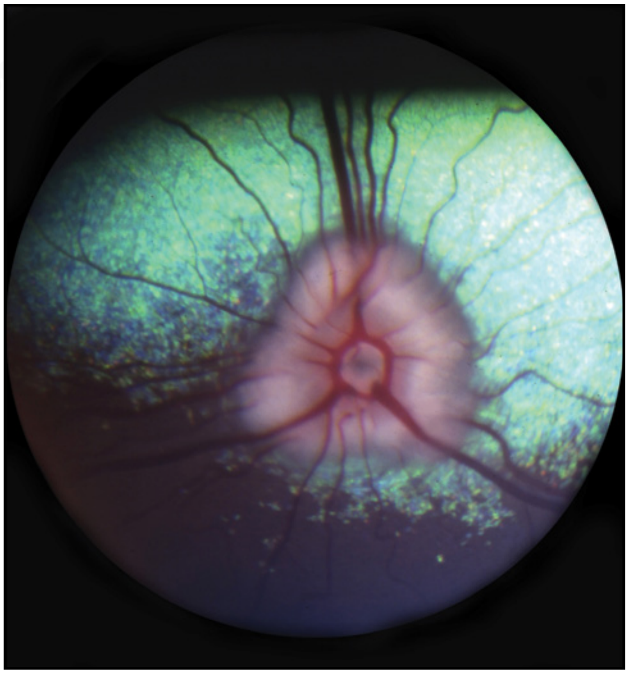


Figure 14.4 Normal moderately to heavily myelinated canine optic nerve head.

Section 3

Pharmacology

CHAPTER 15
DIAGNOSTIC PHARMACOLOGY

Diagnostic pharmacology plays an important role in every ophthalmic examination. Topical anesthetics, vital stains, and mydriatics are invaluable tools in this arena.

Some patients will require the use of tranquilizing/sedative agents and/or topical anesthetics to allow accurate ophthalmic examination. The measurement of tear production, tear film stability and/or the harvesting of samples for culture/sensitivity testing should be performed prior to the instillation of topical anesthetics. The most commonly used topical anesthetic is 0.5% proparacaine hydrochloride. Proparacaine impedes sodium ion permeation to the corneal and conjunctival nerve axons, preventing nerve depolarization. In cats and dogs, the clinical duration of effect of topically applied proparacaine typically lasts between 25 and 45 minutes. One percent tetracaine ophthalmic solution may have a slightly longer duration of action but may cause ocular irritation in some patients.

Vital stains serve to highlight corneal lesions that might otherwise be less visible. The most commonly used vital stain when evaluating corneal integrity is fluorescein sodium. Cobalt blue light causes fluorescence of the dye. Fluorescein is water soluble and will adhere to the hydrophilic corneal stroma, but not to the corneal epithelium or Descemet's membrane. It is therefore used to identify corneal defects involving the loss of corneal epithelium and stroma. Rose Bengal stain may also be used to identify dendritic/herpetic ulcers and devitalized corneal epithelium. Fluorescein can also be used identify aqueous humor leakage from corneal lacerations and perforations (Seidel test). Finally, fluorescein passes though the nasolacrimal puncta, via the nasolacrimal ducts to the external nares and can therefore be used to assess patency of the nasolacrimal system (Jones test).

Thorough fundic examination is most easily achieved through a mydriatic pupil. Mydriatic agents may be either anticholinergics, which paralyze the pupillary sphincter muscles and provide cycloplegia, or sympathomimetics, which stimulate the iris dilator muscles. The most commonly used topical cholinergic antagonists are tropicamide and atropine. Tropicamide can cause dilation in as little as 15 minutes but has a relatively short duration of action, and provides little cycloplegia. Atropine may take 30–60 minutes to cause mydriasis, with a duration of action which can be several days. Atropine does provide cycloplegia, but can also decrease tear production, aggravate lenticular instability, and/or vitreous prolapse and increase intraocular pressure, so should be used with caution. Sympathomimetics like phenylephrine and epinephrine can also be used to promote mydriasis although epinephrine is most effective when administered intracamerally. Topical phenylephrine is commonly used in the diagnosis and localization of sympathetic denervation (Horner's syndrome).

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 16
MODES OF ADMINISTRATION AND DRUG DELIVERY

The eye represents a unique anatomical structure in that it can be treated with both topical and systemic therapies, but it is also well protected (under normal physiologic circumstances) from the effects of some medications administered by both routes. The part of the eye affected, and the specific condition being treated will dictate the most appropriate route of therapy.

Diseases of the eyelids and periocular tissues are generally more responsive to systemic therapy, while simple corneal or conjunctival diseases can often be treated successfully with topical therapy alone. Intraocular diseases may require a combination of modalities.

Topical ophthalmic medications are typically formulated as solutions, suspensions, or ointments. Various vehicles are used to increase absorption, prolong contact time, minimize irritation, and stabilize the active pharmacologic ingredients. In certain circumstances (for example, in the case of a ruptured eye), one formulation (a solution) is preferable to another (an ointment). Topical ophthalmic medications are typically administered two to six times daily depending on the severity of the condition being treated.

Systemic medications are useful when treating adnexal as well as intraocular diseases. The integrity of the blood ocular barriers (blood-aqueous and blood-retinal) is generally decreased in the presence of inflammation, promoting the penetration of certain medications across the blood-ocular barriers. In general, lipophilic drugs penetrate the blood-ocular barriers more effectively than hydrophilic drugs.

Subconjunctival, intracameral, and intravitreal injections are less commonly employed with the potential for significant complication if performed incorrectly. Subconjunctival injections may be used to provide prolonged effects in association with less frequent dosing, particularly when managing corneal and/or anterior segment disease. A surprising array of ophthalmic medications can be safely and effectively administered via the sub-conjunctival route. Medications may diffuse directly into the ocular tissues, may be absorbed by the conjunctival vessels, or may gradually be released from the injection site to enter the eye through corneal absorption.

Intracameral and intravitreal injections are typically performed at the time of intraocular surgery and are most safely performed on sedated or anesthetized patients. Damage to intraocular structures can occur as a result of physical injection-related trauma or as a result of intraocular toxicity associated with some medications and preservatives.

Finally, ocular implants providing lengthy periods of effective ocular therapy are increasingly available.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 17
ANESTHESIA AND SEDATION IN OPHTHALMOLOGY

Planning perioperative care, analgesia, sedation and/or anesthesia for the veterinary ophthalmic patient dictates special considerations. Many ophthalmic patients present with unstable ocular structures, exhibit significant discomfort, and/or may have delicate surgical sites. Forceful restraint, neck leads, significant extension of the neck for jugular venipuncture, and even intubation (and jaw opening) can be hazardous for a patient with significant ocular pathology. Difficult mask or box-inductions should also be avoided wherever possible.

The appropriate use of topical and/or local injectable anesthetic agents may facilitate routine diagnostic and/or therapeutic procedures. The use of topical agents however, may mask clinical symptoms such as those associated with spastic entropion and may additionally alter subsequent diagnostic results, notably Schirmer tear test values.

As with other surgical patients, minimizing stress and anxiety, as well as pain, preoperatively benefits the patient and will reduce the amount of induction agent and the level of maintenance anesthetic required. Dexmedetomidine, diazepam/midazolam, butorphanol, and acepromazine may be safely incorporated into ophthalmic premedication plans. Opioids may also be safely used in ophthalmic patients, providing both analgesia and sedation. Premedication with maropitant can minimize nausea and the risk of vomiting and may also have analgesic effects. Ketamine, however, may increase intraocular pressure and the use of this agent may be disadvantageous in some cases.

Some ophthalmic procedures can be safely performed under injectable sedation/anesthesia.

Dexmedetomidine in combination with butorphanol, or propofol alone (with proper airway/respiratory support if needed) are adequate for selected minor procedures such as facilitating thorough ophthalmic evaluation of fractious or painful animals, corneal debridement/keratotomy, harvesting of surface biopsies, suture removal, or simple conjunctival foreign body removal.

Routine induction with propofol, alfaxalone, or etomidate is recommended for ophthalmic patients. Maintenance anesthesia using isoflurane or sevoflurane mixed with oxygen is standard. Patients undergoing intraocular procedures will usually require the use of a neuromuscular blocking agent (atracurium, rocuronium, pancuronium) to facilitate appropriate globe positioning and minimize the risk of intraoperative complications. Blocked patients require mechanical ventilation and careful and specific monitoring. The use of retrobulbar analgesic blocks may decrease the demand on anesthetic agents and maximize patient comfort during recovery.

During ophthalmic procedures (notably enucleation), it is important to be aware of the oculo-cardiac reflex (OCR), which may result in a significant reduction in heart rate associated with traction on the extraocular muscles and/or compression of the globe. The sensory afferent limb of the OCR is carried by the trigeminal nerve to the central nervous system (CNS) and the efferent limb by the vagus nerve, which may affect the myocardium and/or sinoatrial node potentially causing bradycardia, arrhythmia and in rare instances, arrest. The appropriate use of anti-cholinergic agents can decrease the incidence of OCR.

The recovery period is a time for close monitoring and patient agitation, anxiety, and pain should be well managed (notably using sedative and/or analgesic agents) to avoid postoperative hemorrhage or trauma. Use of an Elizabethan collar to prevent self-trauma is typical in postoperative ophthalmic cases.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 18
ANTI-INFLAMMATORIES AND THE EYE-NSAIDS

Anti-inflammatory medications can generally be divided into two classes based on their mechanism of action: steroidal and nonsteroidal. The non-steroidal anti-inflammatory drugs (NSAIDS) can be differentiated from steroids by their chemical structure which lacks a cholesterol-derived steroid nucleus. NSAIDS exert their anti-inflammatory effects through inhibition of cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2) enzymes which, in turn, inhibits prostaglandin synthesis. Prostaglandins involved in the regulation of cellular homeostasis are typically associated with COX-1 activity, while prostaglandins associated with inflammation are generally associated with COX-2 activity.

NSAIDs are typically divided into groups based on their chemical structure and selectivity with major classes including the salicylates (aspirin), the proprionic acid derivatives (ketoprofen, ibuprofen, flurbiprofen, ketoprofen, naproxen, and carprofen), the acetic acid derivations (diclofenac, ketorolac, nepafenac, indomethacin, and etodolac), the enolic acids (meloxicam, piroxicam), and selective COX-2 inhibitors (celecoxib, etoricoxib).

NSAIDs are usually administered topically and/or systemically.

Common topical NSAIDS include flurbiprofen, diclofenac, ketorolac, bromfenac, and nepafenac. Topical application may increase the ocular bioavailability of the drug and reduce systemic side effects. The pharmacokinetics, strength, cost, use of preservatives, and recommended frequency of application of these drugs vary but most can be used interchangeably. Side effects of topical NSAIDS are rare, but may include ocular irritation, increased intraocular pressure (IOP) and increased risk for corneal ulceration/keratomalacia.

Systemic NSAIDS commonly used in veterinary patients include carprofen, meloxicam, piroxicam, deracoxib, robenacoxib, firocoxib, etodolac, and grapiprant. Side effects associated systemic NSAIDs may include gastrointestinal upset/ulceration (especially when combined with steroids), increased bleeding tendencies, renal and hepatic changes, and rarely cardiovascular changes. In addition, etodolac has been associated with the development of keratoconjunctivitis sicca in canine patients.

Grapiprant, a novel compound, does not inhibit cyclooxygenases, but rather works further along the inflammatory pathway as an EP4 prostaglandin receptor antagonist reducing the activity of that specific prostaglandin. This may reduce its impact on kidney and liver health.

Nonsteroidal anti-inflammatories are frequently utilized in veterinary ophthalmology. By inhibiting prostaglandins, they decrease the disruption of the blood-aqueous barriers as well as minimizing vasodilation and corneal neovascularization. They may be used to reduce inflammation in cases of blepharitis, conjunctivitis, scleritis, keratitis, and uveitis and/or neuro-retinitis. NSAIDS are beneficial in cases in which steroids are contraindicated, such as in diabetic patients. Perioperative use of NSAIDS may reduce intraoperative miosis, manage postoperative inflammation and reduce ocular pain or discomfort. NSAIDS are also occasionally used in chemotherapeutic protocols. Prior to treatment with NSAIDS, a thorough history should be taken, and patients should undergo a complete physical examination, including complete blood count and serum chemistry and urinalysis. Periodic monitoring of laboratory parameters, particularly complete blood count (CBC), hepatic and renal values is advised when patients remain on long-term NSAID therapy.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 19
ANTI-INFLAMMATORIES AND THE EYE-STEROIDS

Naturally derived steroid hormones are classified as mineralocorticoids, which maintain electrolyte balance, and glucocorticoids, which broadly speaking help regulate metabolism and the immune response. Glucocorticoids may also exert mineralocorticoid effects. Both are synthesized in the adrenal cortex from cholesterol.

Most steroid medications are corticosteroids, with a chemical structure similar to the endogenous hormone cortisol. Since the desired effects of corticosteroids are anti-inflammatory and/or immunosuppressive, drugs are usually designed to have greater glucocorticoid effects and minimal mineralocorticoid effects. The anti-inflammatory effects of corticosteroids occur primarily as a result of inhibition of phospholipase A_2, which in turn blocks the conversion of arachidonic acid to prostaglandins (via the cyclooxygenase pathway) and leukotrienes (via the lipoxygenase pathway.)

Steroidal medications may be administered topically, via sub-conjunctival or intraocular routes and/or systemically.

Systemic steroids are an essential part of the management of inflammatory diseases of the ocular adnexa, posterior segment and orbit as therapeutic concentrations cannot be achieved in these tissues using topical administration. Systemic steroids are typically indicated when treating severe ocular inflammatory disorders including blepharitis, scleritis, uveitis, chorioretinitis, optic neuritis, and many forms of ocular neoplasia. Side effects may include gastrointestinal distress, adrenal suppression, polyuria/polydipsia, weight gain, hepatopathy, immunosuppression, and/or muscle wasting.

The effects of topical steroids are significantly dependent on their ability to penetrate the cornea; with acetates (prednisolone acetate) more potent than alcohols, which are in turn more potent than phosphates (dexamethasone sodium phosphate) and hydrocortisone penetrating the cornea poorly. Additionally, the frequency of application and consequently the concentration of the drug significantly affects local therapeutic levels. Side effects potentially associated with topical ophthalmic steroid use may include local irritation, delayed wound healing, suppression of ocular immunity, the development of corticosteroid-keratopathy potential adrenal, and/or effects. Intraocular pressure (IOP) elevation and lenticular pathology as a result of steroid administration are extremely rare in veterinary patients.

Similar to nonsteroidal anti-inflammatory medications, steroids are indispensable when managing the veterinary ophthalmic patient, with perioperative treatment with steroids additionally critical for ophthalmic surgical patients. Careful consideration should be given to patients with underlying disease processes in which steroid use may be contraindicated and routine evaluation of complete blood count (CBC)/chemistry is advised in patients receiving long-term therapy.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 20
IMMUNOTHERAPY, BIOLOGICS, AND CHEMOTHERAPY IN OPHTHALMOLOGY

Glucocorticoids represent the mainstay of immunosuppressive therapy in clinical veterinary medicine; however, in some cases alternate and/or additional systemic immunomodulating therapies may be indicated in order to more ideally manage diseases symptoms as well as to minimize the unwanted side effects associated with long-term steroid administration.

Commonly encountered ophthalmic pathologies requiring potentially long-term immunomodulation include primary (immune-mediated) blepharitis, primary scleritis/episcleritis, chronic/severe uveitis, neuro-retinopathy/optic neuritis, and neoplastic disease.

The following agents are increasingly utilized when managing complex/severe/recurrent ophthalmic pathologies:

Azathioprine ("Imuran"), is an immunosuppressive prodrug which is converted into its active metabolite 6-mercaptopurine in the body and results in both DNA and RNA synthesis interruption and the suppression of WBC synthesis. Azathioprine is frequently used as "steroid-sparing" treatment.

Cyclosporine ("Atopica") exerts an immunosuppressive effect via the inactivation of calcineurin which results in the inhibition of clonal activated T-cell expansion as well as the blocking of downstream B -lymphocyte and macrophage activation.

Mycophenolate mofetil ("Cellcept") is an immunosuppressive which is metabolized to mycophenolic acid and interferes with DNA replication, particularly inhibiting lymphocyte proliferation. Mycophenolate is frequently used as "steroid-sparing" treatment.

Leflunomide ("Arava") is an inactive prodrug that is hydrolyzed to the active metabolite teriflunomide, which interferes with lymphocyte differentiation and proliferation via the inhibition of mitochondrial enzymes necessary for their synthesis.

Cyclophosphamide ("Cytoxan") is a chemotherapeutic alkylating agent which is converted into active metabolites that lead to cellular death via apoptosis, notably in actively proliferating tissues.

Methotrexate ("Trexall") is a chemotherapeutic antifolate antimetabolite which selectively inhibits folic acid (and hence DNA) synthesis, resulting in lymphocyte inhibition.

All of these agents may potentially be associated with a variety of significant and potentially life-threatening side effects including GI distress, hepatopathy, and/or myelosuppression and should only be utilized under close supervision, including diligent complete blood count (CBC)/chemistry monitoring and carefully informed owner consent.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 21
TREATING KERATOCONJUNCTIVITIS SICCA

Keratoconjunctivitis sicca (KCS) or "dry eye" broadly describes a range of conditions associated with tear-film deficiencies. Suboptimal tear production is a common condition in canine patients and is often an underlying cause for eyelid pathology, inflammatory keratoconjunctivitis, ulcerative keratitis, and/or secondary bacterial infections. KCS itself may be caused by congenital or inherited/genetic factors, as a result of trauma, by the administration of certain drugs (notably sulfonamides, etodolac, and atropine), by excision of the gland of the third eyelid(s), by neurogenic factors, and most commonly, by immune mediated disease affecting the lacrimal tissue itself.

The diagnosis of KCS is typically made on the basis of Schirmer (I) Tear Testing and associated clinical signs. In canine patients, Schirmer Tear Tests are performed without topical anesthetic, with normal values typically approximating 15–25 mm wetting/minute. Clinical symptoms may include secondary meibomian gland dysfunction/inflammation/infection, mucoid to mucopurulent discharge, conjunctival hyperemia, a "lackluster" appearance to the cornea fibrovascular and/or pigmentary keratitis, and occasionally secondary ulcerative keratitis.

Historically, KCS has been treated with supplemental lubricants, antibiotics, and anti-inflammatory therapy. The development of ophthalmic immunomodulating agents have however improved the success rate of medical treatment and significantly reduced the frequency of surgical parotid duct transposition.

Cyclosporine and tacrolimus are calcineurin inhibitors derived from fungal cell walls that decrease T-lymphocyte activation and which in turn exert an immunosuppressive effect on the lacrimal glands and improves tear production in many cases of KCS. Topical application of cyclosporine and tacrolimus products has been shown to improve tear film quantity and quality as well as reverse some of the inflammatory changes affecting corneal and conjunctival tissues. Potential side effects associated with these products may include periocular dermatitis/alopecia as well as local corneal irritation.

Pilocarpine is a direct acting parasympathomimetic agent, which may be administered systemically to stimulate lacrimation, particularly in cases of neurogenic KCS. Salivation, nausea and/or gastrointestinal (GI) distress may be associated with the excessive administration of oral pilocarpine.

Supplemental therapy (lubricants, antibiotics, anti-inflammatories) is an important part of KCS therapy as well. Lubricants may be especially useful at the outset of treatment, as it may take several weeks for an increase in lacrimal function to occur. Antibiotics are beneficial when treating secondary meibomian gland inflammation/infection, bacterial conjunctivitis, and/or secondary corneal ulceration. Anti-inflammatory agents (typically topical steroids), may be included in KCS treatment regimens when appropriate. Corneal pathology associated with KCS can be significantly improved in most cases.

Currently, combination medical therapy provides the most ideal clinical outcome when treating KCS.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 22
GLAUCOMA THERAPY

The glaucomas are a group of ophthalmic diseases generally associated with increased intraocular pressure (IOP) and degeneration of the optic nerve. Rarely, glaucomatous changes may be congenital (present at birth), with the prognosis for affected eyes unfortunately poor. The primary glaucomas are associated with a genetic predisposition resulting in a series of initiating events, leading to impaired aqueous outflow and typically acute IOP elevation. The secondary glaucomas are more frequently associated with chronic changes (typically uveitic), and include pathologies associated with phacolytic uveitis, lens instability/luxation, pigmentary/cystic pathology, feline aqueous misdirection, and neoplastic diseases.

Prompt medical management to lower IOP, decrease intraocular inflammation, improve patient comfort, and provide neuroprotection when possible is essential in the initial treatment of glaucoma.

Rapid lowering of IOP may be achieved via the intravenous administration of mannitol, an osmotic agent that results in dehydration and contracture of the vitreous, allowing the iris and lens to move posteriorly, opening the iridocorneal angle. Mannitol is generally considered safe to administer in diabetic patients but is contraindicated in cases of renal failure, dehydration, and significant cardiovascular disease. Glycerin is an osmotic diuretic which can be administered orally. It is metabolized to glucose, however and should not be administered to diabetic patients. Side effects associated with both mannitol and glycerin administration may include vomiting, dehydration, and electrolyte imbalances.

Following the initial stabilization, more definitive control can be achieved by a combination of medical and/or surgical management to decrease aqueous production and maximize its outflow. Potentially beneficial medical agents when treating glaucoma include the prostaglandin analogues (such as latanaprost), the carbonic anhydrase inhibitors (such as dorzolamide), parasympathomimetic agents (such as demacarium bromide), and B-blockers (such as timolol). Prostaglandin analogues are generally considered ineffective in feline patients based on receptor distribution. Medications which may result in miosis should generally be avoided if an anterior lens luxation is suspected. Ongoing medical therapy with or without adjunctive surgical intervention is likely to be required to maintain control over IOP and intraocular inflammation.

Patients presenting with elevated IOP typically also demonstrate some degree of intraocular inflammation. Anti-inflammatory treatment (topically and/or systemic) significantly improves patient comfort and clinical outcomes. Topical non-steroidal anti-inflammatory drugs (NSAIDS) have not been shown to provide suitable analgesia and have been demonstrated to elevate IOPs in some cases.

The glaucomas are neurodegenerative diseases. While medical and/or surgical treatment may control IOP, secondary axonal neurodegenerative effects result in progressive optic nerve damage. Consequently, adjunctive therapy to attempt to provide some degree of neuroprotection should be considered when managing patients. Multiple medications have been proposed to have potentially neuroprotective effects, the most studied of which is the *N*-methyl-D-aspartate (NMDA)-receptor antagonist memantine. Additional therapies under further evaluation for their potentially neuroprotective properties include the topical alpha-2 adrenergic agonist brimonidine, calcium-channel-blockers, tnfα-inhibitors, ET1-antagonists, complement-inhibitors, cholesterol-statins, nitric oxide synthase (NOS)-inhibitors, rho-kinase (ROC)-inhibitors, and ROS-scavengers.

Finally, primary glaucoma is generally a bilateral disease. Agents with the ability to increase aqueous outflow, in combination with anti-inflammatory therapy, have been shown to be beneficial in preventing or delaying the onset of clinical disease in the contralateral eye.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 23
APPROPRIATE ANTIBIOTIC CHOICE

Bacterial infection of the eye and adnexa occurs frequently in association with underlying pathology and represents a common cause for clinical presentation. Accurate pathogen identification, via culture and cytology and/or serology/PCR (polymerase chain reaction), ideally determine the choice of antibiotic; however, clinical signs, history, signalment, and gram-staining can also be used to guide appropriate therapy. Antibiotics can be classified in many ways, but mechanism of action is a helpful way to discuss the types of antibiotics commonly used in when managing ophthalmic disease. In this regard, antibiotics generally fall into one of four categories: drugs that affect cell wall synthesis and cell wall integrity, drugs that affect bacterial protein synthesis, drugs that affect bacterial intermediary metabolic pathways, and drugs that inhibit bacterial DNA synthesis. Antibiotics in each of these categories may be used both topically and/or systemically.

Penicillins, cephalosporins, and bacitracin are drugs that affect cell wall synthesis. Penicillins are most useful when treating gram-positive pathogens. Clavulanate-potentiated amoxicillin and cephalosporins are especially useful when treating blepharitis. Cephalosporins vary in their efficacy against gram-positive and gram-negative bacteria, with first generation drugs being more effective against gram-positive bacteria and third generation drugs being more effective against gram-negative organisms. Cephalosporins may be administered subconjunctivally, intravenously, or may even compounded for topical use. Bacitracin is commonly used topically and is predominantly effective against gram-positive organisms. It may also be utilized in combination with other medications like polymyxin B, which disrupts the integrity of the bacterial cell wall, broadening the spectrum of antimicrobial activity when treating simple ocular surface infections.

Aminoglycosides, tetracyclines, macrolides, chloramphenicol, and clindamycin disrupt bacterial protein synthesis. Commonly available ophthalmic aminoglycosides include gentamicin, tobramycin, and neomycin (with a generally similar spectrum of activity, predominantly against gram-negative organisms).

Neomycin may be utilized in combination with bacitracin, providing a broader spectrum topical of coverage. Hypersensitivity reactions to neomycin may occur. *Rickettsia* and *Chlamydophila* organisms may be sensitive to systemic tetracyclines, macrolides, and/or chloramphenicol. Azithromycin is generally recommended for treatment of feline *bartonellosis* and clindamycin for treatment of ocular *toxoplasmosis*.

Sulfonamides and trimethoprim inhibit bacterial folic acid synthesis. They are synergistic when used in conjunction, with a generally broad spectrum of activity, however may lead to the development of keratoconjunctivitis sicca when administered systemically to canine patients.

Fluoroquinolones inhibit bacterial DNA synthesis and exert a broad spectrum of action against gram-positive and gram-negative bacteria when administered topically and/or systemically. Fluoroquinolones are useful for treatment of conjunctival, corneal, intraocular, and/or orbital bacterial infections. Systemically administered enrofloxacin has been associated with generalized (and irreversible) retinal degeneration in cats, notably at doses in excess of 5 mg/kg and particularly following IV administration.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 24
ANTIVIRALS FOR FELINE HERPES VIRUS

Feline herpes virus frequently affects ocular tissues, causing chronic and recurrent keratoconjunctivitis and/or ulcerative keratitis. Antiviral medications are used most commonly in veterinary medicine to address feline (and occasionally canine) herpes virus. These medications are virostatic, rather than virocidal, and their purpose is to suppress the virus, thereby reducing the severity and duration of outbreaks.

Most antiviral medications are classified as nucleoside analogs, which are further classified as either pyrimidine nucleoside analogs or purine nucleoside analogs. There are several non-nucleoside analog antivirals as well, although their in vivo efficacy against feline herpesvirus is limited. The choice of antiviral agent is based on efficacy, route of administration, required frequency of administration, availability, cost, and patient tolerance.

Idoxuridine and trifluridine are two commonly utilized topical antiviral pyrimidine-nucleoside-analogs. These agents act as analogs of the nucleoside deoxythymidine, necessary for viral DNA synthesis. Idoxuridine is typically compounded solution or ointment, with trifluridine is commercially available as "Viroptic" or similarly compounded. Trifluridine may be more effective, but also potentially more irritating. Both medications are applied topically for days to weeks with frequent application (q 4–6 hours) is advised.

Ganciclovir, penciclovir (the active form of famciclovir), and cidofovir are the most commonly utilized and generally effective purine nucleoside analogs. Ganciclovir is an analog of deoxyguanosine and it both disrupts viral DNA synthesis and inhibits viral DNA polymerases. Ganciclovir is available commercially as an ophthalmic gel ("Zirgan").

Penciclovir, the active form of famciclovir, is also an analog of deoxyguanosine. Famciclovir is used off label in cats. Although generally safe and effective, uncommon side effects can include nausea, vomiting, and diarrhea.

Cidofovir is an analog of deoxycytosine and has been shown to be clinically effective against feline herpesvirus with less frequent topical application (BID) than other antivirals.

The administration of oral L-lysine (available in many different forms) when managing herpes-related disease has been advocated by some authors. L-lysine administered at 250–500 mg PO BID may help to decrease viral replication by competing with arginine, which is required for viral growth/replication.

A combination of antiviral medications and supportive care (as well as the avoidance of anti-inflammatory therapy where possible) is generally the best way to manage patients affected by feline herpes-viral-associated ophthalmic disease.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

CHAPTER 25
ANTIFUNGALS IN VETERINARY OPHTHALMOLOGY

Fungal-associated diseases are relatively less commonly encountered in veterinary ophthalmology.

Dermatophytic infections may result in primary blepharitis. Mycotic blepharitis is most frequently caused by the dermatophytes *Microsporum* and *Trichophyton*, and dermatophyte testing media are commonly used to make the clinical diagnosis.

Keratomycosis occurs when the loss of corneal epithelial integrity allows fungal organisms to gain access to the stroma. Keratomycosis typically results from contact with vegetative material or soil contaminated by fungi.

Ocular manifestations of systemic fungal diseases may manifest as any combination of keratitis, uveitis, chorioretinitis, optic neuritis, and/or periocular cellulitis/abscess formation. Certain fungal pathogens are more prevalent in specific geographic locations, and exposure to soil and vegetative material, as well as immunocompromise may predispose a patient to developing clinical fungal disease. Ocular manifestations of systemic mycoses are most commonly noted in cases of coccidioidomycosis, blastomycosis, cryptococcosis, histoplasmosis, and aspergillosis. Testing for exposure to these diseases is encompassed by many standard diagnostic uveitis (blood) panels; however, false-negative tests may be associated with active fungal infections.

Dermatophytic infections of the eyelids and adjacent tissues may be treated using systemic ketoconazole, fluconazole, itraconazole, griseofulvin, or terbinafine as well as a variety of topical ointments and shampoos.

Effective treatment of keratomycosis is complicated by relative lack of available topical ophthalmic antifungal preparations and treatment of uveal, chorioretinal, and/or systemic mycoses can be challenging due to tissue penetration, potential toxicity as well as cost and availability of appropriate systemic antifungal drugs. Antifungals employed for topical ophthalmic use include the azoles (miconazole, itraconazole, voriconazole, and fluconazole) and natamycin, a polyene antibiotic. Azoles are fungistatic, primarily disrupting both fungal cell membrane synthesis and permeability. Topical 1% solutions of miconazole or voriconazole as well as Itraconazole/dimethyl sulfoxide (DMSO) combinations may be compounded and used to treat keratomycosis. Natamycin is fungicidal and serves to increase fungal cell membrane permeability and is commercially available as a 5% suspension for topical ophthalmic use.

Treatment of systemic mycoses will depend upon the specific pathogen. The polyene antibiotic amphotericin B and the azoles are the most commonly prescribed systemic antifungals in veterinary medicine. Adverse effects, including nephrotoxicity in association with amphotericin B and gastrointestinal and dermal side effects in association with the azoles may occur. Cataracts have been associated with the long-term systemic administration of ketoconazole in dogs. Additional care may also be indicated when treating uveitis, chorioretinitis, and secondary glaucoma in cases affected by ocular manifestations of systemic mycoses.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Section 4

The Ocular Examination

CHAPTER 26
THE OCULAR EXAMINATION

The ophthalmic examination should comprise the following:

HISTORY

Signalment, preexisting medical/surgical history (including travel history), and/or current medications.

PRESENTING COMPLAINT

Identification of the presenting ophthalmic complaint.

DISTANT "HANDS OFF" EXAMINATION

Patient is allowed to move around freely – demonstrating mentation, neurological status, and visual ability.

BRIEF GENERAL PHYSICAL EXAMINATION

Includes assessment of mucous membranes, oral cavity, external ear canals, thoracic auscultation, palpation of lymph nodes, and abdomen and body temperature.

CLOSE UP "HANDS ON" EXAMINATION

Includes careful palpation of the skull and orbits, noting any deformity, asymmetry, crepitus, or discomfort.

NEUROPHTHALMIC EXAMINATION

- Palpebral reflex (closure of eyelids upon tactile stimulus)
- Menace response (eyelid closure and/or head withdrawal in response to menacing hand gesture)
- Dazzle reflex (closure of eyelids in response to bright focal light source being shined into eye)
- Pupillary light reflex (PLR) (direct and consensual reflex pupillary miosis in response to a focal light source)

SEGMENTAL ANTERIOR EXAMINATION

A focal light source is used to examine the eyelids, conjunctival surfaces, third eyelid, sclera, cornea, anterior chamber (AC), iris, lens, and anterior vitreous face.

FUNDIC EXAMINATION

The posterior segment is examined using either a focal light source and handheld lens or an ophthalmoscope.

ANCILLARY DIAGNOSTICS

- Schirmer Tear Test I (STT1) tear test (Schirmer strip placed into ventral fornix for one minute, normal value = 15–20 mm/wetting/min)
- Intraocular Pressure (IOP) estimation (using applanation or rebound tonometer, normal value = 15–25 mmHg)
- Vital Staining (sodium fluorescein dye is applied to cornea to detect retention associated with epithelial defects) may also be used to assess nasolacrimal duct patency ("Jones" test) and/or aqueous leakage form the AC ("Seidel" test).

Where indicated, additional diagnostic aides may include obtaining blood samples for complete blood count (CBC), chemistry, metabolic, endocrine, and/or infectious titer testing, the harvesting of microbial samples for culture and sensitivity testing as well as cytological and/or histological examination and/or advanced imaging (including radiography, B-mode ultrasonography, computed tomography, and/or magnetic resonance imaging).
Where an indirect ophthalmoscope is unavailable, a small flashlight or transilluminator will suffice as a distant focal light source, held adjacent to the examiner's head.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 26.1 Estimation of intraocular pressure (IOP) using a rebound tonometer ("tonovet"). The instrument's probe is gently allowed to contact the axial corneal surface (without the use of local anesthetic) by pressing the measurement button. Several readings are taken, so that aberrant readings may be disregarded.

Figure 26.2 Measurement of lacrimal function using gradated ("Schirmer") tear strips. The tip of each strip is folded and placed into the lower medial fornix for one minute and the resultant Schirmer Tear Test (STT) value recorded. This test should be performed prior to the installation of any topical agents.

Figure 26.3 Vital staining of the corneal surface using fluorescein-impregnated strips. Strips are moistened using physiologic eyewash and gently touched to the scleral limbus. Excess stain is then carefully irrigated away using physiologic eyewash to prevent residual "pooling" of stain.

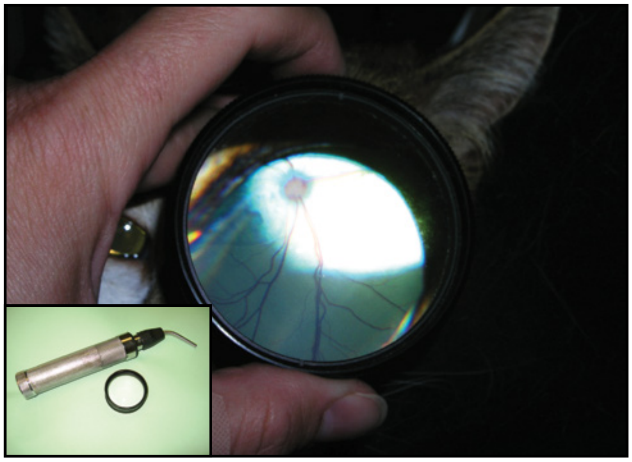


Figure 26.4 Visualization of the fundus using a simple hand-held indirect lens, positioned just in front of the eye and parallel to the posterior segment.

Section 5

Diseases of the Eyelids

CHAPTER 27
EYELID AGENESIS

PRESENTATION

Feline eyelid agenesis describes a congenital condition in which affected animals are born with incomplete eyelid margins. Multiple kittens from a single litter are commonly affected. Typically, the presentation is bilateral and symmetrical with the full thickness defect affecting the superiotemporal eyelid margins; however, variations may occur. The size of the defect may also vary. Additional ocular abnormalities may be present, with persistent pupillary membranes (PPMs) being the most common. Eyelid agenesis commonly results in trichiasis or contact between hairs and the corneal surface. Potential sequelae include fibrosing, vascular and pigmentary keratitis, the formation of corneal sequestra and/or corneal ulceration, which may become severe enough to result in perforation of the globe.

DIAGNOSIS AND TREATMENT

Medical management of this condition includes the frequent application of a topical lubricant to coat and protect the corneal surfaces as well as the treatment of inflammatory and/or ulcerative lesions. The use of immune-suppressive medications and/or corticosteroids should be cautiously considered as a result of their potential to facilitate herpesviral recrudescence and/or aggravate ulcerative lesions. In simple or less severe cases, offending hairs (and their follicles) may be permanently removed via cryo-epilation. More elegant but complex surgical techniques to recreate a functional eyelid margin have been described (generally encompassing the transposition of skin, orbicularis muscle, and conjunctival tissue), the most typically appropriate being that by Dziezyc and Millichamp. These procedures however, may require a multistage surgical approach and limited subsequent cryo-epilation may additionally be necessary. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete.

SELECTED REFERENCES

Dziezyc J and Millichamp NJ. Surgical correction of eyelid agenesis in a cat. *J Am Anim Hosp Assoc*. 1989;**25**, 513–516.

Glaze MB. Congenital and hereditary ocular abnormalities in cats. *Clin Tech Small Anim Pract*. 2005;**20**(2):74–82.

Whittaker CJ, Wilkie DA, Simpson DJ, Deykin A, Smith JS, Robinson CL. Lip commissure to eyelid transposition for repair of feline eyelid agenesis. *Vet Ophthalmol*. 2010;**13**(3):173–178.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 27.1 Feline upper eyelid agenesis. Inset demonstrates the typical bilateral and symmetric nature of the condition.

Figure 27.2 Feline upper eyelid agenesis. Note dorsal fine superficial vascular keratitis as a result of exposure.

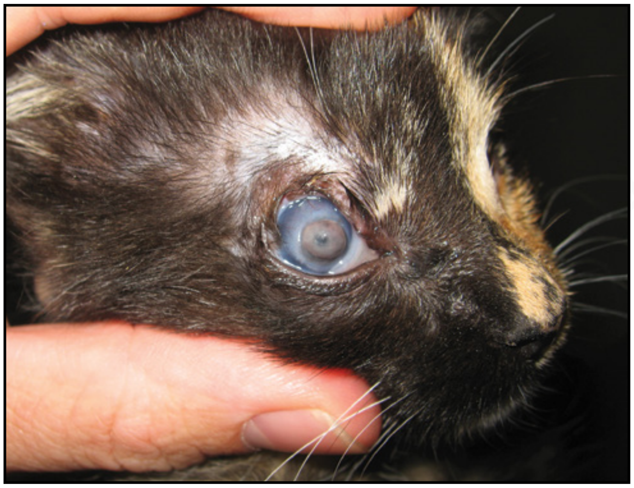


Figure 27.3 Feline upper eyelid agenesis. Note the perforating ulcerative keratitis secondary to chronic trichiasis.

Figure 27.4 Feline upper eyelid agenesis. Note the presence of fine persistent pupillary membranes (PPMs) in the anterior chamber.

CHAPTER 28
EYELID LACERATION

PRESENTATION

The eyelids comprise an inner layer (made up of conjunctiva and underlying connective tissue) and an outer layer (made of up haired skin and underlying orbicularis muscle). The eyelid margins also contain the lipid-secreting meibomian glands. Eyelid lacerations are commonly encountered, as a result of trauma such as bite-wounds. Full-thickness lacerations disrupt normal eyelid structure and function and require accurate reconstruction.

DIAGNOSIS AND TREATMENT

Potentially life-threatening injuries should be addressed and stabilized prior to treating eyelid injuries. Sedation or anesthesia may be required in order to facilitate adequate evaluation and treatment. Affected tissues should be carefully cleansed and copiously irrigated using physiologic saline and foreign material as well as accumulated mucous and blood gently removed in order to assess the extent of injury and tissue viability. Devitalized tissues should be carefully debrided, taking care to retain as much of the eyelid margin as possible as well as to preserve subcutaneous vascular structures. Fresh wounds (24–48 hours) can usually be debrided and repaired immediately. Older wounds may require the treatment of significant inflammation and/or secondary bacterial infection prior to debridement and repair. Ultimately, however, full-thickness eyelid lacerations should be surgically repaired rather than being allowed to heal by second intention in order to ensure accurate apposition and long-term functionality. Full-thickness eyelid lacerations should be repaired in two layers. A continuous absorbable suture material should be used to appose deeper conjunctival and connective tissue layers. Interrupted nonabsorbable sutures should be used to close the outer skin and muscular layers. Careful and accurate apposition of the eyelid margin (ideally by placing a “figure-8” suture) is critical in order to prevent subsequent keratopathy. Where appropriate, dead space should be closed and drains placed. If laceration of the nasolacrimal duct and/or canaliculi is identified, these structures may be stented using silicone or nylon. Postoperative care comprises routine systemic antimicrobial, anti-inflammatory and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete.

SELECTED REFERENCES

Aquino SM. Surgery of the eyelids. *Top Companion Anim Med*. 2008;**23**(1):10–22. doi: 10.1053/j.ctsap.2007.12.003.

Lackner PA. Techniques for surgical correction of adnexal disease. *Clin.Tech Small Anim Pract*. 2001;**16**(1):40–50.

Mandell DC and Holt E. Ophthalmic emergencies. *Vet Clin North Am Small Anim Pract*. 2005;**35**(2):455–480, vii–viii.

Reifler DM. Management of canalicular laceration. *Surv Ophthalmol*. 1991;36(2):113–132.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 28.1 Eyelid lacerations. This acute full-thickness laceration requires prompt two-layer closure.

Figure 28.2 Eyelid lacerations. Careful apposition of the eyelid margin will be necessary to avoid subsequent keratopathy.

Figure 28.3 Eyelid lacerations. The infected wound shown here requires medical treatment prior to surgical repair.

Figure 28.4 Eyelid lacerations. The fibrosed wound edges require resection prior to reconstruction.

CHAPTER 29
DISTICHIASIS

PRESENTATION

Distichiae are abnormal cilia arising from the eyelid margin at/or close to the level of the meibomian gland openings. In most cases, distichiae do not result in significant ocular pathology; however, contact between distichiae and the corneal surface may result in discomfort marked by epiphora and blepharospasm and may lead to secondary keratitis and/or corneal ulceration. Distichiae may affect one or both eyelids, (upper and/or lower). Distichiae occur commonly in dogs and less commonly in cats. Commonly affected breeds include the Cocker Spaniel, Boxer, English Bulldog, Yorkshire Terrier, and Shih Tzu.

DIAGNOSIS AND TREATMENT

Where indicated, the appropriate treatment of distichiae represents removal and the permanent destruction of their associated follicle. Manual epilation alone may provide temporary relief but will result in regrowth. Permanent follicular destruction may be achieved using surgical excision, cryo-epilation in a double freeze-thaw cycle and/or electro-epilation. Cryo-epilation should only be undertaken using an appropriate nitrous-oxide cooled unit. Focal swelling and eyelid depigmentation are common following cryo-epilation; however, affected tissues typically re-pigment with time. Extreme care should be taken to preserve the eyelid margins and to avoid excessive eyelid tissue damage or necrosis and the generation of excessive fibrosis, which may lead to cicatricial entropion. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete.

SELECTED REFERENCES

Aquino SM. Surgery of the eyelids. *Top Companion Anim Med*. 2008;**23**(1):10–22. doi: 10.1053/j.ctsap.2007.12.003.

Ioannides J, Everson R, Matas Riera M, Dawson C. A description of blend electrolysis for treatment of **canine** distichiasis: 78 cases (2012–2017). *Vet Rec*. 2020;**187**(11):e102.

Lackner PA. Techniques for surgical correction of adnexal disease. *Clin Tech Small Anim Pract*. 2001;**16**(1):40–50.

Wheeler CA and Severin GA. Cryosurgical epilation for the treatment of distichiasis in the dog and cat. *J Am Anim Hosp Assoc*. 1984;**20**:877–884.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 29.1 Note epiphora and mucoid ocular discharge as a result of irritation.

Figure 29.2 Note the varying lengths and thickness of the distichia, some of which have been previously manually epilated.

Figure 29.3 Aberrant hairs are most readily identified against the pale conjunctival and episcleral tissues.

Figure 29.4 Although less common, distichiasis is identified in feline patients as well.

CHAPTER 30
ECTOPIC CILLIA

PRESENTATION

Ectopic cilia are abnormal hairs arising from the inner aspect of the eyelid margin and are most commonly encountered within the upper eyelids of young dogs. Either a single or group of ectopic hairs may be present. In most cases, ectopic cilia result in significant ocular pathology with discomfort marked by epiphora and blepharospasm and typically lead to keratitis and/or corneal ulceration. Ectopic cilia may be challenging to identify, particularly in painful or uncooperative patients, those with significant conjunctival swelling and/or light-colored hair coats. Where ectopic cilia are suspected (such as in cases of persistent or recurrent corneal ulceration), sedation and/or magnification may be required in order to confirm their presence. Commonly affected breeds include the Shih Tzu, English Bulldog, French Bulldog, and Golden Retriever.

DIAGNOSIS AND TREATMENT

The appropriate treatment of ectopic cilia represents en bloc removal of both the hair(s) and its associated follicle, with or without adjunctive cryo-therapy in a double freeze-thaw cycle. Manual epilation alone may provide temporary relief but will result in hair regrowth. Cryotherapy should only be undertaken using an appropriate nitrous-oxide cooled cryo unit. Focal swelling and eyelid depigmentation are common following cryoepilation; however, affected tissues typically repigment with time. Extreme care should be taken to preserve the eyelid margins and to avoid excessive eyelid tissue damage or necrosis and the generation of excessive fibrosis, which may lead to cicatricial entropion. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete.

SELECTED REFERENCES

Aquino SM. Surgery of the eyelids. *Top Companion Anim Med*. 2008;**23**(1):10–22.

D'Anna N, Sapienza JS, Guandalini A, Guerriero A. Use of a dermal biopsy punch for removal of ectopic cilia in dogs: 19 cases. *Vet Ophthalmol*. 2007;**10**(1):65–67.

Hase K, Kase S, Noda M, Ohashi T, Shinkuma S, Ishida S. Ectopic cilia: a histopathological study. *Case Rep Dermatol*. 2012;**4**(1):37–40. doi: 10.1159/000336887

Lackner PA. Techniques for surgical correction of adnexal disease. *Clin Tech Small Anim Pract*. 2001;**16**(1):40–50.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 30.1 Typical solitary ectopic cilium within the upper palpebral conjunctiva at the 2 o'clock position. Note vascular and fibrosing keratitis dorsally and mucoid discharge associated with chronic corneal irritation.

Figure 30.2 Solitary, thick, upper eyelid ectopic cilium at the 12 o'clock position.

Figure 30.3 Note the presence of multiple ectopic cilia at the 12 o'clock position. Keratitis as a result of chronic irritation is present.

Figure 30.4 Note the more uncommon presentation in the lower eyelid palpebral conjunctiva at the 6 o'clock position. Secondary ulcerative keratitis is present.

CHAPTER 31
TRICHIASIS

PRESENTATION

Trichiasis describes contact between hairs from normal hair-bearing periocular skin and the corneal surface. This may involve hairs from the upper lid, medial canthus, or nasal fold region. Corneal irritation may result in discomfort, keratitis, and/or corneal ulceration. Commonly affected breeds include the Cocker Spaniel, Shih Tzu, Pug, and Miniature Poodle.

DIAGNOSIS AND TREATMENT

Selected patients may be successfully managed by careful and regular grooming of periocular hair. More definitive treatment involves judicious cryoepilation of offending hairs and/or surgical resection of hair-bearing skin where appropriate. Several surgical techniques have been described including that for partial upper eyelid resection by Stades and that for nasal fold resection by Severin. Cryoepilation should only be undertaken using an appropriate nitrous-oxide cryo unit. Focal depigmentation and swelling are common following cryoepilation; however, affected tissues typically repigment with time. Extreme care should be taken to preserve the eyelid margins and to avoid excessive eyelid tissue damage or necrosis and the generation of excessive fibrosis, which may lead to cicatricial entropion. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete. Keratitis and/or corneal ulceration should be managed using routine topical antimicrobial therapy as well as the judicious use of a lubricating and protective agent.

SELECTED REFERENCES

Gelatt KN *et al*. 2013. *Veterinary Ophthalmology*, Vol. 2, pp. 867–871, Wiley-Blackwell, Oxford, UK.

Lackner PA. Techniques for surgical correction of adnexal disease. *Clin Tech Small Anim Pract*. 2001;**16**(1):40–50.

Stades FC, van de Sandt RR, Boevé MH. Clinical aspects and surgical procedures in trichiasis. *Tijdschr Diergeneeskd*. 1993;**118** Suppl 1: 38S–39S.

Yi NY, Park SA, Jeong MB, Kim MS, Lim JH, Nam TC, Seo K. Medial canthoplasty for epiphora in **dogs**: a retrospective study of 23 cases. *J Am Anim Hosp Assoc*. 2006;**42**(6):435–439.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 31.1 Conformational trichiasis associated with the upper eyelid. Note the excessive epiphora and periocular tear staining.

Figure 31.2 Trichiasis associated with the upper eyelid following inappropriate removal of distichia and cicatricial upper eyelid entropion.

Figure 31.3 Trichiasis in association with prominent nasal folds. Significant secondary keratitis is present.

Figure 31.4 Chronic keratitis secondary to conformational hypertrichiasis.

CHAPTER 32
TEAR FILM WICKING SYNDROME

PRESENTATION

The medial canthal caruncle describes the region of haired tissue at the base of the third eyelid. In many canine breeds, particularly small breeds, this region contains an excessive amount of hair which may contact the corneal surface, acting as a "wick" and drawing the pre-corneal tear film out onto the haired skin surrounding the medial canthus. This may lead to secondary tear-staining and in severe cases, recurrent moist dermatitis. Clinical signs may be exacerbated by stenotic or imperforate nasolacrimal puncta and/or ducts. The patency of the nasolacrimal drainage system may be evaluated via the "Jones Test" during which a small amount fluorescein dye is instilled into the ventral fornix. In animals with adequate nasolacrimal drainage, dye will be noted to exit the ipsilateral nostril within 5–10 minutes. In patients affected by wicking syndrome, fluorescein dye will be noted to stream out onto the haired eyelid skin around the medial canthal region. Commonly affected breeds include the Shih Tzu, Lhasa Apso, Miniature Poodle, and Bichon Frise.

DIAGNOSIS AND TREATMENT

Tear film wicking may be significantly reduced by the permanent removal of hairs in the medial caruncular region. This is most effectively achieved using judicious cryoepilation in a double freeze-thaw cycle. Manual epilation alone may result in temporary relief but will result in regrowth of hairs. Cryotherapy should only be undertaken using an appropriate nitrous-oxide cryo-unit. Significant swelling and eyelid depigmentation are common following cryoepilation; however, affected tissues typically repigment with time. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete. Stenotic nasolacrimal systems may be cannulated and flushing attempted. In rare cases of punctual aplasia, nasolacrimal openings may be surgically re-established. Postoperative indwelling stents using nylon or silicone may help reduce the propensity for recurrent stenosis, which nevertheless remains high. Several techniques have also been described for the creation of alternative nasolacrimal drainage into the oral and sinus spaces; however, these procedures are rarely justified.

SELECTED REFERENCES

Gelatt KN and Gelatt JP. 1995. *Handbook of Small Animal Ophthalmic Surgery: Extraocular Procedures*, Vol. 1. Pergammon Press, Gainesville, FL.

Gelatt KN *et al*. 2013. *Veterinary Ophthalmology,* Vol. 2, pp. 871–873, Wiley-Blackwell, Oxford, UK.

Ny Y *et al*. Medial canthoplasty for epiphora in dogs: a retrospective study of 23 cases. *J Am Anim Hosp Assoc*. (2006);42, 435–439.

Seo KM and Nam TC. Tear formation, the patency and angle of bend of nasolacrimal duct in poodle dogs with tear staining syndrome. *Korean J Vet Res*. (1995);**35**, 383–390

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 32.1 Tear film wicking syndrome. Note that staining is more pronounced in animals with white or light hair coats.

Figure 32.2 Tear film wicking syndrome. Significant tear film wicking in association with trichiasis.

Figure 32.3 Tear film wicking syndrome. Significant tear film wicking and facial staining.

Figure 32.4 Tear film wicking syndrome. Significant tear film wicking and secondary moist dermatitis.

CHAPTER 33
ENTROPION

PRESENTATION

Entropion describes eyelid pathology such that the eyelid margin(s) are inverted toward the globe(s). Most typically, the lower eyelids are involved; however, upper eyelid entropion may also occur. Similarly, the condition is most commonly bilateral; however, unilateral entropion may occur. Primary entropion arises due to a conformational eyelid abnormality and may additionally involve excessive eyelid length and laxity and/or brow-fold redundancy. Primary entropion is common in multiple breeds and may affect juvenile animals (particularly the English Bulldog, Chow Chow, and Shar Pei) as well as older animals (particularly the Labrador Retriever and Rottweiler). Spastic entropion arises as a result of ocular discomfort (such as that arising from corneal ulceration) and subsequent chronic blepharospasm. Spastic entropion may be differentiated from primary conformational entropion by the application of a topical anesthetic agent, which results in resolution of symptoms. Secondary entropion may also result from abnormalities of globe positioning (most commonly enophthalmos due to orbital fat and/or muscle atrophy) or globe size (most commonly due to phthisis or shrinkage of the globe). Cicatricial entropion may arise secondary to post-inflammatory contracture and eyelid scarring. Regardless of the etiology of entropion, corneal pathology generally arises as a result of trichiasis.

DIAGNOSIS AND TREATMENT

Pending more definitive intervention, corneal tissues affected by entropion may be temporarily protected by the liberal application of a petroleum-based ocular lubricants. Both juvenile primary entropion and spastic entropion may be initially treated by temporarily everting the eyelids using either nonabsorbable mattress sutures or surgical staples. Sutures or staples may be left in place (and/or replaced) for weeks to months as appropriate. Entropion in juvenile animals may resolve with increasing age and musculoskeletal conformational maturity. Multiple surgical techniques have been described for the permanent correction of either persistent or primary conformational entropion, including the basic "Hotz-Celsus" procedure and its variations. These techniques are also appropriate for the management of entropion arising secondary to abnormalities of globe position or size as well as cicatricial entropion. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete. Extreme caution is advised when considering the permanent correction of entropion using techniques such as electrocautery, high wavelength laser energy, and/or the injection of subcutaneous materials since the potential for inadequate correction as well as surgical complication associated with these procedures is high.

SELECTED REFERENCES

Lewin GA. Temporary lateral tarsorrhaphy for the treatment of lower lateral eyelid entropion in juvenile dogs. *Vet Rec*. 2000;**146**(15):439–440.

Moore CP and Constantinescu GM. Surgery of the adnexa. *Vet Clin North Am Small Anim Pract*. 1997;**27**(5):1011–1066.

Read RA and Broun HC. Entropion correction in dogs and cats using a combination Hotz-Celsus and lateral eyelid wedge resection: results in 311 eyes. *Vet Ophthalmol*. 2007;**10**(1):6–11.

Williams DL and Kim JY. Feline **entropion**: a case series of 50 affected animals (2003–2008). *Vet Ophthalmol*. 2009;**12**(4):221–226.

van der Woerdt A. Adnexal surgery in dogs and cats. *Vet Ophthalmol*. 2004;**7**(5):284–290.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 33.1 Note the significant trichiasis as a result of lower eyelid entropion.

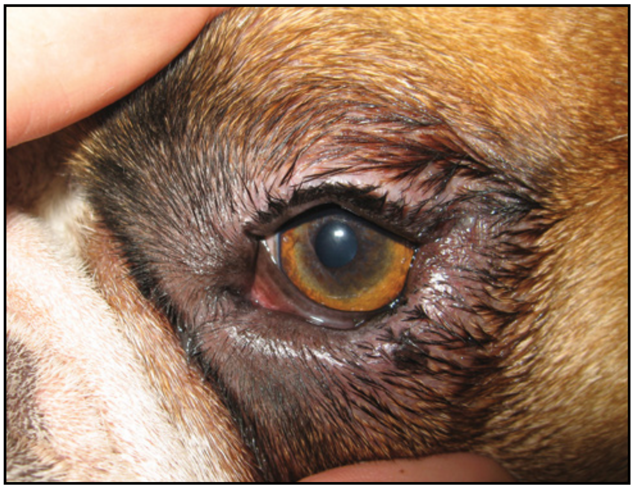


Figure 33.2 Conformational entropion, resulting from excessive lower eyelid laxity. Note the periocular moist dermatitis associated with entropion.

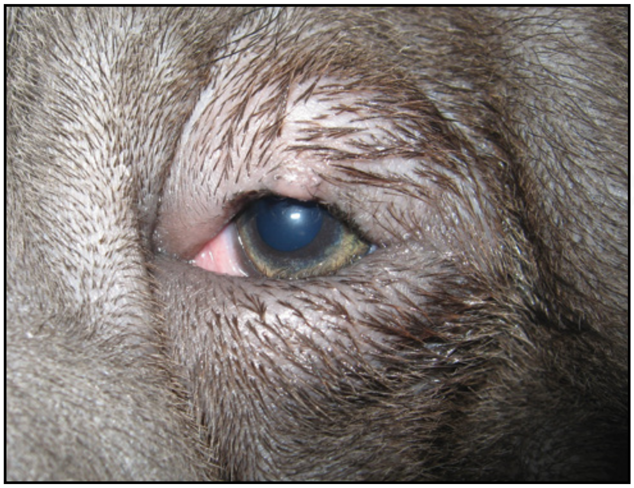


Figure 33.3 This patient displays both upper and lower eyelid entropion.

Figure 33.4 Feline lower eyelid entropion occurs most often in older patients as a result of enophthalmos.

CHAPTER 34
ECTROPION

PRESENTATION

Ectropion describes eversion of the eyelid(s) away from the ocular surface such that increased exposure and resultant keratoconjunctivitis may result. Ectropion is less common and less likely to result in significant corneal pathology than entropion. Ectropion may be the result of conformational abnormalities (particularly in the Bloodhound, St Bernard, Newfoundland and Cocker Spaniel) or may occur secondary to trauma and subsequent cicatricial scarring or the inadvertent surgical overcorrection of entropion.

DIAGNOSIS AND TREATMENT

Ectropion may be medically managed in selected cases using a combination of anti-inflammatory as well as lubricating and protective therapy. Surgical correction of ectropion is only indicated if the condition results in significant secondary pathology. Typically, a wedge resection and/or "V to Y" procedure is employed. Surgical correction should ideally not be undertaken until patients have reached conformational maturity. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete.

SELECTED REFERENCES

Donaldson D, Smith KM, Shaw SC, Sansom J, Hartley C. Surgical management of cicatricial **ectropion** following scarring dermatopathies in two **dogs.** *Vet Ophthalmol*. 2005;**8**(5):361–366. Erratum in: Vet Ophthalmol. 2005 Nov-Dec;8(6):451.

Gelatt KN and Gelatt JN. 2001. *Small Animal Ophthalmic Surgery*, pp. 101–102, Elsevier, Edinburgh.

Lackner PA. Techniques for surgical correction of adnexal disease. *Clin Tech Small Anim Pract*. 2001;**16**(1):40–50.

van der Woerdt A. Adnexal surgery in **dogs** and cats. *Vet Ophthalmol*. 2004;**7**(5):284–290.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 34.1 Breed related conformational lower eyelid ectropion. Moderate third eyelid conjunctival hyperemia and exposure are present.

Figure 34.2 Breed related conformational lower eyelid ectropion. Mild conjunctivitis is present.

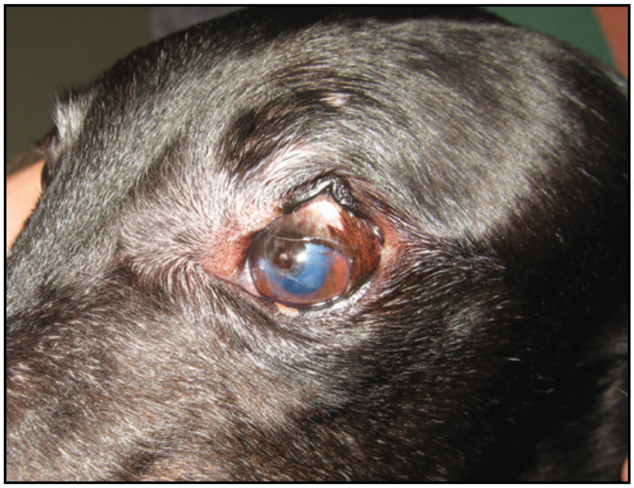


Figure 34.3 Secondary (cicatricial) ectropion; note the pigmentary and fibrosing keratitis as a result of exposure.

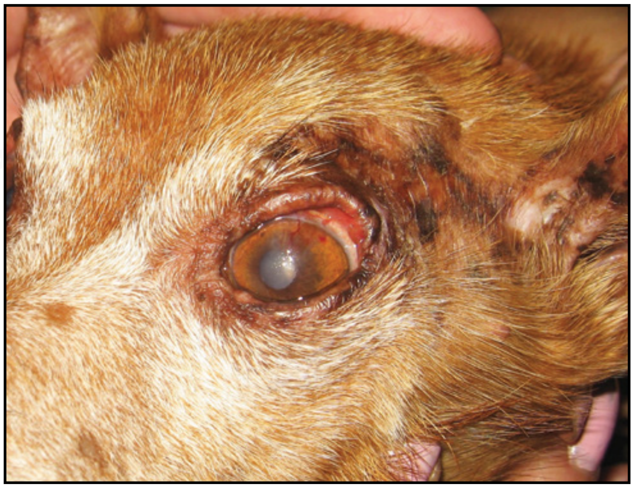


Figure 34.4 Secondary (cicatricial) ectropion caused by injury to upper eyelid and face adjacent to pinna.

CHAPTER 35
COMBINED ENTROPION-ECTROPION

PRESENTATION

Combined entropion/ectropion is commonly referred to as "diamond eye." This conformation comprises lateral and/or medial entropion in combination with central ectropion of the upper and/or lower eyelids. This presentation is common in large breed dogs, particularly those with redundant facial skin (including the St Bernard, Clumber Spaniel and English Mastiff). Secondary pathology associated with both entropic and entropic components may result, including keratoconjunctivitis, trichiasis, and/or corneal ulceration.

DIAGNOSIS AND TREATMENT

Combined entropion/ectropion may be medically managed in selected cases using a combination of anti-inflammatory as well as lubricating and protective therapy. Surgical correction is indicated if the condition results in significant secondary pathology. A number of complex and combined blepharoplasty procedures have been described for the permanent correction of diamond eye, including the generally applicable modified Kuhnt-Szymanowski technique. Surgical correction should ideally not be undertaken until patients have reached conformational maturity. Pending this, temporary eyelid eversion, using nonabsorbable sutures or surgical staples, may be employed in young or skeletally immature animals.

SELECTED REFERENCES

Bedford PG. Technique of lateral canthoplasty for the correction of macropalpebral fissure in the dog. *J Small Anim Pract*. 1998;**39**(3):117–120.

Gelatt KN and Gelatt JN. 2001. *Small Animal Ophthalmic Surgery*, pp. 102–103, Elsevier, Edinburgh.

Steinmetz A. Shared rhytidectomy continued to lateral canthoplasty in a Mastiff with excessive facial folding and macroblepharon. *Tierarztl Prax Ausg K Kleintiere Heimtiere*. 2015;**43**(1):40–44

van der Woerdt A. Adnexal surgery in **dogs** and cats. *Vet Ophthalmol*. 2004;**7**(5):284–290.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

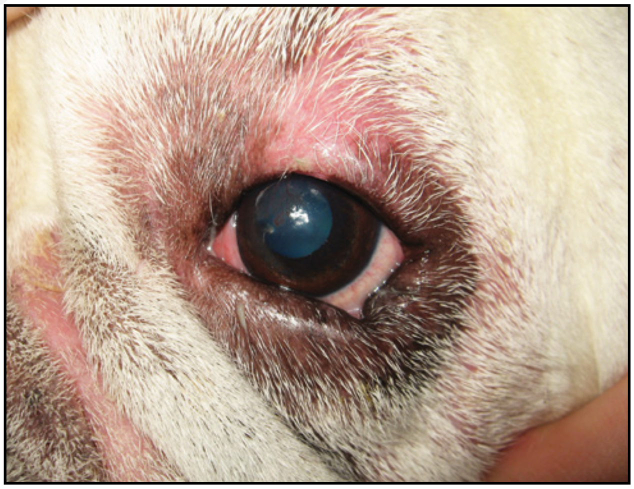


Figure 35.1 Combined entropion-ectropion. Moderate keratitis is present.

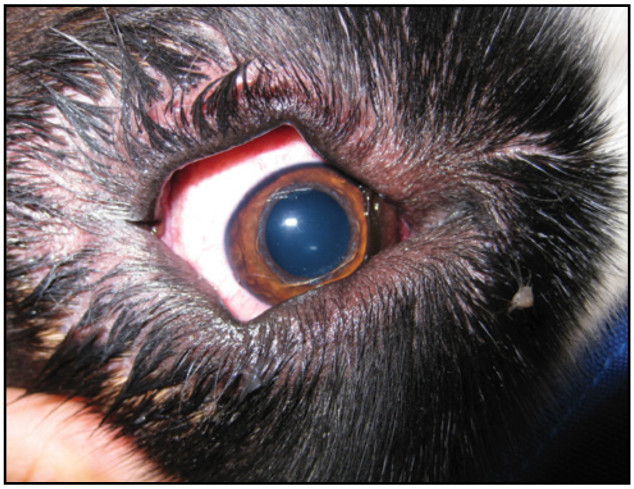


Figure 35.2 Combined entropion-ectropion. Note that the entropic component typically occurs toward the lateral and/or medial canthal regions and the ectropic component typically occurs centrally.

Figure 35.3 Combined entropion-ectropion. Moderate keratoconjunctivitis is present.

Figure 35.4 Combined entropion-ectropion. Mild conjunctivitis is present.

CHAPTER 36
MACROPALPEBRAL FISSURE

PRESENTATION

The term macropalpebral fissure describes a conformation comprising excessively large eyelid fissures and eyelid length. Lagophthalmos or a decreased ability to effectively close the eyelids as well as a tendency to sleep with the eyelids partially open may additionally be present. Keratoconjunctivitis, tear-film abnormalities, ulcerative keratitis, and an increased risk of proptosis result from increased exposure of the globe. Commonly affected breeds including the Pekinese, Japanese Chin, Shih Tzu, Lhasa Apso, and Pug.

DIAGNOSIS AND TREATMENT

Selected patients may be medically managed using lubricating and protective therapy (in combination with anti-inflammatory if indicated). Medications to increase tear production and/or stabilize the pre-corneal tear film may also be of benefit. These include topical cyclosporine (available in various concentrations). Potential adverse effects associated with the use of cyclosporine include hypersensitivity and gastrointestinal (GI) distress. Surgical correction is indicated if the condition results in significant pathology such as exposure keratopathy and/or ulcerative keratitis. Multiple techniques to decease the size of the palpebral fissure have been described, including the generally appropriate Roberts/Jensen medial pocket canthoplasty.

SELECTED REFERENCES

Bedford PG. Technique of lateral canthoplasty for the correction of macropalpebral fissure in the dog. *J Small Anim Pract.* 1998;**39**(3):117–120.

Gelatt KN, Gelatt JN. 2001. *Small Animal Ophthalmic Surgery*, p. 109, Elsevier, Edinburgh.

Krecny M, Tichy A, Rushton J, Nell B. A retrospective survey of ocular abnormalities in pugs: 130 cases. *J Small Anim Pract.* 2015;**56**(2):96–102.

van der Woerdt A. Adnexal surgery in dogs and cats. *Vet Ophthalmol.* 2004;**7**(5):284–290. Review.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 36.1 Conformational macropalpebral fissure. Note severe chronic corneal pathology including vascular, fibrosing, and pigmentary keratitis.

Figure 36.2 Conformational macropalpebral fissure. Mild keratitis is present medially and axially due to significant globe exposure.

Figure 36.3 Conformational macropalpebral fissure. Mild pigmentary keratitis is noted medially.

Figure 36.4 Conformational macropalpebral fissure. Mild vascular keratitis is noted medially and axially.

CHAPTER 37
CHALAZION

PRESENTATION

A Chalazion describes a granulomatous reaction within the (upper or lower) eyelid, specifically affecting the meibomian gland and resulting from glandular inflammation, infection, and/or obstruction. Glandular inflammation and/or infection may be associated with more widespread blepharoconjunctivitis and/or meibomitis. Glandular obstruction is commonly the result of eyelid margin neoplasia. Clinically, these lesions comprise one or more non-painful, firm, pale-yellow swellings at or inside one or both eyelid margins. This finding is common in older dogs.

DIAGNOSIS AND TREATMENT

Topical anti-inflammatory and/or antimicrobial treatment alone is unlikely to be effective. Systemic antimicrobial therapy will address bacterial blepharitis if present; however, the granulomatous reaction which comprises a chalazion, requires physical incision, drainage, and curettage in order to be effectively reduced. Any eyelid-associated neoplasia should be concurrently addressed. Appropriate antimicrobial selections include cephalosporins and tetracyclines. Potential adverse effects associated with cephalosporins include hypersensitivity reactions, gastrointestinal (GI) distress, and renal disease. Potential adverse effects associated with tetracyclines include GI distress, photosensitivity, and hepatic damage. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Postoperatively warm-compressing and/or systemic anti-inflammatory therapy will speed the resolution of clinical signs; however, new or recurrent chalazia may occur.

SELECTED REFERENCES

Dubielzig R *et al*. 2010. *Veterinary Ocular Pathology*, p. 162, Elsevier, Edinburgh.
Leis ML, Sandmeyer LS. Diagnostic ophthalmology. *Can Vet J.* 2019;**60**(1):95–96.
Peiffer RL Jr. Ocular immunology and mechanisms of ocular inflammation. *Vet Clin North Am Small Anim Pract.* 1980;**10**(2):281–302.
Viñas M, Maggio F, D'Anna N, Rabozzi R, Peruccio C. **Meibomian gland dysfunction** (MGD), as diagnosed by non-contact infrared Meibography, in **dogs** with ocular surface disorders (OSD): a retrospective study. *BMC Vet Res.* 2019;**15**(1):443.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 37.1 A chalazion affecting the lower eyelid, note the accumulation of meibum (meibomian gland secretion.)

Figure 37.2 A chalazion affecting the lower eyelid.

Figure 37.3 Chalazia affecting the upper eyelid, note the inflammation of two adjacent meibomian glands.

Figure 37.4 This chalazion has developed secondary to the obstruction of a meibomian gland by a small, benign eyelid tumor.

CHAPTER 38
JUVENILE PYODERMA

PRESENTATION

Juvenile pyoderma (also referred to as "juvenile cellulitis," "juvenile dermatitis," or "puppy-strangles") describes a pyogranulomatous condition affecting young dogs, generally within the first 6 months of life. Clinical symptoms comprise variable combinations of symmetrical pustular dermatitis of the muzzle, face, periocular tissues, and/or ears as well as external ear canal inflammation and/or regional (submandibular) reactive lymphadenopathy. In some cases, more generalized dermatitis may also be present. The disease is postulated to be an immune-mediated reaction; however, secondary bacterial infection is common. Commonly affected breeds include the Golden Retriever, Labrador Retriever, and Dachshund.

DIAGNOSIS AND TREATMENT

The diagnosis of juvenile pyoderma is based on clinical findings, confirmed via histological assessment of skin biopsies if necessary. Bacterial culture and sensitivity testing is frequently unrewarding. Treatment comprises the administration of systemic corticosteroids, with or without adjunctive systemic anti-microbial therapy. Appropriate empirical antimicrobial selections include cephalosporins and tetracyclines (with or without adjunctive niacinamide). Potential adverse effects associated with the use of systemic cortiocosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with cephalosporins include hypersensitivity reactions, gastrointestinal (GI) distress, and renal disease. Potential adverse effects associated with tetracyclines include GI distress, photosensitivity, and hepatic damage. Potential adverse effects associated with niacinamide include GI distress.

SELECTED REFERENCES

Gortel K. Recognizing pyoderma: more difficult than it may seem. *Vet Clin North Am Small Anim Pract.* 2013;**43**(1):1–18. doi: 10.1016/j.cvsm.2012.09.004.

Hutchings SM. Juvenile cellulitis in a puppy. *Can Vet J.* 2003;**44**(5):418–419.

White SD, Rosychuk RA, Stewart LJ, Cape L, Hughes BJ. Juvenile cellulitis in dogs: 15 cases (1979–1988). *J Am Vet Med Assoc.* 1989;**195**(11):1609–1611.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 38.1 Juvenile pyoderma. Findings associated with juvenile pyoderma, marked by symmetrical, facial, and periocular pustular dermatitis (the inset demonstrates typical bilateral presentation).

Figure 38.2 Juvenile pyoderma. Mild presentation.

Figure 38.3 Juvenile pyoderma. Note typical bilateral presentation.

Figure 38.4 Juvenile pyoderma. Note young age of patient.

CHAPTER 39 IMMUNE-MEDIATED BLEPHAROCONJUNCTIVITIS

PRESENTATION

The term "blepharoconjunctivitis" defines a severe, acute, bilateral inflammation of the eyelid tissues which occurs relatively frequently, particularly in small breed dogs. Clinical symptoms may include irregular erythema and/or swelling of the eyelids and marked patient discomfort manifesting as blepharospam and epiphora. An associated conjunctivitis is also typically present. This condition may or may not be associated inflammation or infection of the meibomian glands or "meibomitis" and may additionally coincide with the incidence and severity of more generalized atopic dermatitis. Commonly affected breeds include the Chihuahua, Miniature Pincher, Papillion, and Dachshund.

DIAGNOSIS AND TREATMENT

The diagnosis of immune-mediated blepharoconjunctivitis is made on clinical findings. It is likely that a sterile hypersensitivity reaction to commensal bacterial eyelid flora plays a role in many cases and bacterial culture and sensitivity testing frequently fails to identify the presence of specific pathogens. The concurrent presence of parasitic mites may be ruled out by skin-scraping. Biopsy may be indicated in severe or poorly responsive cases in order to differentiate them from autoimmune and/or neoplastic processes (see also Autoimmune blepharoconjunctivitis). Topical therapy alone is unlikely to result in clinical resolution, and effective treatment usually comprises aggressive and lengthy systemic antimicrobial and anti-inflammatory and/or immune-modulating therapy (typically encompassing steroidal therapy). Appropriate empirical antimicrobial selections include cephalosporins and tetracyclines (with or without niacinamide). Potential adverse effects associated with the use of systemic cortiocosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with cephalosporins include hypersensitivity reactions, gastrointestinal (GI) distress, and renal disease. Potential adverse effects associated with tetracyclines include GI distress, photosensitivity, and hepatic damage. Potential adverse effects associated with niacinamide include GI distress. Warm compressing may help facilitate the opening of inflamed meibomian glands and focal lesions may also be curettaged if necessary. The use of an Elizabethan collar will prevent self-trauma until active inflammation is controlled. Blepharoconjunctivitis may be chronic, frustrating to treat, and/or recurrent.

SELECTED REFERENCES

Bistner S. Allergic- and immunologic-mediated diseases of the eye and adnexae. *Vet Clin North Am Small Anim Pract.* 1994;**24**(4):711–734. Review.

Chambers ED, Severin GA. Staphylococcal bacterin for treatment of chronic staphylococcal blepharitis in the dog. *J Am Vet Med Assoc.* 1984;**185**(4):422–425.

Furiani N, Scarampella F, Martino PA, Panzini I, Fabbri E, Ordeix L. Evaluation of the bacterial microflora of the conjunctival sac of healthy dogs and dogs with atopic dermatitis. *Vet Dermatol.* 2011;**22**(6):490–496. doi: 10.1111/j.1365-3164.2011.00979.x. Epub 2011 May 2.

Peña MA, Leiva M. Canine conjunctivitis and blepharitis. *Vet Clin North Am Small Anim Pract.* 2008;**38**(2):233–249. doi: 10.1016/j.cvsm.2007.12.001. Review.

Sansom J, Heinrich C, Featherstone H. Pyogranulomatous blepharitis in two dogs. *J Small Anim Pract.* 2000;**41**(2):80–83.

Weingart C, Kohn B, Siekierski M, Merle R, Linek M. **Blepharitis** in **dogs**: a clinical evaluation in 102 **dogs**. *Vet Dermatol.* 2019;**30**(3):222–e69.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 39.1 Immune-mediated blepharoconjunctivitis. Inset demonstrates the typical, bilateral, relatively symmetrical presentation.

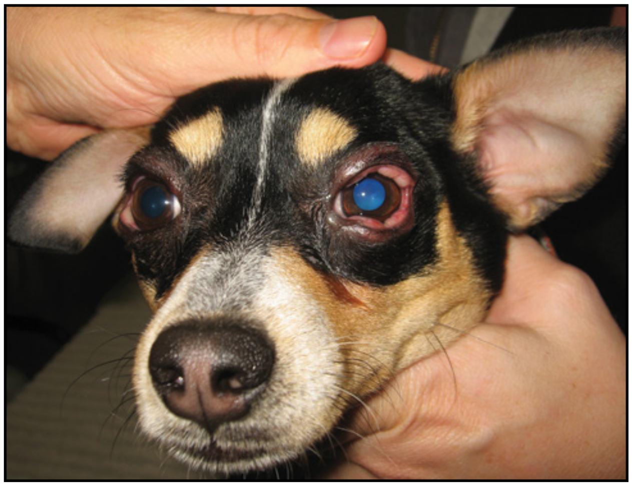


Figure 39.2 Immune-mediated blepharoconjunctivitis. Note significant conjunctival component.

Figure 39.3 Immune-mediated blepharoconjunctivitis. Note bilateral, symmetrical presentation.

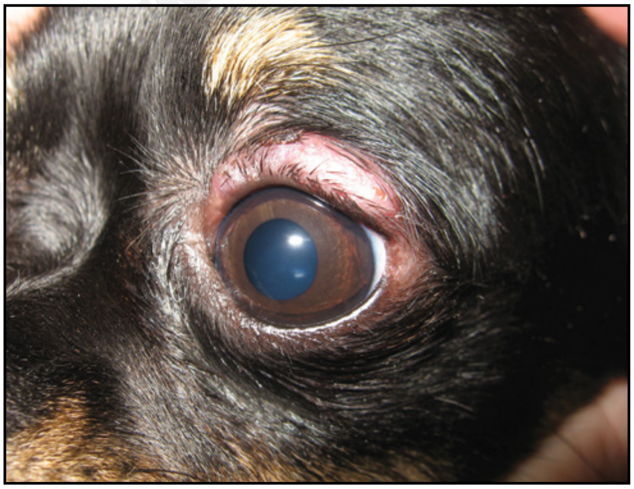


Figure 39.4 Immune-mediated blepharoconjunctivitis. Broken hair shafts are the result of self-trauma.

CHAPTER 40
AUTOIMMUNE BLEPHARITIS

PRESENTATION

Primary dermal autoimmune disease may affect multiple areas including the facial, nasal, and periocular regions, mucocutaneous junctions and/or external pinnae, manifesting as papular erosions, ulceration, dermatitis, and/or depigmentation. The classification of these diseases is complex, encompassing the pemphigus (auto-antibody-mediated acantholysis) diseases, lupus (immune-cellular dysfunction) diseases, vasculitis-related diseases, and uveodermatological syndrome (also described as "Vogt-Koyanagi-Harada-like" or "VKH-like" disease). Of the pemphigus diseases, pemphigus foliaceous (PF) is more commonly encountered, notably affecting the Akita, Chow Chow, and Labrador Retriver. Of the lupus diseases, discoid lupus erythematosis (DLE), also described as "cutaneous lupus erythematosus," is more commonly encountered, notably affecting the Akita, Chow Chow, Siberian Husky, German Shepherd, Australian herding, and Collie-breeds. Uveodermatological syndrome typically involves not only dermal tissues (manifesting as nasal, periocular, and mucocutaneous ulceration and/or depigmentation) but also the uveal tract where it may result in uveitis, retinal detachment, hyphema, and/or glaucoma. Breeds commonly affected by uveodermatological syndrome are the Akita, Chow Chow, and Siberian Husky (see also Uveodermatological associated uveitis and uveodermatological associated chorioretinitis).

DIAGNOSIS AND TREATMENT

The diagnosis of autoimmune blepharitis is made based on clinical findings in conjunction with the interpretation of representative dermal biopsies, ideally taken from the interface of affected/nonaffected tissue. In all cases, treatment comprises long-term immune-modulation, typically encompassing steroidal therapy with or without the adjunctive use of immunosuppressive agents including azathioprine, chlorambucil, mycofenylate, and/or cyclosporine. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of systemic azathioprine and chlorambucil and mycofenylate include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of cyclosporine include hypersensitivity and GI distress. Secondary pyoderma, if present, is treated using appropriate systemic antimicrobials.

SELECTED REFERENCES

Griffin CE. Diagnosis and management of primary autoimmune skin diseases: a review. *Semin Vet Med Surg (Small Anim).* 1987;**2**(3):173–185. Review.

Mueller RS, Krebs I, Power HT, Fieseler KV. Pemphigus foliaceus in 91 dogs. *J Am Anim Hosp Assoc.* 2006;**42**(3):189–196.

Pye CC. Uveodermatologic syndrome in an Akita. *Can Vet J.* 2009;**50**(8):861–864.

Weingart C, Kohn B, Siekierski M, Merle R, Linek M. **Blepharitis** in **dogs**: a clinical evaluation in 102 **dogs**. *Vet Dermatol.* 2019;**30**(3):222–e69.

White SD, Rosychuk RA, Reinke SI, Paradis M. Use of tetracycline and niacinamide for treatment of autoimmune skin disease in 31 **dogs**. *J Am Vet Med Assoc.* 1992;**200**(10):1497–1500.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 40.1 Autoimmune blepharitis. Periocular and facial findings associated with feline pemphigus foliaceous.

Figure 40.2 Autoimmune blepharitis. Periocular and facial findings associated with canine pemphigus foliaceous.

Figure 40.3 Autoimmune blepharitis. Periocular and facial findings associated with discoid lupus erythematosus.

Figure 40.4 Autoimmune blepharitis. Findings associated with uveodermatological syndrome or "VKH-like disease" including blepharitis, vitiligo, and active uveitis (inset demonstrates typical nasal changes).

CHAPTER 41
EOSINOPHILLIC FOLLICULITIS/FURUNCULOSIS

PRESENTATION

Folliculitis describes inflammation of the dermis and hair follicles. Furunculosis describes a deep necrotizing folliculitis. Eosinophillic folliculitis/furunculosis represents a severe, rapid onset, nodular and/or ulcerative dermatitis, typically affecting the muzzle, face, and periocular regions. Occasionally, more generalized dermal lesions may also occur. The etiology of this condition is unclear; however, a hypersensitivity reaction has been proposed. Affected animals are typically young, large-breed dogs, with no breed-predisposition noted.

DIAGNOSIS AND TREATMENT

The diagnosis of eosinophillic folliculitis/furunculosis is made based on clinical findings, supported by histological interpretation of a representative incisional skin biopsy, indicating a mixed inflammatory and marked eosinophillic infiltration. Treatment comprises the systemic administration of prednisone at an anti-inflammatory to immunosuppressive dose. Secondary bacterial dermatitis, if present, should be managed using a systemic antimicrobial agent. Appropriate empirical selections include cephalosporins and tetracyclines. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with cephalosporins include GI distress, hypersensitivity reactions, and renal toxicity. Potential adverse effects associated with tetracyclines may include GI distress, hepatotoxicity, and photosensitivity.

SELECTED REFERENCES

Fraser M. What is your diagnosis? Eosinophilic folliculitis and furunculosis. *J Small Anim Pract.* 2002;**43**(4):150, 187.

Mauldin EA, Palmeiro BS, Goldschmidt MH, Morris DO. Comparison of clinical history and dermatologic findings in 29 dogs with severe eosinophilic dermatitis: a retrospective analysis. *Vet Dermatol.* 2006;**17**(5):338–347.

Van Poucke S. What is your diagnosis? Canine eosinophilic furunculosis (folliculitis) of the face. *J Small Anim Pract.* 2000;**41**(11): 485, 524–525.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

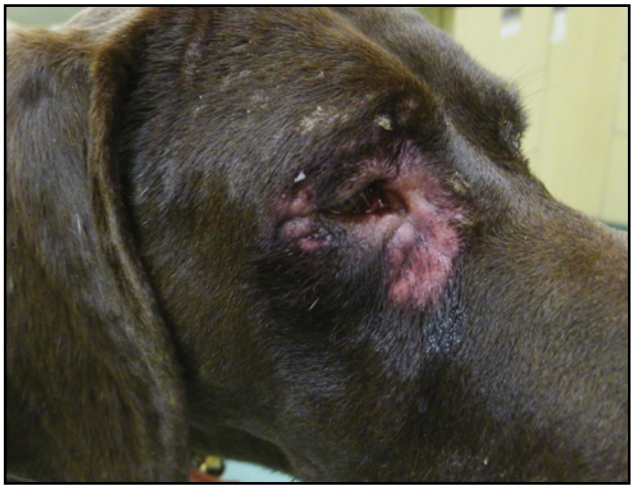


Figure 41.1 Eosinophillic folliculitis/furunculosis. Note the characteristic nodular lesions affecting the periocular region.

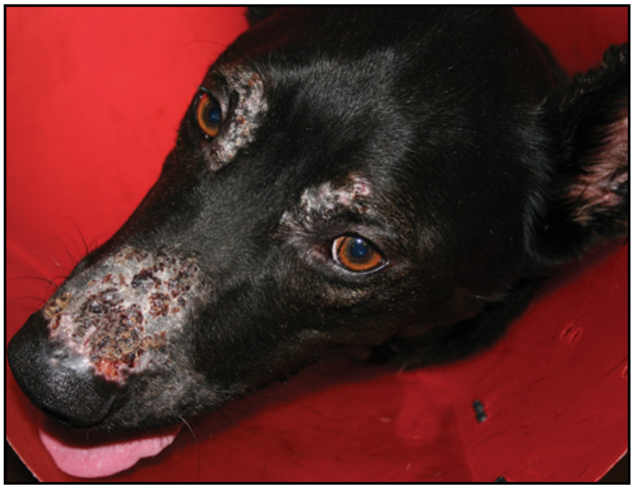


Figure 41.2 Eosinophillic folliculitis/furunculosis. Bilaterally symmetric nodular lesions affecting the muzzle and periocular regions.

Figure 41.3 Eosinophillic folliculitis/furunculosis. Note the characteristic nodular lesions affecting the muzzle and periocular regions.

Figure 41.4 Eosinophillic folliculitis/furunculosis. Note the associated conjunctivitis.

CHAPTER 42
ADVERSE DRUG REACTIONS (ADRs)

PRESENTATION

Adverse (cutaneous) drug reactions (ADRs) may occur as a result of both drug-related factors as well as components of patient's idiosyncratic immune responses. Symptoms may include generalized as well as severe facial and/or periocular erythema, bullous, and/or pustular eruptions. Mechanisms of ADRs include immediate, delayed, and antigen-antibody reactions as well as complement activation and/or mast cell degranulation. Medications which may be more frequently associated with ADRs include sulfa-containing antimicrobials (particularly those potentiated with trimethoprim), penicillins, cephalosporins, neomycin, and cyclosporine. Affected breeds may include the Shetland Sheepdog, Australian Shepherd, Doberman Pincher, and Yorkshire Terrier.

DIAGNOSIS AND TREATMENT

The diagnosis is supported by medical history, histopathologic interpretation of incisional dermal biopsy, and response to drug withdrawal. Differentials include erythema multiforme, toxic epidermal necrolysis, and cutaneous vasculitis as well as auto-immune diseases. Treatment comprises the withdrawal of medications suspected to be resulting in ADR. Glucocorticoid therapy may help speed the resolution of clinical signs. The symptomatic management of secondary bacterial pyoderma if present is also recommended. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Mason KV. Cutaneous drug eruptions. *Vet Clin North Am Small Anim Pract.* 1990;**20**(6):1633–1653. Review.

Niza MM, Félix N, Vilela CL, Peleteiro MC, Ferreira AJ. Cutaneous and ocular adverse reactions in a dog following meloxicam administration. *Vet Dermatol.* 2007;**18**(1):45–49.

Noli C, Koeman JP, Willemse T. A retrospective evaluation of adverse reactions to trimethoprim-sulphonamide combinations in dogs and cats. *Vet Q.* 1995;**17**(4):123–128.

Scott DW, Miller WH Jr. Idiosyncratic cutaneous adverse drug reactions in the dog: literature review and report of 101 cases (1990–1996). *Canine Pract.* 1999;**24**,16–22.

Scott DW, Miller WH Jr. Idiosyncratic cutaneous adverse drug reactions in the cat: literature review and report of 14 cases (1990–1996). *Feline Pract.* 1998;**26**,10–15.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 42.1 Adverse drug reaction. Adverse reaction associated with the topical use of sulphacetamide.

Figure 42.2 Adverse drug reactions. Adverse reaction associated with the topical use of sulphacetamide.

Figure 42.3 Adverse drug reactions. Adverse reaction associated with the topical use of neomycin.

Figure 42.4 Adverse drug reactions. Adverse reaction associated with the topical use of cyclosporine.

CHAPTER 43
DERMATOMYOSITIS

PRESENTATION

Dermatomyositis describes an inflammatory condition affecting the dermis, connective tissues, and to a variable degree, muscular tissues of young dogs. Dermal lesions typically comprise erythema, dermatitis, and/or alopecia of the extremities, digits, ears, and face. Affected animals are typically less than 1 year of age. Many develop subsequent myositis (notably affecting the muscles of mastication and/or locomotion). Commonly affected breeds include the Shetland Sheepdog and the Collie breeds.

DIAGNOSIS AND TREATMENT

The diagnosis of dermatomyositis is made based on clinical findings, supported histopathological interpretation of representative skin and muscle by biopsies. Affected animals may display variably elevated creatine kinase levels. Treatment comprises the systemic administration of corticosteroids, with or without adjunctive pentoxyfiline and/or vitamin E. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of pentoxyfiline include gastrointestinal (GI) distress, CNS (central nervous system) excitement, and/or hypersensitivity reactions. Dermal lesions typically resolve (with the formation of scar tissue), however myopathy may persist.

SELECTED REFERENCES

Evans J, Levesque D, Shelton GD. Canine inflammatory myopathies: a clinicopathologic review of 200 cases. *J Vet Intern Med.* 2004;**18**(5):679–691.

Ferguson EA, Cerundolo R, Lloyd DH, Rest J, Cappello R. Dermatomyositis in five Shetland sheepdogs in the United Kingdom. *Vet Rec.* 2000;**146**(8):214–217.

Hargis AM, Haupt KH, Hegreberg GA, Prieur DJ, Moore MP. Familial **canine dermatomyositis**. Initial characterization of the cutaneous and muscular lesions. *Am J Pathol.* 1984;**116**(2):234–244.

Morris DO. Ischemic dermatopathies. *Vet Clin North Am Small Anim Pract.* 2013;**43**(1):99–111.

Rees CA, Boothe DM. Therapeutic response to pentoxifylline and its active metabolites in dogs with familial canine dermatomyositis. *Vet Ther.* 2003;**4**(3):234–241.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

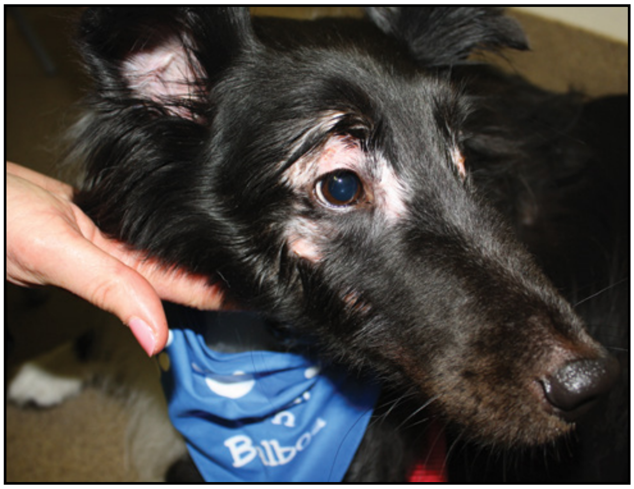


Figure 43.1 Dermatomyositis. Note periocular alopecia.

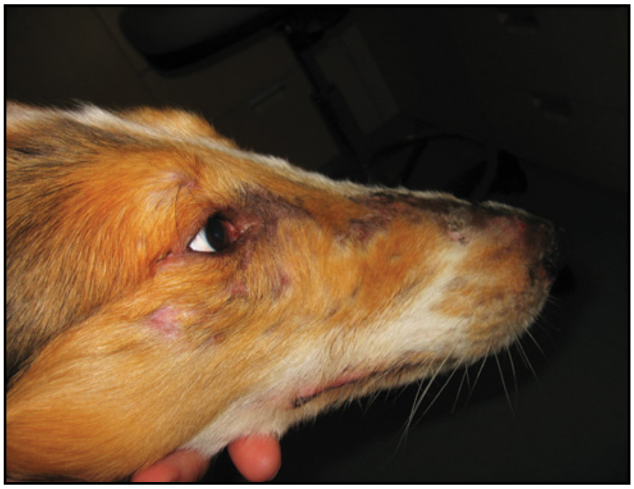


Figure 43.2 Dermatomyositis. Note typical presentation in a Collie.

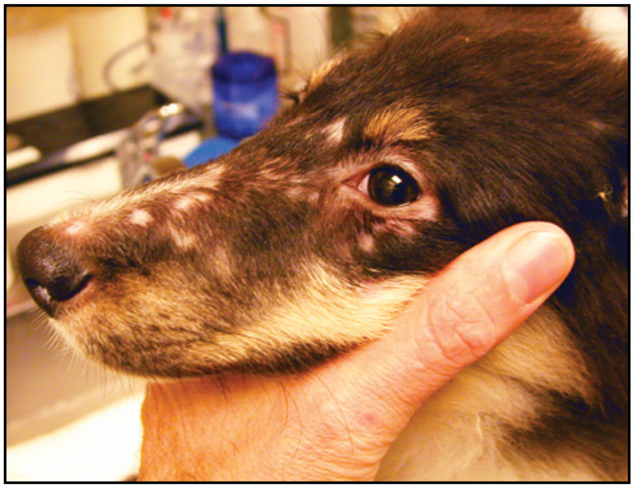


Figure 43.3 Dermatomyositis. Note periocular and muzzle lesions.

Figure 43.4 Dermatomyositis. Note chronic scarring associated with resolved inflammation.

CHAPTER 44
DEMODEX-ASSOCIATED BLEPHARITIS

PRESENTATION

Clinical cases of canine demodecosis arise as a result of significantly increased numbers of commensal *Demodex Canis* or *Demodex Cati* mites within hair follicles. Poor body condition, debilitating disease, stress, corticosteroid-induced immune-suppression, and/or hereditary factors may play a role in the development of clinical disease. Affected patients typically display erythema, moist dermatitis, alopecia, and/or lichinfication and hyperpigmentation in either a localized or generalized pattern, particularly around the periocular regions. Multiple regions of the body may be affected and significant self-trauma as well as secondary bacterial pyoderma may result. Both canine and feline species of any breed or crossed breed may be affected (cats also potentially being affected by *Notoedres cati*). Commonly affected canine breeds include the Old English Sheep Dog, Lhasa Apso, Shih Tzu, and English Bulldog.

DIAGNOSIS AND TREATMENT

The diagnosis of demodecosis is made based on cytological assessment following manual follicular expression in combination with deep skin scraping, demonstrating large numbers of adult parasites and/or an increased ratio of immature parasite forms (ova, larvae, and nymphs). The use of localized, topical treatments in the management of demodecosis is frequently unrewarding. Appropriate therapy comprises mitocidal treatments, most commonly ivermectin, administered at a frequency of Q24–48 hours over the course of 3–6 months, until two negative skin scrapings are achieved with an interval of 2–3 months. Potential adverse reactions associated with use of ivermectins include neurotoxicity and retinal damage, particularly in the Collie breeds. Secondary pyoderma should be treated using appropriate systemic antimicrobials (with or without the adjunctive use of medicated shampoos as indicated).

SELECTED REFERENCES

Kuznetsova E, Bettenay S, Nikolaeva L, Majzoub M, Mueller R. Influence of systemic antibiotics on the treatment of **dogs** with generalized **demodicosis**. *Vet Parasitol.* 2012;**188**(1–2):148–155.

Mueller RS. An update on the therapy of canine **demodicosis**. *Compend Contin Educ Vet.* 2012;**34**(4):E1–E4. Review.

Mueller RS, Bensignor E, Ferrer L, Holm B, Lemarie S, Paradis M, Shipstone MA. Treatment of **demodicosis** in **dogs**: 2011 clinical practice guidelines. *Vet Dermatol.* 2012;**23**(2):86–96, e20–e21. Review.

Ravera I, Ferreira D, Gallego LS, Bardagí M, Ferrer L. Serum detection of IgG antibodies against **Demodex** canis by western blot in healthy **dogs** and **dogs** with juvenile generalized demodicosis. *Res Vet Sci.* 2015;**101**:161–164.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 44.1 Canine demodicosis. Typical clinical presentation.

Figure 44.2 Canine demodicosis. Note periocular inflammation associated with self-trauma.

Figure 44.3 Feline demodicosis. Presentation associated with generalized feline demodicosis.

Figure 44.4 Canine demodicosis. Note lichenification and hyperpigmentation associated with chronic demodicosis.

CHAPTER 45
DERMATOPHYTOSIS

PRESENTATION

Dermatophytosis describes dermal infection with one of a number of fungal organisms. The confusing name "ringworm" has been used as a blanket term to describe these infections. Commonly identified organisms include *Microsporum canis, Microsporum gypseum, and Trichophyton mentagrophytes*. Clinically, affected patients display erythematous and/or crusting dermatitis, with or without secondary bacterial infection. Changes frequently affect the face, periocular tissues, pinnae, and/or forelimbs. Chronic lesions may display alopecia and/or lichenification. Both canine and feline species of any breed or crossed breed may be affected. Commonly affected breeds include the Jack Russell Terrier & Yorkshire Terrier.

DIAGNOSIS AND TREATMENT

The diagnosis of dermatophytosis is made based on clinical findings as well as the results of incubating samples on dermatophyte test media (DTM), with or without adjunctive cytological or histological assessment of representative samples. Infectious organisms represent potentially contagious zoonoses. Treatment typically comprises the systemic administration of fungicidal agents including fluconazole and itraconazole (with or without adjunctive antibiotic agents if secondary bacterial infection is present). Potential adverse effects associated with the use of fluconazole and itraconazole include GI distress and hepatotoxicity.

SELECTED REFERENCES

Boehm TMSA, Mueller RS. **Dermatophytosis** in **dogs** and cats – an update. *Tierarztl Prax Ausg K Kleintiere Heimtiere.* 2019;**47**(4):257–268.

Bond R. Superficial veterinary mycoses. *Clin Dermatol.* 2010;**28**(2):226–236. doi: 10.1016/j.clindermatol.2009.12.012.

Moriello KA, Coyner K, Paterson S, Mignon B. Diagnosis and treatment of **dermatophytosis** in **dogs** and cats. Clinical Consensus Guidelines of the World Association for Veterinary Dermatology. *Vet Dermatol.* 2017;**28**(3):266–e68.

Moriello KA. Treatment of dermatophytosis in dogs and cats: review of published studies. *Vet Dermatol.* 2004;**15**(2):99–107.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 45.1 Dermatophytosis. Severe, diffuse, chronic facial dermatophytosis.

Figure 45.2 Dermatophytosis. Chronic facial and periocular alopecia and crusting.

Figure 45.3 Dermatophytosis. Severe active facial erythema and alopecia.

Figure 45.4 Dermatophytosis. Severe, diffuse, chronic facial dermatophytosis.

CHAPTER 46
APOCRINE HIDROCYSTOMA

PRESENTATION

Apocrine hidrocystomas are relatively common eyelid masses affecting cats, predominantly the Persian and Himalayan breeds. These lesions appear as one or more focal to coalescing, raised, gray, cystic masses affecting one or both eyelid margins (upper and/or lower) and/or the periocular region. Histologically these masses comprise adenomatous tissue and dilated epithelial cysts, which contain viscous tan to brown colored proteinaceous debris.

DIAGNOSIS AND TREATMENT

Apocrine hidrocystomas are benign and generally do not result in significant discomfort or secondary pathology. If necessary, however, theses lesions may be removed by either surgical excision or more ideally by CO_2 laser ablation. In all cases, an attempt should be made to preserve eyelid margins. New or recurrent lesions may occur following removal.

SELECTED REFERENCES

Cantaloube B, Raymond-Letron I, Regnier A. Multiple eyelid apocrine hidrocystomas in two Persian cats. *Vet Ophthalmol.* 2004;**7**(2):121–125.

Chaitman J, van der Woerdt A, Bartick TE. Multiple eyelid cysts resembling apocrine hidrocystomas in three Persian cats and one Himalayan cat. *Vet Pathol.* 1999;**36**(5):474–476.

Giudice C, Muscolo MC, Rondena M, Crotti A, Grieco V. Eyelid multiple cysts of the apocrine gland of Moll in Persian cats. *J Feline Med Surg.* 2009;**11**(6):487–491. doi: 10.1016/j.jfms.2008.11.006. Epub 2008 Dec 21.

Yang SH, Liu CH, Hsu CD, Yeh LS, Lin CT. Use of chemical ablation with trichloroacetic acid to treat eyelid apocrine hidrocystomas in a cat. *J Am Vet Med Assoc.* 2007;**230**(8):1170–1173.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 46.1 Apocrine hidrocystoma. Clinical presentation associated with multiple apocrine hidrocystomas affecting both eyelids.

Figure 46.2 Apocrine hidrocystoma. Large medial canthal apocrine hidrocystoma.

Figure 46.3 Apocrine hidrocystoma. Large medial canthal apocrine hidrocystoma, note the smooth, gray cystic appearance.

Figure 46.4 Apocrine hidrocystoma. Large medial canthal apocrine hidrocystoma, in a typical brachycephalic feline.

CHAPTER 47
SEBACEOUS ADENOMA/EPITHELIOMA

PRESENTATION

Both adenomas and epitheliomas represent relatively common benign neoplastic proliferations of sebaceous (meibomian) glandular tissue. Adenomas generally exhibit histological evidence of glandular differentiation, whereas epitheliomas generally do not; however some degree of histological "overlap" occurs. Both lesions typically appear as pink to gray, form irregular and proliferative eyelid masses arising from the eyelid margin (upper or lower) and display variable degrees of eyelid invasion. Secondary blepharoconjunctivitis and/or keratitis may develop as a result of abrasion and/or self-trauma. Secondary chalazia may also develop in association with these lesions as a result of glandular obstruction.

DIAGNOSIS AND TREATMENT

The diagnosis is based on clinical findings as well as histopathology interpretation of biopsy samples where indicated. Histopathology will define the nature and extent of the neoplastic process and provide an indication as whether complete excision has been achieved. In rare cases malignant adenocarcinomas may be identified. Treatment comprises the surgical removal of these lesions. Small lesions (2–5 mm) may be ablated using a CO_2 laser, preserving eyelid margin function and form and frequently avoiding the need for surgical reconstruction. Larger lesions, however, require full thickness excision and accurate subsequent eyelid repair. Ideally the medial and lateral canthal anatomy should be preserved where possible. Resultant eyelid defects which are less than one third of the eyelid length may be directly apposed. Larger defects typically require blapharoplastic reconstruction such as lateral canthotomy and the excellent split-lid technique described by Lewin. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete.

SELECTED REFERENCES

Bussieres M, Krohne SG, Stiles J, Townsend WM. The use of carbon dioxide laser for the ablation of meibomian gland adenomas in dogs. *J Am Anim Hosp Assoc.* 2005;**41**(4):227–234.

Dubielzig R *et al*. 2010. *Veterinary Ocular Pathology*, pp. 151–152, Elsevier, Edinburgh.

Grahn BH and Sandmeyer LS. Diagnostic ophthalmology. Tarsal gland adenoma. *Can Vet J.* 2009;**50**(11):1199–1200.

Roberts SM, Severin GA, Lavach JD. Prevalence and treatment of palpebral neoplasms in the dog: 200 cases (1975–1983). *J Am Vet Med Assoc.* 1986;**189**(10):1355–1359.

Romkes G, Klopfleisch R, Eule JC. Evaluation of one- vs. two-layered closure after wedge excision of 43 eyelid tumors in **dogs**. *Vet Ophthalmol.* 2014;**17**(1):32–40.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 47.1 Sebaceous epithelioma. This small epithelioma is amenable to CO_2 laser ablation.

Figure 47.2 Sebaceous adenoma. This small adenoma is amenable to CO_2 laser ablation.

Figure 47.3 Sebaceous adenoma. This larger adenoma, with extension beneath the eyelid margin is more appropriately surgically resected.

Figure 47.4 Sebaceous adenoma. This larger adenoma with significant extension beyond the eyelid margin should be surgically resected.

CHAPTER 48
HISTIOCYTOMA

PRESENTATION

Histiocytomas are generally benign and frequently self-limiting dermal tumors, which may affect the eyelids or periocular regions, particularly in young dogs. Lesions present as firm, raised, pink to red, well-circumscribed masses on or close to the eyelid margin, which may or may not ulcerate. Commonly affected breeds include the Cocker Spaniel, Boxer, and Dachshunds.

DIAGNOSIS AND TREATMENT

The diagnosis of histiocytoma is made based on histopathologic interpretation of representative (typically excisional) biopsy. Histiocytomas in young dogs may spontaneously regress over the course of 1–3 months in association with a lymphocytic infiltrate, or may require surgical excision. In surgical cases, an attempt should be made to preserve eyelid margins. While solitary, eyelid histiocytomas are generally benign in their behavior, the spectrum of more generalized cutaneous as well as systemic histiocytosis-associated diseases (notably affecting Bernese Mountain Dogs and Labrador Retrievers) are of more concern and the behavior of much less common histiocytic sarcomas is highly malignant.

SELECTED REFERENCES

Gelatt KN. Histiocytoma of the eyelid of a dog. *Vet Med Small Anim Clin.* 1975;**70**(3):305.
Killick DR, Rowlands AM, Burrow RD, Cripps P, Miller J, Graham P, Blackwood L. Mast cell tumour and cutaneous histiocytoma excision wound healing in general practice. *J Small Anim Pract.* 2011;**52**(9):469–475. doi: 10.1111/j.1748-5827.2011.01093.x.
Schmidt JM, North SM, Freeman KP, Ramiro-Ibañez F. Canine paediatric oncology: retrospective assessment of 9522 tumours in dogs up to 12 months (1993–2008). *Vet Comp Oncol.* 2010;**8**(4):283–292. doi: 10.1111/j.1476-5829.2010.00226.x.
Moore PF. A review of histiocytic diseases of **dogs** and cats. *Vet Pathol.* 2014;**51**(1):167–184.
Fulmer AK and Mauldin GE. Canine histiocytic neoplasia: an overview. *Can Vet J.* 2007;**48**(10):1041–1043, 1046–1050.
Costa D, Ferreira R, Prada J, Queiroga FL, Rodrigues P, Silva F, Pires I. A Role for Angiogenesis in **Canine** Cutaneous **Histiocytoma** Regression: Insights into an Old Clinical Enigma. *in vivo.* 2020;**34**(6):3279–3284.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 48.1 Histiocytomas. Common clinical presentation on the eyelid margin.

Figure 48.2 Histiocytomas. Note the well circumscribed nature of the mass near the eyelid margin.

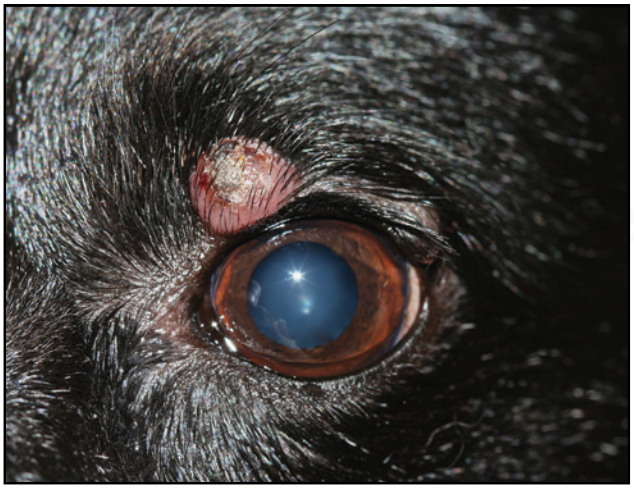


Figure 48.3 Histiocytomas. Larger canine histiocytoma on the upper eyelid margin.

Figure 48.4 Histiocytomas. Note the ulcerated surface of the histiocytoma.

CHAPTER 49
MELANOCYTOMA

PRESENTATION

Eyelid melanocytomas represent a benign proliferation of melanocytes and present as one or frequently more, small, firm, irregular, pigmented slowly proliferative masses along the eyelid margin(s). Commonly affected breeds include the Vizsla and Doberman Pincher.

DIAGNOSIS AND TREATMENT

The diagnosis of melanocytoma is made based on clinical findings, supported where indicated by histopathological interpretation of a representative tissue biopsy. This lesion's predication for the eyelid margin renders the surgical excision of multiple lesions challenging. Options for the management of these lesions include benign neglect, surgical resection, local treatment using cryosurgical application or more ideally CO_2 mediated laser ablation. Local recurrence as well as the development of new lesions may occur postoperatively.

SELECTED REFERENCES

Dubielzig R *et al*. 2010. *Veterinary Ocular Pathology*, p. 150, Elsevier, Edinburgh.

Finn M, Krohne S, Stiles J. Ocular melanocytic neoplasia. *Compend Contin Educ Vet.* 2008;**30**(1):19–25; quiz 26. Review.

Roberts SM, Severin GA, Lavach JD. Prevalence and treatment of palpebral neoplasms in the dog: 200 cases (1975–1983). *J Am Vet Med Assoc.* 1986;**189**(10):1355–1359.

Wang AL and Kern T. Melanocytic ophthalmic neoplasms of the domestic veterinary species: a review. *Top Companion Anim Med.* 2015;**30**(4):148–157.

Zibura AE, Henriksen ML, Rendahl A, Lim CC, Reilly C. Retrospective evaluation of **canine** palpebral masses treated with debulking and cryotherapy: 46 cases. *Vet Ophthalmol.* 2019;**22**(3):256–264.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 49.1 Eyelid melanocytoma. Clinical presentation associated with canine melanocytomas affecting the eyelid margin.

Figure 49.2 Eyelid melanocytoma. Note the multifocal presentation.

Figure 49.3 Eyelid melanocytoma. Clinical presentation associated with melanocytomas diffusely affecting the lower eyelid.

Figure 49.4 Eyelid melanocytoma. Note the common clinical presentation in a Vizsla.

CHAPTER 50
MELANOMA

PRESENTATION

Eyelid melanomas typically present as firm irregular and proliferative masses. These lesions may or not be pigmented (the diagnostic spectrum encompassing amelanotic melanomas) and may occasionally be associated with hemorrhage. Eyelid melanomas typically exhibit malignant behavior including aggressive local infiltration. Commonly affected breeds include the Doberman Pincher and Toy Poodle.

DIAGNOSIS AND TREATMENT

Definitive diagnosis of a melanoma is made by histopathologic evaluation of representative tissue biopsy. Treatment comprises wide surgical excision where possible, as well as adjunctive chemotherapy or external beam (electron) radiation therapy where appropriate. The potential for metastasis exists and presurgical staging via local lymph node aspiration, three-view radiography and complete blood count (CBC)/chemistry analysis is recommended. Cranial soft tissue imaging (MRI or CT) will further facilitate accurate presurgical planning if necessary. Local blepharoplasty reconstruction may be necessary following the excision of large lesions. The potential value of vaccine-mediated immunotherapy in the treatment of eyelid melanomas has not been fully evaluated.

SELECTED REFERENCES

Aquino SM. Management of eyelid neoplasms in the dog and cat. *Clin Tech Small Anim Pract.* 2007;**22**(2):46–54. Review.

Finn M, Krohne S, Stiles J. Ocular melanocytic neoplasia. *Compend Contin Educ Vet.* 2008;**30**(1):19–25; quiz 26. Review.

Gwin RM, Alsaker RD, Gelatt KN. Melanoma of the lower eyelid of a dog. *Vet Med Small Anim Clin.* 1976;**71**(7):929–931.

Roberts SM, Severin GA, Lavach JD. Prevalence and treatment of palpebral neoplasms in the dog: 200 cases (1975–1983). *J Am Vet Med Assoc.* 1986;**189**(10):1355–1359.

Wang AL and Kern T. Melanocytic ophthalmic neoplasms of the domestic veterinary species: a review. *Top Companion Anim Med.* 2015;**30**(4):148–157.

Zibura AE, Henriksen ML, Rendahl A, Lim CC, Reilly C. Retrospective evaluation of **canine** palpebral masses treated with debulking and cryotherapy: 46 cases. *Vet Ophthalmol.* 2019;**22**(3):256–264.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

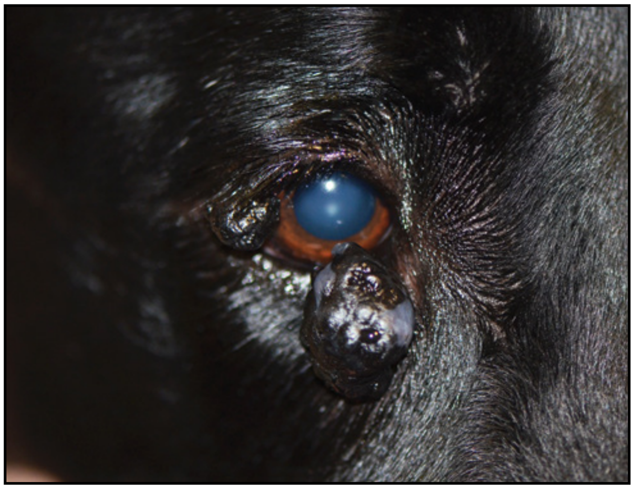


Figure 50.1 Eyelid melanoma. Clinical presentation associated with eyelid melanoma, note the two discrete lesions in this patient.

Figure 50.2 Eyelid melanoma. Clinical presentation associated with a large eyelid melanoma.

Figure 50.3 Eyelid melanoma. Note that the patient also displays the presence of a small (unrelated) upper eyelid adenoma.

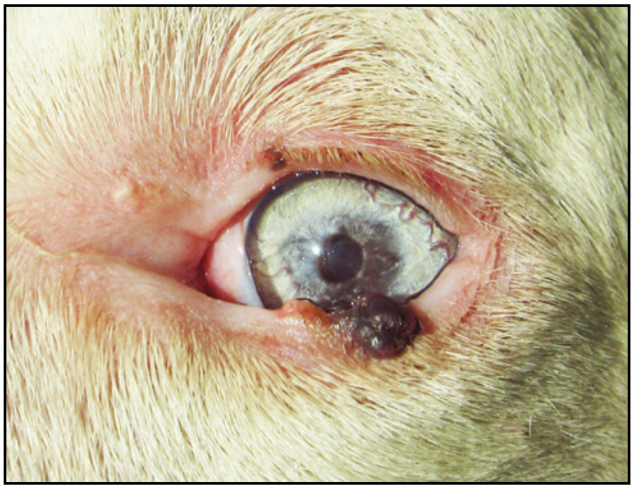


Figure 50.4 Eyelid melanoma. Clinical presentation of eyelid melanoma in a poorly pigmented patient. Note the "amelanotic" melanoma lesion medially.

CHAPTER 51
CUTANEOUS EPITHELIOTROPIC LYMPHOMA (CEL)

PRESENTATION

Cutaneous epitheliotropic lymphoma (CEL) describes a form of lymphoma, which may specifically affect the eyelids and adnexal tissues. The somewhat confusing term "mycosis fungoides" has also been used to refer to this condition. Clinical presentation includes thickened, irregular to nodular/proliferative and/or ulcerative dermatitis/blepharitis, and/or depigmentation, which varies in severity and distribution. Mucocutaneous junctions around the eyes and mouth are commonly affected, as are nail beds and/or paw pads; however, more generalized dermal involvement may also be present. In cases affecting the periocular tissues, associated conjunctivitis and/or ocular discharge may additionally be present. Late stages of the disease may include involvement of other tissues including the lymphatic, hepatic, and/or gastrointestinal systems. Commonly affected breeds include the Golden Retriever, Boxer, and American Eskimo.

DIAGNOSIS AND TREATMENT

The diagnosis of CEL is made by histologic interpretation of samples obtained by incisional biopsy. Wherever lymphoma is suspected, samples should be harvested prior to beginning therapy in order to facilitate the planning of an accurate treatment protocol. The potential for metastasis exists, and staging via local lymph node (and/or organ/bone marrow) aspiration, three-view radiography, and CBC/chemistry analysis is recommended, prior to the initiation of chemotherapy. Treatment comprises systemic chemotherapy as well as the management of secondary bacterial infection where present. Individual chemotherapeutic protocols are most ideally designed by a veterinary oncologist however typically include prednisone and lomustine. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of lomustine include stomatitis, corneal epithelial toxicity, alopecia, GI distress, renal, and/or hepatotoxicity, and/or myelosuppression. The prognosis varies based on the stage of lymphoma as well as extent of involvement representing a period, which may vary from months to years.

SELECTED REFERENCES

Chan CM, Frimberger AE, Moore AS. Clinical outcome and prognosis of **dogs** with histopathological features consistent with **epitheliotropic lymphoma**: a retrospective study of 148 cases (2003–2015). *Vet Dermatol.* 2018;**29**(2):154–e59.

Donaldson D and Day MJ. Epitheliotropic **lymphoma** (mycosis fungoides) presenting as blepharoconjunctivitis in an Irish setter. *J Small Anim Pract.* 2000;**41**(7):317–320.

Fontaine J, Heimann M, Day MJ. Canine cutaneous epitheliotropic T-cell lymphoma: a review of 30 cases. *Vet Dermatol.* 2010;**21**(3): 267–275. doi: 10.1111/j.1365-3164.2009.00793.x. Epub 2010 Feb 5.

Fontaine J, Heimann M, Day MJ. Cutaneous epitheliotropic T-cell lymphoma in the cat: a review of the literature and five new cases. *Vet Dermatol.* 2011;**22**(5):454–461. doi: 10.1111/j.1365-3164.2011.00972.x. Epub 2011 May 2.

Rook KA. **Canine** and Feline Cutaneous **Epitheliotropic Lymphoma** and Cutaneous Lymphocytosis. *Vet Clin North Am Small Anim Pract.* 2019;**49**(1):67–81.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 51.1 Cutaneous epitheliotropic lymphoma. This patient displays erosive periocular and facial lesions.

Figure 51.2 Cutaneous epitheliotropic lymphoma. Clinical presentation displaying generalized dermal changes.

Figure 51.3 Cutaneous epitheliotropic lymphoma. This patient displays severe proliferative lesions primarily affecting the eyelids.

Figure 51.4 Cutaneous epitheliotropic lymphoma. This patient displays more generalized facial proliferative lesions.

CHAPTER 52
SQUAMOUS CELL CARCINOMA (SCC)

PRESENTATION

Squamous cell carcinoma (SCC) of the eyelids is a neoplastic process, which more commonly affects white or minimally pigmented animals. UV sunlight may contribute to the development of these lesions, which progress from focal acanthosis through carcinoma-in-situ to outright squamous cell carcinoma with time. Carcinomas affecting the eyelid are more commonly encountered in feline patients and typically present as irregular, pink to red, erosive, and/or proliferative lesions, which may be associated with local blepharoconjunctivitis, mucoid discharge, and/or hemorrhage. Carcinomas are aggressive and locally invasive tumors, which may extend into surrounding tissues, including the conjunctiva, globe, and orbit.

DIAGNOSIS AND TREATMENT

The diagnosis of SCC is based on clinical findings, supported by representative incisional tissue biopsy. Histopathology defines the degree of malignancy present. Treatment comprises wide surgical excision where possible. Local blepharoplastic reconstruction is typically necessary following excision. The potential for metastasis exists and basic staging via local lymph node aspiration, three-view radiography and complete blood count (CBC)/chemistry analysis is recommended. Cranial soft tissue imaging (MRI or CT) will facilitate accurate presurgical planning where necessary. Superficial lesions may be amenable to strontium therapy. SCC demonstrates a variable sensitivity to external beam radiation.

SELECTED REFERENCES

Dos Anjos DS, Bueno, C., Magalhães, L.F. *et al*. Electrochemotherapy induces tumor regression and decreases the proliferative index in canine cutaneous squamous cell carcinoma. *Sci Rep* **9**, 15819 (2019).

Murphy S. Cutaneous squamous cell carcinoma in the cat: current understanding and treatment approaches. *J Feline Med Surg.* 2013;**15**(5):401–407.

Newkirk KM, Rohrbach BW. A retrospective study of eyelid tumors from 43 cats. *Vet Pathol.* 2009;**46**(5):916–927.

Roberts SM, Severin GA, Lavach JD. Prevalence and treatment of palpebral neoplasms in the dog: 200 cases (1975–1983). *J Am Vet Med Assoc.* 1986;**189**(10):1355–1359.

Schmidt K, Bertani C, Martano M, Morello E, Buracco P. Reconstruction of the lower eyelid by third eyelid lateral advancement and local transposition cutaneous flap after "en bloc" resection of squamous cell carcinoma in 5 cats. *Vet Surg.* 2005;**34**(1):78–82.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 52.1 Squamous cell carcinoma. Erosive lower eyelid squamous cell carcinoma affecting a canine patient.

Figure 52.2 Squamous cell carcinoma. Erosive lower eyelid squamous cell carcinoma affecting a feline patient.

Figure 52.3 Squamous cell carcinoma. Erosive upper eyelid squamous cell carcinoma affecting a feline patient.

Figure 52.4 Squamous cell carcinoma. Proliferative lower eyelid squamous cell carcinoma affecting a feline patient.

CHAPTER 53
MAST CELL TUMOR (MCT)

PRESENTATION

Mast cell tumors (MCTs) are relatively common eyelid neoplasms. Lesions typically present as progressive, firm, raised, masses, which may or may not ulcerate. Local extension into surrounding tissues may occur. Commonly affected breeds include the Boston Terrier, Labrador Retriever, and Boxer.

DIAGNOSIS AND TREATMENT

The diagnosis of MCT is based on representative tissue biopsy. Histopathology will confirm the diagnosis, provide information regarding the completeness of excision as well as degree of malignancy of neoplasia present. Presurgical treatment using an anti-histamine or corticosteroid is considered appropriate in view of the potential for neoplastic degranulation in association with manipulation. The potential for metastasis exists, and staging via local lymph node aspiration, three-view radiography, and complete blood count (CBC)/Chemistry analysis is recommended. Management options include wide surgical excision (where possible), local treatment with strontium, and/or external-beam (electron) radiation where appropriate.

SELECTED REFERENCES

Fife M, Blocker T, Fife T, Dubielzig RR, Dunn K. Canine conjunctival mast cell tumors: a retrospective study. *Vet Ophthalmol.* 2011;**14**(3):153–160. doi: 10.1111/j.1463-5224.2010.00857.x.

Killick DR, Rowlands AM, Burrow RD, Cripps P, Miller J, Graham P, Blackwood L. Mast cell tumour and cutaneous histiocytoma excision wound healing in general practice. *J Small Anim Pract.* 2011;**52**(9):469–475. doi: 10.1111/j.1748-5827.2011.01093.x.

Lewin G. Eyelid reconstruction in seven dogs using a split eyelid flap. *J Small Anim Pract.* 2003;**44**(8):346–351.

Roberts SM, Severin GA, Lavach JD. Prevalence and treatment of palpebral neoplasms in the dog: 200 cases (1975–1983). *J Am Vet Med Assoc.* 1986;**189**(10):1355–1359.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 53.1 Mast cell tumor. Clinical presentation associated with a canine eyelid mast cell tumor.

Figure 53.2 Mast cell tumor. Clinical presentation associated with a feline eyelid mast cell tumor.

Figure 53.3 Mast cell tumor. Note significant surface ulceration.

Figure 53.4 Mast cell tumor. Note significant subcutaneous extension.

CHAPTER 54
FIBROSARCOMA

PRESENTATION

Fibrosarcomas represent an uncontrolled, neoplastic proliferation of fibroblasts. Large/chronic tumors affecting the eyelids frequently also involve adjacent ocular/orbital tissues. Lesions generally present as progressively enlarging, firm, and poorly mobile subcutaneous swellings, which may additionally display surface ulceration. Sarcomas display varying morphological features, however typically behave in an aggressive and locally invasive manner. Commonly affected breeds include the Golden Retriever, Doberman Pincher, and Rottweiler.

DIAGNOSIS AND TREATMENT

The diagnosis of fibrosarcoma is made by histologic interpretation of a representative incisional tissue biopsy. Histopathology will additionally define the degree of malignancy present. The potential for metastasis exists, and staging via local lymph node aspiration, three-view radiography, and complete blood count (CBC)/chemistry analysis is recommended. Treatment comprises wide surgical excision where possible, with or without adjunctive chemotherapy. These neoplasms are generally poorly responsive to external beam radiation therapy. Cranial soft tissue imaging (MRI or CT) will facilitate accurate presurgical planning where indicated. Local blepharoplastic reconstruction may be necessary following excision.

SELECTED REFERENCES

Al-Dissi AN, Haines DM, Singh B, Kidney BA. Immunohistochemical expression of vascular endothelial growth factor and vascular endothelial growth factor receptor in canine cutaneous fibrosarcomas. *J Comp Pathol.* 2009;**141**(4):229–236.

Bostock DE and Dye MT. Prognosis after surgical excision of canine fibrous connective tissue sarcomas. *Vet Pathol.* 1980;**17**(5):581–588.

Dubielzig R, *et al*. 2010. *Veterinary Ocular Pathology*, pp. 127–128, Elsevier, Edinburgh.

Nordio L, Fattori S, Giudice C. **Fibrosarcoma** of the eyelid in two sibling Czech wolfdogs. *Open Vet J.* 2017;**7**(2):95–99.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 54.1 Fibrosarcoma. Clinical presentation associated with a large, invasive canine eyelid fibrosarcoma.

Figure 54.2 Fibrosarcoma. Clinical presentation associated with a firm, ulcerated, canine eyelid fibrosarcoma.

Figure 54.3 Fibrosarcoma. Clinical presentation associated with a large, invasive feline eyelid fibrosarcoma.

Figure 54.4 Fibrosarcoma. Clinical presentation associated with a large, ulcerated, and invasive feline eyelid fibrosarcoma.

Section 6

Diseases of the Conjunctiva, Nasolacrimal System and Third Eyelid

CHAPTER 55
ALLERGIC CONJUCTIVITIS

PRESENTATION

The conjunctival surfaces are richly supplied with the cellular components of the immune system and represent a first line of antigenic response. As a consequence, allergic conjunctivitis is not uncommon, particularly in those patients affected by generalized dermal allergic or atopic disease. Symptoms are generally mild to moderate in severity and may include conjunctival hyperemia, conjunctivitis, chemosis, follicular proliferation, epiphora, and/or mucoid discharge. Additionally, symptoms may fluctuate with generalized allergic symptoms and or seasonal allergen exposure. Commonly affected breeds include the English Bulldog and West Highland White terrier.

DIAGNOSIS AND TREATMENT

The diagnosis of allergic conjunctivitis is typically made based on a combination of clinical findings as well as the absence of significant underlying ocular abnormalities. If necessary, a conjunctival biopsy will support the diagnosis, frequently containing significantly increased numbers of mast cells. Treatment comprises the avoidance of inciting allergens where possible, the systemic management of generalized atopy wherever appropriate and the application of topical anti-inflammatory therapy, the frequency and duration of application being determined by the clinical picture. Topical steroidal therapy is frequently required although topical nonsteroidal and/or anti-histamines may also be employed in the management of this condition. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration.

SELECTED REFERENCES

Bistner S. Allergic- and immunologic-mediated diseases of the eye and adnexae. *Vet Clin North Am Small Anim Pract.* 1994;**24**(4):711–734.

Glaze MB. Ocular allergy. *Semin Vet Med Surg (Small Anim).* 1991;**6**(4):296–302.

Lourenço-Martins AM, Delgado E, Neto I, Peleteiro MC, Morais-Almeida M, Correia JH. Allergic conjunctivitis and conjunctival provocation tests in atopic dogs. *Vet Ophthalmol.* 2011;14(4):248–56. doi: 10.1111/j.1463-5224.2011.00874.x. Epub 2011 Apr 18.

Varandas C, Cartaxeiro C, Lourenço AM, Delgado E, Gil S. Selected cytokine expression in **dogs** with allergic **conjunctivitis**: Correlation with disease activity. *Res Vet Sci.* 2020;**130**, 33–40.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 55.1 Allergic conjunctivitis. Note follicular proliferation on anterior third eyelid conjunctiva.

Figure 55.2 Allergic conjunctivitis. Note significant thickening of the third eyelid associated with excessive follicular proliferation.

Figure 55.3 Allergic conjunctivitis. Note follicular proliferation and associated mucoid discharge/periocular crusting.

Figure 55.4 Allergic conjunctivitis. Typical clinical presentation associated with allergic conjunctivitis.

CHAPTER 56
DACRYOCYSTITIS

PRESENTATION

Dacryocystitis describes inflammation of the nasolacrimal drainage structures, which comprise the eyelid puncta, lacrimal canaliculi, lacrimal cavern, and the sino-nasal ducts that exit to the external nares. Inflammation of the nasolacrimal system may be the result of bacterial infection, extension of inflammation from adjacent structures, neoplasia, or entrapment of foreign material. Primary aplasia or hypoplasia of any part of this system may also occur, typically manifesting in young animals. Post-inflammatory scarring and fibrosis is a common cause of nasolacrimal non-patency, particularly in cats previously affected by herpesviral-associated inflammation. Chronic inflammation or obstruction of the nasolacrimal ducts may also result in cystic dilation. Clinical symptoms of dacryocystitis may include conjunctivitis, fornix-based mucoid to purulent discharge, and localized swelling and/or discomfort over any part of the nasolacrimal duct system, particularly around the medial canthal region. Symptoms are typically unilateral. Both canine and feline species of any breed or mixed breed may be affected.

DIAGNOSIS AND TREATMENT

The diagnosis of dacryocystitis is made based on clinical symptoms, particularly the presence of mucopurulent material noted to be arising from the nasolacrimal puncta. Nasolacrimal drainage may be impaired or absent on the affected side, marked by a negative "Jones test" such that fluorescein dye instilled into the fornix fails to exit at the ipsilateral external nares within 10–15 minutes. Further investigation of dacryocystitis may include microbial sampling for cytology as well as culture and sensitivity testing, contrast radiography, and/or soft tissue imaging via magnetic resonance imaging (MRI) or computed tomography (CT). Medical therapy using topical and/or systemic antimicrobial and/or anti-inflammatory agents as well as canulation, irrigation, stenting, and/or surgical exploration may be indicated based on the severity of disease, diagnostic findings, and response to therapy. In extreme cases, the creation of alternate drainage via surgical conjunctivo-rhinostomy or conjunctivo-buccostomy is possible, however is rarely indicated.

SELECTED REFERENCES

Giuliano EA, Pope ER, Champagne ES, Moore CP. Dacryocystomaxillorhinostomy for chronic dacryocystitis in a dog. *Vet Ophthalmol*. 2006;**9**(2):89–94.

Murphy JM, Severin GA, Lavach JD. Nasolacrimal catheterization for treating chronic dacryocystitis. *Vet Med Small Anim Clin*. 1977;**72**(5):883–887.

Singh A, Cullen CL, Gelens H, Grahn BH. Diagnostic Ophthalmology. Left dacryocystitis with naso-lacrimal duct obstruction. *Can Vet J*. 2004;**45**(11):953–955.

van der Woerdt A, Wilkie DA, Gilger BC, Smeak DD, Kerpsack SJ. Surgical treatment of dacryocystitis caused by cystic dilatation of the nasolacrimal system in three dogs. *J Am Vet Med Assoc*. 1997;**211**(4):445–447.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 56.1 Dacryocystitis. Inset indicates a negative "Jones test" with fluorescein dye failing to exit the external nares on the affected side as a result of nasolacrimal obstruction.

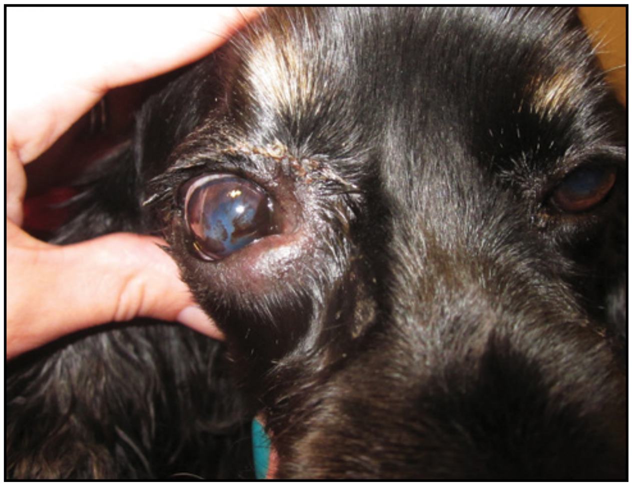


Figure 56.2 Dacryocystitis. A fluctuant swelling of the medial canthus in association with inflammation of the lacrimal sac is present.

Figure 56.3 Dacryocystitis. Clinical presentation associated with dacryocystitis, note the fluctuant swelling of the medial canthus.

Figure 56.4 Dacryocystitis. Note the discharge arising from the nasolacrimal puncta.

CHAPTER 57
SYMBLEPHARON

PRESENTATION

Symblepharon describes varying degrees of adhesion between adjacent conjunctival surfaces (including palpebral and/or third eyelid surfaces) and/or the cornea. Clinically, varying degrees of fibrosing and/or pigmentary areas of tissue are noted to extend across palpebral and/or corneal surfaces. Conjunctivitis, chemosis, fibrosis and or ocular discharge may additionally be present. Significant narrowing of the palpebral fissure and/or occlusion of the corneal surface may also result. Symblepharon results from loss of epithelial continuity, most commonly arising secondary to feline herpesviral disease but potentially arising secondary to any severe feline or canine conjunctival inflammation (see also Herpesviral-associated conjunctivitis).

DIAGNOSIS AND TREATMENT

Treatment comprises addressing conjunctival inflammation as well as underlying viral and/or bacterial disease. Surgical resection of conjunctival adhesions is relatively straightforward; however, the tendency for the aggressive (potentially aggravated) reformation of these adhesions is extremely high. The potential benefits of surgical intervention should consequently be carefully considered before proceeding. Surgery should not be undertaken in actively inflamed or infected eyes. The use of silicone surgical barriers to prevent re-adhesion prior to re-epithelialization as well as the judicious use of antimicrobial and/or anti-inflammatory agents (generally non-steroidal in nature) may help maximize long-term success.

SELECTED REFERENCES

Andrew SE. Ocular manifestations of feline herpesvirus. *J Feline Med Surg*. 2001;**3**(1):9–16.

Jacobi S and Dubielzig RR. Feline early life ocular disease. *Vet Ophthalmol.*. 2008;**11**(3):166–169.

Kim Y, Kang S, Seo K. Application of superficial keratectomy and soft contact lens for the treatment of **symblepharon** in a cat: a case report. *J Vet Sci*. 2021;**22**(2):e19.

Nasisse MP. Feline herpesvirus ocular disease. *Vet Clin North Am Small Anim Pract*. 1990;**20**(3):667–680.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 57.1 Symblepharon. Clinical presentation associated with severe symblepharon demonstrating narrowing of the palpebral fissure and significant visual impairment.

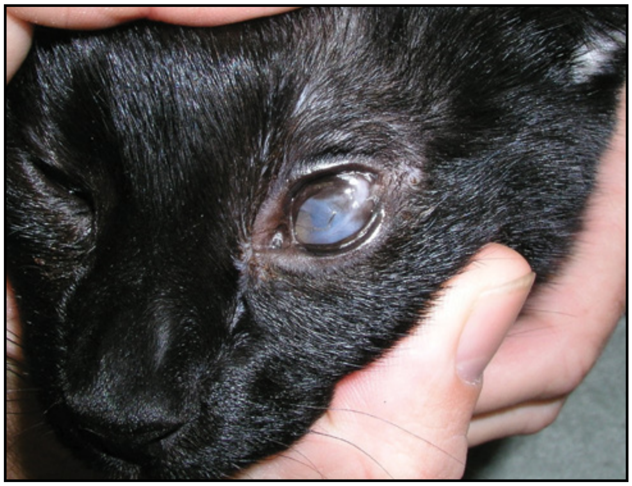


Figure 57.2 Symblepharon. Typical clinical presentation associated with feline symblepharon. Note fibrosing and pigmentary keratitis.

Figure 57.3 Symblepharon. Note elevation of the third eyelid as a result of adhesions associated with symblepharon.

Figure 57.4 Symblepharon. An unusual clinical presentation associated with canine symblepharon.

CHAPTER 58
HERPESVIRAL-ASSOCIATED CONJUNCTIVITIS

PRESENTATION

Herpesviral ocular disease is highly prevalent, resulting predominantly from animal to animal contact, frequently via aerosolization of sneezed particles. Affected animals may display a range of symptoms including sneezing, coughing, nasal discharge, pyrexia, inappetance, lethargy, conjunctivitis, chemosis, keratitis, and/or secondary corneal ulceration (see also Herpesviral-associated keratitis). Young kittens are generally most severely affected, immune-competent adult cats generally exhibiting less severe symptoms. Feline patients may additionally be infected with secondary pathogens including calicivirus, *chlamydophila felis*, *mycoplasma sp*., and/or bacterial pathogens. Herpes viral disease (CHV-1) should also be considered when investigating similar symptoms in canine patients. Other canine pathogens including Canine Adenovirus 2 (CAV-2), Bordetella bronchiseptica, Canine Influenza virus (H3N8), Canine Parainfluenza virus, and *mycoplasma sp* should also be considered when investigating the potential role of canine herpes virus in ocular surface disease. Once a patient becomes infected, alphaherpes viral particles remain latent with the ability to recrudesce under conditions of physiologic or pharmacologic stress (including the inappropriate administration of immunosuppressive agents). As a consequence, particular caution should be employed when considering the administration of long acting or "depo" formulations of corticosteroids to patients which may be affected by herpesviral disease.

DIAGNOSIS AND TREATMENT

Accurate testing for the presence of herpesviral disease and its correlation with ophthalmic disease is challenging; however, polymerase-chain-reaction assays, based on tissue samples, are currently considered most reliable. Ultimately healthy patients recover spontaneously from outbreaks over the course of several weeks; however, treatment is frequently indicated. Topical and/or systemic antiviral agents may be administered, including; trifluorothymidine, idoxuridine, cidofovir, and gancoclovir and/or famciclovir (metabolized to penciclovir), respectively. Potential complications associated with the use of topical antiviral agents include local irritation. Potential complications associated with the use of systemic antiviral medications include GI distress, myelosuppression, and/or hepatopathy. The concurrent administration of systemic L-lysine may decrease the severity of symptoms in cats, potentially via the inhibition of herpesviral metabolism. Extreme caution is indicated when considering the use of anti-inflammatory therapy to treat herpesviral-associated disease. Nonsteroidal agents are generally considered less likely to result in complication and the use of corticosteroids which are considered contra-indicated in most cases. Tetracyclines, azithromycin, and fluroquinolones represent appropriate antimicrobial choices when treating potentially complicated cases. Potential adverse effects associated with tetracyclines and azithromycin include GI distress, photosensitivity, and hepatic damage. Potential adverse effects associated with fluroquinolones include gastrointestinal (GI) distress, hepatic disease, neurologic disease, and/or retinal toxicity.

SELECTED REFERENCES

Gaskell R, Dawson S, Radford A, Thiry E. Feline herpesvirus. *Vet Res*. 2007;**38**(2):337–354.

Gould D. **Feline herpesvirus**-1: ocular manifestations, diagnosis and treatment options. *J Feline Med Surg*. 2011;**13**(5):333–346.

Ledbetter EC. **Canine herpesvirus-1** ocular diseases of mature dogs. *N Z Vet J*. 2013;**61**(4):193–201.

Thomasy SM and Maggs DJ. A review of antiviral drugs and other compounds with activity against **feline herpesvirus** type 1. *Vet Ophthalmol*. 2016;**19** Suppl 1:119–130.

Thomasy SM, Shull O, Outerbridge CA, Lim CC, Freeman KS, Strom AR, Kass PH, Maggs DJ. Oral administration of famciclovir for treatment of spontaneous ocular, respiratory, or dermatologic disease attributed to **feline herpesvirus** type 1: 59 cases (2006–2013). *J Am Vet Med Assoc*. 2016;**249**(5):526–538.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 58.1 Herpesviral-associated conjunctivitis. Note significant chemosis and ocular discharge associated with herpesviral disease.

Figure 58.2 Herpesviral-associated conjunctivitis. Note chemosis and epiphora associated with feline herpesviral disease.

Figure 58.3 Herpesviral-associated conjunctivitis. Clinical presentation associated with herpesviral disease in a young feline patient. Note significant ocular discharge, potentially associated with secondary bacterial infection.

Figure 58.4 Herpesviral-associated conjunctivitis. Clinical presentation associated with herpesviral disease in a canine patient. Inset shows serous nasal discharge.

CHAPTER 59
THIRD EYELID GLAND PROLAPSE ("CHERRY EYE")

PRESENTATION

Prolapse of the lacrimal gland of the third eyelid ("cherry eye") is relatively commonly encountered. Glandular prolapse arises as a result of defective connective tissue development and consequent glandular laxity in predisposed breeds. Partial or complete prolapse of the lacrimal gland of the third eyelid, presenting as a smooth pink to red tissue mass, which protrudes from the posterior surface of one or both third eyelids. Third eyelid gland prolapse typically affects young animals. Associated symptoms may include conjunctivitis, ocular discharge, and/or decreased lacrimometic function as well as secondary ulcerative keratitis. Severe secondary inflammation and or bacterial infection may develop, particularly in cases of chronic glandular prolapse. Commonly affected canine breeds include the English Bulldog, Cocker Spaniel, and Shih Tzu. This condition may also affect feline patients, with the Burmese being most frequently noted.

DIAGNOSIS AND TREATMENT

The ideal treatment represents surgical repositioning of prolapsed tissue. The excision of prolapsed glandular tissue should always be avoided wherever possible, in order to avoid subsequent complications. Several techniques for repair have been described with the "Morgan Pocket" technique being most generally appropriate. Sufficient blunt dissection and the creation of a pocket into which glandular tissue can be replaced as well as the use of absorbable suture material and avoidance of knots on the bulbar surface of the third eyelid will maximize the likelihood of surgical success. More challenging cases such as those associated with chronic or severe prolapse, severe inflammation, and/or previously failed surgical intervention may be repaired via a variety of techniques which anchor prolapsed tissue to scleral or orbital tissues. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until third eyelid healing is complete.

SELECTED REFERENCES

Chahory S, Crasta M, Trio S, Clerc B. Three cases of prolapse of the nictitans gland in cats. *Vet Ophthalmol*. 2004;**7**(6):417–419.

Dugan SJ, Severin GA, Hungerford LL, Whiteley HE, Roberts SM. Clinical and histologic evaluation of the prolapsed third eyelid gland in dogs. *J Am Vet Med Assoc.*. 1992;**201**(12):1861–1867.

Mazzucchelli S, Vaillant MD, Wéverberg F, Arnold-Tavernier H, Honegger N, Payen G, Vanore M, Liscoet L, Thomas O, Clerc B, Chahory S. Retrospective study of 155 cases of prolapse of the nictitating membrane gland in dogs. *Vet Rec*. 2012;**170**(17):443.

Multari D, Perazzi A, Contiero B, De Mattia G, Iacopetti I. Pocket technique or pocket technique combined with modified orbital rim anchorage for the replacement of a **prolapsed gland** of the **third eyelid** in dogs: 353 dogs. *Vet Ophthalmol*. 2016;**19**(3):214–219.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 59.1 Third eyelid gland prolapse (cherry eye). Inset demonstrates the frequently bilateral presentation of this condition.

Figure 59.2 Third eyelid gland prolapse (cherry eye). Note the inflamed, follicular presentation of the gland.

Figure 59.3 Third eyelid gland prolapse (cherry eye). Clinical presentation associated with third eyelid gland prolapse in a feline patient.

Figure 59.4 Third eyelid gland prolapse (cherry eye). Note the common presentation in an English Bulldog.

CHAPTER 60
KERATOCONJUNCTIVITIS SICCA ("DRY EYE")

PRESENTATION

The pre-corneal tear film (PCTF) comprises an outer lipid, central aqueous, and inner mucoid component. Lipid and mucoid components are secreted by the eyelid and conjunctival glands, respectively. The aqueous portion of the PCTF is secreted by the lacrimal glands of the orbit and third eyelid. Keratoconjunctivitis sicca (KCS) may represent a qualitative deficiency resulting from insufficient lipid and/or mucous secretion and/or quantitative deficiency arising secondary to insufficient lacrimal gland secretion. Glandular dysfunction may arise secondary immune-mediated, traumatic, inflammatory, toxic, or neurological etiologies. Pharmacological agents with the potential to cause or exacerbate KCS include etodolac and sulpha-containing anti-microbial compounds. Symptoms of KCS typically include conjunctivitis, vascular/pigmentary keratitis, secondary meibomian gland inflammation/infection, and the accumulation of green/yellow-colored mucous in and around the fornices. Symptoms may be unilateral or bilateral, their severity correlating with the degree of tear film dysfunction. Commonly affected breeds include the English Bulldog, West Highland White terrier, and King Charles Cavalier spaniel.

DIAGNOSIS AND TREATMENT

Qualitative KCS is diagnosed on the basis of abnormal tear film break-up times (TFBUTs). This represents the time taken for surface tension to break down across the corneal surface following the application of fluorescein dye and the manual opening of the eyelids. A TFBUT of less than 20 seconds, is generally considered to be abnormally shortened. Quantitative KCS is diagnosed based on decreased Schirmer tear test (STT) values, measured by placing graded strips into the ventral fornix for 1 minute. Values of less than 15 mm/wetting per minute are generally considered suspect and those than 10 mm, generally considered diagnostic of KCS. Values of 0mm/wetting define "absolute" KCS and represent particularly challenging clinical cases (see also Neurogenic KCS and Xeromycteria). Treatment comprises a combination of lacrimostimulent and/or anti-inflammatory agents and long-term therapy is typically necessary. Lacrimostimulent agents include the immunosuppressive fungal cell wall extracts cyclosporine and tarcolimus. The administration of oral pilocarpine may help re-establish lacrimomimetic function in cases of neurologic dysfunction. Anti-inflammatory agents typically encompass topical steroid agents. Secondary eyelid and/or meibomian gland inflammation/infection should be addressed wherever present. The ruling out of underlying endocrinopathies has been advocated. Potential adverse effects associated with the use of topical cyclosporine and tacrolimus include periocular dermatitis and alopecia. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential side effects associated with the use of oral pilocarpine may include hypersalivation, nausea, panting and diarrhea. In usually severe cases or those which fail to respond to appropriate therapy, surgical intervention in the form of parotoid duct transposition may be considered, however this procedure may be associated with a range of postoperative complications.

SELECTED REFERENCES

Dodi PL. Immune-mediated **keratoconjunctivitis sicca** in **dogs**: current perspectives on management. *Vet Med (Auckl)*. 2015;**6**:341–347.

Haeussler DJ Jr. Improving the diagnosis of canine **keratoconjunctivitis sicca**. *Vet Rec*. 2019;**184**(22):676–677.

Hendrix DV, Adkins EA, Ward DA, Stuffle J, Skorobohach B. An investigation comparing the efficacy of topical ocular application of tacrolimus and cyclosporine in dogs. *Vet Med Int*. 2011;**2011**:487592. doi: 10.4061/2011/487592.

Klauss G, Giuliano EA, Moore CP, Stuhr CM, Martin SL, Tyler JW, Fitzgerald KE, Crawford DA. Keratoconjunctivitis sicca associated with administration of etodolac in dogs: 211 cases (1992–2002). *J Am Vet Med Assoc*. 2007;**230**(4):541–547.

Matheis FL, Walser-Reinhardt L, Spiess BM. Canine neurogenic Keratoconjunctivitis sicca: 11 cases (2006-2010). *Vet Ophthalmol*. 2012;**15**(4):288–290. doi: 10.1111/j.1463-5224.2011.00968.x.

Williams DL. Immunopathogenesis of keratoconjunctivitis sicca in the dog. *Vet Clin North Am Small Anim Pract*. 2008;**38**(2):251–268, vi. doi: 10.1016/j.cvsm.2007.12.002.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 60.1 Keratoconjunctivitis sicca (dry eye). Typical clinical presentation associated with keratoconjunctivitis sicca, including accumulated mucoid discharge, conjunctivitis, and significant pigmentary keratitis.

Figure 60.2 Keratoconjunctivitis sicca (dry eye). Note the significant accumulated discharge, conjunctivitis, and pigmentary and vascular keratitis.

Figure 60.3 Keratoconjunctivitis sicca (dry eye). Mild clinical presentation associated with keratoconjunctivitis sicca. Note minimal conjunctivitis and early corneal changes, including dorsal vascular and fibrosing keratitis.

Figure 60.4 Keratoconjunctivitis sicca (dry eye). Note discoloration of discharge, suggesting secondary bacterial infection.

CHAPTER 61
SCROLLED THIRD EYELID CARTILAGE

PRESENTATION

The third eyelid is supported by a "T-shaped" cartilaginous structure. Occasionally, this cartilage may be malformed, typically resulting in bending or "scrolling" of the leading edge of the third eyelid and consequent eversion of these tissues away from the ocular surface. This condition is generally hereditary and may be unilateral or bilateral. Secondary conjunctivitis and/or partial prolapse of the third eyelid lacrimal gland ("cherry eye") may be associated with scrolling of the third eyelid cartilage. Commonly affected breeds include the Great Dane.

DIAGNOSIS AND TREATMENT

The diagnosis is made based on careful inspection of the third eyelid structures. When indicated, treatment comprises the surgical resection of malformed cartilage. In skilled hands, the prognosis for repair is excellent; however, significant potential exists for postoperative complications associated with inappropriate cartilage resection or apposition as well as the subsequent abrasion of incorrectly placed suture material against the corneal surface. Postoperative care comprises routine systemic antimicrobial (if indicated), anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete.

SELECTED REFERENCES

Allbaugh RA and Stuhr CM. Thermal cautery of the canine third eyelid for treatment of cartilage eversion. *Vet Ophthalmol*. 2013;**16**(5):392–395.

Mañé MC, Vives MA, Barrera R, Bascuas JA. Results and histological development of various surgical techniques for correcting eversion of the third eyelid in dogs. *Histol Histopathol*. 1990;**5**(4):415–425.

Michel J, Lazard P, Vigan M, Albaric O. Treatment of prolapsed gland and **cartilage** deformity of the **nictitating membrane** with pocket technique and chondrectomy alone, or combined with a wedge conjunctivectomy: 132 dogs (1998–2018). *Vet Ophthalmol*. 2020;**23**(2):305–313.

Williams D, Middleton S, Caldwell A. Everted third eyelid cartilage in a cat: a case report and literature review. *Vet Ophthalmol*. 2012;**15**(2):123–127. doi: 10.1111/j.1463-5224.2011.00945.x.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 61.1 Scrolled third eyelid cartilage. Note mild associated conjunctivitis.

Figure 61.2 Scrolled third eyelid cartilage. Significant bending of the third eyelid can occur in some patients.

Figure 61.3 Scrolled third eyelid cartilage. Common clinical presentation associated with scrolled third eyelid cartilage in a Great Dane.

Figure 61.4 Scrolled third eyelid cartilage. Unusual presentation associated with scrolled third eyelid cartilage in a feline patient.

CHAPTER 62
MEDIAL CANTHAL POCKET SYNDROME

PRESENTATION

Medial canthal pocket syndrome describes a relatively common presentation, particularly associated with breeds displaying deep orbits and narrow skulls such that micro-debris collects in the ventral conjunctival fold or "pocket." Clinically, this condition presents as low-grade, chronic, follicular conjunctivitis affecting both the ventral palpebral fornix and tissues of the third eyelid. Mild to moderate associated mucoid ocular discharge is typically present and dried discharge may additionally accumulate around the medial canthus. Symptoms may be exacerbated by seasonal/atopic dermatitis. Commonly affected breeds include the Standard Poodle and Afghan Hound.

DIAGNOSIS AND TREATMENT

Treatment of this condition typically involves management rather than cure. Frequent flushing of debris and discharge from the conjunctival fornix using an ophthalmic irrigating solution as well as the application of topical anti-inflammatory agent (such as cyclosporine and/or corticosteroids) is indicated. Potential adverse effects associated with the use of cyclosporine include hypersensitivity and gastrointestinal (GI) distress. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Treatment may need to be continued for a significant period of time or indefinitely in select cases.

SELECTED REFERENCES

Hendrix DVH. 1991. Diseases and surgery of the canine conjunctiva In: *Veterinary Opthalmology*, 3rd ed., Gelatt KN (Ed.), Lippincott Williams & Wilkins, Baltimore.

Rubin LF. 1989. *Inherited Eye Diseases in Purebred Dogs*, Williams & Wilkins, Baltimore.

Turner SM. 2008. Medial canthal pocket syndrome In: *Small Animal Ophthalmology*.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 62.1 Medial canthal pocket syndrome. Typical presentation associated with medial canthal pocket syndrome.

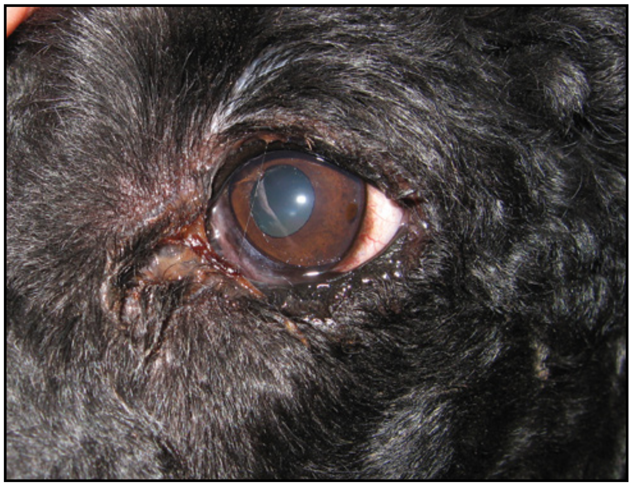


Figure 62.2 Medial canthal pocket syndrome. Note the accumulation of mucoid discharge and debris in the ventral conjunctival fold.

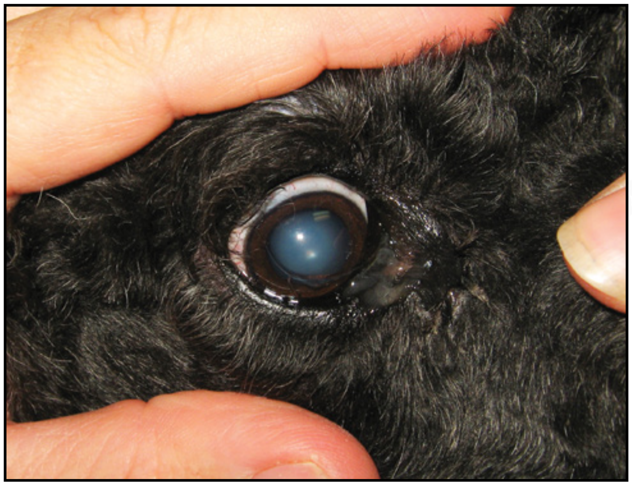


Figure 62.3 Medial canthal pocket syndrome. Common clinical presentation associated with medial canthal pocket syndrome in the Standard Poodle.

Figure 62.4 Medial canthal pocket syndrome. Note that in this chronic and severe case, the patient has developed secondary erosive blepharitis.

CHAPTER 63
ONCHOCERCA

PRESENTATION

Onchocerciasis represents a filarial nematode parasitic infection of the ocular and periocular tissues, which may affect dogs. Infection with O. Lupi and O. Lienalis has been described. This parasite cycles through a definitive wild host, adults mating to produce microfilariae which may be vectored by either simulium sp or culicoides sp flies. Clinically, onchocerciasis may present as any combination of ocular discharge, blepharospasm, conjunctivitis, chemosis, episclerokeratitis, uveitis, and/or parasite-containing granulomas affecting the episcleral or periocular tissues.

DIAGNOSIS AND TREATMENT

The diagnosis of onchocerciasis is made based on the identification of parasites and/or biopsy specimens. Treatment comprises

- surgical removal of granulomas (with or without adult parasites)
- systemic anti-inflammatory therapy (typically using corticosteroids)
- systemic antimicrobial therapy to destroy endosymbiotic *Wolbachia* sp (typically using tetracyclines)
- adulticide therapy (typically using melarsomine daily for 2 days immediately postoperatively)
- microfilaricide therapy (typically using a single dose of ivermectin 1 month following surgery, representing off-label use of this product).

SELECTED REFERENCES

Hassan HK, Bolcen S, Kubofcik J, Nutman TB, Eberhard ML, Middleton K, Wekesa JW, Ruedas G, Nelson KJ, Dubielzig R, De Lombaert M, Silverman B, Schorling JJ, Adler PH, Unnasch TR, Beeler ES. Isolation of **Onchocerca** lupi in **Dogs** and Black Flies, California, USA. *Emerg Infect Dis*. 2015;**21**(5):789–796.

Labelle AL, Daniels JB, Dix M, Labelle P. Onchocerca lupi causing ocular disease in two cats. *Vet Ophthalmol*. 2011;**14** Suppl 1:105–110.

Labelle AL, Maddox CW, Daniels JB, Lanka S, Eggett TE, Dubielzig RR, Labelle P. Canine ocular onchocercosis in the United States is associated with Onchocerca lupi. *Vet Parasitol*. 2013;**193**(1–3):297–301.

Sréter T and Széll Z. Onchocercosis: a newly recognized disease in dogs. *Vet Parasitol*. 2008;**151**(1):1–13.

Zarfoss MK, Dubielzig RR, Eberhard ML, Schmidt KS. Canine ocular onchocerciasis in the United States: two new cases and a review of the literature. *Vet Ophthalmol*. 2005;**8**(1):51–57.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 63.1 Onchocerca. Typical presentation associated with onchocerciasis.

Figure 63.2 Onchocerca. Note the large episcleral granuloma and associated conjunctivitis.

Figure 63.3 Onchocerca. Note the characteristic granuloma, a common finding associated with canine onchocerciasis.

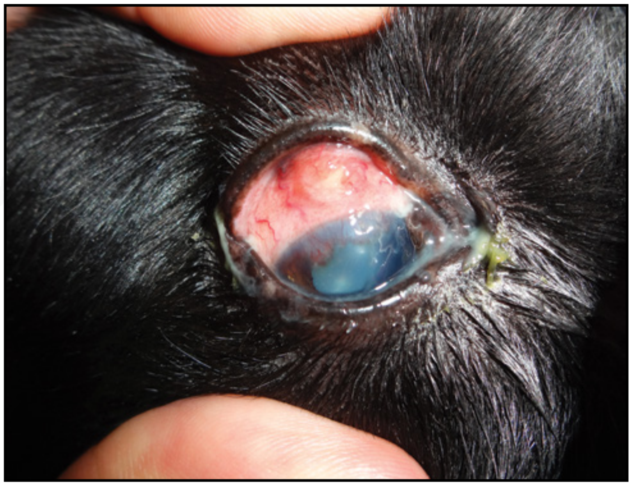


Figure 63.4 Onchocerca. Clinical presentation demonstrating associated conjunctivitis, episcleritis, keratitis, and mucoid ocular discharge.

CHAPTER 64
THELAZIA

PRESENTATION

Thelazia represent a genus of nematode parasites, (sometimes referred to as "eye worms"), which may affect the ocular and adnexal tissues of dogs and cats. The life cycle of these parasites comprises a definitive host (comprising a range of wild mammals including horses, ruminants, and numerous wild carnivores) and an intermediate host (notably dipteran flies). After mating, adult female parasites produce immature (first stage) larvae, which are released into the pre-corneal tear film of the definitive host. Immature larvae are subsequently ingested by flies, where they develop to third stage larvae before again being released into the tear film. Adult parasites may be found in the conjunctival fornix, nasolacrimal system, under the third eyelid or in rare cases, inside the globe. Associated symptoms may include blepharospasm, conjunctivitis, chemosis, ocular discharge, third eyelid elevation, and/or uveitis, all of which may be aggravated by self-trauma.

DIAGNOSIS AND TREATMENT

The diagnosis of this condition is made based on clinical findings and the identification of parasites in association with ocular and or adnexal tissues. Adult worms are thin, linear, white to translucent in color and approximately 1–2 cm in length. Treatment includes the removal of adult parasites, the topical administration of demecarium bromide and/or systemic anthelminthic agents including the avermectins, praziquantel, and/or mebendazole. Potential complications associated with the use of demecarium bromide include ocular discomfort and gastrointestinal (GI) distress. Potential complications associated with the use of avermectins include neurologic abnormalities and retinal toxicity. Potential complications associated with the use praziquantal and mebendazole include GI distress.

SELECTED REFERENCES

Burnett HS, Lee RD, Parmelee WE, Wagner ED. A survey of Thelazia californiensis, a mammalian eye worm, with new locality records. *J Am Vet Med Assoc*. 1956;**129**(7):325–327.

Calero-Bernal R, Sánchez-Murillo JM, Alarcón-Elbal PM, Sánchez-Moro J, Latrofa MS, Dantas-Torres F, Otranto D. Resolution of canine ocular thelaziosis in avermectin-sensitive Border Collies from Spain. *Vet Parasitol*. 2014;**200**(1–2):203–206. doi: 10.1016/j.vetpar.2013.12.014.

Marino V, Gálvez R, Mascuñán C, Domínguez I, Sarquis J, Montoya A, Barrera JP, Zenker C, Checa R, Hernández M, Miró G. Update on the treatment and prevention of ocular thelaziosis (**Thelazia**callipaeda) in naturally infected **dogs** from Spain. *Int J Parasitol*. 2021;**51**(1):73-81.

Motta B, Schnyder M, Basano FS, Nägeli F, Nägeli C, Schiessl B, Mallia E, Lia RP, Dantas-Torres F, Otranto D. Therapeutic efficacy of milbemycin oxime/praziquantel oral formulation (Milbemax®) against Thelazia callipaeda in naturally infested dogs and cats. *Parasit Vectors*. 2012;**5**:85. doi: 10.1186/1756-3305-5-85.

Otranto D and Traversa D. Thelazia eyeworm: an original endo- and ecto-parasitic nematode. *Trends Parasitol*. 2005;**21**(1):1–4.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 64.1 Thelazia. Clinical presentation associated with canine thelazia.

Figure 64.2 Thelazia. An adult Thelazia parasite is present ventrally, note the presence of associated conjunctivitis and mucoid discharge.

Figure 64.3 Thelazia. Parasites can be difficult to identify (as in this case, parasite is adjacent to the leading edge of the third eyelid) however, may be stimulated into vigorous movement by the installation of topical demecarium bromide.

Figure 64.4 Thelazia. An adult Thelazia parasite is apparent on the cornea medially, note the presence of associated conjunctivitis.

CHAPTER 65
PAPILLOMA

PRESENTATION

Squamous papillomas are benign neoplasms, which may affect the conjunctival and mucosal surfaces. Young dogs may be affected by significant numbers of viral papillomas, typically developing on the conjunctival and eyelid surfaces as well as the mucous membranes of the oral cavity. Adult animals more frequently display single discrete papillary eyelid lesions, which are pink to tan in color and often sessile with a "stalk-like" base.

DIAGNOSIS AND TREATMENT

A tentative diagnosis of papilloma may be made based on clinical presentation; however, confirmation requires incisional or excisional biopsy and adjunctive histopathology. Papillomas may be managed via benign neglect or surgical excision where indicated, the condition frequently resolving spontaneously over the course of 6–24 months. If present in large numbers, lesions may be ablated effectively using a CO_2 laser. The administration of patient-specific auto-antigenic vaccines has also been advocated. Although often a frustrating, condition to manage initially, viral papillomas typically regress as the animal achieves maturity.

SELECTED REFERENCES

Bonney CH, Koch SA, Confer AW, Dice PF. A case report: a conjunctivocorneal papilloma with evidence of a viral etiology. *J Small Anim Pract*. 1980;**21**(3):183–188.

Bonney CH, Koch SA, Dice PF, Confer AW. Papillomatosis of conjunctiva and adnexa in **dogs.** *J Am Vet Med Assoc*. 1980;**176**(1):48–51.

Munday JS, Thomson NA, Luff JA. Papillomaviruses in **dogs** and cats. *Vet J*. 2017;**225**:23–31.

Wiggans KT, Hoover CE, Ehrhart EJ, Wobeser BK, Cohen LB, Gionfriddo JR. Malignant transformation of a putative eyelid papilloma to squamous cell carcinoma in a dog. *Vet Ophthalmol*. 2013;**16** Suppl 1:105–112. doi: 10.1111/j.1463-5224.2012.01062.x.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 65.1 Papilloma. Clinical presentation associated with a viral papilloma in a young dog, inset demonstrates oral lesions.

Figure 65.2 Papilloma. Patient affected by significant number of viral papillomas, inset demonstrates associated oral lesions.

Figure 65.3 Papilloma. Patient affected by multiple viral papillomas, inset demonstrates oral lesions.

Figure 65.4 Papilloma. A sessile conjunctival papilloma in an adult dog.

CHAPTER 66
MELANOMA

PRESENTATION

Melanomas are not uncommonly associated with the mucocutaneous junction, typically presenting as firm irregular and proliferative masses. Lesions may or not be pigmented (the diagnostic spectrum encompassing amelanotic melanomas) and may occasionally be associated with hemorrhage. Conjunctival melanomas typically exhibit malignant behavior including aggressive local infiltration. Canine and feline species of any breed or cross breed may be affected.

DIAGNOSIS AND TREATMENT

The diagnosis of melanoma is made by histopathological interpretation of a representative biopsy sample. The potential for metastasis exists and basic staging via local lymph node aspiration, three-view radiography, and complete blood count (CBC)/Chemistry analysis is recommended. Treatment comprises surgical excision wherever possible (sometimes extending to excenturation), with or without adjunctive chemotherapy, external beam radiation, and/or "metronomic therapy" (the combination of an NSAID and alkylating agent to inhibit vascular growth) where indicated. Cranial soft tissue imaging (MRI or CT) will facilitate accurate presurgical planning if necessary. The value of vaccine-mediated immunotherapy in the treatment of periocular lesions has not been fully evaluated.

SELECTED REFERENCES

Finn M, Krohne S, Stiles J. Ocular melanocytic neoplasia. *Compend Contin Educ Vet*. 2008;**30**(1), 19–25; quiz 26.

Roberts SM, Severin GA, Lavach JD. Prevalence and treatment of palpebral neoplasms in the dog: 200 cases (1975-1983). *J Am Vet Med Assoc*. 1986;**189**(10):1355–1359.

Munger RJ, Gourley IM. Cross lid flap for repair of large upper eyelid defects. *J Am Vet Med Assoc*. 1981;**178**(1):45–48.

Gwin RM, Alsaker RD, Gelatt KN. Melanoma of the lower eyelid of a dog. *Vet Med Small Anim Clin*. 1976;**71**(7):929–931.

Aquino SM. Management of eyelid neoplasms in the dog and cat. *Clin Tech Small Anim Pract* 2007;**22**(2):46–54.

Wang AL and Kern T. Melanocytic ophthalmic neoplasms of the domestic veterinary species: a review. *Top Companion Anim Med*. 2015;**30**(4):148–157.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 66.1 Melanoma. Clinical presentation of a conjunctival melanoma in a canine patient, note the amelanotic portion of the lesion.

Figure 66.2 Melanoma. Typical darkly pigmented presentation of a conjunctival melanoma.

Figure 66.3 Melanoma. The lesion in this case affects the palpebral surface of the third eyelid.

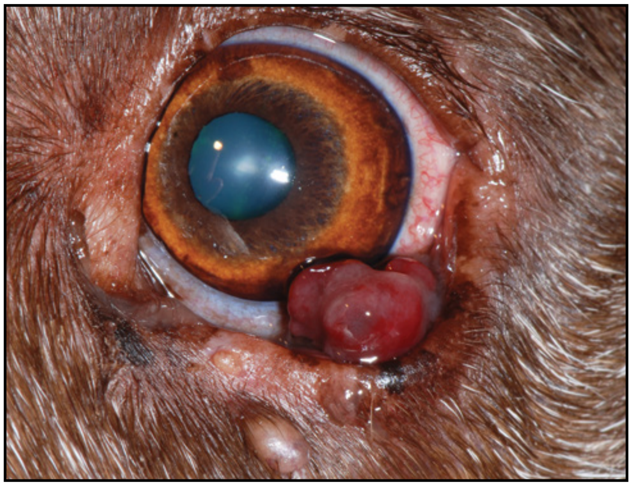


Figure 66.4 Melanoma. The lesion in this case represents an amelanotic conjunctival melanoma.

CHAPTER 67
HEMANGIOMA/HEMANGIOSARCOMA

PRESENTATION

Tumors of vascular endothelial origin may affect the corneoscleral and/or overlying conjunctival tissues (see also Corneoscleral hemangioma/hemangiosarcoma). These lesions typically present as bright red, smooth, raised, irregular to nodular masses at or near the corneoscleral limbus on the palpebral conjunctival surfaces or on the palpebral surfaces of the third eyelid. Tumors enlarge locally and may invade surrounding tissues. Exposure to UV radiation has been proposed to play a factor in the development of adnexal hemangioma/hemangiosarcoma. Commonly affected breeds include the Australian Shepherd and the Collie breeds.

DIAGNOSIS AND TREATMENT

A tentative diagnosis of hemangioma/hemangiosarcoma may be made based on the characteristic clinical appearance of these lesions; however, confirmation as well as characterization of malignancy requires incisional or excisional biopsy and histopathology. Systemic health and/or involvement should be evaluated via local lymph node aspiration, three-view radiography and complete blood count (CBC)/chemistry analysis, although lesions are frequently localized to the adnexal tissues only. Treatment typically encompasses surgical resection where possible (with or without tectonic defect repair using biological or synthetic graft material) and/or the application of adjunctive cryotherapy. Cryotherapy should only be undertaken using an appropriate nitrous-oxide cooled unit. Focal swelling is common following cryotherapy, however inflammation rapidly resolves. Postoperative care comprises topical and or systemic anti-inflammatory and/or anti-microbial therapy as appropriate and the use of an Elizabethan collar to prevent self-trauma.

SELECTED REFERENCES

Chandler HL, Newkirk KM, Kusewitt DF, Dubielzig RR, Colitz CM. Immunohistochemical analysis of ocular hemangiomas and hemangiosarcomas in dogs. *Vet Ophthalmol*. 2009;**12**(2):83–90. doi: 10.1111/j.1463-5224.2008.00684.x.

Hargis AM, Ihrke PJ, Spangler WL, Stannard AA. A retrospective clinicopathologic study of 212 dogs with cutaneous hemangiomas and hemangiosarcomas. *Vet Pathol*. 1992;**29**(4):316–328.

Pirie CG and Dubielzig RR. Feline **conjunctival** hemangioma and **hemangiosarcoma**: a retrospective evaluation of eight cases (1993–2004). *Vet Ophthalmol*. 2006;**9**(4):227–231.

Pirie CG, Knollinger AM, Thomas CB, Dubielzig RR. Canine conjunctival hemangioma and hemangiosarcoma: a retrospective evaluation of 108 cases (1989–2004). *Vet Ophthalmol*. 2006;**9**(4):215–226.

Richardson S and Deykin AR. Surgical treatment of conjunctival hemangioma and **hemangiosarcoma**: a retrospective study of 52 **dogs**. *Vet Ophthalmol*. 2021;**24**(5):432–441.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 67.1 Hemangioma/hemangiosarcoma. Clinical presentation associated with a conjunctival hemangioma/ hemangiosarcoma, note the associated conjunctivitis and mild hemorrhage.

Figure 67.2 Hemangioma/hemangiosarcoma. Typical presentation associated with conjunctival hemangioma/ hemangiosarcoma.

Figure 67.3 Hemangioma/hemangiosarcoma. Note the significant associated hemorrhage in this case.

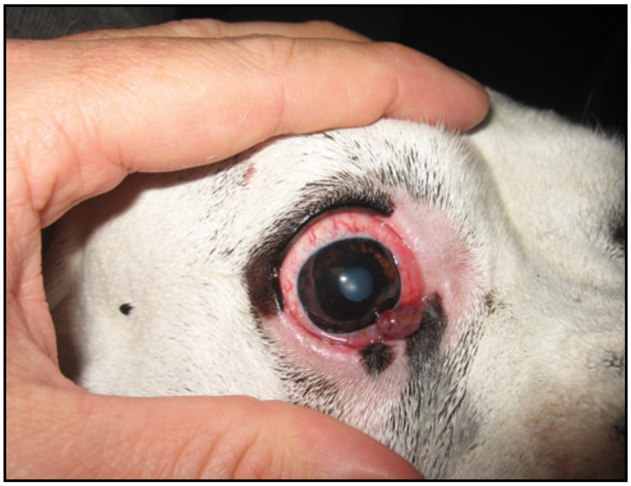


Figure 67.4 Hemangioma/hemangiosarcoma. Note the common clinical presentation in a lightly pigmented patient.

CHAPTER 68
ADENOMA/ADENOCARCINOMA

PRESENTATION

Neoplasia of the third eyelid is not uncommon, the lacrimal gland of the third eyelid frequently representing the site of neoplastic proliferation. These tumors chiefly represent adenoma/adenocarcinoma, the distinction being made histologically. Clinically, neoplasms present as a slowly progressive enlargement, thickening, displacement, and hyperemia of the third eyelid. Secondary conjunctivitis and/or ocular discharge may additionally be present.

DIAGNOSIS AND TREATMENT

The diagnosis is made histologically following incisional or excisional biopsy. Systemic health and/or involvement should be evaluated via local lymph node aspiration, three-view radiography and complete blood count (CBC)/Chemistry analysis, although lesions frequently appear to be localized to the tissues of the third eyelid only. Treatment comprises surgical excision of affected tissue, typically involving removal of the entire third eyelid. Postoperative care comprises routine antimicrobial, anti-inflammatory, and analgesic therapy as well as the use of a protective Elizabethan collar to prevent self-trauma. In addition, an effort should be made to avoid secondary complications associated with decreased corneal protection well as decreased lacrimomimetic function, potentially including the use of topical cyclosporine or tacrolimus and/or the long-term use of a topical lubricating and protective agent. Potential adverse effects associated with the use of topical cyclosporine and tacrolimus include periocular dermatitis and alopecia.

SELECTED REFERENCES

Dees DD, Schobert CS, Dubielzig RR, Stein TJ. **Third eyelid gland** neoplasms of dogs and cats: a retrospective histopathologic study of 145 cases. *Vet Ophthalmol*. 2016;**19**(2):138–143.

Komaromy AM, Ramsey DT, Render JA, Clark P. Primary adenocarcinoma of the gland of the nictitating membrane in a cat. *J Am Anim Hosp Assoc*. 1997;**33**(4):333–336.

Schäffer EH, Pfleghaar S, Gordon S, Knödlseder M. Malignant nictitating membrane tumors in dogs and cats. *Tierarztl Prax*. 1994;**22**(4):382–391.

Wang FI, Ting CT, Liu YS. Orbital adenocarcinoma of lacrimal gland origin in a dog. *J Vet Diagn Invest*. 2001;**13**(2):159–161.

Wilcock B and Peiffer R Jr. Adenocarcinoma of the gland of the third eyelid in seven dogs. *J Am Vet Med Assoc*. 1988;**193**(12):1549–1550.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 68.1 Adenoma/adenocarcinoma. Clinical presentation associated with conjunctival adenoma/adenocarcinoma of the third eyelid gland.

Figure 68.2 Adenoma/adenocarcinoma. Note the subtle swelling associated with the third eyelid.

Figure 68.3 Adenoma/adenocarcinoma. Clinical presentation associated with conjunctival adenoma/adenocarcinoma of the third eyelid. This patient displays the presence of an unrelated, benign, and upper eyelid adenoma.

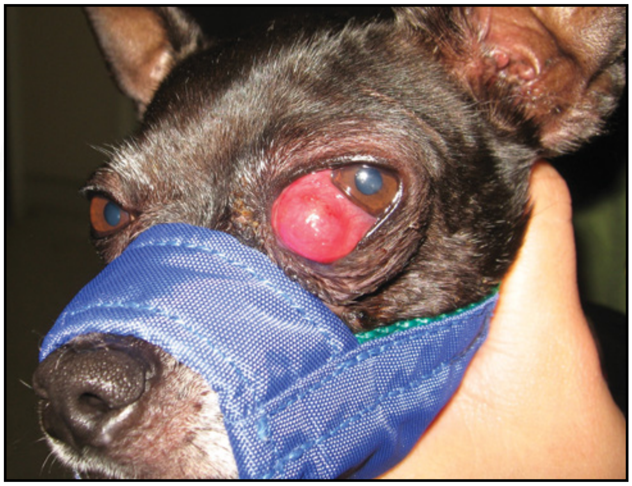


Figure 68.4 Adenoma/adenocarcinoma. Note the significant swelling associated with the third eyelid in this case.

CHAPTER 69
CONJUNCTIVAL LYMPHOMA

PRESENTATION

Conjunctival/third eyelid (TEL) tissues may be affected by lymphoma (see also cutaneous epitheliotropic, corneoscleral, uveal and chorioretinal lymphoma, and retrobulbar neoplasia). In some cases, ocular and/or adnexal symptoms may be the initial presentation for more generalized disease; however, conjunctival/TEL lymphoma may also represent localized disease. Clinical findings may encompass a spectrum of changes from conjunctival/TEL hyperemia to tissue infiltration and/or nodular and/or diffuse cream-colored mass formation. Both canine and feline species of any breed or crossed-breed may be affected.

DIAGNOSIS AND TREATMENT

A diagnosis of conjunctival lymphoma is based on histopathologic interpretation of representative tissue biopsy. Wherever lymphoma is suspected, samples should be harvested prior to the institution of therapy in order to facilitate the planning of an accurate treatment protocol. Treatment comprises topical anti-inflammatory therapy (typically utilizing a corticosteroid) as well as systemic chemotherapy. Associated ophthalmic changes such as elevated intraocular pressure should be addressed as appropriate. Staging via local lymph node (and/or organ/bone marrow) aspiration, three-view radiography and complete blood count (CBC)/chemistry analysis is recommended, prior to initiation of systemic chemotherapy. Diagnostic testing for the presence of infectious viruses, notably FelV and FIV is additionally recommended in feline patients. Individual chemotherapeutic protocols are most ideally designed by a veterinary oncologist however typically include a variable combination of prednisone, vincristine, cyclophosphamide, and/or doxyrubicin. Potential adverse effects associated with the use of systemic cortiocosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of vincristine include stomatitis, GI distress, neuropathy, hepatopathy, and myelosuppression. Potential adverse effects associated with the use of cyclophosphamide include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of doxorubicin include hypersensitivity, gastrointestinal (GI) distress, cardiac dysfunction, and myelosuppression. The prognosis varies depending on the extent of disease present prior to initiation of therapy.

SELECTED REFERENCES

Hong IH, Bae SH, Lee SG, Park JK, Ji AR, Ki MR, Han SY, Lee EM, Kim AY, You SY, Kim TH, Jeong KS. Mucosa-associated lymphoid tissue lymphoma of the third eyelid conjunctiva in a dog. *Vet Ophthalmol*. 2011;**14**(1):61–65.

Kang S, Jeong M, Seo K. Corneoconjunctival manifestations of **lymphoma** in three **dogs**. *J Vet Sci*. 2019;**20**(1):98–101.

McCowan C, Malcolm J, Hurn S, O'Reilly A, Hardman C, Stanley R. Conjunctival lymphoma: immunophenotype and outcome in five dogs and three cats. *Vet Ophthalmol*. 2014;**17**(5):351–357.

Nola M, Lukenda A, Bollmann M, Kalauz M, Petrovecki M, Bollmann R. Outcome and prognostic factors in ocular adnexal lymphoma. *Croat Med J*. 2004;**45**(3):328–332.

Olbertz L, Lima L, Langohr I, Werner J, Teixeira L, Montiani-Ferreira F. Supposed primary conjunctival lymphoma in a dog. *Vet Ophthalmol*. 2013;**16** Suppl 1:100–104. doi: 10.1111/j.1463-5224.2012.01027.x.

Wiggans KT, Skorupski KA, Reilly CM, Frazier SA, Dubielzig RR, Maggs DJ. Presumed solitary intraocular or **conjunctival lymphoma** in **dogs** and cats: 9 cases (1985–013). *J Am Vet Med Assoc*. 2014;**244**(4):460–470.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 69.1 Conjunctival lymphoma. Note perilimbal conjunctival thickening and hyperemia as well as associated corneal changes.

Figure 69.2 Conjunctival lymphoma. Note significant conjunctival chemosis, hyperemia, and thickening as well as associated corneal changes.

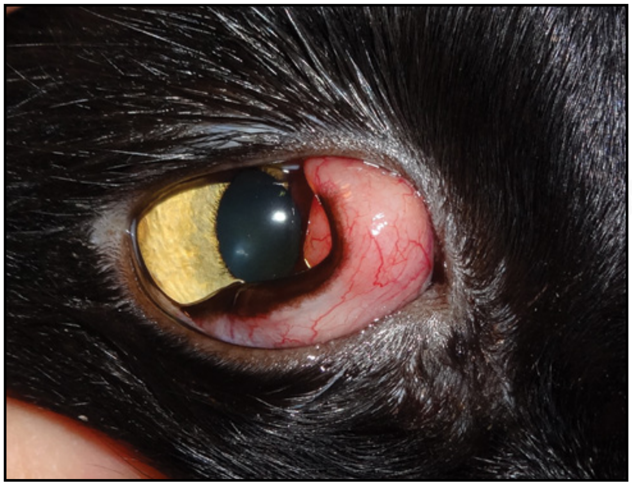


Figure 69.3 Conjunctival lymphoma. Conjunctival thickening and hyperemia are predominantly associated with the tissues of the third eyelid.

Figure 69.4 Conjunctival lymphoma. Again, in this case, conjunctival thickening and hyperemia are predominantly associated with the tissues of the third eyelid.

CHAPTER 70
SQUAMOUS CELL CARCINOMA

PRESENTATION

Conjunctival and/or third eyelid squamous cell carcinoma (SCC) may develop secondary to chronic inflammation with affected cells undergoing various degrees of anisocytosis, anisokaryosis, and/or dyskeratosis, before progressing through low-grade or "carcinomain-situ" to outright SCC. Clinically, SCC presents as irregular, pink colored proliferative to erosive lesions, tissue. Commonly affected breeds include the Pug and the English Bulldog.

DIAGNOSIS AND TREATMENT

Diagnosis is made by histopathology following incisional or excisional biopsy, with SCC frequently appearing clinically similar to (and even encompassing) chronic corneal inflammation/granulation. Systemic health and/or involvement should be evaluated via local lymph node aspiration, three-view radiography, and complete blood count (CBC)/chemistry analysis. Treatment comprises resection of neoplastic tissue wherever possible, with or without the adjunctive application of cryotherapy and/or B-radiation. Cryotherapy should only be undertaken using an appropriate nitrous-oxide cooled unit. Focal swelling is common postoperatively; however, inflammation rapidly resolves. Postoperative care comprises topical and/or systemic anti-inflammatory and/or anti-microbial therapy as appropriate and the use of an Elizabethan collar to prevent self-trauma.

SELECTED REFERENCES

Dees DD, Schobert CS, Dubielzig RR, Stein TJ. **Third eyelid** gland neoplasms of **dogs** and cats: a retrospective histopathologic study of 145 cases. *Vet Ophthalmol.* 2016;**19**(2):138–143.

Dreyfus J, Schobert CS, Dubielzig RR. Superficial corneal squamous cell carcinoma occurring in dogs with chronic keratitis. *Vet Ophthalmol.* 2011;**14**(3):161–168. doi: 10.1111/j.1463-5224.2010.00858.x.

Lavach JD, *et al*. Squamous cell carcinoma of the third eyelid in a dog. *J Am Vet Med Assoc.* 1984.

Montiani-Ferreira F, Kiupel M, Muzolon P, Truppel J. Corneal squamous cell carcinoma in a dog: a case report. *Vet Ophthalmol.* 2008;**11**(4):269–272. doi: 10.1111/j.1463-5224.2008.00622.x.

Takiyama N, Terasaki E, Uechi M. Corneal squamous cell carcinoma in two dogs. *Vet Ophthalmol.* 2010;**13**(4):266–269. doi: 10.1111/j.1463-5224.2010.00792.x.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

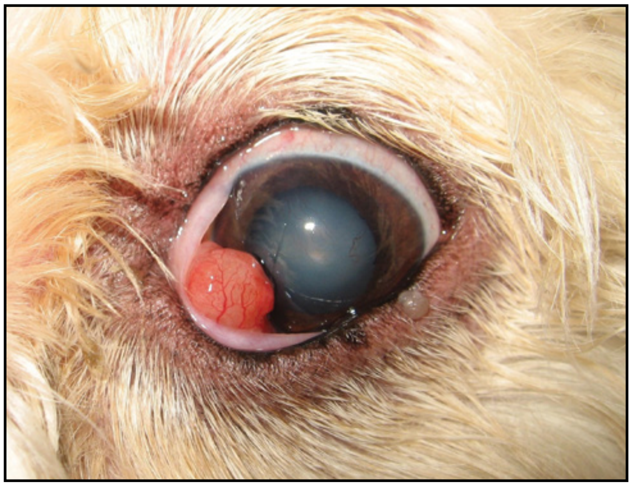


Figure 70.1 Squamous cell carcinoma. Clinical presentation associated with conjunctival/third eyelid squamous cell carcinoma in a canine patient.

Figure 70.2 Squamous cell carcinoma. Clinical presentation associated with conjunctival/third eyelid squamous cell carcinoma in a feline patient.

Figure 70.3 Squamous cell carcinoma. Proliferative squamous cell carcinoma lesion in the third eyelid of a feline patient.

Figure 70.4 Squamous cell carcinoma. Early presentation of a third eyelid conjunctival squamous cell carcinoma in a canine patient.

Section 7

Corneoscleral Disease

CHAPTER 71
DERMOID

PRESENTATION

A dermoid (also sometimes referred to as a "choristoma") represents an area of normal haired skin in an abnormal location on the eye or adnexal tissue. Lesions may affect the cornea and/or adjacent structures including the limbus, conjunctival surfaces, third eyelid and/or eyelid. Changes are congenital so are typically noted soon after the eyes open at two weeks of age. Secondary conjunctivitis and discomfort may manifest as blepharospasm and/or ocular discharge. Commonly affected breeds include the German Shepherd, the Dachshund, and the St Bernard.

DIAGNOSIS AND TREATMENT

The treatment of choice is surgical excision of the abnormal tissue by superficial peritomy and/or keratectomy. Once excised these lesions do not recur. The medial and lateral canthal anatomy should be preserved where possible. Tectonic corneal grafting may or may not be necessary after dermoid removal, based on the depth of the resultant defect. Similarly, resultant eyelid defects may require blepharoplastic reconstruction. Postoperative care comprises routine systemic antimicrobial, anti-inflammatory, and analgesic care. An Elizabethan collar should be placed in order to prevent self-trauma, and corneal health should be frequently monitored until eyelid healing is complete.

SELECTED REFERENCES

Badanes Z and Ledbetter EC. Ocular dermoids in dogs: a retrospective study. *Vet Ophthalmol*. 2019;**22**(6):760–766.

Brudenall DK, Bernays ME, Peiffer RL Jr. Central corneal dermoid in a Labrador retriever puppy. *J Small Anim Pract*. 2007;**48**(10):588–590.

Kalpravidh M, Tuntivanich P, Vongsakul S, Sirivaidyapong S. Canine amniotic membrane transplantation for corneal reconstruction after the excision of dermoids in dogs. *Vet Res Commun*. 2009;**33**(8):1003–1012.

Saraiva IQ and Delgado E. Congenital **ocular** malformations in **dogs** and cats: 123 cases. *Vet Ophthalmol*. 2020;**23**(6):964–978.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

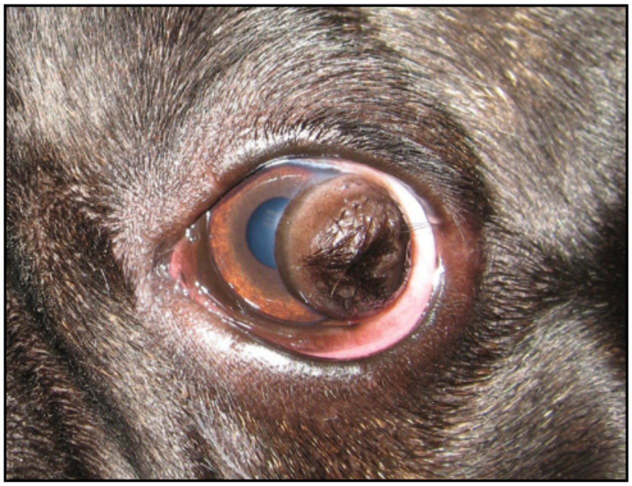


Figure 71.1 Dermoid. Clinical presentation associated with a large corneoscleral dermoid in a canine patient.

Figure 71.2 Dermoid. Typical presentation associated with a corneoscleral dermoid at the lateral limbus.

Figure 71.3 Dermoid. Corneoscleral dermoid with long hairs causing significant discomfort.

Figure 71.4 Dermoid. Corneoscleral dermoid located at the lateral limbus.

CHAPTER 72
CORNEAL DYSTROPHY

PRESENTATION

Corneal dystrophy describes a group of corneal disorders, likely hereditary in etiology, in which cholesterol, phospholipids, and free fatty acids accumulate within the cornea(s). Lesions appear as subepithelial to stromal gray/white, crystalline, relucencies within the corneal stroma. This disorder is bilateral in presentation (although not always symmetrical) and is not associated with any preexisting or concurrent corneal or ocular inflammation. Multiple patterns of corneal dystrophy have been described, many with breed-associations. These may include axial or peripheral, ovoid to circular lesions. Occasionally, corneal dystrophic changes may reflect underlying metabolic storage disease. Commonly affected breeds include the King Charles Cavalier Spaniel, Siberian Husky, Beagle, and Shetland Sheepdog.

DIAGNOSIS AND TREATMENT

Evaluation of underlying thyroid and adrenocortical function as well as systemic triglyceride and cholesterol levels, and/or glucose, calcium, and phosphorus may be indicated in select cases, in order to rule out underlying metabolic abnormalities. Abnormal serum chemistry values may be addressed by dietary modification using low-fat foods. Corticosteroid based anti-inflammatory medications may exacerbate changes. Keratectomy may be indicated in rare cases of secondary ulceration and/or mineralization associated with epithelial discontinuity and discomfort.

SELECTED REFERENCES

Barsotti G, Pasquini A, Busillo L, Senese M, Cardini G, Guidi G. Corneal crystalline stromal dystrophy and lipidic metabolism in the dog. *Vet Res Commun.* 2008;**32** (Suppl 1):S227–S2229

Cooley PL, Dice PF. Corneal dystrophy in the dog and cat. *Vet Clin North Am Small Anim Pract.* 1990;**20**(3):681–692.

Crispin SM. Crystalline corneal dystrophy in the dog. Histochemical and ultrastructural study. *Cornea.* 1988;**7**(2):149–161.

Spangler WL, Waring GO, Morrin LA. Oval lipid corneal opacities in beagles. *Vet Pathol.* 1982;**19**(2):150–159.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 72.1 Corneal dystrophy. Clinical presentation of corneal dystrophy. These changes are typically bilateral and are not associated with clinical inflammation.

Figure 72.2 Corneal dystrophy. Note the typical axial location and circular appearance of the lesion.

Figure 72.3 Corneal dystrophy. This case demonstrates a significant corneal leukoma, inset displays bilateral nature of condition.

Figure 72.4 Corneal dystrophy. Note the typical axial location and the lack of any clinical inflammation.

CHAPTER 73
ANTERIOR SEGMENT DYSGENESIS (ASD)

PRESENTATION

Anterior segment dysgenesis describes a failure of the normal development of the anterior tissues and structures of the eye (predominantly the cornea, anterior uvea, and lens), with strong similarities to "Peters Anomaly" in humans. This condition is occasionally encountered in clinical practice. Pathology is thought to arise as a result of faulty separation of the embryonic lens vesicle in association with altered mesenchymal neural crest cellular migration and differentiation. Features of this developmental disorder typically encompass any combination of microcornea, corneal thinning and/or leukoma (opacity), anterior uveal adhesions, iris hypoplasia, lens pathology (most commonly cataract formation), and/or microphthalmos. This condition may encompass iridocorneal angle abnormalities and an increased risk for the development of glaucoma. Changes are usually bilateral (though frequently asymmetrical) and are commonly noted in juvenile animals. Dog and cats of any breed or crossed breed may be affected.

DIAGNOSIS AND TREATMENT

The treatment of animals affected by anterior segment dysgenesis (ASD) comprises the monitoring and management of secondary pathology including keratitis, uveitis, cataracts, and/or intraocular pressure (IOP) elevation, often comprising the judicious use of topical non-steroidal anti-inflammatory drugs (NSAIDS) as well as topical lubricating and protective agents. The prognosis for successful management of affected animals is typically considered positive.

SELECTED REFERENCES

Laratta LJ, Riis RC, Kern TJ, Koch SA. Multiple congenital ocular defects in the Akita dog. *Cornell Vet.* 1985;**75**(3):381–392.

Peiffer RL Jr, Fischer CA. Microphthalmia, retinal dysplasia, and anterior segment dysgenesis in a litter of Doberman Pinschers. *J Am Vet Med Assoc.* 1983;**183**(8):875–878.

Shaw GC, Tse MPY, Miller AD. Microphthalmia with multiple anterior segment defects in Portuguese water dogs. *Vet Pathol.* 2019;**56**(2):269–273. doi: 10.1177/0300985818794248. Epub 2018 Aug 21. PMID: 30131012.

Swanson HL, Dubielzig RR, Bentley E, Murphy CJ, Collins BK, Collier LL, Johnson GS, Shibuya H, Moore CP, da Silva Curiel JM. A case of Peters' anomaly in a springer spaniel**.** *J Am Vet Med Assoc.* 1992;**200**(10):1485–1491. PMID: 1612983.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

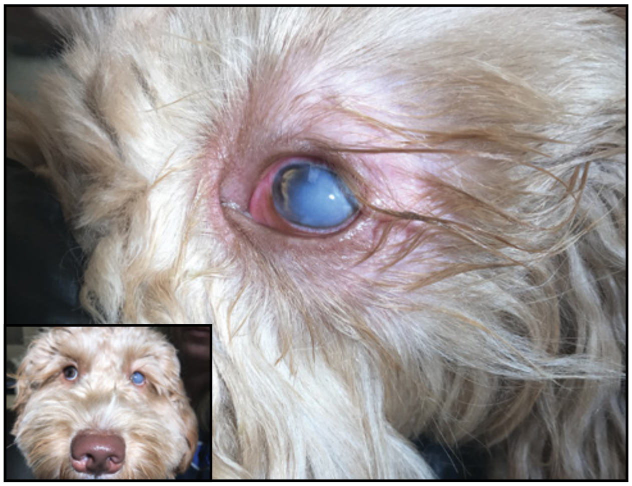


Figure 73.1 Anterior segment dysgenesis. Clinical presentation associated with anterior segment dysgenesis, inset demonstrates bilateral clinical changes.

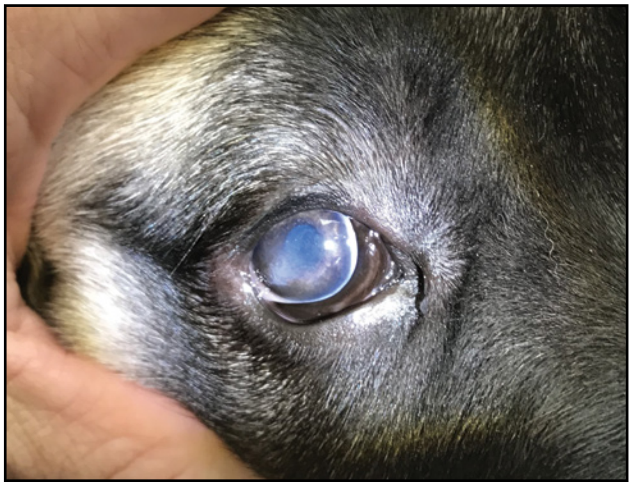


Figure 73.2 Anterior segment dysgenesis. Note paraxial corneal thinning and fibrosis.

Figure 73.3 Anterior segment dysgenesis. Note multifocal endothelial opacities.

Figure 73.4 Anterior segment dysgenesis. Note central diffuse endothelial opacities.

CHAPTER 74
CORNEAL DEGENERATION

PRESENTATION

Corneal degeneration represents pathological changes occurring in the cornea secondary to preexisting ocular and/or systemic inflammation. Degenerative changes appear as irregular, asymmetric gray/white deposits within the cornea. Lesions may be unilateral or bilateral and are typically associated with neovascularization. Concurrent ulceration and/or secondary infection may additionally be present and unstable regions of degenerative and mineralized tissue may spontaneously slough. Variable discomfort may be manifested by epiphora, blepharospasm, and/or mucoid discharge. Many breeds may be affected, particularly with significantly advanced age.

DIAGNOSIS AND TREATMENT

Evaluation of thyroid and adrenocortical function, complete blood count (CBC)/chemistry values as well as systemic lipid and cholesterol levels is indicated, in order to rule out underlying metabolic abnormities. Mineral deposits may be removed wherever indicated via diamond-burr keratectomy, chemical keratectomy (using dilute trichloroacetic acid), or surgical keratectomy. Tectonic corneal grafting may or may not be necessary based on the depth of the resultant defect. Exposed mineral may also be partially chelated using topical EDTA solution until re-epithelialization is complete. Associated ulceration should be treated medically using topical antimicrobial agents or surgically if appropriate. In general, topical corticosteroids should be avoided. The use of an Elizabethan collar will prevent self-trauma until corneal healing is complete.

SELECTED REFERENCES

Crispin SM, Barnett KC. Dystrophy, degeneration and infiltration of the canine cornea. *Journal of Small Animal Practice* 1983; **24**: 63–83.

Laus JL, dos Santos C, Talieri IC, Oriá AP, Bechara GH. Combined corneal lipid and calcium degeneration in a dog with hyperadrenocorticism: a case report. *Vet Ophthalmol.* 2002;**5**(1):61–64.

Nevile JC, Hurn SD, Turner AG, Morton J. Diamond burr debridement of 34 canine corneas with presumed **corneal** calcareous **degeneration**. *Vet Ophthalmol.* 2016;**19**(4):305–312.

Sansom J, Blunden T. Calcareous degeneration of the canine cornea. *Vet Ophthalmol.* 2010;**13**(4):238–243.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 74.1 Corneal degenaration. Clinical presentation associated with corneal degeneration. Active inflammation as well as ulcerative keratitis are present.

Figure 74.2 Corneal degenaration. Note the associated vascular keratitis.

Figure 74.3 Corneal degenaration. The central corneal defect likely resulted from mineral sloughing.

Figure 74.4 Corneal degenaration. Note the associated vascular keratitis and irregular nature of the lesions.

CHAPTER 75
CORNEAL ENDOTHELIAL DECOMPENSATION

PRESENTATION

Corneal endothelial decompensation represents a progressive loss of corneal endothelial cell function and density and results in slowly progressive corneal edema. Corneal endothelial cells do not regenerate and have only a limited ability to compensate. Clinically, this disease presents as progressive, avascular, corneal edema, marked by blue/gray discoloration and thickening. Changes typically begin within the superiotemporal corneal quadrant. The disease is bilateral though not always symmetrical. Initially, changes do not result in significant visual disturbance or discomfort; however with progression, a cycle of painful bullous and/or ulcerative keratopathy may develop. Commonly affected breeds include the Boston Terrier, the Dachshund, Chihuahua, and the Miniature Poodle.

DIAGNOSIS AND TREATMENT

When indicated, definitive surgical treatment includes limited thermo or CO_2 laser keratoplasty, the placement of a "Gunderson" conjunctival pedicle graft or ultimately penetrating keratoplasty. The palliative use of sodium chloride ointment has been reported to increase patient comfort however will not adequately address underlying endothelial dysfunction. The palliative use of topical anti-inflammatory agents has also been advocated by some authors.

SELECTED REFERENCES

Giannikaki S, Escanilla N, Sturgess K, Lowe RC. A modified technique of keratoleptynsis ("letter-box") for treatment of canine **corneal** edema associated with **endothelial** dysfunction. *Vet Ophthalmol.* 2020;**23**(6):930–942.

Gwin, R., Pollack, F., Warren, J *et al* (1982) Primary canine corneal endothelial cell dystrophy: specular microscopic evaluation, diagnosis and therapy. *J Am Anim Hosp Assoc.*, **18**, 471–479.

Leonard BC, Kermanian CS, Michalak SR, Kass PH, Hollingsworth SR, Good KL, Maggs DJ, Thomasy SM. A retrospective study of corneal **Endothelial Dystrophy** in **Dogs** (1991–2014). *Cornea.* 2021;**40**(5):578–583.

Martin, C & Dice, P (1982) Corneal endothelial dystrophy in the dog. J Am Anim Hosp Assoc., **18**, 327–336.

Michau TM, Gilger BC, Maggio F, Davidson MG. Use of thermokeratoplasty for treatment of ulcerative keratitis and bullous keratopathy secondary to corneal endothelial disease in dogs: 13 cases (1994–2001). *J Am Vet Med Assoc.* 2003;**222**(5):607–12.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

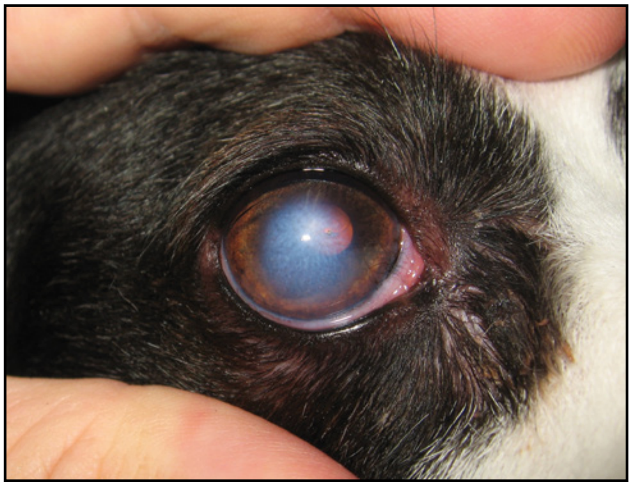


Figure 75.1 Corneal endothelial decompensation. Clinical presentation associated with early corneal endothelial decompensation and secondary corneal edema.

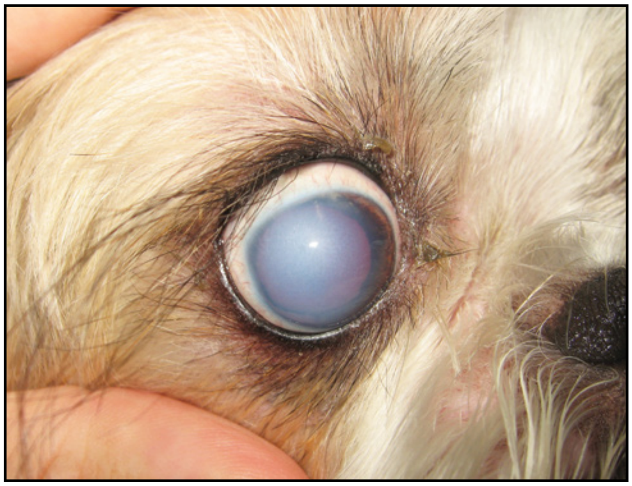


Figure 75.2 Corneal endothelial decompensation. Note significant generalized secondary corneal edema in this advanced case.

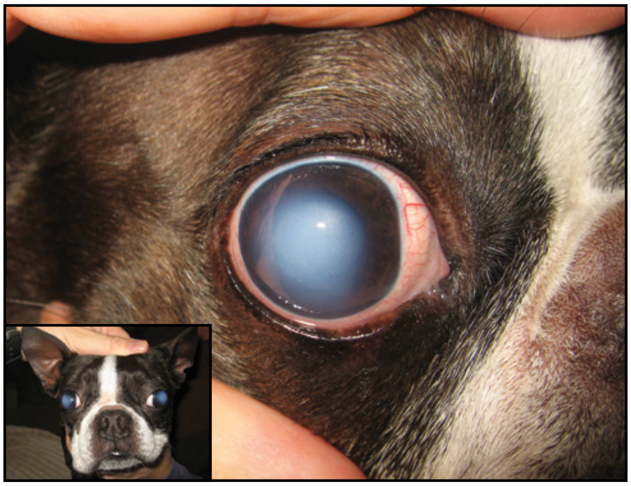


Figure 75.3 Corneal endothelial decompensation. Typical presentation associated with corneal endothelial decompensation in a Boston Terrier, inset demonstrates the bilateral nature of the disease.

Figure 75.4 Corneal endothelial decompensation. Typical presentation associated with corneal endothelial decompensation in a Chihuahua, inset demonstrates the bilateral nature of the disease.

CHAPTER 76
SCLERITIS

PRESENTATION

Scleritis describes a primary inflammatory disorder of the canine scleral tissues which is thought to be immune-mediated in origin. Scleritis presents as inflammatory changes comprising thickening, hyperemia, and/or vascular congestion of the scleral tissues. An inflammatory infiltrate around part or all of the corneoscleral limbus, affecting one or both eyes may be present. Associated discomfort may be manifested by blepharospasm and/or ocular discharge. Symptoms are generally bilateral however frequently asymmetric with one eye commonly affected before the other. Adjacent structures including the uveal tract may be involved and demonstrate associated inflammation. In rare cases, scleral necrosis may develop. Secondary complications associated with this severe form of scleritis may include uveitis, hyphema, retinal detachment, scleral rupture, and/or globe loss. Dogs of any breed or crossed breed may be affected. Commonly affected breeds include the Golden Retriever, Siberian Husky, Australian Shepherd, and the Spaniel breeds (American Cocker, English Cocker, English Springer, and Brittany).

DIAGNOSIS AND TREATMENT

The diagnosis of scleritis is made based on clinical findings, confirmed if necessary by tissue biopsy and histopathology, which typically reveals a significant lymphoplasmacytic infiltrate. Necrotizing scleritis is specifically marked by the presence of collagen lysis. Systemic screening for immune-mediated disorders may be indicated in severe cases. Management of scleritis may comprise topical, subconjunctival, and/or systemic immunomodulating therapy. The combination of chronic surface inflammation and steroidal agents may predispose affected eyes to significant secondary mineral degeneration. Appropriate systemic agents include prednisone, cyclosporin, azathioprine, leflunomide, cyclophosphamide, and/or methotrexate. In addition, areas of scleral tectonic weakness/perforation may require surgical repair. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of cyclosporine include hypersensitivity and GI distress. Potential adverse effects associated with the use of systemic azathioprine and chlorambucil include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Scleritis can typically be managed; however in most cases, ongoing therapy is required in order to maintain the patient in a disease-free state.

SELECTED REFERENCES

Breaux CB, Sandmeyer LS, Grahn BH. Immunohistochemical investigation of canine episcleritis. *Vet Ophthalmol.* 2007;**10**(3):168–172.

Denk N, Sandmeyer LS, Lim CC, Bauer BS, Grahn BH. A retrospective study of the clinical, histological, and immunohistochemical manifestations of 5 dogs originally diagnosed histologically as necrotizing scleritis. *Vet Ophthalmol.* 2012;**15**(2):102–109.

Grahn BH, Sandmeyer LS. Canine episcleritis, nodular episclerokeratitis, scleritis, and necrotic scleritis. *Vet Clin North Am Small Anim Pract.* 2008;**38**(2):291–308, vi. doi: 10.1016/j.cvsm.2007.11.003.

Sandmeyer LS, Breaux CB, Grahn BH. What are your clinical diagnosis, differential diagnoses, therapeutic plan, and prognosis? Diffuse episcleritis of the right eye. *Can Vet J.* 2008;**49**(1):89–90.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 76.1 Scleritis. Clinical presentation associated with primary scleritis.

Figure 76.2 Scleritis. Note limbal infiltration visible laterally.

Figure 76.3 Scleritis. Note characteristic dilated episcleral vasculature.

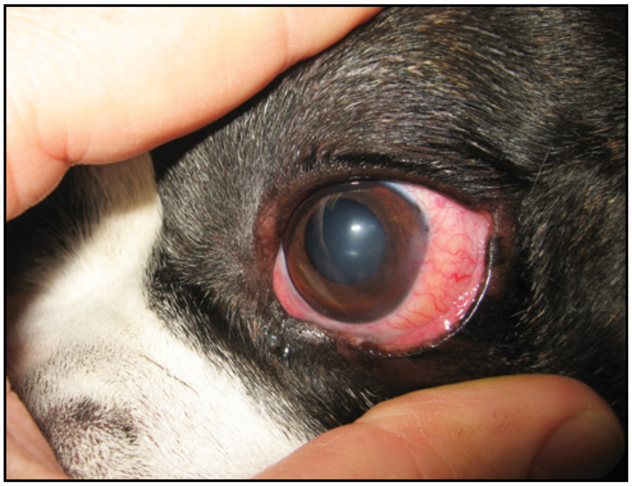


Figure 76.4 Scleritis. Limbal infiltration is visible laterally and ventrally in this milder presentation of primary scleritis.

CHAPTER 77
NODULAR GRANULOMATOUS EPISCLERITIS

PRESENTATION

Nodular granulomatous episcleritis (NGE) describes a proliferative inflammatory disorder of the canine scleral and/or episcleral tissues, which is thought be immune-mediated in origin. Multiple terms have been used to describe this condition, including nodular fasciitis, fibrous histiocytoma, pseudotumor, and collie-granuloma. Clinically, affected patients display one or more smooth, firm, mass(es) affecting the corneoscleral limbus of one or both eyes, arising most commonly in the superior-temporal region. Associated discomfort may be manifested by blepharospasm and/or ocular discharge. Commonly affected breeds include the Collie-breeds, Spaniel-breeds, and Terrier-breeds.

DIAGNOSIS AND TREATMENT

The diagnosis of NGE is made based on clinical findings, confirmed where indicated by tissue biopsy and histopathology (which typically reveals a proliferation of fibroblasts and a significant infiltration of lymphocytes, plasma cells and histiocytes). Treatment comprises topical and/or systemic immune-modulatory therapy using corticosteroids and or adjunctive agents such as azathioprine, chlorambucil, and cyclosporine. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of systemic azathioprine and chlorambucil include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of cyclosporine include hypersensitivity, and GI distress. In severe cases, lesions may initially be managed by the injection of repository corticosteroids, surgical resection, cryotherapy, and/or the application of B-irradiation. NGE can typically be managed; however, in most cases, ongoing therapy is required in order to maintain the patient in a disease-free state.

SELECTED REFERENCES

Barnes LD, Pearce JW, Berent LM, Fox DB, Giuliano EA. Surgical management of orbital nodular granulomatous episcleritis in a dog. *Vet Ophthalmol.* 2010;**13**(4):251–258. doi: 10.1111/j.1463-5224.2010.00781.x.

Day MJ, Mould JR, Carter WJ. An immunohistochemical investigation of canine idiopathic granulomatous scleritis. *Vet Ophthalmol.* 2008;**11**(1):11–17. doi: 10.1111/j.1463-5224.2007.00592.x.

Grahn BH, Sandmeyer LS. Canine episcleritis, nodular episclerokeratitis, scleritis, and necrotic scleritis. *Vet Clin North Am Small Anim Pract.* 2008;**38**(2):291–308, vi. doi: 10.1016/j.cvsm.2007.11.003. Review.

Hamzianpour N, Heinrich C, Jones RG, McElroy P, Wilson N, Scurrell E. Clinical and pathological findings in three **dogs** with a corneocentric presentation of nodular granulomatous episcleritis. *Vet Ophthalmol.* 2019;**22**(4):529–537.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 77.1 Nodular granulomatous episcleritis. Clinical presentation associated with nodular granulomatous episcleritis. Note the discrete, solitary lesion in this case.

Figure 77.2 Nodular granulomatous episcleritis. Note the large, smooth, and firm limbal lesion.

Figure 77.3 Nodular granulomatous episcleritis. This patient displays a more generalized episcleral inflammation in addition to several smooth and firm nodules.

Figure 77.4 Nodular granulomatous episcleritis. Typical presentation affecting the temporal limbus.

CHAPTER 78
CHRONIC SUPERFICIAL KERATITIS (CSK)

PRESENTATION

Chronic superficial keratitis (CSK) describes a bilateral inflammatory condition, which predominantly affects the corneal tissues. Changes encompass vascular proliferation, inflammatory cell infiltration, and secondary pigment deposition. Cellular infiltration comprises lymphocytes, plasma cells, neutrophils, and melanocytes. These changes most commonly arise from the lateral limbal region and progress across the central corneas. Adjacent eyelid margin, conjunctival and third eyelid inflammation are frequently associated with this process. Secondary corneal degeneration and mineralization may ensue in chronic and severe cases. This disease has been also referred to "pannus" and "Uberreiter's syndrome". CSK is considered to represent an immune-mediated process with an underlying hereditary basis. Exposure to UV sunlight likely exacerbates clinical symptoms. Commonly affected breeds include the German Shepherd, Belgian Malinois, and Greyhound.

DIAGNOSIS AND TREATMENT

The diagnosis of CSK is made based on clinical findings, supported if necessary by tissue biopsy. Untreated, CSK has the potential to result in blindness. Additionally, chronic corneal inflammation may be risk a factor for the development of corneal squamous cell carcinoma. Inflammatory changes typically respond to topical anti-inflammatory therapy using corticosteroids (+/− topical immune-suppressive agents including cyclosporine and/or tacrolimus). Occasionally, repository sub-conjunctival corticosteroids may be indicated. In severe cases, systemic therapy using corticosteroids and/or azathioprine additionally may be required. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of systemic Azothioprine include GI distress, pancreatitis, hepatotoxicity, and myelo-suppression. Potential adverse effects associated with the use of topical cyclosporine and tacrolimus include periocular dermatitis and alopecia. CSK is easily controlled in most cases; however, ongoing topical therapy is typically required in order to prevent a recurrence of symptoms. In unusually severe and chronic cases, surgical superficial keratectomy +/− the use of adjunctive B-radiation may be indicated.

SELECTED REFERENCES

Allgoewer I, Hoecht S. Radiotherapy for canine chronic superficial keratitis using soft X-rays (15 kV). *Vet Ophthalmol.* 2010;**13**(1):20–25. doi: 10.1111/j.1463-5224.2009.00750.x.

Balicki I. Clinical study on the application of tacrolimus and DMSO in the treatment of chronic superficial keratitis in dogs. *Pol J Vet Sci.* 2012;**15**(4):667–676.

Chavkin MJ, Roberts SM, Salman MD, Severin GA, Scholten NJ. Risk factors for development of **chronic superficial keratitis** in **dogs**. *J Am Vet Med Assoc.* 1994;**204**(10):1630–1634.

Drahovska Z, Balicki I, Trbolova A, Mihalova M, Holickova M. A retrospective study of the occurrence of **chronic superficial keratitis** in 308 German Shepherd **dogs**: 1999–2010. *Pol J Vet Sci.* 2014;**17**(3):543–546.

Peiffer RL Jr, Gelatt KN, Gwin RM. Chronic superficial keratitis. *Vet Med Small Anim Clin.* 1977;**72**(1):35–37.

Williams DL. Histological and immunohistochemical evaluation of canine chronic superficial keratitis. *Res Vet Sci.* 1999;**67**(2):191–195.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 78.1 Chronic superficial keratitis. Associated infiltration of the third eyelid tissues is notable. Inset demonstrates the typically bilateral presentation of the condition.

Figure 78.2 Chronic superficial keratitis. Secondary corneal pigmentation is notable in this case. Inset demonstrates the typically bilateral presentation of the condition.

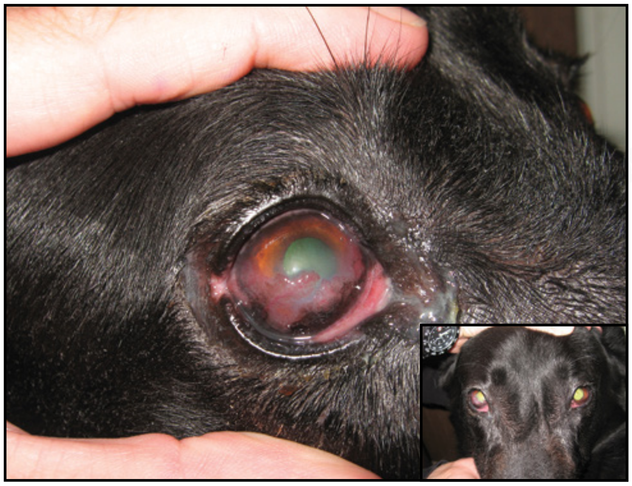


Figure 78.3 Chronic superficial keratitis. Typical presentation associated with chronic superficial keratitis demonstrating both corneal and third eyelid tissue infiltration.

Figure 78.4 Chronic superficial keratitis. Typical presentation associated with chronic superficial keratitis, primarily demonstrating significant vascular proliferation.

CHAPTER 79
EOSINOPHILLIC KERATOCONJUNCTIVITIS

PRESENTATION

Eosinophilic keratoconjunctivitis (EKC) describes a feline condition comprising infiltration of the corneal and/or conjunctival tissues with an eosinophil-rich inflammatory infiltrate. Clinically, affected cats display a variable degree of cream to white-colored infiltration and "plaque-like" deposition, frequently in association with vascular keratitis. Changes typically originate from the corneoscleral limbus and may be uni or bilateral. Associated discomfort may be manifested by blepharospasm and/or ocular discharge.

DIAGNOSIS AND TREATMENT

The diagnosis of eosinophilic keratoconjunctivitis is confirmed by the cytologic or histopathologic evaluation of samples harvested from affected tissues. Treatment comprises topical and/or systemic anti-inflammatory therapy, typically comprising corticosteroids. Adjunctive cyclosporine therapy may also beneficial in selected cases. In severe cases, a prompt clinical response is typically noted following the institution of systemic megesterol acetate therapy; however, the significant potential adverse effects which may be associated with its use, dictate clinical caution and close monitoring of treated patients. Finally, the relationship between feline herpesviral disease and EKC remains unclear and topical and/or systemic antiviral therapy may additionally be warranted in selected cases. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of topical cyclosporine include periocular dermatitis and alopecia. Potential adverse effects associated with the use of megestrol acetate include the development of diabetes mellitus, mammary hyperplasia, and neoplasia. EKC is easily controlled in most cases; however, ongoing therapy is typically required in order to control the condition and prevent a recurrence of symptoms.

SELECTED REFERENCES

Andrew SE. Immune-mediated canine and feline keratitis. *Vet Clin North Am Small Anim Pract.* 2008;**38**(2):269–290, vi. doi: 10.1016/j.cvsm.2007.11.007.

Dean E, Meunier V. Feline eosinophilic keratoconjunctivitis: a retrospective study of 45 cases (56 eyes). *Feline Med Surg.* 2013;**15**(8):661–666. doi: 10.1177/1098612X12472181. Epub 2013 Jan 15.

Nasisse MP, Glover TL, Moore CP, Weigler BJ. Detection of feline herpesvirus 1 DNA in corneas of cats with eosinophilic keratitis or corneal sequestration. *Am J Vet Res.* 1998;**59**(7):856–858.

Spiess AK, Sapienza JS, Mayordomo A. Treatment of proliferative feline **eosinophilic keratitis** with topical 1.5% cyclosporine: 35 cases. *Vet Ophthalmol.* 2009;**12**(2):132–137.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 79.1 Eosinophilic keratoconjunctivitis. Clinical presentation associated with eosinophilic keratoconjunctivitis, comprising cream-colored corneal infiltration and vascular changes.

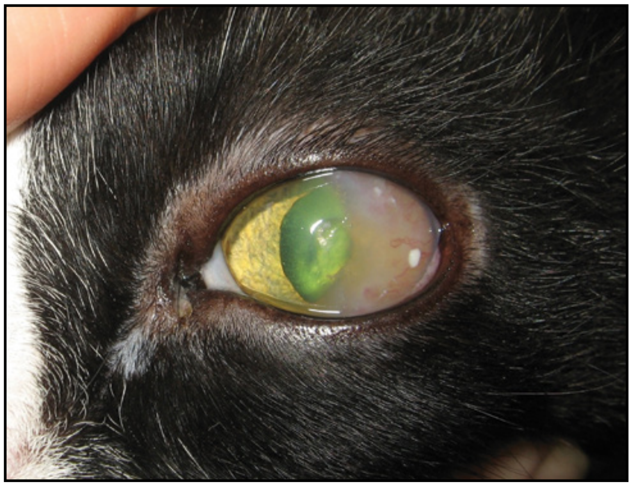


Figure 79.2 Eosinophilic keratoconjunctivitis. Note the focal eosinophilic plaques and significant vascular keratitis.

Figure 79.3 Eosinophilic keratoconjunctivitis. Mild clinical presentation associated with early eosinophilic keratoconjunctivitis.

Figure 79.4 Eosinophilic keratoconjunctivitis. Advanced clinical presentation associated with chronic eosinophilic keratoconjunctivitis.

CHAPTER 80
HERPESVIRAL-ASSOCIATED KERATITIS

PRESENTATION

Feline herpes virus-1 (FHV-1) is a widespread and prevalent cause of ophthalmic disease, affecting both kittens and mature cats (particularly shelter animals and those from multi-cat households). Initial infection occurs via contact with the virus (typically through cat-to-cat contact) or contaminated fomites. Once infected, cats become latent carriers of the disease, which may recrudesce under conditions of physiologic or pharmacologic immunosuppression. Symptoms may include pyrexia, lethargy, and inappetence, signs of upper respiratory disease (such as coughing or sneezing), conjunctivitis, chemosis, symblepharon and superficial, dendritic, stromal, and/or ulcerative keratitis (see also Herpesviral-associated conjunctivitis). Discomfort may be manifested as blepharospasm and/or ocular discharge. Symptoms may be unilateral or bilateral.

DIAGNOSIS AND TREATMENT

Accurate testing for the presence of FHV-1 and its correlation with ophthalmic disease is problematic; however, polymerase-chain-reaction assays, based on tissue samples, are currently considered most reliable. Ultimately healthy cats recover spontaneously from outbreaks over the course of several weeks; however, treatment is frequently indicated. Topical and/or systemic antiviral agents may be administered, including; trifluorothymidine, idoxuridine, cidofovir, ganciclovir, and/or famciclovir (metabolized to penciclovir), respectively. Potential complications associated with the use of topical antiviral agents include local irritation. Potential complications associated with the use of systemic antiviral medications include gastrointestinal (GI) distress, myelosuppression, and/or hepatopathy. The concurrent administration of systemic L-lysine may decrease the severity of symptoms, potentially via the inhibition of herpesviral metabolism. Caution is indicated when considering the use of anti-inflammatory therapy to treat herpesviral-associated disease. Non-steroidal agents are generally considered less likely to result in complication, and the use of corticosteroids generally considered contra-indicated in most cases. Corneal ulcers should be treated medically or surgically as indicated. Herpesviral-associated conjunctivitis may be complicated by underlying systemic disease (such as FelV and FIV), concurrent viral pathogens (such as Calicivrus), and/or secondary infection with Chlamdophila, Mycoplasma, and/or bacterial pathogens. As a consequence, tetracyclines, azithromycin, and fluoroquinolones represent appropriate antimicrobial choices when treating potentially complicated cases. Potential adverse effects associated with tetracyclines and azithromycin include GI distress, photosensitivity, and hepatic damage. Potential adverse effects associated with fluoroquinolones include GI distress, hepatic disease, neurologic disease, and/or retinal toxicity.

SELECTED REFERENCES

Drazenovich TL, Fascetti AJ, Westermeyer HD, Sykes JE, Bannasch MJ, Kass PH, Hurley KF, Maggs DJ. Effects of dietary lysine supplementation on upper respiratory and ocular disease and detection of infectious organisms in cats within an animal shelter. *Am J Vet Res.* 2009;**70**(11):1391–1400. doi: 10.2460/ajvr.70.11.1391.

Fontenelle JP, Powell CC, Veir JK, Radecki SV, Lappin MR. Effect of topical ophthalmic application of cidofovir on experimentally induced primary ocular feline herpesvirus-1 infection in cats. *Am J Vet Res.* 2008;**69**(2):289–293. doi: 10.2460/ajvr.69.2.289.

Maggs DJ. Antiviral therapy for feline herpesvirus infections. *Vet Clin North Am Small Anim Pract.* 2010;**40**(6):1055–1062. doi: 10.1016/j.cvsm.2010.07.010.

Maggs DJ. Update on pathogenesis, diagnosis, and treatment of feline herpesvirus type 1. *Clin Tech Small Anim Pract.* 2005;**20**(2):94–101.

Thomasy SM, Lim CC, Reilly CM, Kass PH, Lappin MR, Maggs DJ. Evaluation of orally administered famciclovir in cats experimentally infected with feline herpesvirus type-1. *Am J Vet Res.* 2011;**72**(1):85–95. doi: 10.2460/ajvr.72.1.85.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 80.1 Herpesviral-associated keratitis. Vascular and fibrosing keratitis associated with herpesviral infection.

Figure 80.2 Herpesviral-associated keratitis. Typical presentation of keratitis associated with herpesviral infection.

Figure 80.3 Herpesviral-associated keratitis. Vascular and fibrosing keratitis associated with herpesviral infection. Note periocular discharge and rhinitis, also likely associated with active herpesviral infection in this case.

Figure 80.4 Herpesviral-associated keratitis. Chronic vascular keratitis in an adult feline patient associated with herpesviral infection.

CHAPTER 81
CANINE MULTIFOCAL IMMUNE-MEDIATED PUNCTATE KERATITIS

PRESENTATION

Multifocal immune-mediated punctate keratitis describes a canine condition that results in multiple, sites of discrete corneal ulceration and which is suspected to be immune-mediated in etiology. Lesions are typically oval to circular, well-defined, and may be associated with arborizing vascular response. Secondary infection may result in rapid progression of these lesions. Commonly affected breeds include the Shetland Sheepdog and Dachshund.

DIAGNOSIS AND TREATMENT

The diagnosis of immune-mediated punctate keratitis is made based on clinical findings. Ulcerative keratitis, thought to be immune-mediated in origin, should be managed with a cautious combination of topical (+/− systemic) antimicrobial as well as anti-inflammatory therapy. Appropriate anti-inflammatory agents may include glucocorticoids and/or cyclosporine. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of topical cyclosporine and tacrolimus include periocular dermatitis and alopecia. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. The potential for acute clinical deterioration is initially high and patients should be frequently monitored until corneal inflammation is adequately controlled. The prognosis for controlling this disease is generally good; however, many cases require long-term, low-dose maintenance therapy in order to prevent a recurrence of symptoms.

SELECTED REFERENCES

Andrew SE. Immune-mediated canine and feline keratitis. *Vet Clin North Am Small Anim Pract.* 2008;**38**(2):269–290.

Clerc, C. and Jegou, J. Superficial punctuate keratitis. *Canine Pract.* 1996;**21**, 6–11.

Dice PF. Primary corneal disease in the dog and cat. *Vet Clin North Am Small Anim Pract.* 1980;**10**(2):339–356.

Peiffer RL Jr. Ocular immunology and mechanisms of ocular inflammation. *Vet Clin North Am Small Anim Pract.* 1980;**10**(2):281–302.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

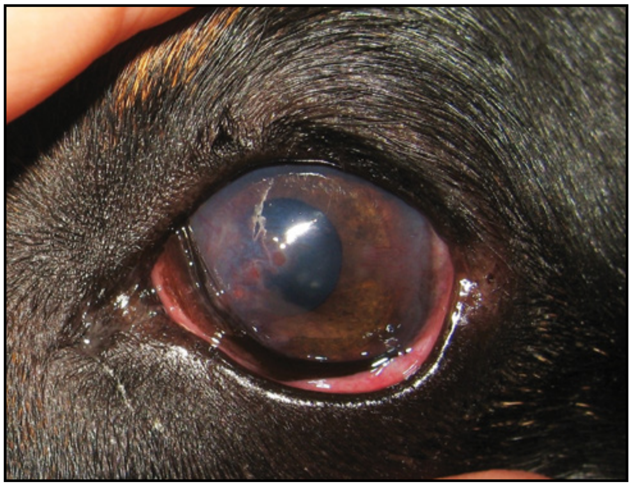


Figure 81.1 Canine multifocal immune-mediated punctate keratitis. Clinical presentation associated with canine multifocal immune-mediated punctate keratitis.

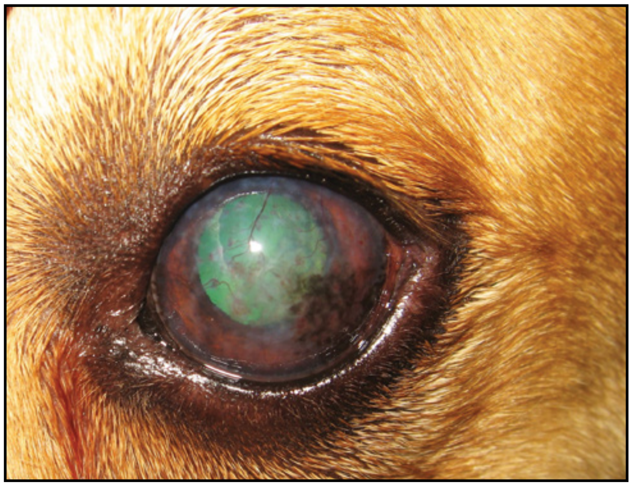


Figure 81.2 Canine multifocal immune-mediated punctate keratitis. Note the associated pigmentary keratitis, suggesting chronic disease in this case.

Figure 81.3 Canine multifocal immune-mediated punctate keratitis. Note the multifocal, round, punctate areas of fibrosis, likely representing a patient that is well controlled on anti-inflammatory medications.

Figure 81.4 Canine multifocal immune-mediated punctate keratitis. Vascular keratitis in this case suggests a more acute presentation demonstrating active disease.

CHAPTER 82
ENDOTHELIITIS

PRESENTATION

Infection with Canine adenovirus type-1 (CAV-1) and/or vaccination with modified live CAV-1 strains and/or anterior uveitis may be associated with endotheliitis (see also vaccine-associated uveitis). Ocular symptoms may include the presence of aqueous flare, pupillary miosis, and/or corneal edema (sometimes described as "blue eye") secondary to endothelial cell dysfunction. Potential sequella include bullous keratopathy, ulcerative keratitis, and/or secondary glaucoma. The Afghan hound has been described as being at increased risk.

DIAGNOSIS AND TREATMENT

The diagnosis of endotheliitis is made based on clinical findings in conjunction with history (including that of recent vaccination), routine CBC/chemistry analysis, and/or testing for the presence of infectious organisms. Treatment typically encompasses the administration of topical and systemic (steroidal) anti-inflammatory therapy (unless contra-indicated). In many cases, symptoms will resolve if appropriately treated early in the course of the disease. Secondary pathologies including corneal ulceration and/or glaucoma should be managed as appropriate if present (see also Secondary glaucoma). Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Aguirre G, Carmichael L, Bistner S. Corneal endothelium in viral induced anterior uveitis. Ultrastructural changes following canine adenovirus type 1 infection. *Arch Ophthalmol.* 1975;**93**(3):219–224.

Andrew SE. Immune-mediated canine and feline keratitis. *Vet Clin North Am Small Anim Pract.* 2008;**38**(2):269–290.

Curtis R and Barnett KC. Canine adenovirus-induced ocular lesions in the Afghan hound. *Cornell Vet.* 1981;**71**(1):85–95.

Curtis R and Barnett KC. The 'blue eye' phenomenon. *Vet Rec.* 1983;**112**(15):347–353.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 82.1 Endotheliitis. Clinical presentation associated with endotheliitis secondary to a vaccine reaction in a young canine.

Figure 82.2 Endotheliitis. Mild diffuse corneal edema associated with endotheliitis secondary to a vaccine reaction.

Figure 82.3 Endotheliitis. Typical clinical appearance of "blue-eye", corneal edema associated with endotheliitis.

Figure 82.4 Endotheliitis. Note the associated conjunctival hyperemia, suggesting more significant ocular inflammation.

CHAPTER 83
BULLOUS KERATOPATHY

PRESENTATION

Bullous keratopathy describes an acute decompensation of corneal stromal tissue. Severe, acute, focal, or generalized corneal edema may lead to the development of one or more large, unstable cornea bullae (see also Keratomalacia). Changes may develop extremely rapidly (typically within 24–48 hours), coalesce and/or rupture. Clinically, this condition appears as grossly distorted edematous corneal tissue, which typically bulges forward. Secondary bacterial or fungal keratitis may complicate bullous keratopathy. This process is mediated by proteolytic enzymes which may derive from corneal cells, inflammatory cells and/or bacterial pathogens and may or may not be associated with preexisting corneal inflammation and/or ulceration. Bullous keratopathy may occur in canine and feline species as well as any breed or crossed-breed; however, brachycephalic animals are frequently affected.

DIAGNOSIS AND TREATMENT

Bullous keratopathy is diagnosed based on clinical findings. Treatment comprises the stabilization of the metalloprotease enzymes which mediate this process. Appropriate topical therapeutic agents include serum (fresh or frozen), tetracyclines, EDTA solution, and/or N-acetyl cysteine. Topical anti-collagenases should be applied frequently. The use of corticosteroids is generally contraindicated. Secondary bacterial infection may be treated using appropriate antimicrobials. In severe cases, or those associated with existing or impending corneal rupture, surgical resection of diseased tissue and/or tectonic corneal grafting procedures may be indicated. The use of an Elizabethan collar will prevent self-trauma during the healing process.

SELECTED REFERENCES

Cooley PL, Dice PF 2nd. Corneal dystrophy in the dog and cat. *Vet Clin North Am Small Anim Pract.* 1990;**20**(3):681–692. Review.

Moore PA. Feline corneal disease. *Clin Tech Small Anim Pract.* 2005;**20**(2):83–93. Review.

Ollivier FJ, Gilger BC, Barrie KP, Kallberg ME, Plummer CE, O'Reilly S, Gelatt KN, Brooks DE. Proteinases of the cornea and preocular tear film. *Vet Ophthalmol.* 2007;**10**(4):199-206. Review.

Pattullo K. Acute bullous keratopathy in a domestic shorthair. *Can Vet J.* 2008;49(2):187–189.

Pederson SL, Pizzirani S, Andrew SE, Pate DO, Stine JM, Michau TM. Use of a nictitating membrane flap for treatment of feline acute corneal hydrops-21 eyes. *Vet Ophthalmol.* 2016;**19** Suppl 1:61–68.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 83.1 Bullous keratopathy. Clinical presentation associated with acute bullous keratopathy.

Figure 83.2 Bullous keratopathy. Bullous lesions as a result of keratitis and uncontrolled protease-mediated tissue destruction.

Figure 83.3 Bullous keratopathy. Severe, coalescing bullous keratopathy affecting most of the corneal surface.

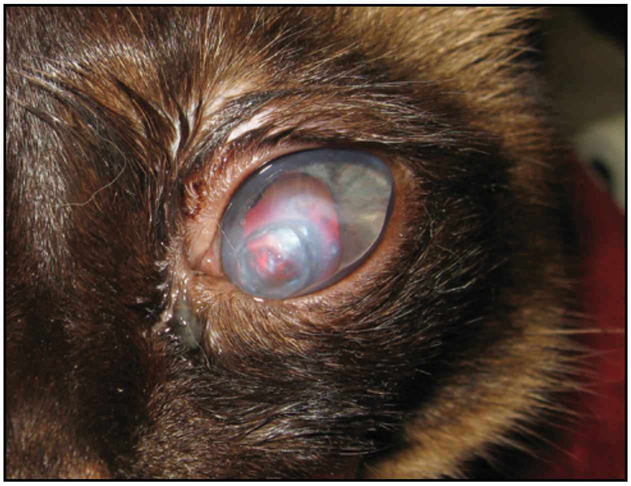


Figure 83.4 Bullous keratopathy. Focal bullous keratopathy demonstrating one large, unstable corneal bulla.

CHAPTER 84
FELINE CORNEAL SEQUESTRUM

PRESENTATION

The feline corneal sequestrum represents a localized region of corneal necrosis and associated inflammation. Clinically, corneal sequestrums appear as tan to brown region of corneal discoloration. Lesions may vary significantly in both size and depth and may or may not be associated with concurrent ulceration. The condition may be unilateral or bilateral. Associated discomfort may be manifested as blepharospasm and/or ocular discharge. Chronic preexisting keratitis, corneal ulceration, the performing of inappropriate "grid keratotomies" and/or the presence of FHV-1 as well as breed predispositions are potential contributory factors. Commonly affected breeds include the Persian and Himalayan.

DIAGNOSIS AND TREATMENT

The treatment of choice for corneal sequestrums is excision via surgical keratectomy, facilitated by operating microscopy. The size and depth of keratectomy is determined by the extent of the lesion and tectonic corneal grafting may or may not additionally be warranted following the removal of diseased tissue. Routine topical and/or systemic antimicrobials as well as topical and/or systemic antivirals may or may not be indicated on a case-by-case basis. The routine use of an Elizabethan collar postoperatively will prevent self-trauma.

SELECTED REFERENCES

Cullen CL, Wadowska DW, Singh A, Melekhovets Y. Ultrastructural findings in feline corneal sequestra. *Vet Ophthalmol.* 2005;**8**(5):295–303.

Featherstone HJ, Sansom J. Feline corneal sequestra: a review of 64 cases (80 eyes) from 1993 to 2000. *Vet Ophthalmol.* 2004;**7**(4):213–227.

Grahn BH, Sisler S, Storey E. Qualitative tear film and conjunctival goblet cell assessment of cats with corneal sequestra. *Vet Ophthalmol.* 2005;**8**(3):167–170.

Laguna F, Leiva M, Costa D, Lacerda R, Peña Gimenez T. **Corneal** grafting for the treatment of **feline corneal sequestrum**: a retrospective study of 18 eyes (13 **cats**). *Vet Ophthalmol.* 2015;**18**(4):291–296.

Volopich S, Benetka V, Schwendenwein I, Möstl K, Sommerfeld-Stur I, Nell B. Cytologic findings, and feline herpesvirus DNA and Chlamydophila felis antigen detection rates in normal cats and cats with conjunctival and corneal lesions. *Vet Ophthalmol.* 2005;**8**(1):25–32.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 84.1 Feline corneal sequestrum. Clinical presentation associated with a feline corneal sequestrum. Note significant fibrovascular keratitis in this case.

Figure 84.2 Feline corneal sequestrum. Note the diffuse nature of this lesion, which affects nearly the entire corneal surface.

Figure 84.3 Feline corneal sequestrum. Focal, axial, and superficial feline corneal sequestrum.

Figure 84.4 Feline corneal sequestrum. Typical presentation associated with a feline corneal sequestrum in a Persian cat.

CHAPTER 85
PIGMENTARY KERATITIS

PRESENTATION

Pigmentary keratitis describes a relatively common presentation comprising the progressive deposition of pigment on the corneal and associated conjunctival surface(s). Clinically, this process is apparent as a smooth to irregular brown opacification, affecting a variable surface area. Concurrent inflammatory keratitis, marked by neovascularization may additionally be present. Severe cases may exhibit complete pigmentary keratitis resulting in significant visual impairment or even functional blindness. Contributing factors may include exposure as a result of conformational lagophthalmos, chronic or severe keratitis, quantitative and/or qualitative tear film disorders, trichiasis, and/or genetic factors. Commonly affected breeds include the Pug, The Pekinese, Shih Tzu, and Lhasa Aphso.

DIAGNOSIS AND TREATMENT

Treatment comprises addressing any underlying disorders including corneal inflammation and/or tear film disorders, typically using, topical corticosteroids, cyclosporine, tacrolimus and/or tear film replacements/stabilizers, Long-term medication may be required in order to minimize progression of disease. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of topical cyclosporine and tacrolimus include periocular dermatitis and alopecia. Additionally, increased corneal protection and tear film coverage via reduction of the palpebral fissure through limited medial and/or lateral canthoplasty may help to palliate progression of disease. Multiple techniques to decrease the size of palpebral fissure have been described, including the generally appropriate Roberts/Jensen medial pocket canthoplasty. Although corneal pigment is readily removed via keratectomy, the likelihood of its recurrence is high. The judicious application of cryotherapy or B-radiation may help to limit recurrence in selected cases.

SELECTED REFERENCES

Azoulay T. Adjunctive cryotherapy for **pigmentary keratitis** in **dogs**: a study of 16 corneas. *Vet Ophthalmol.* 2014;**17**(4):241–249.

Bedford PG. Technique of lateral canthoplasty for the correction of macropalpebral fissure in the dog. *J Small Anim Pract.* 1998;**39**(3):117–120.

Labelle AL, Dresser CB, Hamor RE, Allender MC, Disney JL. Characteristics of, prevalence of, and risk factors for corneal pigmentation (pigmentary keratopathy) in Pugs. *J Am Vet Med Assoc.* 2013;**243**(5):667–674.

Maini S, Everson R, Dawson C, Chang YM, Hartley C, Sanchez RF. **Pigmentary keratitis** in pugs in the United Kingdom: prevalence and associated features. *BMC Vet Res.* 2019;**15**(1):384.

van der Woerdt A. Adnexal surgery in dogs and cats. *Vet Ophthalmol.* 2004;**7**(5):284–290. Review.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 85.1 Pigmentary keratitis. Clinical presentation associated with mild pigmentary keratitis.

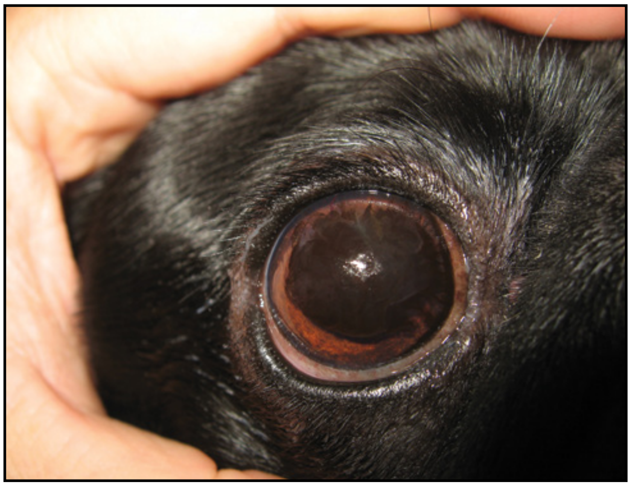


Figure 85.2 Pigmentary keratitis. Dorsal pigment associated with conformational lagophthalmos and poor tear film quality and distribution.

Figure 85.3 Pigmentary keratitis. Typical pigment distribution, most commonly progressing from medial to lateral.

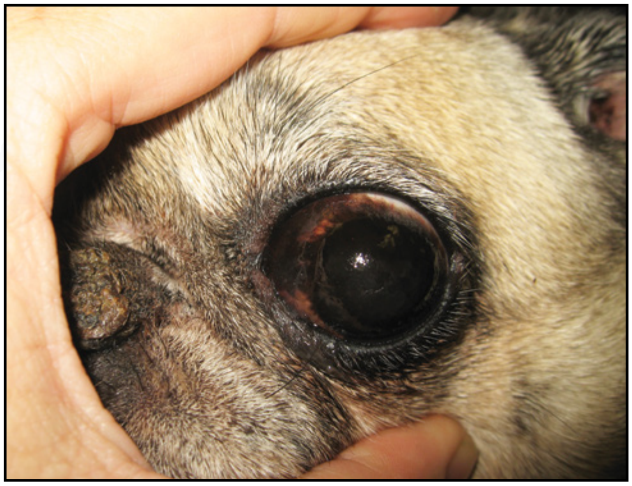


Figure 85.4 Pigmentary keratitis. Severe, generalized pigmentary keratitis in a pug.

CHAPTER 86
CORNEAL HEMORRHAGE

PRESENTATION

Intracorneal hemorrhage may occasionally result in association with the presence of corneal blood vessels. Clinically, this condition presents as variable red to tan discoloration, arising in a fan-like pattern from an adjacent corneal vessel, occurring at any depth within the cornea and typically affecting one eye only.

DIAGNOSIS AND TREATMENT

The diagnosis of intracorneal hemorrhage is made based on clinical findings. Most cases of discrete corneal hemorrhage will resolve spontaneously. The presence of underlying coagulopathy/vasculopathy, metabolic disease, and/or hypertension should be ruled out in affected cases. Neoplastic vasculopathies represent the most important differential diagnosis (see also Corneoscleral hemangioma/hemangiosarcoma). Additionally, the abnormal presence of blood vessels within the cornea should be evaluated, specifically identifying and treating associated surface or intraocular disease. Topical steroidal or nonsteroidal agents will help to speed the resorption of blood; however, discoloration may be present for months to years and some degree or permanent opacification may remain at the site of hemorrhage following its resorption, particularly in cases of deep stromal hemorrhage.

SELECTED REFERENCES

Matas M, Donaldson D, Newton RJ. **Intracorneal hemorrhage** in 19 **dogs** (22 eyes) from 2000 to 2010: a retrospective study. *Vet Ophthalmol.* 2012;**15**(2):86–91.

McDonnell PJ, Green WR, Stevens RE, Bargeron CB, Riquelme JL. Blood staining of the cornea. Light microscopic and ultrastructural features. *Ophthalmology.* 1985;**92**(12):1668–1674.

Swanson JF. Ocular manifestations of systemic disease in the dog and cat. Recent developments. *Vet Clin North Am Small Anim Pract.* 1990;**20**(3):849–867.

Violette NP, Ledbetter EC. Intracorneal stromal hemorrhage in dogs and its associations with ocular and systemic disease: 39 cases. *Vet Ophthalmol.* 2017;**20**(1):27–33.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 86.1 Corneal hemorrhage. Clinical presentation associated with the presence of intracorneal hemorrhage.

Figure 86.2 Corneal hemorrhage. Note discrete nature of hemorrhage in this case.

Figure 86.3 Corneal hemorrhage. Note the fanlike pattern of hemorrhage associated with several adjacent corneal vessels.

Figure 86.4 Corneal hemorrhage. Note diffusion of hemorrhage around adjacent corneal vessel.

CHAPTER 87
CORNEAL ABSCESSATION

PRESENTATION

A corneal abscess may form following a breakdown in the epithelial (or rarely endothelial) barrier, allowing bacterial and/or fungal organisms entry into the corneal stroma, resulting in the formation of a nidus of infectious and/or inflammatory material. Clinically, this condition presents as a variably sized, focal tan to yellow area of stromal corneal discoloration, which is typically associated with intense discomfort as well as an aggressive vascular response. Fluorescein staining frequently fails to demonstrate concurrent corneal ulceration. Discomfort may be manifested as blepharospasm and/or ocular discharge.

DIAGNOSIS AND TREATMENT

The diagnosis of corneal abscessation is made based on clinical findings. Treatment includes the exposure and removal of the infective focus via debridement/curettage as well as the institution of aggressive systemic an/or topical antimicrobial therapy. Intact epithelial surfaces and/or significant accumulations of biologic material are significant impediments to antimicrobial penetration and healing. Ideally, therapy should be based on cytological interpretation and gram's staining as well as the subsequent culture and sensitivity testing of microbial samples. Concurrent topical and/or systemic (nonsteroidal) anti-inflammatory therapy may be indicated in selected cases. Affected corneas should be monitored closely and repeatedly during the healing process. The routine use of an Elizabethan collar postoperatively will prevent self-trauma.

SELECTED REFERENCES

Bussieres M, Krohne SG, Stiles J, Townsend WM. The use of porcine small intestinal submucosa for the repair of full-thickness **corneal** defects in **dogs**, cats and horses. *Vet Ophthalmol.* 2004;**7**(5):352–359.

Hewitt JS, Allbaugh RA, Kenne DE, Sebbag L. Prevalence and antibiotic susceptibility of **bacterial** isolates from **dogs** with ulcerative **keratitis** in Midwestern United States. *Front Vet Sci.* 2020;**7**:583965.

Ollivier FJ. Bacterial corneal diseases in dogs and cats. *Clin Tech Small Anim Pract.* 2003;**18**(3):193–198.

Sherman A, Daniels JB, Wilkie DA, Lutz E. Actinomyces bowdenii ulcerative keratitis in a **dog**. *Vet Ophthalmol.* 2013;**16**(5):386–391.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 87.1 Corneal abscessation. Clinical presentation associated with corneal abscessation.

Figure 87.2 Corneal abscessation. Note significant vascular infiltration.

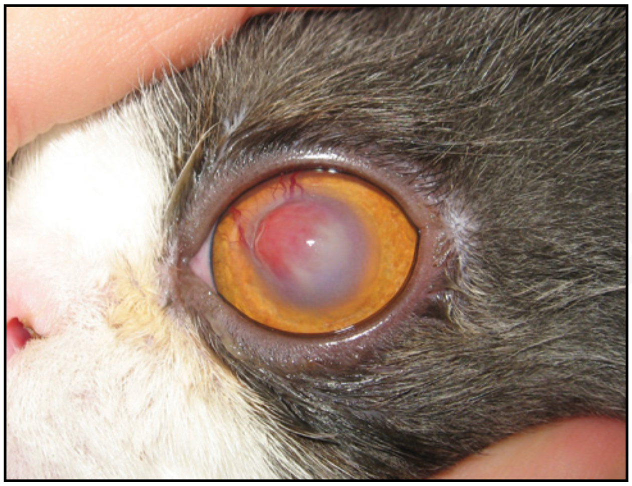


Figure 87.3 Corneal abscessation. Resolving corneal abscessation with aggressive vascular response.

Figure 87.4 Corneal abscessation. Note associated pigmentary keratitis suggesting chronic disease.

CHAPTER 88
CORNEOSCLERAL LACERATION

PRESENTATION

Traumatic corneal laceration is relatively commonly encountered and constitutes an ocular emergency. Cat-claw injuries are frequently the cause of these injuries. Corneal laceration is typically marked by extreme discomfort, blepharospasm, ocular discharge, hemorrhage, the accumulation of fibrin/mucous at the wound site, anterior chamber shallowing or collapse, and/or the prolapse of uveal tissue.

DIAGNOSIS AND TREATMENT

The diagnosis of corneoscleral laceration is made based on clinical findings in conjunction with clinical history. Lacerated globes are tectonically fragile, and affected patients should be handled with extreme caution in order to minimize the risk of further intraocular pathology including iris prolapse and/or retinal detachment. The mechanical manipulation of tissue, including attempted debridement or "wiping away" of accumulated fibrin, prior to surgical repair is contraindicated. Systemic (steroidal or nonsteroidal) anti-inflammatory and/or antimicrobial therapy are generally appropriate. Surgical repair should be performed as soon as possible, with a variety of techniques described. The use of an Elizabethan collar will prevent self-trauma prior to referral and postoperatively. An important consideration is the potential that lenticular structures may also have been damaged. Failure to identify and address this potential complication will significantly increase the risk of potentially catastrophic, secondary pathology including glaucoma as a result of uncontrolled phacolastic uveitis (see also Phacolastic uveitis and FPTS).

SELECTED REFERENCES

Belknap EB. **Corneal** emergencies. *Top Companion Anim Med.* 2015;**30**(3):74–80.
Braus BK, Tichy A, Featherstone HJ, Renwick PW, Rhodes M, Heinrich CL. Outcome of phacoemulsification following **corneal** and lens **laceration** in cats and **dogs** (2000–2010). *Vet Ophthalmol.* 2017;**20**(1):4–10.
Giuliano EA. Feline ocular emergencies. *Clin Tech Small Anim Pract.* 2005;**20**(2):135–141.
Mandell DC. Ophthalmic emergencies. *Clin Tech Small Anim Pract.* 2000;**15**(2):94–100. Review.
Paulsen ME, Kass PH. Traumatic corneal laceration with associated lens capsule disruption: a retrospective study of 77 clinical cases from 1999 to 2009. *Vet Ophthalmol.* 2012;**15**(6):355–368.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 88.1 Corneoscleral laceration. Clinical presentation associated with an acute corneoscleral laceration. Pupillary dyscoria in association with iris prolapse is present.

Figure 88.2 Corneoscleral laceration. Note the hyphema in association with limbal iris prolapse.

Figure 88.3 Corneoscleral laceration. Iritis, as well as a triangular "flap" of lacerated corneal tissue, typical of a cat-claw injury, is present.

Figure 88.4 Corneoscleral laceration. Note the presence of intracameral hemorrhage and fibrin.

CHAPTER 89
SPONTANEOUS CHRONIC CORNEAL EPITHELIAL DEFECTS (SCCEDs)

PRESENTATION

Spontaneous chronic corneal epithelial defects (SCCEDs) are a relatively common cause of canine corneal pathology. These lesions are frequently described as "indolent" or "Boxer" ulcers. SCCEDs likely arise as a result of corneal micro-trauma in association with pre-existing structural and/or physiological corneal abnormalities. Clinically, SCCEDs present as chronic superficial corneal ulcerations, typically surrounded by a visible rim of poorly adherent epithelial tissue, which is easily under-run by fluorescein creating a "halo" effect following corneal staining. Significant associated corneal inflammation, vascularization, and/or granulation may develop in association with longstanding lesions. Discomfort is marked by blepharospasm and/or ocular discharge. Commonly affected breeds include the Boxer, Boston Terrier, French Bulldog, and Labrador Retriever.

DIAGNOSIS AND TREATMENT

The diagnosis of a SCCED is made based on its typical clinical appearance and staining pattern. SCCEDs are treated by physical debridement of both loose superficial epithelial tissue as well as the underlying stromal surface. Medical therapy alone is unlikely to result in corneal healing. Similarly, the placement of a "third eyelid flap" is unlikely to address underlying pathology and result in adequate healing. Debridement may be performed using sterile cotton-tipped applicators, a sterile spatula, a scalpel blade, or a diamond-tipped burr. A linear or punctate keratotomy may additionally be performed following corneal debridement. In challenging or refractory cases, a superficial keratectomy may ultimately be required. Routine topical anti-microbial therapy is indicated pending corneal healing and the postoperative use of an Elizabethan-collar will prevent self-trauma. Dogs with a history of SCCEDs are prone to the development of similar lesions within the contralateral eye.

SELECTED REFERENCES

Bentley E, Abrams GA, Covitz D, Cook CS, Fischer CA, Hacker D, Stuhr CM, Reid TW, Murphy CJ. Morphology and immunohistochemistry of spontaneous chronic corneal epithelial defects (SCCED) in dogs. *Invest Ophthalmol Vis Sci.* 2001;**42**(10):2262–2269.

Bentley E. Spontaneous chronic corneal epithelial defects in dogs: a review. *J Am Anim Hosp Assoc.* 2005;**41**(3):158–165. Review.

Boutin MP, Coutellier M, Ollivier FJ. Cotton-tip debridement, scalpel blade debridement, and superficial grid keratotomy for treatment of spontaneous chronic corneal epithelial defects (SCCED): A retrospective evaluation of 308 cases. *Vet Ophthalmol.* 2020;**23**(6):979–986.

Hung JH, Leidreiter K, White JS, Bernays ME. Clinical characteristics and treatment of spontaneous chronic corneal epithelial defects (SCCEDs) with **diamond burr** debridement. *Vet Ophthalmol.* 2020;**23**(4):764–769.

Levitt S, Osinchuk SC, Bauer B, Sandmeyer LS. Bacterial isolates of **indolent ulcers** in 43 **dogs**. *Vet Ophthalmol.* 2020;**23**(6):1009–1013.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 89.1 Spontaneous chronic corneal epithelial defects. A rim of poorly adherent epithelial tissue is noted to surround the lesion.

Figure 89.2 Spontaneous chronic corneal epithelial defects. Significant neovascularization/corneal granulation has developed in this case.

Figure 89.3 Spontaneous chronic corneal epithelial defects. Note associated corneal edema, conjunctivitis, epiphora, and mucoid ocular discharge.

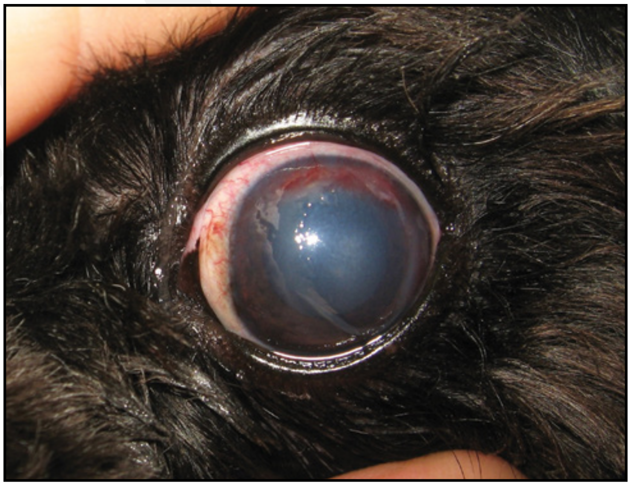


Figure 89.4 Spontaneous chronic corneal epithelial defects. Note rim of poorly adherent epithelial tissue and early vascular keratitis.

CHAPTER 90
STROMAL ULCERATIVE KERATITIS

PRESENTATION

Corneal ulceration may arise secondary to a multitude of etiologies including conformational abnormalities, tear-film deficiencies (quantitative and/or qualitative), neurological dysfunction, trauma, and/or microbial contamination. Stromal ulcers are identified as deep defects of variable size in the cornea, which stain positive with fluorescein dye and which may be associated with edema as well as a variable vascular response. Discomfort may be marked by blepharospasm and/or mucoid ocular discharge. Commonly affected breeds include the Shi Tzu, Pug, Lassa Apso, and Boston Terrier.

DIAGNOSIS AND TREATMENT

Bacterial keratitis should be treated using appropriate antimicrobial therapy. Ideally, treatment should be based on cytological interpretation and gram's staining as well as the subsequent culture and sensitivity testing of microbial samples. In cases of deep stromal ulceration, or those associated with existing or impending corneal rupture, surgical tectonic corneal grafting may be indicated. A variety of appropriate techniques have been described including the placement of a conjunctival pedicle graft (CPG), conjunctival island graft (CIG), or the advancement of a corneoscleral transposition (CCT), facilitated by operating microscopy. The use of corticosteroids is generally contraindicated in the management of stromal ulcerative keratitis. The use of an Elizabethan collar will prevent self-trauma during the healing process.

SELECTED REFERENCES

Gogova S, Leiva M, Ortillés Á, Lacerda RP, Seruca C, Laguna F, Crasta M, Ríos J, Peña MT. Corneoconjunctival transposition for the treatment of deep **stromal** to full-thickness **corneal** defects in **dogs**: A multicentric retrospective study of 100 cases (2012–2018). *Vet Ophthalmol*. 2020;**23**(3):450–459.

Jaksz M, Fischer MC, Fenollosa-Romero E, Busse C. Autologous **corneal graft** for the treatment of deep **corneal** defects in **dogs**: 15 cases (2014–2017). *J Small Anim Pract*. 2021;**62**(2):123–130.

Ollivier FJ. Bacterial corneal diseases in dogs and cats. *Clin Tech Small Anim Pract*. 2003;**18**(3):193–198.

Scagliotti RH. Tarsoconjunctival island graft for the treatment of deep corneal ulcers, desmetocoeles, and perforations in 35 dogs and 6 cats. *Semin Vet Med Surg (Small Anim)*. 1988;**3**(1):69–76.

Wilkie DA and Whittaker C. Surgery of the cornea. *Vet Clin North Am Small Anim Pract*. 1997;**27**(5):1067–1107.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 90.1 Stromal ulcerative keratitis. Clinical presentation associated with malacic stromal ulcerative keratitis. Note significant vascular response.

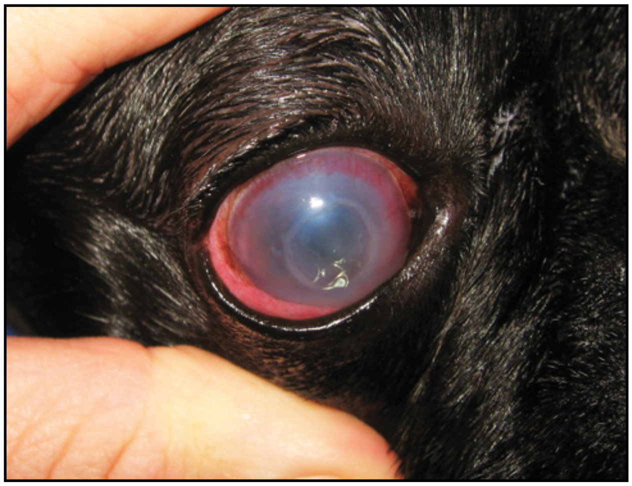


Figure 90.2 Stromal ulcerative keratitis. Note early vascular response and associated corneal edema.

Figure 90.3 Stromal ulcerative keratitis. Clinical presentation associated with deep stromal ulcerative keratitis. Surgical repair is indicated due to the depth of the lesion.

Figure 90.4 Stromal ulcerative keratitis. Note the deep stromal ulcerative keratitis and the lack of vascular response. Surgical repair is indicated.

CHAPTER 91
DESCEMETOCELE

PRESENTATION

A descemetocele describes a deep corneal ulcer in which the overlying epithelium and stroma are no longer present so that only descemet's membrane prevents globe rupture. This thin layer may be appreciated to be bulging forward in the center of the ulcerative lesion. The presence of a descemetocele is typically marked by discomfort, blepharospasm, and/or ocular discharge.

DIAGNOSIS AND TREATMENT

A descmetocele may be diagnosed as a corneal ulcer, the center of which fails to retain fluorescein stain. Copiously irrigating deep corneal lesions following fluorescein staining will differentiate deep stromal ulcers form descemetocele since stain will not be rinsed away from exposed corneal stream in the absence of a descemetocele. Affected globes are tectonically fragile and affected patients should be handled with extreme caution in order to minimize the risk of globe rupture. Covering a descemetocele with a "third eyelid flap" does not provide significant wound support and is generally contraindicated. Surgical repair should be performed as soon as possible. A variety of appropriate techniques have been described including the placement of a conjunctival pedicle graft (CPG), conjunctival island graft (CIG), or the advancement of a corneoscleral transposition (CCT), facilitated by operating microscopy. The use if an Elizabethan collar will prevent self-trauma prior to referral and post-operatively.

SELECTED REFERENCES

Gogova S, Leiva M, Ortillés Á, Lacerda RP, Seruca C, Laguna F, Crasta M, Ríos J, Peña MT. Corneoconjunctival transposition for the treatment of deep stromal to full-thickness corneal defects in **dogs**: A multicentric retrospective study of 100 cases (2012–2018). *Vet Ophthalmol*. 2020;**23**(3):450–459.

Hansen PA and Guandalini A. A retrospective study of 30 cases of frozen lamellar corneal graft in dogs and cats. *Vet Ophthalmol*. 1999;**2**(4):233–241.

Jaksz M, Fischer MC, Fenollosa-Romero E, Busse C. Autologous corneal graft for the treatment of deep corneal defects in **dogs**: 15 cases (2014–2017). *J Small Anim Pract*. 2021;**62**(2):123–130.

Mandell DC. Ophthalmic emergencies. *Clin Tech Small Anim Pract*. 2000;**15**(2):94–100.

Wilkie DA and Whittaker C. Surgery of the cornea. *Vet Clin North Am Small Anim Pract*. 1997;**27**(5):1067–1107.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 91.1 Descemetocele. Clinical presentation associated with a corneal descemetocele.

Figure 91.2 Descemetocele. Note the surrounding corneal fibrosis, but lack of vascular response.

Figure 91.3 Descemetocele. Note the surrounding mineral degeneration, which may have sloughed leaving a descemetocele.

Figure 91.4 Descemetocele. Note the surrounding malacic corneal tissue and significant vascular response.

CHAPTER 92
KERATOMALACIA ("MELTING ULCER")

PRESENTATION

Corneal ulceration may arise secondary to a multitude of etiologies including conformational abnormalities, tear-film deficiencies (quantitative and/or qualitative), neurological dysfunction, trauma and/or microbial contamination. Tissue dissolution through the action of enzymatic proteases is a normal part of corneal metabolism as well as the healing and remodeling process; however, uncontrolled lysis or "melting" of corneal tissue as a result of excessive protease activity has the potential to result in significant pathology (*see also Bullous keratopathy). Excessive enzymatic activity may stem from corneal or inflammatory cells as well pathological microorganisms. Affected tissue becomes edematous and soft, manifesting as corneal thickening and gray discoloration. An associated corneal vascular response may additionally be present. Keratomalacia is frequency complicated by severe secondary bacterial infection. Discomfort may be marked by blepharospasm and/or mucoid ocular discharge. Commonly affected breeds include the Shi Tzu, Pug, Lassa Apso, and Boston Terrier.

DIAGNOSIS AND TREATMENT

The diagnosis of keratomalacia is made based on clinical findings. Treatment comprises the stabilization of the metalloprotease enzymes which mediate this process. Appropriate topical therapeutic agents include serum (fresh or frozen), tetracyclines, EDTA solution, and/or N-acetyl cysteine. Anti-collagenases should be applied frequently. Bacterial keratitis should be treated using appropriate antimicrobials. Ideally, therapy should be based on cytological interpretation and gram's staining as well as the subsequent culture and sensitivity testing of microbial samples. In severe cases, or those associated with existing or impending corneal rupture, surgical resection of diseased tissue and/or tectonic corneal grafting procedures may be indicated. The use of corticosteroids is generally contraindicated. The use of an Elizabethan collar will prevent self-trauma during the healing process.

SELECTED REFERENCES

Guyonnet A, Desquilbet L, Faure J, Bourguet A, Donzel E, Chahory S. Outcome of medical therapy for keratomalacia in **dogs**. *J Small Anim Pract*. 2020;**61**(4):253–258.

Kimmitt BA, Moore GE, Stiles J. Comparison of the efficacy of various concentrations and combinations of serum, ethylenediaminetetraacetic acid, tetracycline, doxycycline, minocycline, and N-acetylcysteine for inhibition of collagenase activity in an in vitro **corneal**-degradation model. *Am J Vet Res*. 2018;**79**(5):555–561.

Ollivier FJ, Gilger BC, Barrie KP, Kallberg ME, Plummer CE, O'Reilly S, Gelatt KN, Brooks DE. Proteinases of the cornea and preocular tear film. *Vet Ophthalmol*. 2007;**10**(4):199–206.

Tsvetanova A, Powell RM, Tsvetanov KA, Smith KM, Gould DJ. **Melting corneal** ulcers (keratomalacia) in **dogs**: a 5-year clinical and microbiological study (2014–2018). *Vet Ophthalmol*. 2021;**24**(3):265–278.

Vanore M, Chahory S, Payen G, Clerc B. Surgical repair of deep **melting** ulcers with porcine small intestinal submucosa (SIS) graft in **dogs** and cats. *Vet Ophthalmol*. 2007;**10**(2):93–99.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 92.1 Keratomalacia. Clinical presentation associated with corneal ulceration and keratomalacia. Note significant early vascular response.

Figure 92.2 Keratomalacia. Note that most of the cornea appears to be affected by tissue lysis, edema, and bacterial infection in this case.

Figure 92.3 Keratomalacia. Note the common presentation in a brachycephalic patient with significant conformational lagophthalmos.

Figure 92.4 Keratomalacia. Note associated discharge, inflammation, and probable secondary bacterial infection.

CHAPTER 93
CORNEAL FOREIGN BODY

PRESENTATION

The potential presence of foreign material should always be considered when evaluating a painful eye. Foreign material is commonly plant-based in origin. Foreign bodies may affect either the corneo-scleral surface (where they may become deeply embedded as a result of enzymatic activity) or the bulbar fornix behind the eyelid or third eyelid. Ocular foreign bodies typically result in significant blepharospasm, chemosis, conjunctivitis, and/or ocular discharge.

DIAGNOSIS AND TREATMENT

The diagnosis of a corneal foreign body is made based on clinical findings. The application of topical anesthesia may facilitate adequate examination; however, painful cases may require sedation or even anesthesia in order to facilitate thorough examination. Appropriate treatment comprises the removal of foreign material. Care should be taken to ensure complete removal, to prevent the loss of biologic material into the anterior chamber (which may lead to endophthalmitis) and also to medically manage and/or tectonically repair any defects, which remain following foreign body removal. The postoperative use of a protective Elizabethan collar will prevention self-trauma until corneal healing is complete.

SELECTED REFERENCES

Cullen CL and Grahn BH. Diagnostic ophthalmology. Right corneal foreign body, secondary ulcerative keratitis, and anterior uveitis. *Can Vet J*. 2005;**46**(11):1054–1055.

Dowler KK, Scott EM, Teixeira LBC, Vallone LV. Cactus-induced keratoconjunctivitis in Texas: a case series of three **dogs** and one cat. *Vet Ophthalmol*. 2020;**23**(2):374–385.

Gelatt KN. Organic corneal foreign bodies in the dog. *Vet Med Small Anim Clin*. 1974;**69**(11):1423–1428.

Rebhun WC. Conjunctival and corneal foreign bodies. *Vet Med Small Anim Clin*. 1973;**68**(8):874–877.

Tetas Pont R, Matas Riera M, Newton R, Donaldson D. **Corneal** and anterior segment **foreign body** trauma in **dogs**: a review of 218 cases. *Vet Ophthalmol*. 2016;**19**(5):386–397.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 93.1 Corneal foreign body. Clinical presentation associated with the presence of a plant-based corneal foreign body.

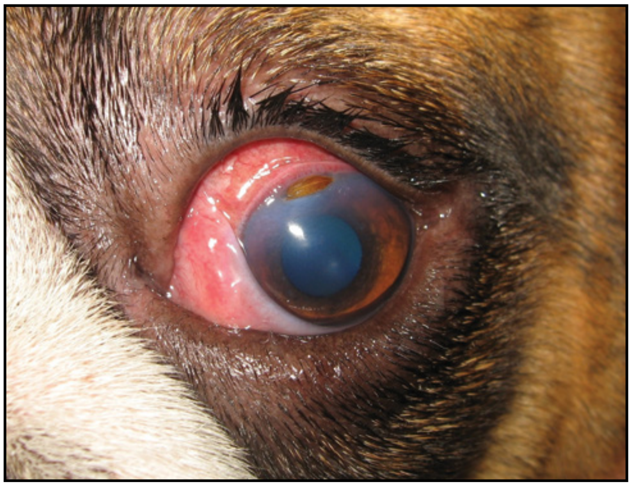


Figure 93.2 Corneal foreign body. Note that this plant-based corneal foreign body has become superficially embedded in the dorsal cornea.

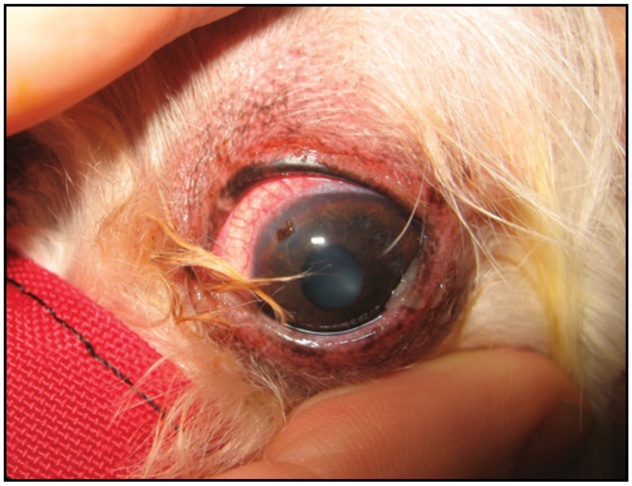


Figure 93.3 Corneal foreign body. Note the periocular erythema associated with self-trauma.

Figure 93.4 Corneal foreign body. Note the presence of a vascular response, suggesting a more chronic corneal foreign body.

CHAPTER 94
CORNEAL PERFORATION

PRESENTATION

Corneal perforation as a result of trauma, severe, rapidly progressive corneal ulceration or a ruptured descemetocele constitutes an ocular emergency. Corneal perforation is typically marked by extreme discomfort, blepharospasm, discharge, hemorrhage, anterior chamber shallowing or collapse, and the accumulation of fibrin/mucous at the wound site.

DIAGNOSIS AND TREATMENT

Perforated globes are tectonically fragile and affected patients should be handled with extreme caution to minimize the risk of further intraocular pathology including iris prolapse and/or retinal detachment. The mechanical manipulation of tissue, including attempted debridement, is contraindicated until surgical repair can be performed. Covering a perforated cornea with a “third eyelid flap” does not provide significant wound support and is generally contraindicated. Surgical repair should be performed as soon as possible, with a variety of techniques described. Systemic (steroidal or nonsteroidal) anti-inflammatory and/or antimicrobial therapy are generally indicated. The use if an Elizabethan collar will prevent self-trauma prior to referral and postoperatively.

SELECTED REFERENCES

Belknap EB. **Corneal** Emergencies. *Top Companion Anim Med*. 2015;**30**(3):74–80.

Gogova S, Leiva M, Ortillés Á, Lacerda RP, Seruca C, Laguna F, Crasta M, Ríos J, Peña MT. Corneoconjunctival transposition for the treatment of deep stromal to full-thickness **corneal** defects in **dogs**: a multicentric retrospective study of 100 cases (2012–2018). *Vet Ophthalmol*. 2020;**23**(3):450–459.

Lacerda RP, Peña Gimenez MT, Laguna F, Costa D, Ríos J, Leiva M. **Corneal** grafting for the treatment of full-thickness **corneal** defects in **dogs**: a review of 50 cases. *Vet Ophthalmol*. 2017;**20**(3):222–231.

Scagliotti RH. Tarsoconjunctival island graft for the treatment of deep corneal ulcers, desmetocoeles, and perforations in 35 dogs and 6 cats. *Semin Vet Med Surg (Small Anim)*. 1988;**3**(1):69–76.

Wilkie DA and Whittaker C. Surgery of the cornea. *Vet Clin North Am Small Anim Pract*. 1997;**27**(5):1067–1107.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 94.1 Corneal perforation. Clinical presentation associated with corneal perforation. An organized fibrin "plug" is visible at the site of the lesion.

Figure 94.2 Corneal perforation. Large area of perforation with a large fibrin "plug" at the site of the lesion.

Figure 94.3 Corneal perforation. Note associated hemorrhage and hyphema.

Figure 94.4 Corneal perforation. Note that iris prolapse is visible within the fibrin "plug" at the site of the lesion.

CHAPTER 95
EPITHELIAL INCLUSION CYSTS

PRESENTATION

Occasionally, following corneal healing, epithelial cells may become trapped beneath the epithelial surface and organize into small cysts which gradually enlarge as they accumulate proteinaceous debris. Clinically, these lesions appear as one or more focal, relatively well-circumscribed, smooth, raised yellow to tan swellings, protruding from the corneal surface at the site of prior trauma. A mild superficial vascular response is typically associated with the presence of inclusion cysts. These lesions do not however, result in significant discomfort.

DIAGNOSIS AND TREATMENT

Diagnosis of epithelial inclusion cysts is made based on clinical findings. If problematic or significantly progressive, these lesions may be removed either by surgical keratectomy or more simply by palliative CO_2 laser ablation. Postoperative topical antimicrobial and anti-inflammatory therapy is indicated and the use of a protective Elizabethan collar will prevent self-trauma during corneal healing.

SELECTED REFERENCES

Cassagnes C, Cognard SA, Nicolier A, Cazalot G, Dossin E, Durieux P, Goulle F, Michel J, Pilorge P, Verneuil M, Isard PF, Mathieson I, Dulaurent T. Corneal **epithelial inclusion cysts** in 12 **dogs** (13 eyes) from 2010 to 2019: a multicentric retrospective study. *Vet Ophthalmol*. 2020;**23**(5):856–862.

Choi US, Labelle P, Kim S, Kim J, Cha J, Lee KC, Lee HB, Kim NS, Kim MS. Successful treatment of an unusually large corneal epithelial inclusion cyst using equine amniotic membrane in a dog. *Vet Ophthalmol*. 2010;**13**(2):122–125.

Cullen CL and Grahn BH. Diagnostic ophthalmology. Epithelial inclusion cyst of the right cornea. *Can Vet J*. 2001;**42**(3):230–231.

Simonazzi B, Castania M, Bosco V, Giudice C, Rondena M. A case of multiple unilateral corneal epithelial inclusion cysts in a dog. *J Small Anim Pract*. 2009;**50**(7):373–376.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 95.1 Epithelial inclusion cysts. Clinical presentation associated with the presence of a corneal epithelial inclusion cyst at the site of a previous trauma.

Figure 95.2 Epithelial inclusion cysts. Note that this large epithelial inclusion cyst demonstrates an associated mild vascular response.

Figure 95.3 Epithelial inclusion cysts. Note that this small, focal epithelial inclusion cyst causes no significant discomfort.

Figure 95.4 Epithelial inclusion cysts. Note the presence of a mild vascular response associated with this multifocal epithelial inclusion cyst.

CHAPTER 96
FUNGAL KERATITIS

PRESENTATION

Fungal keratitis is uncommon in dogs and cats. Environmental contamination, commensal overgrowth, epithelial trauma, and/or immunosuppression (notably using antibiotic/corticosteroid combinations) may facilitate the entry of fungal organisms. These may include *penecillium, fusarium, cladosporium, curvularia, aspergillus,* and *candida* sp. Lesions may be ulcerative or nonulcerative, often resulting in the presence of raised, gray to tan colored corneal plaques with an irregular surface. Progression, keratomalacia, and/or secondary bacterial contamination may ultimately result in corneal perforation.

DIAGNOSIS AND TREATMENT

The diagnosis of fungal keratitis is made based on clinical findings, supported by cytological or histological interpretation of representative material as well as ancillary diagnostics including microbial culture and sensitivity, serologic, and/or PCR-testing. Therapy comprises the topical and/or systemic administration of antifungal agents including natamycin, fluconazole, miconazole, and/or vorikonazole. The adjunctive use of antibiotics may also be indicated. Potential adverse effects associated with the use of antifungal agents include local irritation as well as GI distress and/or hepatopathy.

SELECTED REFERENCES

Ben-Shlomo G, Plummer C, Barrie K, Brooks D. Curvularia keratomycosis in a dog. *Vet Ophthalmol*. 2010;**13**(2):126–130.

Ledbetter EC, Norman ML, Starr JK. in vivo confocal microscopy for the detection of canine fungal keratitis and monitoring of therapeutic response. *Vet Ophthalmol*. 2016;**19**(3):220–229.

Nevile JC, Hurn SD, Turner AG. **Keratomycosis** in five **dogs**. *Vet Ophthalmol*. 2016;**19**(5):432–438.

Pucket JD, Allbaugh RA, Rankin AJ. Treatment of dematiaceous fungal keratitis in a dog. *J Am Vet Med Assoc*. 2012;**240**(9):1104–1108.

Scott EM and Carter RT. Canine keratomycosis in 11 dogs: a case series (2000–2011). *J Am Anim Hosp Assoc*. 2014;**50**(2):112–118.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 96.1 Fungal keratitis. Clinical presentation associated with fungal keratitis. Note the associated severe peripheral vascular keratitis.

Figure 96.2 Fungal keratitis. Note the raised, irregular, and gray corneal plaque axially.

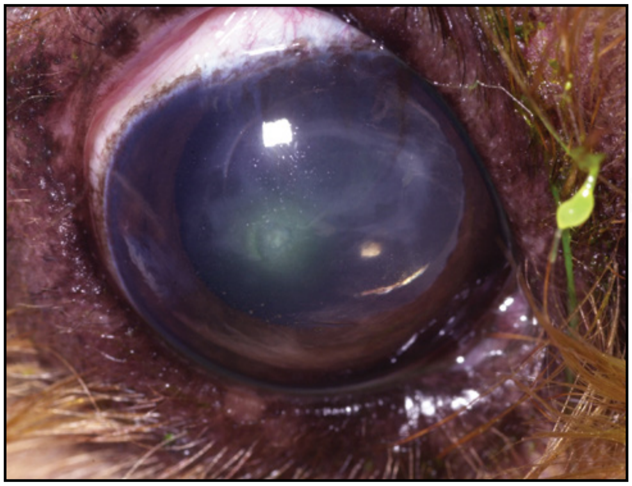


Figure 96.3 Fungal keratitis. Note the associated inflammation, discharge, and vascular keratitis.

Figure 96.4 Fungal keratitis. Hypopyon is visible in the anterior chamber as a result of secondary anterior uveitis.

CHAPTER 97
LIMBAL MELANOCYTOMA

PRESENTATION

Limbal ("epibulbar") melanocytomas represent a relatively common neoplastic proliferation of melanocytic cells at the corneoscleral limbus and may affect dogs or cats. In rare instances, these lesions may display malignant characteristics including significant anaplasia and/or metastasis. Lesions typically present as well defined, raised, and pigmented masses with a smooth surface. Progressive enlargement typically results in corneoscleral and/or intraocular invasion and may ultimately lead to glaucoma and/or retinal detachment. Limbal melanocytomas should be differentiated from extension of more malignant uveal melanomas. Commonly affected breeds include the Labrador Retriever, Golden Retriever, and German Shepherd.

DIAGNOSIS AND TREATMENT

The diagnosis of melanocytoma is made based on clinical findings, supported by histopathology, following incisional or excisional biopsy. These lesions are typically relatively benign and frequently appear to be localized to ocular tissues only. Nevertheless, the evaluation of systemic health and/or involvement via local lymph node aspiration, three-view radiography and complete blood count (CBC)/chemistry analysis, is encouraged. Treatment typically encompasses surgical resection where possible (with or without adjunctive tectonic grafting procedures using either biologic or synthetic materials). Pigment-rich melanocytomas are also relatively susceptible to the application of cryotherapy. The prognosis following prompt surgical intervention is generally good; however, recurrence is possible.

SELECTED REFERENCES

Donaldson D, Sansom J, Adams V. Canine limbal melanoma: 30 cases (1992–2004). Part 2. Treatment with lamellar resection and adjunctive strontium-90β plesiotherapy–efficacy and morbidity. *Vet Ophthalmol*. 2006;**9**(3):179–185.

Norman JC, Urbanz JL, Calvarese ST. Penetrating keratoscleroplasty and bimodal grafting for treatment of limbal melanocytoma in a dog. *Vet Ophthalmol*. 2008;**11**(5):340–345. doi: 10.1111/j.1463-5224.2008.00645.x.

Spangler WL and Kass PH. The histologic and epidemiologic bases for prognostic considerations in canine melanocytic neoplasia. *Vet Pathol*. 2006;**43**(2):136–149.

Wang AL and Kern T. Melanocytic ophthalmic neoplasms of the domestic veterinary species: a review. *Top Companion Anim Med*. 2015;**30**(4):148–157.

Wilkie DA and Wolf ED. Treatment of epibulbar melanocytoma in a dog, using full-thickness eyewall resection and synthetic graft. *J Am Vet Med Assoc*. 1991;**198**(6):1019–1022.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 97.1 Limbal melanocytomas. Clinical presentation associated with the presence of a large, invasive limbal melanocytoma.

Figure 97.2 Limbal melanocytomas. Note the extension into the scleral tissue.

Figure 97.3 Limbal melanocytomas. Note the presence of associated corneal mineral degeneration at the leading edge of the lesion.

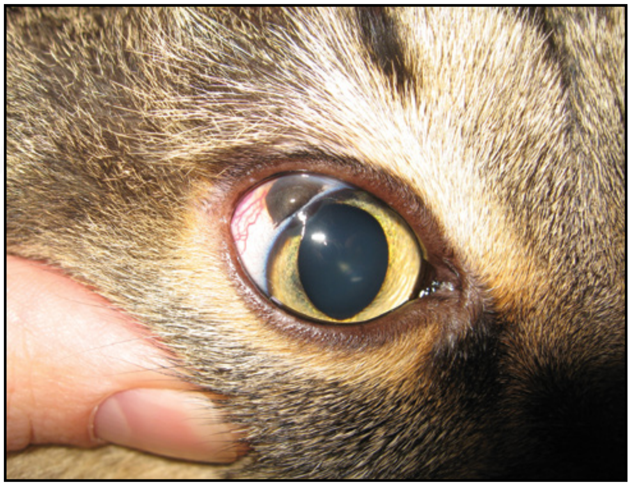


Figure 97.4 Limbal melanocytomas. Less-common clinical presentation associated with the presence of a feline limbal melanocytoma.

CHAPTER 98
CORNEOSCLERAL HEMANGIOMA/HEMANGIOSARCOMA

PRESENTATION

Tumors of vascular endothelial origin may affect the corneoscleral tissues and/or overlying conjunctival tissues (see also Conjunctival hemangioma/hemangiosarcoma). These lesions typically present as bright red, smooth, raised, irregular to nodular masses at or near the corneoscleral limbus. Tumors enlarge locally and may invade surrounding tissues. Exposure to UV radiation has been proposed to play a factor in the development of adnexal hemangioma/hemangiosarcoma. Commonly affected breeds include the Australian Shepherd and the Collie breeds.

DIAGNOSIS AND TREATMENT

A tentative diagnosis of hemangioma/hemangiosarcoma may be made based on the characteristic clinical appearance of these lesions; however, confirmation as well as characterization of malignancy requires incisional or excisional biopsy and histopathology. Systemic health and/or involvement should be evaluated via local lymph node aspiration, three-view radiography, and complete blood count (CBC)/chemistry analysis, although lesions are frequently localized to the adnexal tissues only. Treatment typically encompasses surgical resection where possible and/or the application of adjunctive cryotherapy. Cryotherapy should only be undertaken using an appropriate nitrous-oxide cooled unit. Focal swelling is common following cryotherapy; however, inflammation rapidly resolves. Postoperative care comprises topical and/or systemic anti-inflammatory and/or anti-microbial therapy as appropriate and the use of an Elizabethan collar to prevent self-trauma.

SELECTED REFERENCES

Chandler HL, Newkirk KM, Kusewitt DF, Dubielzig RR, Colitz CM. Immunohistochemical analysis of ocular hemangiomas and hemangiosarcomas in dogs. *Vet Ophthalmol*. 2009;**12**(2):83–90. doi: 10.1111/j.1463-5224.2008.00684.x.

Haeussler DJ Jr, Rodríguez LM, Wilkie DA, Premanandan C. Primary central **corneal hemangiosarcoma** in a **dog**. *Vet Ophthalmol*. 2011;**14**(2):133–136.

Pirie CG, Knollinger AM, Thomas CB, Dubielzig RR. Canine conjunctival hemangioma and hemangiosarcoma: a retrospective evaluation of 108 cases (1989–2004). *Vet Ophthalmol*. 2006;**9**(4):215–226.

Shank AMM, Teixeria LBC, Dubielzig RR. Canine, feline, and equine **corneal** vascular neoplasia: a retrospective study (2007–2015). *Vet Ophthalmol*. 2019;**22**(1):76–87.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 98.1 Corneoscleral hemangioma/hemangiosarcoma. Clinical presentation associated with corneoscleral hemangioma/hemangiosarcoma. Note both corneal and scleral invasion.

Figure 98.2 Corneoscleral hemangioma/hemangiosarcoma. Note the limbal location with early invasion of the cornea.

Figure 98.3 Corneoscleral hemangioma/hemangiosarcoma. This lesion appears to be confined to the sclera.

Figure 98.4 Corneoscleral hemangioma/hemangiosarcoma. Note the raised, nodular lesion in this case.

CHAPTER 99
CORNEOSCLERAL LYMPHOMA

PRESENTATION

Corneoscleral tissues are not infrequently affected by lymphoma. In some cases, ocular and/or adnexal symptoms may be the initial presentation for the disease, which may also remain localized to the adnexal region. Clinical findings may encompass a spectrum of lesions from mild peripheral infiltration around the corneoscleral limbus, to localized nodular and/or diffuse cream-colored tissue infiltration. Other ocular tissues and/or more generalized involvement may also develop. Both canine and feline species as well as any breed or crossed-breed may be affected (see also cutaneous epitheliotropic, conjunctival. uveal and choroidal lymphoma and retrobulbar neoplasia).

DIAGNOSIS AND TREATMENT

A definitive diagnosis of lymphoma is based on tissue biopsy and wherever lymphoma is suspected, samples should be harvested prior to the institution of therapy in order to facilitate the planning of an accurate treatment protocol. Treatment comprises topical anti-inflammatory therapy (typically utilizing a corticosteroid) as well as systemic chemotherapy. Associated ophthalmic changes such as elevated intraocular pressure should be addressed as appropriate (*see also Secondary glaucoma). Staging via local lymph node (and/or organ/bone marrow) aspiration, three-view radiography and complete blood count (CBC)/chemistry analysis is recommended, prior to initiation of systemic chemotherapy. Diagnostic testing for the presence of infectious viruses including FelV, FIV, and FIP is additionally recommended in feline patients. Individual chemotherapeutic protocols are most ideally designed by a veterinary oncologist, however typically include a variable combination of prednisone, vincristine, cyclophosphamide, and/or doxorubicin. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of vincristine include stomatitis, GI distress, neuropathy, hepatopathy, and myelosuppression. Potential adverse effects associated with the use of cyclophosphamide include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of doxyrubicin include hypersensitivity, GI distress, cardiac dysfunction, and myelosuppression. The prognosis varies depending on the extent of disease present prior to initiation of therapy.

SELECTED REFERENCES

Kang S, Jeong M, Seo K. Corneoconjunctival manifestations of **lymphoma** in three **dogs**. *J Vet Sci*. 2019;**20**(1):98–101.

Nola M, Lukenda A, Bollmann M, Kalauz M, Petrovecki M, Bollmann R. Outcome and prognostic factors in ocular adnexal lymphoma. *Croat Med J*. 2004;**45**(3):328–332.

Ota-Kuroki J, Ragsdale JM, Bawa B, Wakamatsu N, Kuroki K. Intraocular and periocular lymphoma in dogs and cats: a retrospective review of 21 cases (2001–2012). *Vet Ophthalmol*. 2013. doi: 10.1111/vop.12106.

Rutley M and MacDonald V. Managing the canine lymphosarcoma patient in general practice. *Can Vet J*. 2007;**48**(9):977–979.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 99.1 Corneoscleral lymphoma. Clinical presentation associated with corneoscleral lymphoma.

Figure 99.2 Corneoscleral lymphoma. Note the cream-colored corneal infiltration.

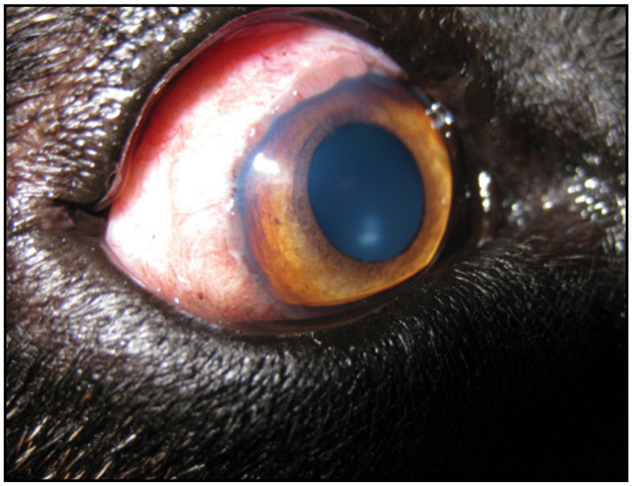


Figure 99.3 Corneoscleral lymphoma. Note the mild peripheral infiltration around the corneoscleral limbus.

Figure 99.4 Corneoscleral lymphoma. Note the diffuse peripheral limbal and corneal infiltration.

CHAPTER 100
SQUAMOUS CELL CARCINOMA

PRESENTATION

Corneal squamous cell carcinoma (SCC) may develop secondary to chronic corneal inflammation with affected cells undergoing various degrees of anisocytosis, anisokaryosis, and/or dyskeratosis, before progressing through low-grade or "carcinoma-in-situ" (which has not yet invaded the corneal stroma) to outright SCC. Clinically, SCC presents as a smooth, raised, gray to pink colored proliferative tissue, typically associated with a vascular supply and extending across the corneal surface in a variable pattern, ranging from focal/nodular to diffuse/lamellar. Commonly affected breeds include the Pug and the English Bulldog.

DIAGNOSIS AND TREATMENT

The diagnosis of SCC is made by histopathology following incisional or excisional biopsy, with SCC frequently appearing clinically similar to (and even encompassing) chronic corneal inflammation/granulation. Systemic health and/or involvement should be evaluated via local lymph node aspiration, three-view radiography and complete blood count (CBC)/ chemistry analysis. Treatment comprises resection of neoplastic tissue with or without the adjunctive application of cryotherapy and/or B-radiation. Cryotherapy should only be undertaken using an appropriate nitrous-oxide cooled unit. Focal swelling is common postoperatively, however inflammation rapidly resolves. Postoperative care comprises topical and or systemic anti-inflammatory and/or anti-microbial therapy as appropriate and the use of an Elizabethan collar to prevent self-trauma.

SELECTED REFERENCES

Bernays ME, *et al*. Primary corneal papilloma and squamous cell carcinoma associated with pigmentary keratitis in four dogs. *J Am Vet Med Assoc*. 1999;**241**(2):215–217.

Dorbandt DM, Driskell EA, Hamor RE. Treatment of **corneal squamous cell carcinoma** using topical 1% 5-fluorouracil as monotherapy. *Vet Ophthalmol*. 2016;**19**(3):256–261.

Dreyfus J, Schobert CS, Dubielzig RR. Superficial corneal squamous cell carcinoma occurring in dogs with chronic keratitis. *Vet Ophthalmol*. 2011;**14**(3):161–168. doi: 10.1111/j.1463-5224.2010.00858.x.

Montiani-Ferreira F, Kiupel M, Muzolon P, Truppel J. Corneal squamous cell carcinoma in a dog: a case report. *Vet Ophthalmol*. 2008;**11**(4):269–272. doi: 10.1111/j.1463-5224.2008.00622.x.

Takiyama N, Terasaki E, Uechi M. Corneal squamous cell carcinoma in two dogs. *Vet Ophthalmol*. 2010;**13**(4):266–269. doi: 10.1111/j.1463-5224.2010.00792.x.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 100.1 Squamous cell carcinoma. Clinical presentation associated with corneal squamous cell carcinoma.

Figure 100.2 Squamous cell carcinoma. Note the vascular supply extending across the corneal surface in a variable pattern.

Figure 100.3 Squamous cell carcinoma. Note the focal, raised proliferative tissue.

Figure 100.4 Squamous cell carcinoma. Note the diffuse vascular supply and the smooth raised corneal lesion.

Section 8

Diseases of the Uvea

CHAPTER 101
PERSISTENT PUPILLARY MEMBRANES (PPMs)

PRESENTATION

Persistent pupillary membranes (PPMs) occur secondary to the incomplete resorption of embryonic lenticular vasculature, a process which is usually complete by several weeks after birth but which may continue for several months in some dogs. Occasionally, the remnants of this process may be recognized as a collection of fine, punctate, tan to brown spots on the axial anterior lens capsule. PPMs may extend from iris to iris, iris to lens, iris to corneal endothelium, and/or extend to float freely into the anterior chamber. The condition may be unilateral but is more commonly bilateral. Large or multiple PPMs may result in corneal opacification (due to endothelial damage, traction, and fibrosis) and/or secondary cataract formation. Commonly affected dog breeds include the Basenji, Pembroke Welsh Corgie, Mastiff, and Chow Chow.

DIAGNOSIS AND TREATMENT

The diagnosis of PPMs is made based on characteristic clinical findings. It may occasionally be challenging to differentiate hereditary PPMs from post-inflammatory synechiae, the latter potentially being associated with other signs of prior ocular inflammation or trauma including adjacent corneal fibrosis, iridal synechiation, pigment deposition, and/or cataract formation. Treatment is not typically necessary, although in rare cases the surgical or laser-mediated resection of PPMs and treatment of associated inflammation maybe indicated. Similarly, significant cataracts, if present, may be surgically removed.

SELECTED REFERENCES

Collins BK, Collier LL, Johnson GS, Shibuya H, Moore CP, da Silva Curiel JM. Familial cataracts and concurrent ocular anomalies in chow chows. *J Am Vet Med Assoc*. 1992;**200**(10):1485–1491.

Glaze MB. Congenital and hereditary ocular abnormalities in cats. *Clin Tech Small Anim Pract*. 2005;**20**(2):74–82.

James RW. Persistent pupillary membrane in basenji dogs. *Vet Rec*. 1991;**128**(12):287–288.

Saraiva IQ and Delgado E. Congenital ocular malformations in **dogs** and cats: 123 cases. *Vet Ophthalmol*. 2020;**23**(6):964–978.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 101.1 Persistent pupillary membranes. Clinical presentation associated with iris to lens persistent pupillary membranes.

Figure 101.2 Persistent pupillary membranes. Clinical presentation associated with iris to iris and iris to cornea persistent pupillary membranes.

Figure 101.3 Persistent pupillary membranes. Clinical presentation associated with iris to cornea persistent pupillary membranes.

Figure 101.4 Persistent pupillary membranes. Clinical presentation associated with iris to cornea persistent pupillary membranes.

CHAPTER 102
IRIS COLOBOMAS

PRESENTATION

Iris colobomas are defects occurring in of any/all layers of developing iridal tissue, as a result of inappropriate embryonic differentiation. Coloboma(s) located within the iridal body may result in the false appearance of multiple pupillary opening or "pseudpolycoria." The complete absence of iris tissue (termed "aniridia") is rare. Clinically, most iris colobomas typically appear as "notch" defects within the iris. Commonly affected breeds include the Australian Shepherd and Collie breeds.

DIAGNOSIS AND TREATMENT

The diagnosis of iris coloboma is made based on characteristic clinical findings. Treatment of iris colobomas is not typically indicated.

SELECTED REFERENCES

Barnett KC and Knight GC. Persistent pupillary membrane and associated defects in the Basenji. *Vet Rec*. 1969;**85**(9):242–248.
Belhorn RW, Barnett KC, Henkind P. Ocular **colobomas** in domestic **cats**. *J Am Vet Med Assoc*. 1971;**159**(8):1015–1021.
Cook CS. Embryogenesis of congenital eye malformatons. *Vet Comp Ophthalmol*. 1995;**5**, 109–123.
Startup FG. Congenital abnormalities of the iris of the dog. *J Small Anim Pract*. 1966;**7**(1):99–100.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

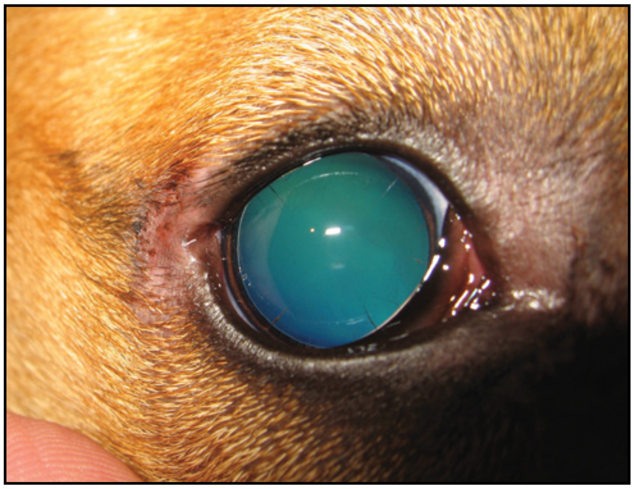


Figure 102.1 Iris colobomas. Rare clinical presentation associated with complete aniridia.

Figure 102.2 Iris colobomas. Clinical presentation associated with colobomatous "notch" defect within iris tissue.

Figure 102.3 Iris colobomas. Note dyscoria as a result of iris coloboma.

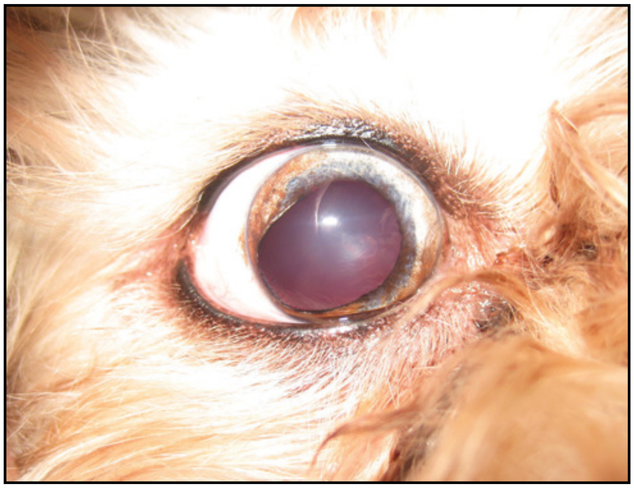


Figure 102.4 Iris colobomas. Note the presentation in heterochromic iris.

CHAPTER 103
SENILE IRIS ATROPHY

PRESENTATION

Senile iris atrophy results from the progressive loss of iridal tissue, which may occur with advanced age. Clinical findings may include thinning/translucency of iridal tissue (notably at the pupillary margin), resultant irregularities of the pupillary opening, outright pupilary dyscoria, the optical illusion of an apparently mydriatic (and poorly responsive) pupil and/or the presence of full-thickness iris defects resulting in the appearance of multiple apparent pupillary openings or "pseudopolycoria." Senile iris atrophy is typically bilateral, although not always symmetrical. Senile atrophy of iridal tissues is common in small breed dogs.

DIAGNOSIS AND TREATMENT

The diagnosis of senile iris atrophy is made based on clinical findings. It may be challenging to differentiate iridal atrophy from congenital pupillary abnormalities (notably iridal colobomas) and/or neurological disease (notably pathology resulting in mydriasis) (see also iris colobomas). Pharmacologic testing using topical pilocarpine will differentiate mechanical from neurologic mydriasis. No specific treatment is indicated; however, significant iridal atrophy and the resultant enlargement of the pupillary orifice may increase the relative risk of degenerate vitreous herniation and/or lenticular (sub)luxation in predisposed breeds, so that the long-term administration of miotic agents may occasionally be indicated.

SELECTED REFERENCES

Grahn BH and Cullen CL. Diagnostic ophthalmology. *Iris atrophy Can Vet J*. 2004;**45**(1):77–78.

Petrick SW. The incidence of eye disease in dogs in a veterinary academic hospital: 1772 cases. *J S Afr Vet Assoc*. 1996;**67**(3):108–110.

Whiting RE, Yao G, Narfström K, Pearce JW, Coates JR, Dodam JR, Castaner LJ, Katz ML. Quantitative assessment of the canine pupillary light reflex. *Invest Ophthalmol Vis Sci*. 2013;**54**(8):5432–5440.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

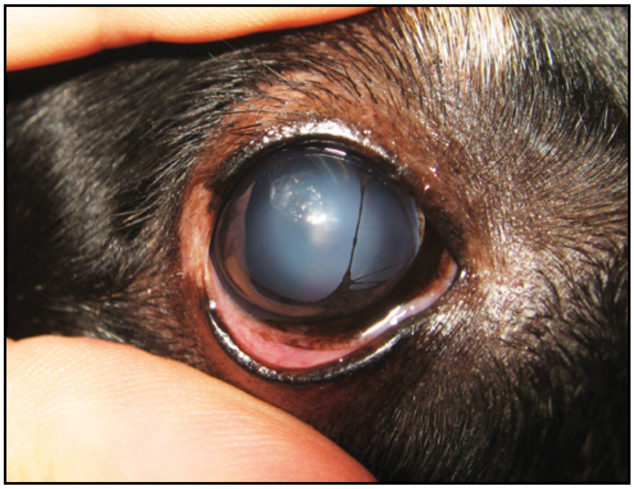


Figure 103.1 Senile iris atrophy. Clinical presentation associated with senile iris atrophy.

Figure 103.2 Senile iris atrophy. Note the "pseudopolycoria" laterally.

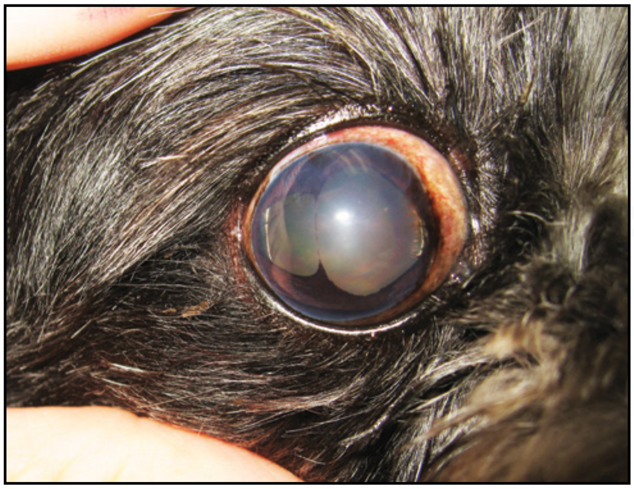


Figure 103.3 Senile iris atrophy. Advanced atrophy associated with the appearance of mydriasis.

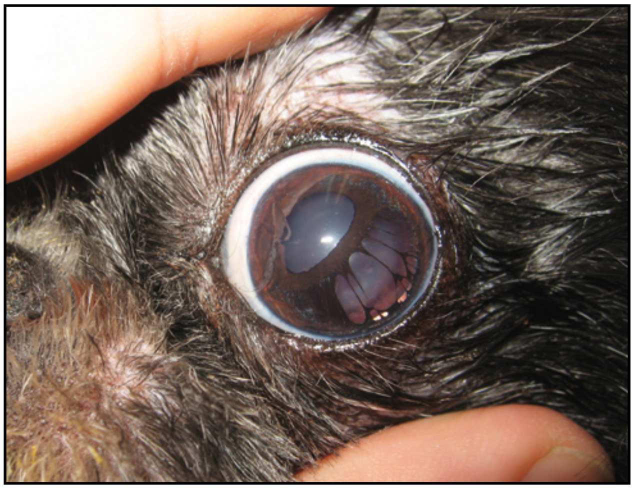


Figure 103.4 Senile iris atrophy. Note pupillary dyscoria and "pseudopolycoria."

CHAPTER 104
UVEAL CYSTS

PRESENTATION

Uveal cysts (sometimes also referred to as "iris cysts") may arise from the posterior iridal or ciliary body epithelium as a result of hereditary factors, cystic hyperplasia, and/or chronic inflammation. Clinically, uveal cysts comprise oval to round, thin-walled, translucent to dark brown structures which may be attached to the ciliary tissue, float freely within the anterior or posterior chambers (or rarely within degenerate vitreous), and/or rupture and adherer against intracameral surfaces including the lens capsule and/or corneal endothelium. Uveal cysts may occur unilaterally or bilaterally and may vary significantly in both size and number. Commonly affected breeds include the Labrador Retriever, Golden Retriever, Boston Terrier, Great Dane, and American Bulldog.

DIAGNOSIS AND TREATMENT

The diagnosis of uveal cyst(s) is made based on clinical findings, supported if necessary by B-mode ultrasonography. Occasionally, intracameral neoplasia (particularly melanocytoma/melanoma) may represent challenging differentials (see also uveal melanoma). Most uveal cysts are benign and do not require treatment. The presence of uveal cysts in Golden Retrievers should raise concern for the presence of additional ocular findings, which may suggest the development of more serious breed-associated pigmentary and cystic uveitis/glaucoma (see Golden Retriever associated pigmentary uveitis and glaucoma). Additionally, the presence of uveal cysts has been associated with other ocular changes and elevated intraocular pressure in some breeds including the American Bulldog and Great Dane (see cystic glaucoma). The administration of systemic topical and/or anti-inflammatory agents (either steroidal or nonsteroidal) may be indicated in cases affected by chronic intracameral inflammation. Surgical intervention may be indicated in cases where cystic proliferation results in visual impairment and/or compromised aqueous outflow. Where indicated, cysts may be removed by surgical extraction, surgical aspiration, and/or diode laser photoablation. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration.

SELECTED REFERENCES

Corcoran KA, Koch SA. **Uveal cysts** in **dogs**: 28 cases (1989–1991). *J Am Vet Med Assoc*. 1993;**203**(4):545–546.

Delgado E, Pissarra H, Sales-Luís J, Peleteiro MC. Amelanotic uveal cyst in a Yorkshire terrier dog. *Vet Ophthalmol* 2010;**13**(5):343–347. doi: 10.1111/j.1463-5224.2010.00825.x.

Gemensky-Metzler AJ, Wilkie DA, Cook CS. The use of semiconductor diode laser for deflation and coagulation of anterior uveal cysts in dogs, cats and horses: a report of 20 cases. *Vet Ophthalmol*. 2004;**7**(5):360–368.

Townsend WM and Gornik KR. Prevalence of uveal cysts and pigmentary uveitis in Golden Retrievers in three Midwestern states. *J Am Vet Med Assoc*. 2013;**243**(9):1298–1301. doi: 10.2460/javma.243.9.1298.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 104.1 Uveal cysts. Single, heavily pigmented uveal cyst in a feline patient.

Figure 104.2 Uveal cysts. Multiple, free-floating uveal cysts in the anterior chamber of a Boston Terrier.

Figure 104.3 Uveal cysts. Single translucent uveal cyst attached to the pupillary margin.

Figure 104.4 Uveal cysts. Single free-floating pigmented uveal cyst.

CHAPTER 105
MERLE OCULAR DYSGENESIS (MOD)

PRESENTATION

Merle ocular dysgenesis (MOD) is a hereditary, congenital condition, which may affect animals displaying merled coat coloration. MOD results from abnormal embryonic differentiation of tissues and may lead to multiple ocular abnormalities including any combination of microphthalmos, microcornea, scleral staphyloma, persistent pupillary membranes (PPMs), heterochromia iris/irides hypoplasia/coloboma (with resultant dyscoria/chorectopia), pseudopolycoria, iridocorneal angle dysplasia, lenticular coloboma/cataract/subluxation, choroidal hypoplasia, optic nerve coloboma, retinal dysplasia and/or retinal detachment (with or without hyphema). Changes are bilateral but not always symmetrical. Affected dogs (notably homozygous “white” merles) may also be congenitally deaf. Commonly affected breeds include the Australian Shepherd, (merled) Collies, Shetland Sheepdog, Harlequin Great Dane, and (merled) Long-haired Dachshund.

DIAGNOSIS AND TREATMENT

The diagnosis of MOD is made based on a combination of signalment and clinical findings. Primary treatment of this disorder is not possible; however, secondary complications, such as hyphema or cataract development, may be treated symptomatically. Surgical intervention in order to address scleral, lenticular, and/or retinal pathology may also be indicated in select cases. Selective breeding reduces the incidence and severity of disease.

SELECTED REFERENCES

Bauer BS, Sandmeyer LS, Grahn BH. Diagnostic ophthalmology. Microphthalmos and multiple ocular anomalies (MOA) OU consistent with **merle** ocular dysgenesis (MOD). *Can Vet J*. 2015;**56**(7):767–768.

Gelatt KN and McGill LD. Clinical characteristics of microphthalmia with colobomas of the Australian Shepherd Dog. *J Am Vet Med Assoc*. 1973;**162**(5):393–396.

Gelatt KN, Powell NG, Huston K. Inheritance of microphthalmia with coloboma in the Australian shepherd dog. *Am J Vet Res*. 1981;**42**(10):1686–1890.

Hédan B, Corre S, Hitte C, Dréano S, Vilboux T, Derrien T, Denis B, Galibert F, Galibert MD, André C. Coat colour in **dogs**: identification of the **merle** locus in the Australian shepherd breed. *BMC Vet Res*. 2006**27**;2:9.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 105.1 Merle ocular dysgenesis. Clinical presentation associated with merle ocular dysgenesis. Inset demonstrate the presence of a mature cataract.

Figure 105.2 Merle ocular dysgenesis. Clinical presentation associated with merle ocular dysgenesis. Inset demonstrate the presence of an immature cataract.

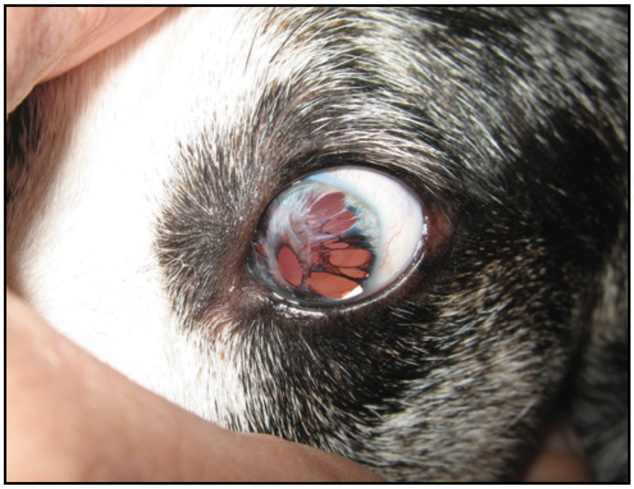


Figure 105.3 Merle ocular dysgenesis. Note the presence of multiple iris colobomas.

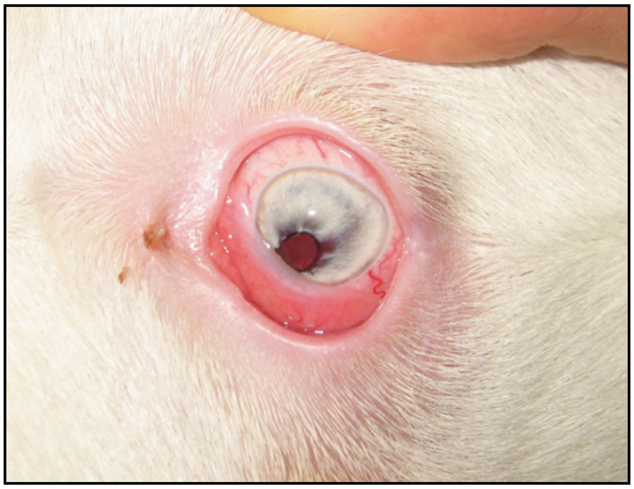


Figure 105.4 Merle ocular dysgenesis. Note corectopia and iris hypoplasia.

CHAPTER 106
FELINE ANTERIOR UVEITIS

PRESENTATION

The uveal tract is composed of anterior (iris and ciliary body) and posterior (choroidal) tissues. These tissues contain components of the "blood-ocular barrier" which regulates the passage of protein into the aqueous humor. The term uveitis describes inflammation of any of these tissues and is typically associated with variable breakdown of the blood-ocular barrier. Inflammation of the anterior uveal tissues, may occur with or without significant clinical involvement of posterior uveal tissue (see also Feline chorioretinitis). The spectrum of clinical symptoms potentially associated with feline anterior uveitis may include: vascular keratitis, corneal edema, and/or the accumulation of proteinaceous keratic precipitates, aqueous proteinaceous flare, iridal thickening/hyperemia/infiltration (diffuse or nodular) or hemorrhage, iridal adhesions (anterior or posuertior), outright it is bombe', secondary cataract formation, vitritis and variable degrees of chorioretinitis (with or without retinal detachment). Initially, intraocular pressures are typically decreased in cases of uveitis; however, secondary glaucoma may occur as a later complication (see also Secondary glaucoma). One or both eyes may be affected. Bilateral uveitis should prompt concern for the presence of systemic disease.

DIAGNOSIS AND TREATMENT

The diagnosis of anterior uveitis is made based on clinical findings. Potential etiologies include hereditary factors, lens-associated inflammation (see also phacolytic and phacoclastic uveitis), trauma, systemic disease, exposure to infectious organisms, and/or the presence of (local or systemic) neoplasia. Infectious etiologies which may be associated with the development of anterior uveitis in cats include viral (FelV, FIV, FIP, FHV), protozoal (*Toxoplasma gondii*), bacterial (notably *Bartonealla* spp), and fungal (*crytococcus, cocciodiomycosis, aspergillus, blastomyces*, and *histoplasmosis*) organisms. Frustratingly, the etiology of uveitis remains unclear in a significant proportion of cases. Additionally, clinical uveitis may be chronic and/or recurrent in its behavior. Treatment encompasses addressing underlying systemic, infectious, or neoplastic disease. Additionally, topical and/or systemic anti-inflammatory therapy (nonsteriodal or steroidal) is typically warranted. Long-term treatment may be indicated in order to minimize the risk of secondary glaucoma. Mydriatic agents (atropine/tropicamide) should be used with caution and only where specifically indicated (i.e., in the presence painful ciliary spasm and/or significant BAB breakdown). Clindamycin generally represents a suitable initial, empirical antimicrobial choice when treating feline patients. Potential side effects associated with the use of Clindamycin include gastrointestinal (GI) distress.

SELECTED REFERENCES

Colitz CM. Feline uveitis: diagnosis and treatment. *Clin Tech Small Anim Pract*. 2005;**20**(2):117–20.

Crossfield L, Fortuna L, Carling R, Scurrell E, Matas Riera M, Chang YM, Dawson C, Pittaway R, Yeung IY, Thaung C, Priestnall SL, Garden OA. Immunohistochemical characterization of **feline** lymphoplasmacytic anterior **uveitis**. *Vet Ophthalmol*. 2019;**22**(2):206–212.

Shukla AK and Pinard CL. Feline uveitis. *Compend Contin Educ Vet*. 2012;**34**(9):E1.

Townsend WM. Canine and feline uveitis. *Vet Clin North Am Small Anim Pract*. 2008;**38**(2):323–346, vii. doi: 10.1016/j.cvsm.2007.12.004.

van der Woerdt A. Management of intraocular inflammatory disease. *Clin Tech Small Anim Pract*. 2001;**16**(1):58–61.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 106.1 Feline anterior uveitis. Clinical presentation associated with feline anterior uveitis. Note nodular iridal proliferation and the accumulation of fibrin within the anterior chamber.

Figure 106.2 Feline anterior uveitis. Note nodular iridal proliferation, mild iritis, and endothelial keratic precipitate formation.

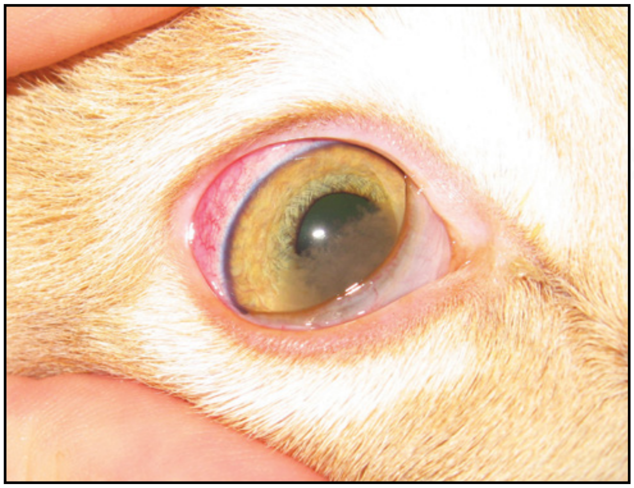


Figure 106.3 Feline anterior uveitis. Note the accumulation of fibrin within the anterior chamber.

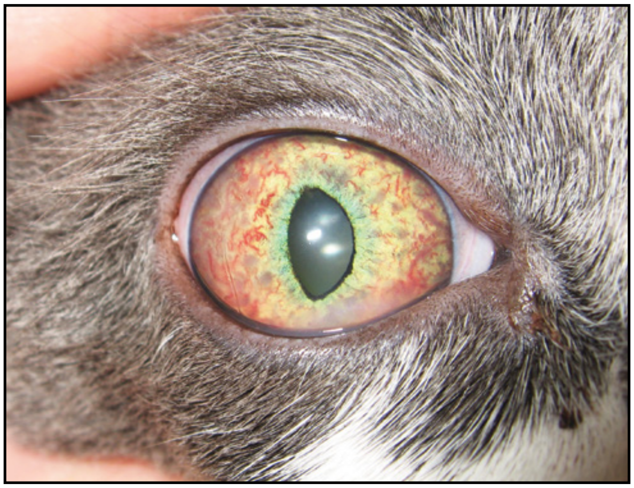


Figure 106.4 Feline anterior uveitis. Note the nodular iridal proliferation, severe iritis, and endothelial keratic precipitate formation.

CHAPTER 107
CANINE ANTERIOR UVIEITIS

PRESENTATION

The uveal tract is composed of the anterior (iris and ciliary body) and posterior (choroidal) tissues. These tissues contain components of the "blood-ocular barrier" which regulates the passage of protein into the aqueous humor. The term uveitis describes inflammation of any of these tissues and is typically associated with variable breakdown of the blood-ocular barrier. Inflammation of the anterior uveal tissues may occur with or without significant clinical involvement of posterior uveal tissue (see also canine chorioretinitis). The spectrum of clinical symptoms potentially associated with canine anterior uveitis may include vascular keratitis, corneal edema, and/or the accumulation of proteinaceous keratic precipitates, aqueous proteinacious flare, iridal thickening/hyperemia/infiltration (diffuse or nodular) or hemorrhage, iridal adhesions (anterior or posterior), outright it is bombe', secondary cataract formation, vitritis and variable degrees of chorioretinitis (with or without retinal detachment). Initially, intraocular pressures are typically decreased in cases of uveitis; however, secondary glaucoma may occur as a later complication (see also Secondary glaucoma). One or both eyes may be affected. Bilateral uveitis should prompt concern for the presence of systemic disease.

DIAGNOSIS AND TREATMENT

The diagnosis of anterior uveitis is made based on clinical findings. Potential etiologies include hereditary factors, trauma, lens-associated inflammation (*see also phacolytic and phacoclastic uveitis), systemic disease, exposure to infectious organisms, and/or the presence of (local or systemic) neoplasia. Infectious etiologies which may be associated with the development of anterior uveitis in dogs include viral (canine adenovirus-1 and canine parvovirus), protozoal (*Toxoplasma gondii*), bacterial (notably *Ehrlichia canis, Rickettsia rickettsia, Leptospira* spp, and *Borellia* spp), and fungal (*coccidiomycosis, aspergillus, blastomyces*, and *histoplasmosis*) organisms. Frustratingly, the precise etiology of uveitis remains unclear in a significant proportion of cases. Additionally, clinical uveitis may be chronic and/or recurrent in its behavior. Treatment encompasses addressing underlying systemic, infectious, or neoplastic disease. Additionally, topical and/or systemic anti-inflammatory therapy (nonsteroidal or steroidal) is typically warranted. Long-term treatment may be indicated in order to minimize the risk of secondary glaucoma. Mydriatic agents (atropine/tropicamide) should be used with caution and only where specifically indicated (i.e., in the presence of painful ciliary spasm and/or significant BAB breakdown). Doxycycline generally represents a suitable initial, empirical antimicrobial choice when treating canine patients. Potential side effects associated with the use of doxycycline, include gastrointestinal (GI) distress.

SELECTED REFERENCES

Bergstrom BE, Stiles J, Townsend WM. **Canine** panuveitis: a retrospective evaluation of 55 cases (2000–2015). *Vet Ophthalmol*. 2017;**20**(5):390–397.

Massa KL, Gilger BC, Miller TL, Davidson MG. Causes of uveitis in dogs: 102 cases (1989–2000). *Vet Ophthalmol*. 2002;**5**(2):93–98.

Townsend WM. Canine and feline uveitis. *Vet Clin North Am Small Anim Pract*. 2008;**38**(2):323–346, vii. doi: 10.1016/j.cvsm.2007.12.004.

Wasik B and Adkins E. Canine anterior uveitis. *Compend Contin Educ Vet*. 2010;**32**(11):E1.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 107.1 Canine anterior uveitis. Clinical presentation associated with canine anterior uveitis, including conjunctival hyperemia, aqueous flare, and iritis.

Figure 107.2 Canine anterior uveitis. Note significant iritis.

Figure 107.3 Canine anterior uveitis. Note iritis and hyphema.

Figure 107.4 Canine anterior uveitis. Note aqueous flare, iritis, hypopyon, and hyphema.

CHAPTER 108
ASYMMETRIC UVEITIS

PRESENTATION

Canine asymmetric uveitis represents a less common but severe form of intraocular inflammation which may affect dogs. Symptoms may include any combination of corneal edema, corneal neovascularization, aqueous flare, iritis, keratic precipitate deposition, cataract formation, glaucoma, retinal detachment, and/or endophthalmitis. Presentation is typically unilateral with the contralateral eye at significant risk for the subsequent development of similar symptoms over a variable period extending from weeks to years. Affected animals may be diabetic or nondiabetic. Small breed dogs are primarily affected by this condition, with Poodles reported to be at increased risk and females potentially overrepresented.

DIAGNOSIS AND TREATMENT

The diagnosis of asymmetric uveitis is made based on clinical findings, supported by standard ancillary diagnostics, the exclusion of alternate etiologies and, in some cases, diagnostic histopathology from enucleated globes. The characteristic histopathological finding associated with this disease is the development of a pyogranulomatous infiltrate affecting the uveal tract. Aggressive topical and/or systemic anti-inflammatory therapy (steroidal or nonsteroidal) is indicated in affected animals. Wherever possible, steroidal agents provide greater efficacy with systemic therapy more likely to effectively penetrate affected uveal tissues. Long-term therapy may additionally include the use of prednisone-sparing (azathioprine) and/or additional immunosuppressive agents (including cyclosporine, leflunomide, and methotrexate). Long-term (indefinite) treatment is indicated in patients affected by asymmetric uveitis, with potential for complications including endophthalmitis, retinal detachment, and/or glaucoma considered significant.

SELECTED REFERENCES

Dubielzig RR. 2010. *Veterinary Ocular Pathology*, pp. 245–322. doi: 10.1016/B978-0-7020-2797-0.00009-6.

Håkanson N and Forrester SD. Uveitis in the dog and cat. *Vet Clin North Am Small Anim Pract*. 1990;**20**(3):715–735. doi: 10.1016/s0195-5616(90)50059-5.

Wasik B and Adkins E. Canine anterior uveitis. *Compend Contin Educ Vet*. 2010;**32**(11):E1.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

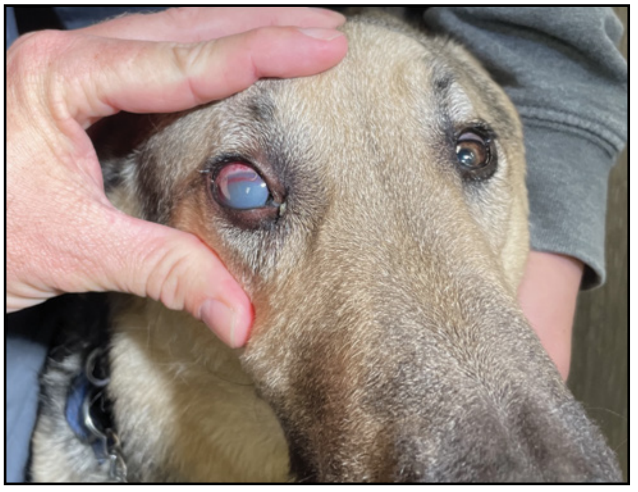


Figure 108.1 Asymmetric uveitis. Clinical presentation associated with asymmetric uveitis. Note diffuse corneal edema and perilimbal corneal vascular brush border.

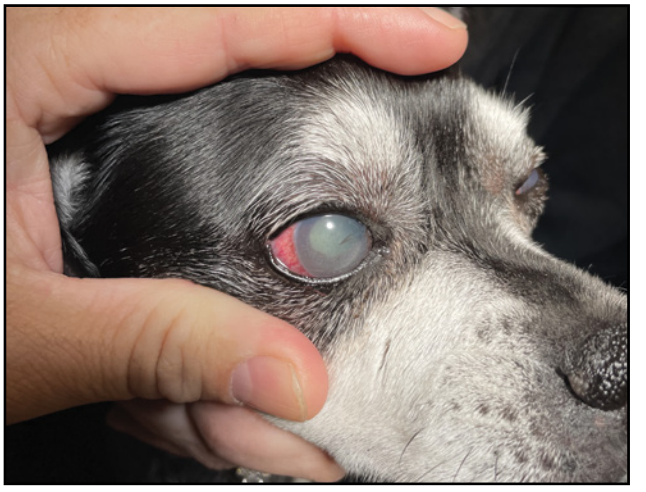


Figure 108.2 Asymmetric uveitis. Note severe aqueous flare and areas of posterior synechia.

Figure 108.3 Asymmetric uveitis. Note uveal thickening and dyscoria.

Figure 108.4 Asymmetric uveitis. Note diffuse generalized corneal edema.

CHAPTER 109
GOLDEN RETRIEVER ASSOCIATED UVEITIS AND GLAUCOMA

PRESENTATION

A syndrome comprising slowly progressive intraocular changes, typically culminating in secondary glaucoma, is well recognized within the Golden Retriever breed. This syndrome has been variably described as "Pigmentary Uveitis," "Golden Retriever Uveitis," and "Pigmentary and Cystic Glaucoma of Golden Retrievers." Findings may include any combination of conjunctival and/or episcleral hyperemia, corneal decompensation, thin-walled uveal cysts within the anterior or posterior chambers, proteinaceous exudation within the anterior chamber, anterior and/or posterior iridal adhesions, pigment dispersion within the anterior chamber and/or onto the anterior lens capsule, cataract formation, hyphema and/or secondary glaucoma (see also Golden Retriever associated glaucoma). Uveal cysts may detach and move into the anterior chamber where they may rupture against corneal endothelial and/or iridal surfaces or collapse and rupture within the iridocorneal angle. The hallmark of this syndrome has been described as the appearance of pigment on the anterior lens capsule, typically distributed in a radial orientation. Initial symptoms are frequently noted around middle age and are usually bilateral, although not necessarily symmetrical. The etiology of this syndrome is poorly understood; however, genetic factors have been proposed based on breed predilection as well as the absence of demonstrable infectious or neoplastic causes.

DIAGNOSIS AND TREATMENT

The diagnosis of Golden Retriever associated uveitis is made based on a combination of signalment and clinical findings. Treatment is generally empirical, frequently comprising topical and/or systemic anti-inflammatory (either steroidal or non-steroidal), immune-modulating (azathioprine or cyclosporine) as well as anti-glaucoma agents; however, secondary glaucoma is frequently the end point of this disease. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of azathioprine include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of cyclosporine include hypersensitivity and gastrointestinal (GI) distress. Patients affected by advanced disease represent high-risk candidates for either cataract or glaucoma surgery based on the frequency and severity of complications encountered. Ultimately, a procedure of comfort (enucleation, cryosurgery, intrascleral prosthesis placement, or chemical ciliary body ablation) may be necessary in order to address blind and/or painful eyes.

SELECTED REFERENCES

Deehr AJ and Dubielzig RR. A histopathological study of iridociliary cysts and glaucoma in Golden Retrievers. *Vet Ophthalmol*. 1998;**1**(2–3):153–158.

Esson D, Armour M, Mundy P, Schobert CS, Dubielzig RR. The histopathological and immunohistochemical characteristics of pigmentary and cystic glaucoma in the Golden Retriever. *Vet Ophthalmol*. 2009;**12**(6):361–368.

Sapienza JS, Simó FJ, Prades-Sapienza A. Golden Retriever uveitis: 75 cases (1994–1999). *Vet Ophthalmol*. 2000;**3**(4):241–246.

Townsend WM and Gornik KR. Prevalence of uveal cysts and pigmentary uveitis in Golden Retrievers in three Midwestern states. *J Am Vet Med Assoc*. 2013;**243**(9):1298–1301. doi: 10.2460/javma.243.9.1298.

Townsend WM, Huey JA, McCool E, King A, Diehl KA. Golden retriever pigmentary **uveitis**: challenges of diagnosis and treatment. *Vet Ophthalmol*. 2020;**23**(5):774–784.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 109.1 Golden Retriever associated uveitis and glaucoma. Clinical presentation associated with golden retriever uveitis. Note increased iridal pigmentation and radial pigment deposition on the anterior lens capsule.

Figure 109.2 Golden Retriever associated uveitis and glaucoma. Note increased iridal pigmentation, posterior synechia, and secondary cataract.

Figure 109.3 Golden Retriever associated uveitis and glaucoma. Note increased iridal pigmentation and anterior chamber protein exudation.

Figure 109.4 Golden Retriever associated uveitis and glaucoma. Note increased iridal pigmentation and thin-walled uveal cysts.

CHAPTER 110
UVEODERMATOLOGIC SYNDROME (UDS) ASSOCIATED UVEITIS

PRESENTATION

Canine uveodermatologic syndrome (UDS) represents an immune-mediated disease affecting melanocytic tissue, which likely has a hereditary etiology and which exhibits many similarities to Vogt-Koyanagi-Harada (VKH) disease in humans. Ocular symptoms associated with UDS may include inflammation marked by any combination of anterior uveitis, chorioretinitis, or panuveitis. Secondary changes may include any combination of cataract formation, retinal detachment, hyphema, and/or glaucoma. Typically, both eyes are affected to a variable degree. Additional symptoms may include any combination of periocular, mucocutaneous oral, and/or nasal vitiligo (loss of pigment), poliosis (whitening of hair), and/or ulcerative dermatitis. Typically, these symptoms are relatively bilaterally symmetrical in appearance (see also Auto-immune blepharitis and uveodermatologic associated chorioretinitis). Commonly affected breeds include the Akita, Siberian Husky, Samoyed, Chow Chow, German Shepherd, and Shetland Sheepdog; however, many other breeds may be affected.

DIAGNOSIS AND TREATMENT

The diagnosis of UDS is made based on a combination of signalment and clinical findings. Wherever representative adnexal or dermal changes are present, the harvesting of a small incisional skin biopsy for histopathological interpretation may support the diagnosis. Treatment usually comprises aggressive, long-term anti-inflammatory, and/or immune-modulating therapy. Typically, this involves the topical administration of corticosteroids and the systemic administration of corticosteroids with or without the adjunctive use of additional agents including cyclosporine and/or azathioprine. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight-gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of azathioprine include gastrointestinal (GI) distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of cyclosporine include hypersensitivity and GI distress.

SELECTED REFERENCES

Angles JM, Famula TR, Pedersen NC. Uveodermatologic (VKH-like) syndrome in American Akita dogs is associated with an increased frequency of DQA1*00201. *Tissue Antigens*. 2005;**66**(6):656–665.

Carter WJ, Crispin SM, Gould DJ, Day MJ. An immunohistochemical study of uveodermatologic syndrome in two Japanese Akita dogs. *Vet Ophthalmol*. 2005;**8**(1):17–24.

Horikawa T, Vaughan RK, Sargent SJ, Toops EE, Locke EP. Pathology in practice. Uveodermatologic syndrome. *J Am Vet Med Assoc*. 2013;**242**(6):759–761. doi: 10.2460/javma.242.6.759.

Zarfoss MK, Tusler CA, Kass PH, Montgomery K, Lim CC, Mowat F, Thomasy SM. Clinical findings and outcomes for **dogs** with **uveodermatologic syndrome**. *J Am Vet Med Assoc*. 2018;**252**(10):1263–1271.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 110.1 Uveodermatologic syndrome (UDS) associated uveitis. Endothelial keratic precipitates are notable in this case.

Figure 110.2 Uveodermatologic syndrome (UDS) associated uveitis. Note iritis and miosis.

Figure 110.3 Uveodermatologic syndrome (UDS) associated uveitis. Note aqueous flare, iritis, and miosis.

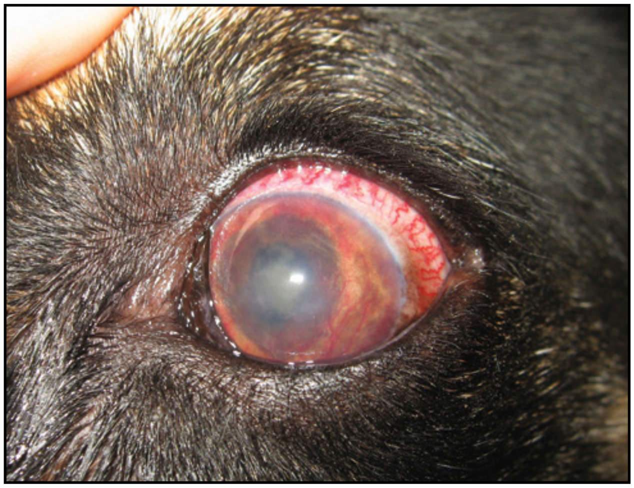


Figure 110.4 Uveodermatologic syndrome (UDS) associated uveitis. Iritis, hyphema, and secondary cataract formation are notable in this case.

CHAPTER 111
VACCINE-ASSOCIATED UVEITIS

PRESENTATION

Infection with Canine adenovirus type-1 (CAV-1) and/or vaccination with modified live CAV-1 strains may be associated with anterior uveitis and/or endothelitis (see also Endothelitis). Ocular symptoms may include the presence of aqueous flare, pupillary miosis, and/or corneal edema secondary to endothelial cell dysfunction. Potential ocular sequella include bullous keratopathy, ulcerative keratitis, and/or secondary glaucoma. The Afghan hound has been described as being at increased risk.

DIAGNOSIS AND TREATMENT

The diagnosis is made based on history (including that of recent vaccination), clinical examination, routine complete blood count (CBC)/chemistry analysis, and/or testing for the presence of infectious organisms. Treatment typically encompasses the administration of topical and systemic (steroidal) anti-inflammatory therapy, unless contraindicated. In many cases, symptoms will resolve if appropriately treated early in the course of the disease. Secondary pathologies including corneal ulceration and/or glaucoma should be managed as appropriate if present. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Aguirre G, Carmichael L, Bistner S. Corneal endothelium in viral induced anterior uveitis. Ultrastructural changes following canine adenovirus type 1 infection. *Arch Ophthalmol*. 1975;**93**(3):219–224.

Carmichael LE, Medic BL, Bistner SI, Aguirre GD. Viral-antibody complexes in **canine** adenovirus type 1 (**CAV**-1)ocular lesions: leukocyte chemotaxis and enzyme release. *Cornell Vet*. 1975;**65**(3):331–351.

Curtis R and Barnett KC. Canine adenovirus-induced ocular lesions in the Afghan hound. *Cornell Vet*. 1981;**71**(1):85–95.

Curtis R and Barnett KC. The “blue eye” phenomenon. *Vet Rec*. 1983;**112**(15):347–353.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 111.1 Vaccine-associated uveitis. Clinical presentation as a result of vaccine-associated uveitis. Corneal edema is notable.

Figure 111.2 Vaccine-associated uveitis. Note signs of inflammation including conjunctival hyperemia, a corneal vascular brush border, and corneal edema.

Figure 111.3 Vaccine-associated uveitis. Note that early buphthalmos has developed as a result of secondary glaucoma.

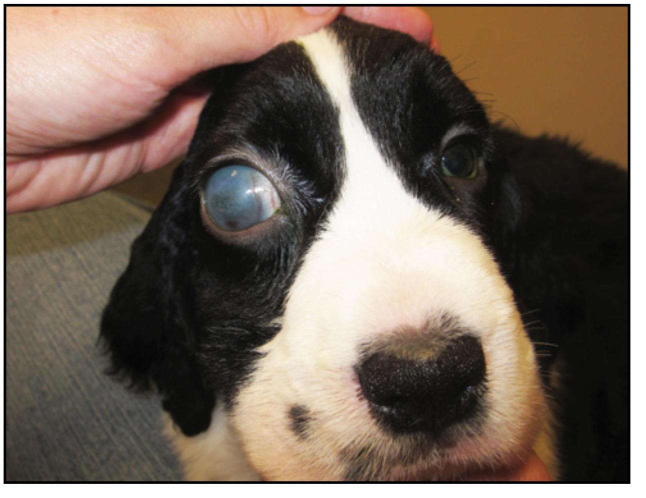


Figure 111.4 Vaccine-associated uveitis. Note significant buphthalmos as a result of secondary glaucoma.

CHAPTER 112
HYPHEMA

PRESENTATION

Hyphema describes the presence of free blood within the anterior chamber (AC). Clinically, the appearance of affected eyes may include varying degrees of tan to red discoloration of the AC (or in the case of longstanding hemorrhage, purple to black discoloration – sometimes referred to as "8-ball" hemorrhage). Hyphema presents a significant diagnostic challenge, potential causes including

- Congenital anomalies (including CEA, RD, MOD, and persistent hyaloid vasculature)
- Trauma
- Coagulopathies (including hereditary, immune-mediated, toxic)
- Vasculopathies
- Severe uveitis
- Systemic disease (including infectious disease and/or hypertension)
- Neoplasia (particularly lymphoma)
- Retinal detachment

DIAGNOSIS AND TREATMENT

The diagnosis of hyphema is made based on clinical findings. Potentially life-threating injuries/disease should be addressed, and the patient stabilized prior to further ophthalmic investigation/treatment. B-mode ultrasonography permits assessment of intraocular structures and scleral integrity in the presence of severe hyphema. Routine complete blood count (CBC), chemistry infectious-titer testing, coagulation times, and blood-pressure measurement are generally indicated when pursuing the diagnostic algorithm for hyphema. Radiography will rule out the presence of penetrating metallic foreign bodies such as air gun pellets and/or the presence of orbital fractures. Bilateral hyphema should raise suspicion of systemic disease/toxicity and/or neoplasia. Medical treatment largely comprises topical and/or systemic anti-inflammatory therapy (typically steroidal, unless contra indicated) as well as addressing underlying pathology. The administration of mydriatic agents when treating severe hyphema is controversial, shorter-acting agents such as tropicamide, potentially being safer than longer acting agents like atropine. The intracameral injection of a fibrinolytic agent (tissue plasminogen activator) may be indicated in some cases, generally being administered within 72 hours of fibrin organization. Although, mild to moderate hyphema typically responds well to medical therapy, secondary glaucoma remains a persistent danger in cases of significant hyphema. Even if vision cannot be salvaged, the appropriate management of hemorrhage will minimize the risk of secondary glaucoma and globe loss (see also Secondary glaucoma).

SELECTED REFERENCES

Bayón A, Tovar MC, Fernández del Palacio MJ, Agut A. Ocular complications of persistent hyperplastic primary vitreous in three dogs. *Vet Ophthalmol*. 2001;**4**(1):35–40.

Mandell DC. Ophthalmic emergencies. *Clin Tech Small Anim Pract*. 2000;**15**(2):94–100.

Nelms SR, Nasisse MP, Davidson MG, Kirschner SE. Hyphema associated with retinal disease in dogs: 17 cases (1986–1991). *J Am Vet Med Assoc*. 1993;**202**(8):1289–1292.

Telle MR and Betbeze C. **Hyphema**: considerations in the small animal patient. *Top Companion Anim Med*. 2015;**30**(3):97–106.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 112.1 Hyphema. Hyphema as a result of warfarin-toxicity. Inset demonstrates multiple mucous membrane-associated petechial hemorrhages.

Figure 112.2 Hyphema. Hyphema as a result of retinal detachment secondary to chronic lens-induced uveitis in a diabetic patient.

Figure 112.3 Hyphema. Hyphema due to a penetrating foreign body (air gun pellet.)

Figure 112.4 Hyphema. Hyphema in association with systemic hypertension.

CHAPTER 113
AQUEOUS LIPIDOSIS

PRESENTATION

Aqueous lipidosis describes a condition in which elevated levels of lipoproteins move across an incompetent and/or overloaded blood-ocular-barrier (BAB) into the anterior chamber. Changes may occur spontaneously as a result of primary hyperlipoproteinemia, following significant dietary indiscretion, in association with underlying metabolic disease and/or as a result of anterior uveitis. The condition may be either unilateral or bilateral and typically develops acutely. Commonly affected breeds include the Beagle, Miniature Schnauzer, and Shetland Sheepdog.

DIAGNOSIS AND TREATMENT

The diagnosis of aqueous lipidosis is made based on clinical findings, with affected anterior chambers appearing cloudy/white. Focal, illuminated examination may reveal the presence of "rivulets" of clear aqueous within the anterior chamber. In some cases, lipid accumulation within the corneal tissue and/or lipemia of the retinal vasculature may also be noted. Routine complete blood count (CBC)/chemistry analysis is typically supportive of the clinical diagnosis; frequently reflecting elevated serum cholesterol and/or triglyceride levels. More specific "lipid panels" as well as further evaluation of hepatic, renal, pancreatic, and/or thyroid function may provide additional diagnostic information. Addressing underlying metabolic and/or dietary imbalances as well as concurrent uveitis, if present, typically results in rapid resolution of symptoms. In selected cases, treatment using fibrates (such as gemfibrozil) and/or statins (such as atorvastatin) may be indicated; however, the use of these products in canine patients has not been approved. Treatment typically encompasses the topical administration of (steroidal or nonsteroidal) anti-inflammatory agents as well as the judicious use of a short-term mydriatic agent such as tropicamide in order to stabilize the BAB and minimize the risk of secondary glaucoma. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration.

SELECTED REFERENCES

Bauer JE. Lipoprotein-mediated transport of dietary and synthesized lipids and lipid abnormalities of dogs and cats. *J Am Vet Med Assoc*. 2004;**224**(5):668–675.

Crispin S. Ocular lipid deposition and hyperlipoproteinaemia. *Prog Retin Eye Res*. 2002;**21**(2):169–224.

Mori N, Lee P, Muranaka S, Sagara F, Takemitsu H, Nishiyama Y, Yamamoto I, Yagishita M, Arai T. Predisposition for primary hyperlipidemia in Miniature Schnauzers and Shetland sheepdogs as compared to other canine breeds. *Res Vet Sci*. 2010;**88**(3):394–399. doi: 10.1016/j.rvsc.2009.12.003. Epub 2010 Jan 12.

Violette NP and Ledbetter EC. Lipemic **uveitis** and its etiologies in **dogs**: 75 cases. *Vet Ophthalmol*. 2019;**22**(5):577–583.

Xenoulis PG and Steiner JM. Lipid metabolism and hyperlipidemia in dogs. *Vet J*. 2010;**183**(1):12–21. doi: 10.1016/j.tvjl.2008.10.011. Epub 2009 Jan 23.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 113.1 Aqueous lipidosis. Clinical presentation associated with aqueous lipidosis. Inset demonstrates the frequently bilateral nature of the condition.

Figure 113.2 Aqueous lipidosis. Typical clinical presentation associated with bilateral aqueous lipidosis.

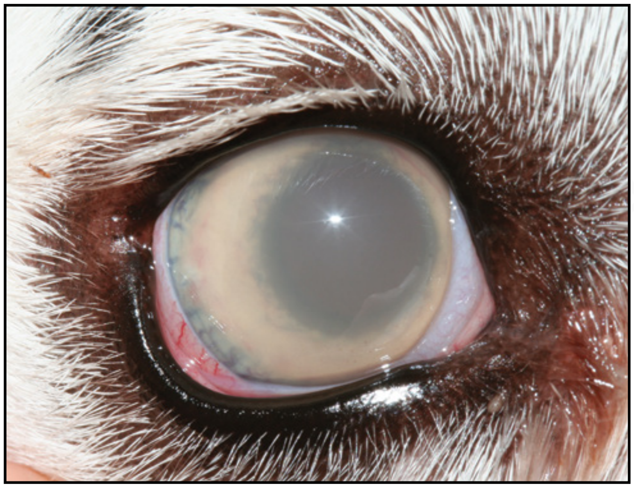


Figure 113.3 Aqueous lipidosis. Note signs of active anterior uveitis, including conjunctival hyperemia and iritis in addition to lipid aqueous.

Figure 113.4 Aqueous lipidosis. Note the characteristic "milky" appearance of the anterior chamber in this case.

CHAPTER 114
IRIS BOMBÉ

PRESENTATION

Iris bombé is not a specific diseases entity, rather a manifestation of post-inflammatory changes. Presentation comprises 360 synechiation around the pupillary opening, between the posterior iris and anterior lens capsule. As a result of impaired aqueous flow, iridal tissue "balloons" forward. Associated findings may include varying degrees of uveitis, hypopion, fibrin, hyphema, pigment deposition, cataract formation, and/or glaucoma. Both canine and feline species as well as any breed or crossed-breed may be affected by iris bombé.

DIAGNOSIS AND TREATMENT

The diagnosis of iris bombé is made based on clinical findings. Active uveitis should be managed if present (see also Feline and canine anterior uveitis). Similarly, elevated intraocular pressure (IOP) should be managed if present (see feline and canine secondary glaucoma). Surgical resection of iridal adhesions, laser iridotomy, and/or lenticular removal may be attempted in selected cases; however, the likelihood of recurrent iridal adhesion and/or secondary postoperative glaucoma is high. The presence if iris bombé reflects chronic pre-exiting intraocular inflammation and as a consequence, the prognosis for affected eyes is considered guarded.

SELECTED REFERENCES

Bentley E, Miller PE, Diehl KA. Use of high-resolution ultrasound as a diagnostic tool in veterinary ophthalmology. *J Am Vet Med Assoc*. 2003;**223**(11):1617–1622, 1599.

Deehr AJ and Dubielzig RR. A histopathological study of iridociliary cysts and glaucoma in Golden Retrievers. *Vet Ophthalmol*. 1998;**1**(2–3):153–158.

Sigle KJ, McLellan GJ, Haynes JS, Myers RK, Betts DM. Unilateral uveitis in a dog with uveodermatologic syndrome. *J Am Vet Med Assoc*. 2006;**228**(4):543–548.

Strubbe T. Uveitis and pupillary block glaucoma in an aphakic dog. *Vet Ophthalmol*. 2002;**5**(1):3–7.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

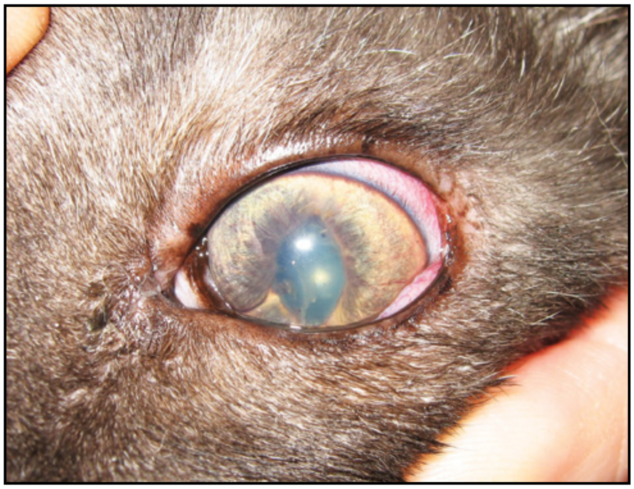


Figure 114.1 Iris bombé. Clinical presentation associated with feline iris bombé.

Figure 114.2 Iris bombé. Note associated hyphema in this case.

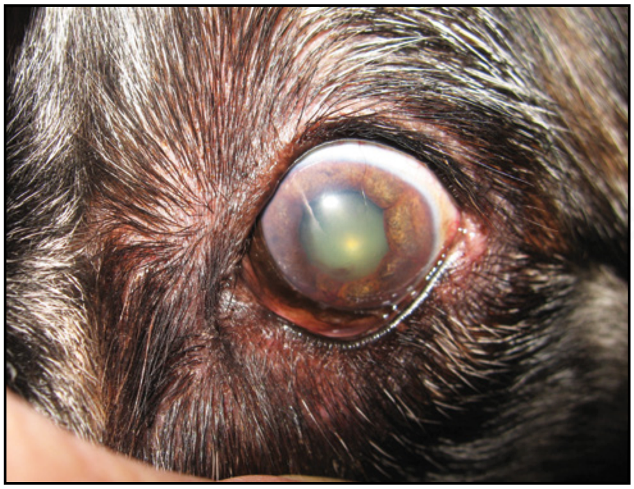


Figure 114.3 Iris bombé. Note early cataract formation secondary to chronic inflammation.

Figure 114.4 Iris bombé. Note posterior synechia and hyphema.

CHAPTER 115
FELINE IRIS MELANOSIS (FIM)

PRESENTATION

Clinically, feline iris melanosis (FIM) presents as one or more tan to brown, focal to coalescing iris nevi (areas of iridal pigmentation). Generally, the extent and density of these lesions increases slowly over time (typically over months to years). One or both eyes may be affected. Associated clinical uveitis is not usually present. Histologically, this process comprises the accumulation of pigmented melanocytes within the anterior iridal tissue. With time, some cases display deeper iris invasion and may additionally develop histologic characteristics of malignancy, representing true feline (diffuse) iris melanomas (FDIM), harboring the potential for metastatic spread of neoplastic cells to other regions of the body and potentially even death. Additionally, melanomas may result in progressive effacement of normal iridal tissue and/or invasion into iridocorneal drainage structures and/or adjacent uveal tissues and may result in severe secondary pathology, most commonly glaucoma.

DIAGNOSIS AND TREATMENT

The management of FIM (and its differentiation from iridis melanoma) represents a challenge to the practitioner. Options include

- benign neglect/clinical monitoring only (particularly relevant in cases displaying only minimally progressive focal nevi which are not elevated above the level of the iris)
- transcorneal diode laser photoablation of affected tissues (particularly relevant in cases showing relatively rapid progression or beginning to invade iridocorneal angle (ICA) structures)
- enucleation (particularly relevant in cases displaying changes suggestive of outright uveal neoplastic progression and/or secondary glaucoma). Clinical changes suggestive of neoplastic proliferation may include the development of "velvety" appearance to focal areas of pigmentation, followed by obvious elevation of pigmented tissue above the iridal surface and resultant pupillary dyscoria

In cases where neoplastic progression suspected, the evaluation of systemic health and/or involvement via local lymph node aspiration, three-view radiography and complete blood count (CBC)/chemistry analysis, is encouraged prior to enucleation.

SELECTED REFERENCES

Featherstone HJ, Scurrell EJ, Rhodes M, Pinheiro de Lacerda R. **Iris** biopsy to investigate **feline iris** hyperpigmentation. *Vet Ophthalmol.* 2020;**23**(2):269–276.

Grahn BH, Peiffer RL, Cullen CL, Haines DM. Classification of feline intraocular neoplasms based on morphology, histochemical staining, and immunohistochemical labeling. *Vet Ophthalmol.* 2006;**9**(6):395–403.

Kalishman JB, Chappell R, Flood LA, Dubielzig RR. A matched observational study of survival in cats with enucleation due to diffuse iris melanoma. *Vet Ophthalmol.* 1998;**1**(1):25–29.

Planellas M, Pastor J, Torres MD, Peña T, Leiva M. Unusual presentation of a metastatic uveal melanoma in a cat. *Vet Ophthalmol* 2010;**13**(6):391–394. doi: 10.1111/j.1463-5224.2010.00839.x.

Wiggans KT, Reilly CM, Kass PH, Maggs DJ. Histologic and immunohistochemical predictors of clinical behavior for **felinediffuse iris melanoma**. *Vet Ophthalmol.* 2016;**19** Suppl 1:44–55.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 115.1 Feline iris melanosis. Focal iris melanosis ("iris nevus".)

Figure 115.2 Feline iris melanosis. Diffuse iris melanosis.

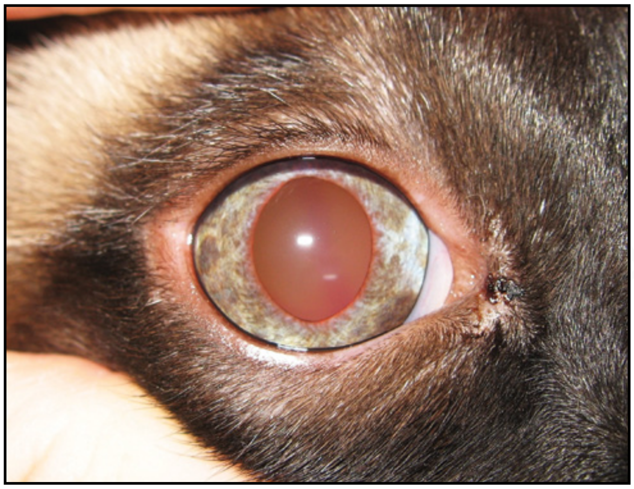


Figure 115.3 Feline iris melanosis. Diffuse iris melanosis demonstrating a "velvety" appearance.

Figure 115.4 Feline iris melanosis. Diffuse iris melanosis demonstrating iridal thickening.

CHAPTER 116
UVEAL MELANOMA

PRESENTATION

Melanocytic neoplasia of the uveal tract encompasses a spectrum extending from relatively benign melanocytoma to malignant melanoma (see also feline iris melanosis). Melanocytomas typically affect the anterior uveal tissues and only rarely extend beyond the globe or metastasize to other tissues. Malignant melanomas occur less frequently and may be more likely to metastasize or extend beyond the tissues of the globe. Less commonly, these tumors may arise from choroidal tissue. Clinically, uveal melanocytic tumors present as focal or multifocal areas of progressive iridal thickening and increased pigmentation. Rarely, amelanotic melanocytomas/melanomas may appear tan to pink in color. Secondary changes may include pupillary dyscoria, hyphema, retinal detachment, keratitis, and/or secondary glaucoma. Both feline and canine eyes may be affected by melanocytoma/melanoma. Commonly affected canine breeds include the Labrador Retriever and Golden Retriever.

DIAGNOSIS AND TREATMENT

A tentative diagnosis of uveal melanoma may be made based on clinical findings. Soft tissue imaging modalities including ultrasonography, computed tomography (CT), and/or magnetic resonance imaging (MRI) may also contribute to the clinical diagnosis; however, definitive diagnosis and the assessment of biologic behavior, is made based on histopatholgical evaluation of representative tissue, often following enucleation or evisceration. Treatment options include diode laser photoablation of neoplastic tissue where indicated, surgical resection of neoplastic tissue wherever possible or enucleation/evisceration of the globe. The evaluation of systemic health and/or potential metastasis via complete blood count (CBC)/chemistry analysis, local lymph node aspiration, and/or three-view radiography and is recommended prior to surgical intervention. The potential value of vaccine-mediated immunotherapy in the treatment of uveal melanomas has not been fully evaluated.

SELECTED REFERENCES

Badanes Z, Espinheira Gomes F, Ledbetter EC. **Choroidal** melanocytic tumors in **dogs**: A retrospective study. *Vet Ophthalmol*. 2020;**23**(6):987–993.

Galán A, Martín-Suárez EM, Molleda JM, Raya A, Gómez-Laguna J, Martín De Las Mulas J. Presumed primary uveal melanoma with brain extension in a dog. *J Small Anim Pract*. 2009;**50**(6):306–310.

Grahn BH, Sandmeyer LS, Bauer B. Diagnostic ophthalmology. extrascleral extension of an uveal melanoma. *Can Vet J*. 2008;**49**(7):723–724.

Yi NY, Park SA, Park SW, Jeong MB, Kang MS, Jung JH, Choi MC, Kim DY, Nam TC, Seo KM. Malignant ocular melanoma in a dog. *J Vet Sci*. 2006;**7**(1):89–90.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 116.1 Uveal melanoma. Clinical presentation associated with canine uveal melanocytoma/melanoma.

Figure 116.2 Uveal melanoma. Note edema at site of corneal endothelial contact with the melanocytoma/melanoma.

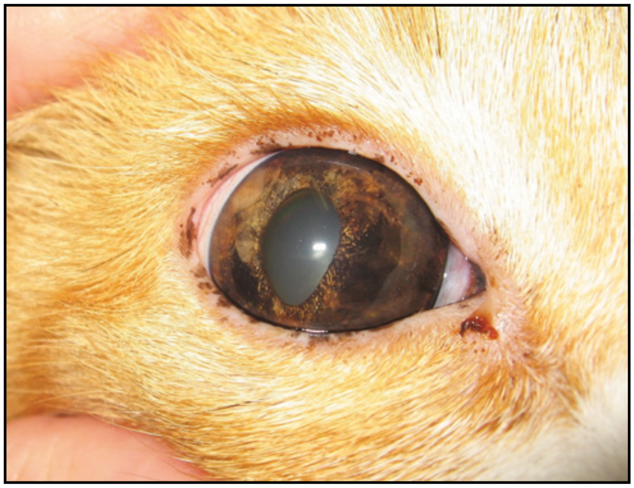


Figure 116.3 Uveal melanoma. Clinical presentation associated with feline uveal melanocytoma/melanoma.

Figure 116.4 Uveal melanoma. Note pupillary dyscoria and pigment on anterior lens capsule.

CHAPTER 117
UVEAL ADENOMA/ADENOCARCINOMA

PRESENTATION

Although not as common as uveal melanoma, iridal epithelial tumors are relatively common intraocular neoplasms. These tumors arise from the ciliary epithelium and typically present as slowly enlarging well-defined pink to tan colored masses, which may be noted to arise to emerge behind the pupil and/or deform the iridal tissue. Secondary intraocular complications may include uveitis, retinal detachment, hyphema, and/or glaucoma. The behavior of these tumors encompasses the spectrum from relatively benign adenomas (which although unlikely to metastasize typically result in globe loss through secondary complications) to adenocarcinomas, which behave in a more malignant fashion and may be more likely to metastasize to distant tissues. Both dogs and cats may be affected. Commonly affected canine breeds include the Labrador Retriever and Golden Retriever.

DIAGNOSIS AND TREATMENT

A tentative diagnosis may be made based on clinical findings. Definitive diagnosis and the assessment of biologic behavior however, is made based on histopathological evaluation of representative tissue, typically either following enucleation or evisceration. Treatment options include diode laser photoablation of neoplastic tissue where indicated, surgical resection of neoplastic tissue where indicated or enucleation/evisceration of the globe where indicated. The evaluation of systemic health and/or involvement via local lymph node aspiration, three-view radiography and complete blood count (CBC)/ Chemistry analysis, is encouraged prior to surgical intervention.

SELECTED REFERENCES

Davis RL, Reilly CM, Moore PA. Surgical excision of iridociliary tumors using a postero-anterior cyclo-iridectomy and thermocautery in two **dogs**. *Vet Ophthalmol*. 2020;**23**(3):579–587.

Dubielzig RR, Steinberg H, Garvin H, Deehr AJ, Fischer B. Iridociliary epithelial tumors in 100 dogs and 17 cats: a morphological study. *Vet Ophthalmol*. 1998;**1**(4):223–231.

Duke FD, Strong TD, Bentley E, Dubielzig RR. Canine ocular tumors following ciliary body ablation with intravitreal gentamicin. *Vet Ophthalmol*. 2013;**16**(2):159-162. doi: 10.1111/j.1463-5224.2012.01050.x. Epub 2012 Jul 19.

Zarfoss MK and Dubielzig RR. Metastatic iridociliary adenocarcinoma in a labrador retriever. *Vet Pathol*. 2007;**44**(5):672–676.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 117.1 Uveal adenoma/adenocarcinoma. Clinical presentation associated with uveal adenoma/adenocarcinoma.

Figure 117.2 Uveal adenoma/adenocarcinoma. Note vascular nature of mass.

Figure 117.3 Uveal adenoma/adenocarcinoma. Note deformation of iridal tissue.

Figure 117.4 Uveal adenoma/adenocarcinoma. Note pupillary dyscoria.

CHAPTER 118
UVEAL LYMPHOMA

PRESENTATION

Uveal lymphoma is a relatively common presentation, frequently occurring in combination with systemic disease, although ocular changes may precede systemic symptoms. One, or more commonly both, eye(s) may be affected and associated ocular findings may include any combination of corneal edema, anterior uveitis, chorioretinitis or panuveitis, hypopyon, hyphema, diffuse or nodular iridal thickening, retinal hemorrhage and/or detachment and/or glaucoma (see also Feline chorioretinitis and canine chorioretinitis, choroidal lymphoma and retrobulbar neoplasia). Systemic symptoms may include any combination of weight loss, lethargy, inappetence, gastrointestinal (GI) distress, pyrexia, lymphadenopathy, organomegally, hypercalcemia, and/or anemia. Both feline and canine species of any breed or crossed breed may be affected.

DIAGNOSIS AND TREATMENT

The diagnosis of lymphoma may be made based on clinical findings, ancillary imaging, cytological/histological interpretation, PCR analysis of samples obtained by aqueocentesis as well as lymph node and/or affected organ aspiration/biopsy. Treatment comprises topical anti-inflammatory therapy (typically utilizing a corticosteroid) as well as systemic chemotherapy. Associated ophthalmic changes such as elevated intraocular pressure should be addressed as appropriate. Staging via local lymph node (and/or organ/bone marrow) aspiration, three-view radiography, and complete blood count (CBC)/ chemistry analysis should be undertaken, prior to initiation of systemic chemotherapy. Diagnostic testing for the presence of infectious viruses including FelV, FIV, and FIP is additionally recommended in feline patients. Individual chemotherapeutic protocols are most ideally designed by a veterinary oncologist however typically include a variable combination of prednisone, vincristine, cyclophosphamide, and/or doxorubicin. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of vincristine include stomatitis, GI distress, neuropathy, hepatopathy, and myelosuppression. Potential adverse effects associated with the use of cyclophosphamide include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of doxyrubicin include hypersensitivity, GI distress, cardiac dysfunction, and myelosuppression. The prognosis varies depending on the extent of disease present prior to initiating of therapy.

SELECTED REFERENCES

Lanza MR, Musciano AR, Dubielzig RD, Durham AC. Clinical and pathological classification of canine intraocular **lymphoma**. *Vet Ophthalmol*. 2018;**21**(2):167–173.

Massa KL, Gilger BC, Miller TL, Davidson MG. Causes of uveitis in dogs: 102 cases (1989-2000). *Vet Ophthalmol*. 2002;**5**(2):93–98.

Nerschbach V, Eule JC, Eberle N, Höinghaus R, Betz D. Ocular manifestation of lymphoma in newly diagnosed cats. *Vet Comp Oncol*. 2013. doi: 10.1111/vco.12061.

Ota-Kuroki J, Ragsdale JM, Bawa B, Wakamatsu N, Kuroki K. Intraocular and periocular lymphoma in dogs and cats: a retrospective review of 21 cases (2001-2012). *Vet Ophthalmol*. 2013. doi: 10.1111/vop.12106.

Rutley M and MacDonald V. Managing the canine lymphosarcoma patient in general practice. *Can Vet J*. 2007;**48**(9):977–979.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 118.1 Uveal lymphoma. Diffuse iridal thickening and aqueous flare are present in this case.

Figure 118.2 Uveal lymphoma. Nodular iridal thickening, aqueous flare, and iritis are present in this case.

Figure 118.3 Uveal lymphoma. Note significant nodular iridal thickening.

Figure 118.4 Uveal lymphoma. Note diffuse iridal thickening, iritis, and fibrin/hypopyon accumulation.

CHAPTER 119
SPINDELOID TUMORS OF BLUE-EYED DOGS/ SCHWANNOMA

PRESENTATION

Primary canine intraocular spindeloid tumors are relatively uncommon. Despite this, a morphologically distinct spindle cell tumor has been identified which is associated with partial or complete blue eye coloration. Any breed or cross-breed may be affected; however, the Siberian Husky, Australian Shepherd, Border Collie, Labrador Retriever, and German Shepherd are overrepresented. Affected animals are usually late to middle-aged, with presentation typically unilateral. Initial clinical findings may include uveitis, pupillary dyscoria, and/or a uveal mass-effect with subsequent changes potentially including keratitis, cataract formation, hyphema, retinal detachment, glaucoma, and/or extrascleral extension. Neoplastic changes typically originate in iridociliary tissues and comprise a focal expansion of spindeloid neoplastic cells. These tumors are morphologically and immunohistochemically most consistent with schwannoma.

DIAGNOSIS AND TREATMENT

The diagnosis of a spindeloid tumor in a blue-eyed dogs/schwannoma is made based on a combination of clinical presentation and histopathologic interpretation (either via incisional biopsy or more commonly via evaluation of eviscerated/enucleated tissues), with enucleation frequently becoming indicated during the course of the disease. The potential for extension beyond orbital tissues as well as metastasis exists and presurgical staging (via local lymph node aspiration, three-view radiography and complete blood count [CBC]/chemistry evaluation) is warranted. If indicated, postoperative management may include chemo/metronomic therapy. The prognosis for affected animals following appropriately timed enucleation is typically positive.

SELECTED REFERENCES

Chijiwa K, Uchida K, Tateyama S. Immunohistochemical evaluation of canine peripheral nerve sheath tumors and other soft tissue sarcomas. *Vet Pathol*. 2004;**41**(4):307–318. doi: 10.1354/vp.41-4-307

Duke FD, Brudenall DK, Scott EM, Teixeira LB, Dubielzig RR. Metastatic uveal schwannoma of blue-eyed dogs. *Vet Ophthalmol*. 2013;**16** Suppl 1:141–144. doi: 10.1111/vop.12022. Epub 2013 Jan 2

Zarfoss MK, Klauss G, Newkirk K, Kiupel M, Jones Y, Colitz CM, Dubielzig RR. Uveal spindle cell tumor of **blue**-eyed dogs: an immunohistochemical study. *Vet Pathol*. 2007;**44**(3):276–284. doi: 10.1354/vp.44-3-276

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 119.1 Spindeloid tumors of blue-eyed dogs/schwannoma. Clinical presentation demonstrating uveal thickening and ectropion uvea.

Figure 119.2 Spindeloid tumors of blue-eyed dogs/schwannoma. Note diffuse iridal thickening.

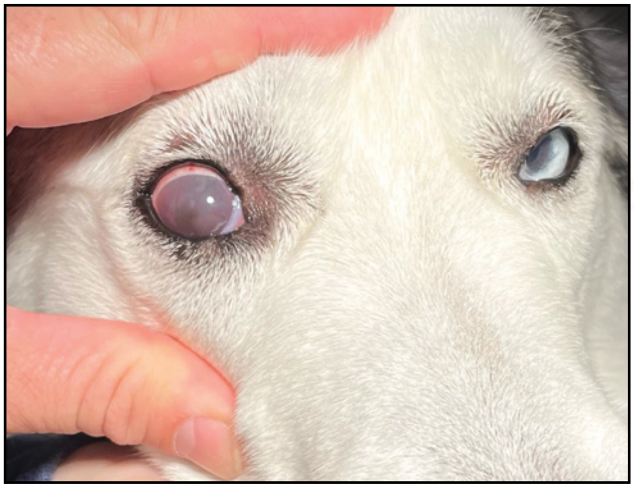


Figure 119.3 Spindeloid tumors of blue-eyed dogs/schwannoma. Note the presence of secondary uveitis, hyphema, and glaucoma

Figure 119.4 Spindeloid tumors of blue-eyed dogs/schwannoma. Note iridal thickening and infiltration, as well as iritis.

CHAPTER 120
HISTIOCYTIC OCULAR DISEASE

PRESENTATION

The histiocytic cell lineage represents two major immune components: phagocytosis (via macrophages) and antigen presentation (via dendritic cells). Histiocytic proliferative/neoplastic disorders are relatively commonly observed in dogs, less so in cats. The classification and nomenclature associated with histiocytic pathology can be confusing and their detailed description is beyond the scope of this text; however, the following broad categories are recognized.

- **Benign histiocytoma** This condition represents localized benign dermal proliferation affecting young dogs which may occur in close proximity to the ocular adnexa and is described in a previous chapter.
- **Cutaneous (proliferative) histiocytosis.** This condition represents the formation of multifocal dermal lesions which may or may not metastasize to regional lymph nodes and/or internal organs.
- Cats may specifically be affected by two variants of histiocytic disease; **feline pulmonary Langerhans cell histiocytosis (LCH)** and **feline progressive histiocytosis (FPH)** have been recognized as a cause of respiratory failure and dermal/multiorgan disease, respectively.
- **Systemic canine reactive histiocytosis** describes a complex inflammatory of diseases which occur secondary to underlying immune dysregulation. Affected animals may display peri-orbital lesions as well as keratouveitis of varying severity which may progress to secondary cataract formation and/or glaucoma and may or may not be accompanied by systemic disease (involving the lymphoid organs, lungs, liver, and/or bone marrow). Lesions are dominated by vasculitis/vasocentric infiltrates. Bernese mountain dogs are overrepresented.
- **Malignant histiocytic sarcoma** predominantly affects dogs (and occasionally cats) and typically originate within ocular, splenic, lymphoid, appendicular and/or marrow-associated tissues. Metastasis/spread is common. Neoplastic changes typically display marked anaplastic and mitotic activity. Affected globes typically display any combination of keratouveitis, retinal detachment, and/or glaucoma. Commonly affected breeds include the Bernese mountain dog, but also Rottweiler, Golden Retriever, Labrador, and Flat-coated Retrievers.

DIAGNOSIS AND TREATMENT

The diagnosis of histiocytic disease is made based on a combination of clinical presentation, systemic evaluation and the evaluation of samples obtained via aqueocentesis, aspiration, biopsy, or enucleation/evisceration. The enucleation of globes containing histiocytic sarcomas typically becomes indicated. The potential for local extension and/or metastasis exists and as a consequence, presurgical staging is appropriate. The management of systemic histiocytosis typically encompasses immunosuppressive agents including cyclosporin, leuflunomide, and/or azathioprine. A chemotherapeutic treatment protocol for the management of patients affected by malignant histiocytic sarcoma, where indicated, is most appropriately designed by a veterinary oncologist; however, typically includes agents like doxorubicin, vinblastine, CCNU and/or palladia. The prognosis following the enucleation of globes affected by localized disease is typically positive; however, the long-term prognosis for animals affected by systemic histiocytic disease is typically guarded to poor.

SELECTED REFERENCES

Moore PF, Affolter VK, Vernau W. Canine hemophagocytic histiocytic sarcoma: a proliferative disorder of CD11d+ macrophages. *Vet Pathol.* 2006;**43**(5):632–645. doi: 10.1354/vp.43-5-632. PMID: 16966440.

Naranjo C, Dubielzig RR, Friedrichs KR. Canine ocular histiocytic sarcoma. *Vet Ophthalmol.* 2007;**10**(3):179–185. doi: 10.1111/j.1463-5224.2007.00534.x. PMID: 17445080.

Rosin A, Moore P, Dubielzig R. Malignant histiocytosis in Bernese Mountain dogs. *J Am Vet Med Assoc.* 1986;**188**(9):1041–1045. PMID: 3710888.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

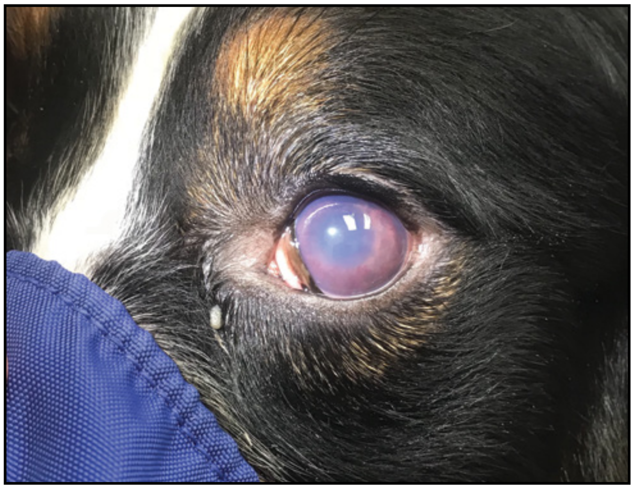


Figure 120.1 Histiocytic ocular disease. Clinical presentation associated with systemic histiocytic disease, note diffuse iridal thickening.

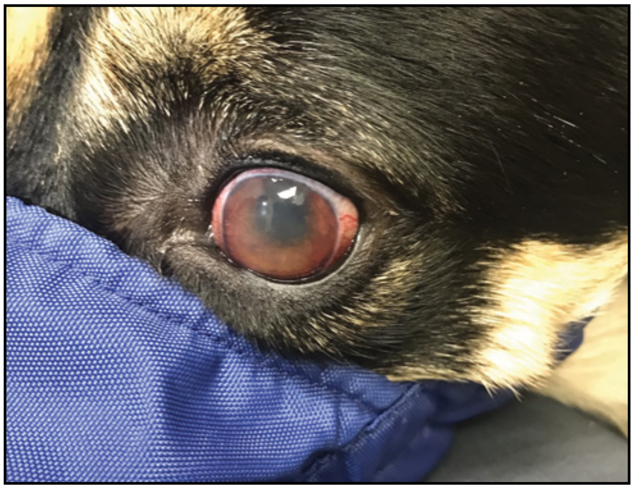


Figure 120.2 Histiocytic ocular disease. Note diffuse iridal thickening and anterior uveitis associated with systemic histiocytic disease.

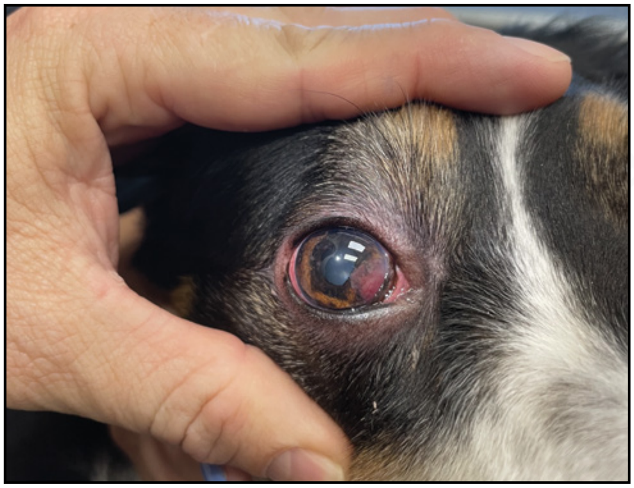


Figure 120.3 Histiocytic ocular disease. Clinical presentation associated with an intraocular histiocytic sarcoma.

Figure 120.4 Histiocytic ocular disease. Note deep perilimbal corneal neovascular infiltrate associated with severe intraocular inflammation and systemic histiocytic disease.

Section 9

Diseases of the Lens

CHAPTER 121
MICROPHAKIA/SPHEROPHKIA

PRESENTATION

Congenital microphakia describes an abnormally small lens. This condition results from abnormal embryonic differentiation and frequently occurs in combination with spherophakia (a spherically shaped lens). Other potential abnormalities include lentiglobus (a globoid shaped lens), lenticonus (a cone shaped lens), lenticular subluxation, and/or cataracts. Affected animals may additionally develop glaucoma. Commonly affected breeds include the Beagle, Doberman pincher, and Miniature Schnauzer and Burmese.

TREATMENT

The diagnosis of microphakia (and/or spherophakia) is made based on clinical findings, supported if necessary by B-mode ultrasonography. Treatment may include benign neglect, medical management using a long-term miotic agent in order to minimize the risk of complete lenticular luxation and/or surgical lens removal/replacement. Appropriate long-term miotic agents include demecarium bromide and the prostaglandin analogs (such as latanoprost). Potential complications associated with the use of demecarium bromide include ocular discomfort and gastrointestinal (GI) distress.

SELECTED REFERENCES

Arnbjerg J and Jensen O. Spontaneous microphthalmia in two Doberman puppies with anterior chamber cleavage syndrome. *J Am Anim Hos Assoc*. 1982;**18**, 481–484.

Binder DR, Herring IP, Gerhard T. Outcomes of nonsurgical management and efficacy of demecarium bromide treatment for primary lens instability in dogs: 34 cases (1990–2004). *J Am Vet Med Assoc*. 2007;**231**(1):89–93.

Curtis R. Lens luxation in the dog and cat. *Vet Clin North Am Small Anim Pract*. 1990;**20**(3):755–773.

Sebbag L, Riggs A, Carnevale J. Oculo-skeletal dysplasia in five Labrador Retrievers. *Vet Ophthalmol*. 2020;**23**(2):386–393.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 121.1 Microphakia/spherophakia. Clinical presentation associated with microphakia/spherophakia, note incipient cataract.

Figure 121.2 Microphakia/spherophakia. Note clearly visible stretched ciliary processes.

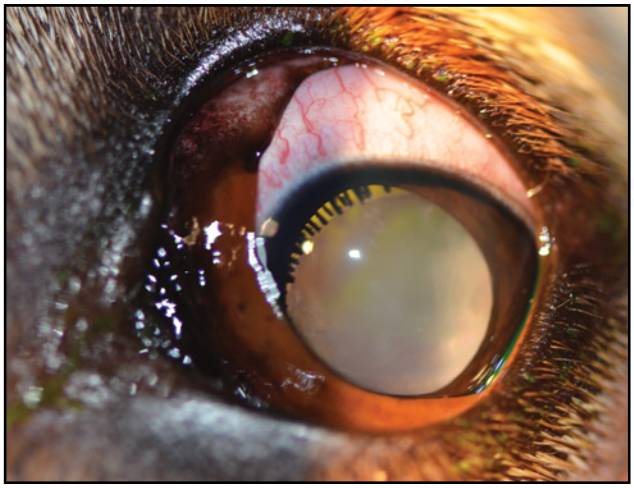


Figure 121.3 Microphakia/spherophakia. Note lens subluxation.

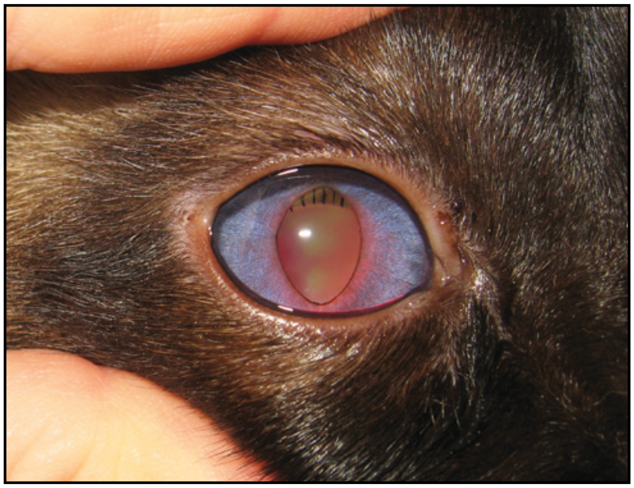


Figure 121.4 Microphakia/spherophakia. Note common feline clinical presentation in a Burmese.

CHAPTER 122
PERSISTANT HYALOID VASCULATURE (PHV)

PRESENTATION

During embryogenesis, the vascular supply to the developing lens regresses. Failure of this process results in the presence of visible (and in some cases patent) vascular remnants, associated with the lens and/or lens capsule, typically leading to cataract formation and visual impairment. Findings associated with this condition have been described as persistent hyperplastic tunica vasculosa lentis/persistent hyperplastic primary vitreous (PHTVL/PHPV). Commonly affected breeds include the Doberman Pincher, Greyhound, and Staffordshire Bull terrier.

TREATMENT

The diagnosis of PHV is made based on clinical findings (confirmed by B-mode ultrasonography where necessary). Where indicated, the lens and opacified lens capsule as well as associated vascular tissue may be surgically removed and patent vessels cauterized; however, the risk of intraoperative and postoperative complication associated with this procedure is significant.

SELECTED REFERENCES

Bayón A, Tovar MC, Fernández del Palacio MJ, Agut A. Ocular complications of persistent hyperplastic primary vitreous in three dogs. *Vet Ophthalmol*. 2001;**4**(1):35–40.

Boevé MH, van der Linde-Sipman JS, Stades FC. Early morphogenesis of the canine lens capsule, tunica vasculosa lentis posterior, and anterior vitreous body. A transmission electron microscopic study. *Graefes Arch Clin Exp Ophthalmol*. 1989;**227**(6):589–594.

Cullen CL and Grahn BH. Diagnostic ophthalmology. Persistent hyperplastic tunica vasculosa lentis and primary vitreous. *Can Vet J*. 2004;**45**(5):433–434.

Gemensky-Metzler AJ and Wilkie DA. Surgical management and histologic and immunohistochemical features of a cataract and retrolental plaque secondary to persistent hyperplastic tunica vasculosa lentis/persistent hyperplastic primary vitreous (PHTVL/PHPV) in a Bloodhound puppy. *Vet Ophthalmol*. 2004;**7**(5):369–375.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

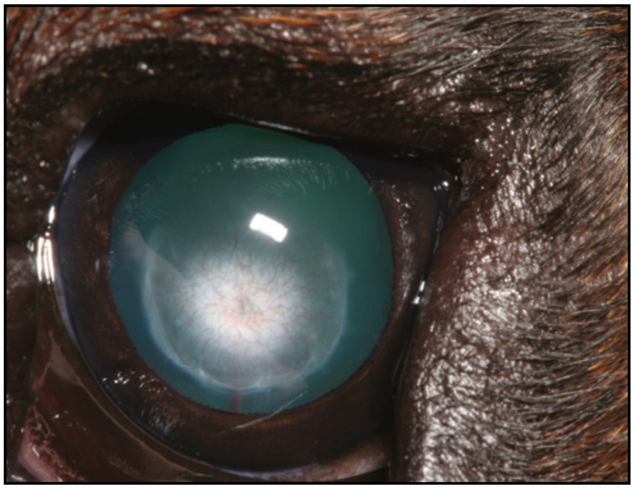


Figure 122.1 Persistant hyaloid vasculature. Note fine vessels radiating across the posterior lens capsule.

Figure 122.2 Persistant hyaloid vasculature. Note free hemorrhage within the cataractous lens.

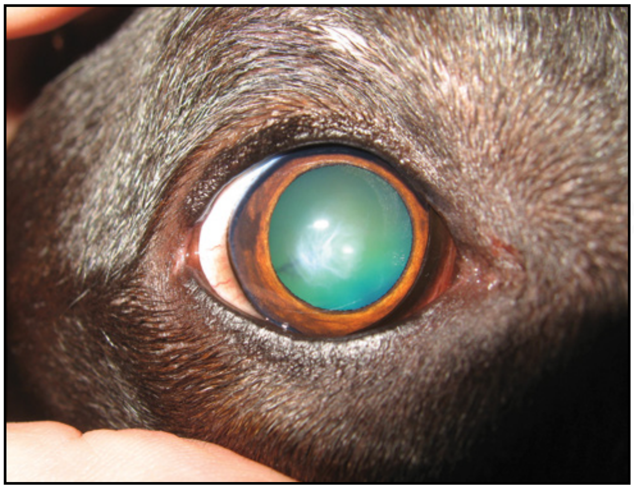


Figure 122.3 Persistant hyaloid vasculature. Note the single vessel extending to the posterior lens capsule and the focal posterior lens capsule cataract.

Figure 122.4 Persistant hyaloid vasculature. Note the fine vessels radiating across the posterior lens capsule.

CHAPTER 123
NUCLEAR SCLEROSIS

PRESENTATION

Nuclear sclerosis (or "lenticular sclerosis") describes the progressive thickening of the canine and feline lens due to ongoing (age-associated) lens-fiber deposition and resultant compression of the lenticular nucleus. Clinically, this process imparts a gray-blue appearance to the nucleus, typically becoming apparent after 7–8 years of age.

DIAGNOSIS AND TREATMENT

The diagnosis of nuclear sclerosis is made based on clinical findings. The major differential is a lenticular cataract; however, (unlike a cataract) nuclear sclerosis does not prevent the uninterrupted observation of a complete tapetal reflex. Although a degree of refractive error may be associated with aging changes, significant visual impairment is not generally noted clinically and neither medical nor surgical treatment of this condition is typically warranted.

SELECTED REFERENCES

Fischer CA. Geriatric ophthalmology. *Vet Clin North Am Small Anim Pract*. 1989;**19**(1):103–123.

Murphy CJ, Zadnik K, Mannis MJ. Myopia and refractive error in dogs. *Invest Ophthalmol Vis Sci*. 1992;**33**(8):2459–2463.

Poulos P Jr. Selenium-tocopherol treatment of senile lenticular sclerosis in dogs (four case reports). *Vet Med Small Anim Clin*. 1966;**61**(10):986–988.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 123.1 Nuclear sclerosis. Clinical presentation of age associated nuclear sclerosis. Note that tapetal reflection is clearly visible through sclerosis.

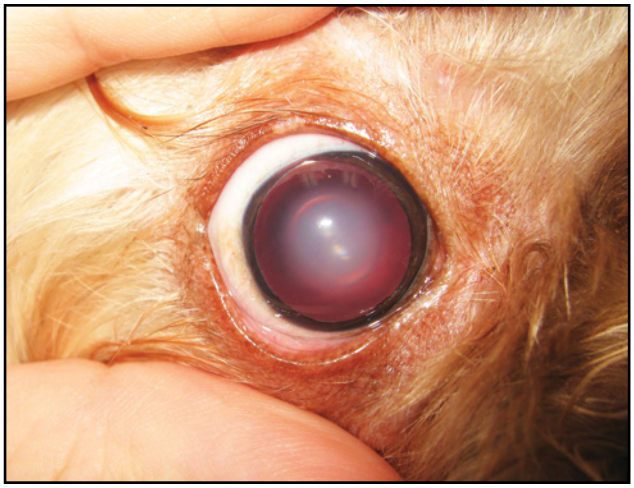


Figure 123.2 Nuclear sclerosis. Note focal, translucent clouding of the nucleus only.

Figure 123.3 Nuclear sclerosis. Advanced sclerosis in a geriatric patient.

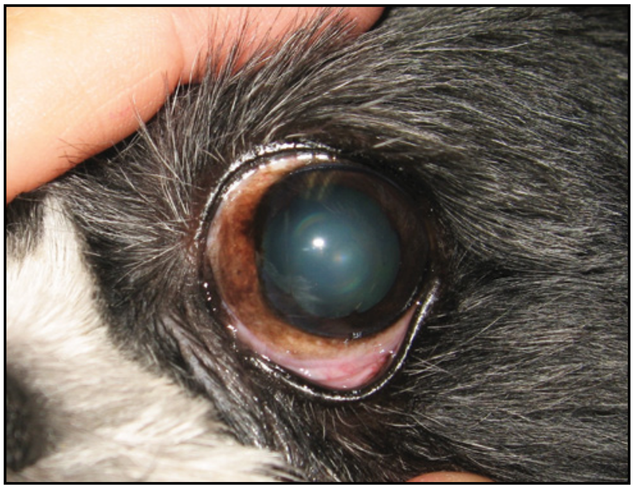


Figure 123.4 Nuclear sclerosis. Note the characteristic gray-blue appearance of the nucleus.

CHAPTER 124
IMMATURE CATARACT

PRESENTATION

A cataract describes any opacity of the normally transparent and avascular lens. Cataractogenesis occurs most commonly as a result of hereditary factors, however may also result from systemic disease (notably diabetes mellitus), lenticular trauma/inflammation, dietary deficiencies (notably associated with the use of milk-replacers), exposure to pharmacologic agents (notably ketoconazole), as a sequela to the retinal atrophies (RAs) and following exposure to therapeutic radiation. Changes may be unilateral or bilateral, symmetrical or asymmetrical, and may develop over varying timeframes. An "incipient" cataract describes opacification of less than 10% of the lens volume. An "immature" cataract describes a variable degree of lenticular opacification; however, the tapetal reflex remains present as does a variable degree of functional vision. Incipient/immature cataracts are not typically associated with overt uveitis. Many breeds (and mixed breeds) may be affected by hereditary cataracts and multiple breed-related features including age of onset and rate of progression have been described. Commonly affected breeds include the Boston Terrier, Bichon Frise, Cocker Spaniel, Labrador Retriever, miniature Schnauzer, miniature Poodle, and Siberian Husky.

TREATMENT

The diagnosis is made based on clinical findings. Treatment of immature cataracts is not typically necessary. Lens-associated uveitis, if present, should be treated with topical anti-inflammatory agents in order to minimize the risk of subsequent glaucoma (see also Phacolytic uveitis). If significant visual impairment is present, cataract surgery may be indicated (pending the results of presurgical diagnostic testing including electro-retinal function).

SELECTED REFERENCES

Basher AW and Roberts SM. Ocular manifestations of diabetes mellitus: diabetic cataracts in dogs. *Vet Clin North Am Small Anim Pract*. 1995;**25**(3):661–676.

Gelatt KN and Mackay EO. Prevalence of primary breed-related cataracts in the dog in North America. *Vet Ophthalmol*. 2005;**8**(2): 101–111.

Lim CC, Bakker SC, Waldner CL, Sandmeyer LS, Grahn BH. Cataracts in 44 dogs (77 eyes): a comparison of outcomes for no treatment, topical medical management, or phacoemulsification with intraocular lens implantation. *Can Vet J*. 2011;**52**(3):283–288.

Maehara S, Itoh N, Wakaiki S, Yamasaki A, Tsuzuki K, Izumisawa Y. The effects of **cataract** stage, lens-induced uveitis and **cataract** removal on ERG in **dogs** with **cataract**. *Vet Ophthalmol*. 2007;**10**(5):308–312.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 124.1 Immature cataract. Clinical presentation associated with an incipient/immature cataract. The tapetal reflex remains visible.

Figure 124.2 Immature cataract. Unilateral immature juvenile cataract in a young patient.

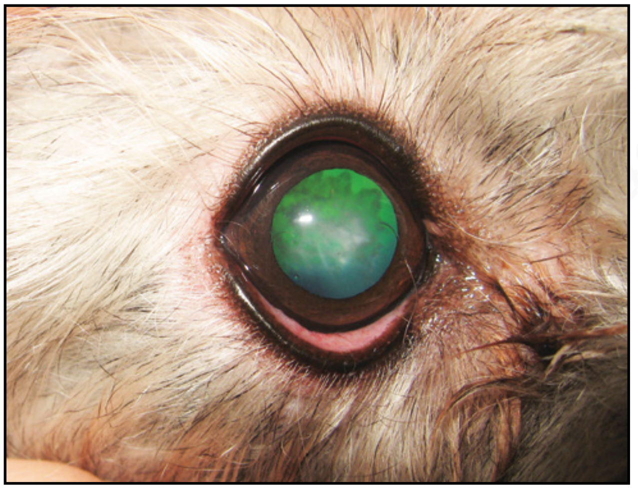


Figure 124.3 Immature cataract. Developing immature cataract associated with progressive retinal atrophy.

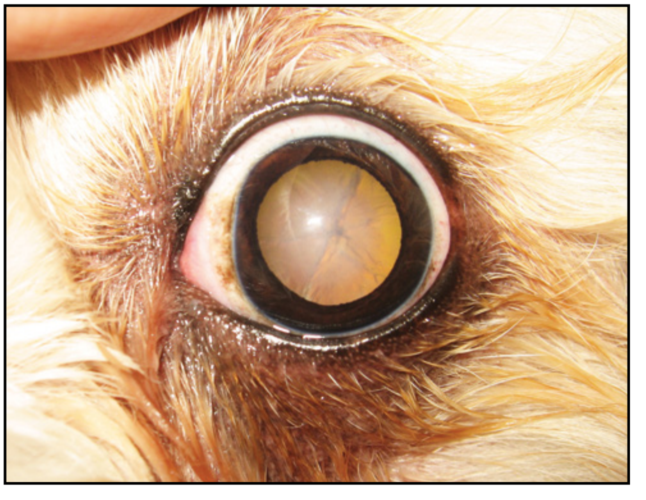


Figure 124.4 Immature cataract. Note that the tapetal reflex remains visible even in this more advanced immature cataract.

CHAPTER 125
MATURE CATARACT

PRESENTATION

A cataract describes any opacity of the normally transparent and avascular lens. Cataractogenesis occurs most commonly as a result of hereditary factors, however may also result from systemic disease (notably diabetes mellitus), lenticular trauma/inflammation, dietary deficiencies (notably associated with the use of milk-replacers), exposure to pharmacologic agents (notably ketoconazole), as a sequela to the retinal atrophies (RAs) and following exposure to therapeutic radiation. Changes may be unilateral or bilateral, symmetrical or asymmetrical, and may develop over varying timeframes. A “mature” cataract describes complete lenticular opacification, such that the tapetal reflex is obscured, and is associated with complete loss of functional vision. Leakage of lens proteins results in a variable degree of lens-associated inflammation (see Phacolytic uveitis). Osmotic action may result in fluid accumulation within the lens and subsequent enlargement of overall lenticular size (described as an “intumescent” cataract). Rapidly forming intumescent cataracts, such as those associated with uncontrolled diabetes, may result in equatorial lens-capsule rapture and severe, acute inflammation (see Phacolastic uveitis). Many breeds (and mixed breeds) may be affected by hereditary cataracts and multiple breed-related features including age of onset and rate of progression are described. Commonly affected breeds include the Boston Terrier, Bichon Frise, Cocker Spaniel, Labrador Retriever, miniature Schnauzer, miniature Poodle, and Siberian Husky.

TREATMENT

The diagnosis is made based on clinical findings. Lens-associated uveitis, if present, should be treated with topical anti-inflammatory agents in order to minimize the risk of subsequent glaucoma (see also Lens induced uveitis). Chronic, uncontrolled LIU may result in secondary complications including retinal detachment and/or glaucoma. Cataract surgery may be indicated (pending the results of presurgical diagnostic testing including electroretinal function).

SELECTED REFERENCES

Fischer MC and Meyer-Lindenberg A. Progression and complications of canine **cataracts** for different stages of development and aetiologies. *J Small Anim Pract*. 2018;**22**, doi: 10.1111/jsap.12910.

Gelatt KN and Mackay EO. Prevalence of primary breed-related cataracts in the dog in North America. *Vet Ophthalmol*. 2005;**8**(2):101–111.

Lim CC, Bakker SC, Waldner CL, Sandmeyer LS, Grahn BH. Cataracts in 44 dogs (77 eyes): a comparison of outcomes for no treatment, topical medical management, or phacoemulsification with intraocular lens implantation. *Can Vet J*. 2011;**52**(3):283–288.

Wilcock BP and Peiffer RL Jr. The pathology of lens-induced uveitis in dogs. *Vet Pathol*. 1987;**24**(6):549–553.

Wilkie DA and Colitz CM. Update on veterinary cataract surgery. *Curr Opin Ophthalmol*. 2009;**20**(1):61–68. doi: 10.1097/ICU.0b013e32831a98aa.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 125.1 Mature cataract. Clinical presentation associated with a mature cataract. The tapetal reflex is obscured completely.

Figure 125.2 Mature cataract. Note mild "clefting" of lens material associated with early intumescence.

Figure 125.3 Mature cataract. Note mydriasis associated with progressive retinal atrophy.

Figure 125.4 Mature cataract. Note significant "clefting" of lens material in association with intumescence.

CHAPTER 126
HYPERMATURE CATARACT

PRESENTATION

A cataract describes any opacity of the normally transparent and avascular lens. Cataractogenesis occurs most commonly as a result of hereditary factors, however may also result from systemic disease (notably diabetes mellitus), lenticular trauma/inflammation, dietary deficiencies (notably associated with the use of milk-replacers), exposure to pharmacologic agents (notably ketoconazole), as a sequela to the retinal atrophies (RAs) and following exposure to therapeutic radiation. Changes may be unilateral or bilateral, symmetrical or asymmetrical. A "hypermature" (or "Morgagnian") cataract describes a cataract in which the lens cortex has begun to liquefy and resorb. The lens nucleus may sink and the capsule shrink, fibrose, and calcify. Hypermature cataracts typically have a crystalline appearance and are frequently associated with symptoms of chronic uveitis. Variable degrees of tapetal reflectivity and/or visual impairment may be present. Many breeds (and mixed breeds) may be affected by hereditary cataracts and many breed-related features including age of onset and rate of progression are described. Commonly affected breeds include the Boston Terrier, Bichon Frise, Cocker Spaniel, Labrador Retriever, miniature Schnauzer, miniature Poodle, and Siberian Husky.

TREATMENT

The diagnosis is made based on clinical findings. Lens-associated uveitis, should be treated with topical anti-inflammatory agents (see also Phacolytic uveitis). Chronic, uncontrolled LIU may result in secondary complications including retinal detachment and/or glaucoma. Typically, steroidal agents are avoided when treating diabetic patients. Cataract surgery may be indicated (pending the results of presurgical diagnostic testing including electroretinal function). The potential for complications associated with the removal of hypermature cataracts is considered significant.

SELECTED REFERENCES

Gelatt KN and Mackay EO. Prevalence of primary breed-related cataracts in the dog in North America. *Vet Ophthalmol*. 2005;**8**(2):101–111.

Gonzalez-Alonso-Alegre E and Rodriguez-Alvaro A. Spontaneous resorption of a diabetic cataract in a geriatric dog. *J Small Anim Pract*. 2005;**46**(8):406–408.

Lim CC, Bakker SC, Waldner CL, Sandmeyer LS, Grahn BH. Cataracts in 44 dogs (77 eyes): a comparison of outcomes for no treatment, topical medical management, or phacoemulsification with intraocular lens implantation. *Can Vet J*. 2011;**52**(3):283–288.

Renzo R, Ribeiro AP, da Silva ML, da Silva GA, Ortencio KP, Barros Sobrinho AA, Mineo TW, Laus JL. Intraocular pressure, specular microscopy, and prostaglandin E2 concentration in **dogs** with mature and **hypermature cataract**. *Vet Ophthalmol*. 2014;**17**(4):280–285.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

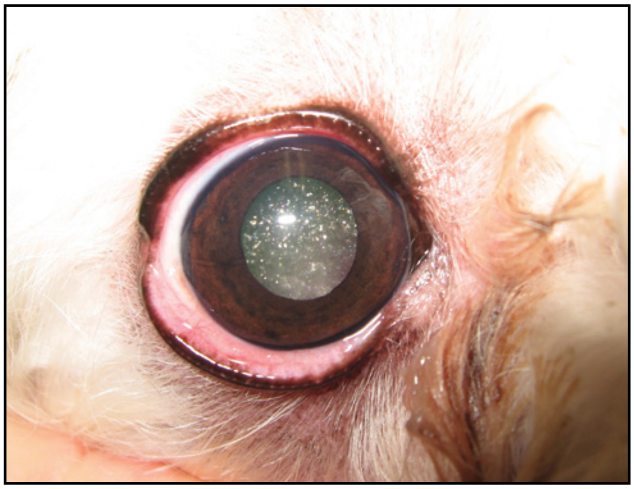


Figure 126.1 Hypermature cataract. Clinical presentation associated with the development of a hypermature cataract. Note crystalline lens material within liquified cortical lens material.

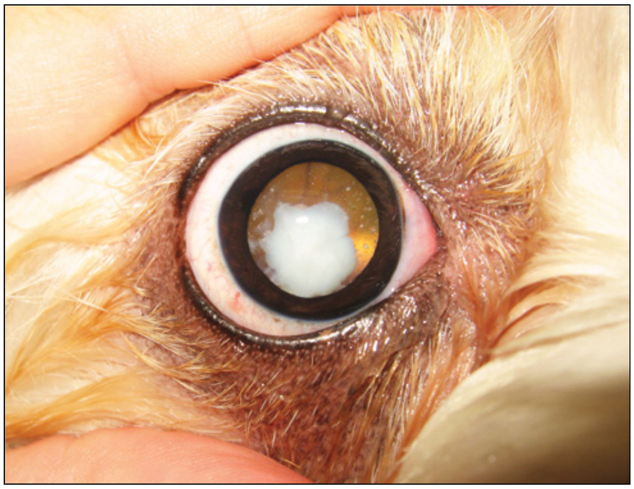


Figure 126.2 Hypermature cataract. Note that the nucleus is visible within liquified cortical lens material.

Figure 126.3 Hypermature cataract. Note multiple areas of iridal adhesion secondary to lens‐associated inflammation.

Figure 126.4 Hypermature cataract. Note shrunken lens capsule characteristic of hypermature cataracts.

CHAPTER 127
PHACOLYTIC UVEITIS

PRESENTATION

Phacolytic (or "lens induced") uveitis describes inflammation of the uveal tract as a result of the leakage of lens-associated proteins across an intact lens capsule (notably in the cases of rapidly forming cataracts such as those as associated with diabetes mellitus). Clinically, phacolytic uveitis may manifest as any combination of conjunctival/episcelral hyperemia, corneal edema, endothelial keratic precipitates, aqueous flare, iritis, iridal adhesions, pupillary miosis, uveal cyst formation, ectropion uvea and/or decreased intraocular pressure (IOP). Any breed or cross-breed may be affected.

TREATMENT

The diagnosis of phacolytic uveitis is made based on clinical findings, in conjunction with the presence of a cataract. Treatment of phacolytic uveitis should be undertaken in order to minimize the risk of secondary complications associated with chronic intraocular inflammation (notably retinal detachment and/or glaucoma). Treatment typically encompasses topical (and where necessary systemic) (steroidal or non-steroidal) anti-inflammatory therapy. Where possible the long-term use of steroidal agents is avoided in the management of diabetic patients. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Maehara S, Itoh N, Wakaiki S, Yamasaki A, Tsuzuki K, Izumisawa Y. The effects of cataract stage, **lens-induced uveitis** and cataract removal on ERG in **dogs** with cataract. *Vet Ophthalmol*. 2007;**10**(5):308–312.

Massa KL, Gilger BC, Miller TL, Davidson MG. Causes of uveitis in dogs: 102 cases (1989-2000). *Vet Ophthalmol*. 2002;**5**(2):93–98.

Wilcock BP and Peiffer RL Jr. The pathology of lens-induced uveitis in dogs. *Vet Pathol*. 1987;**24**(6):549–553.

van der Woerdt A, Nasisse MP, Davidson MG. Lens-induced uveitis in dogs: 151 cases (1985-1990). *J Am Vet Med Assoc*. 1992;**201**(6):921–926.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 127.1 Phacolytic uveitis. Notable is "ectropion uvea," visible as a crenelated pupillary margin.

Figure 127.2 Phacolytic uveitis. Endothelial keratic precipitates are present.

Figure 127.3 Phacolytic uveitis. Aqueous flare and endothelial keratic precipitates are present.

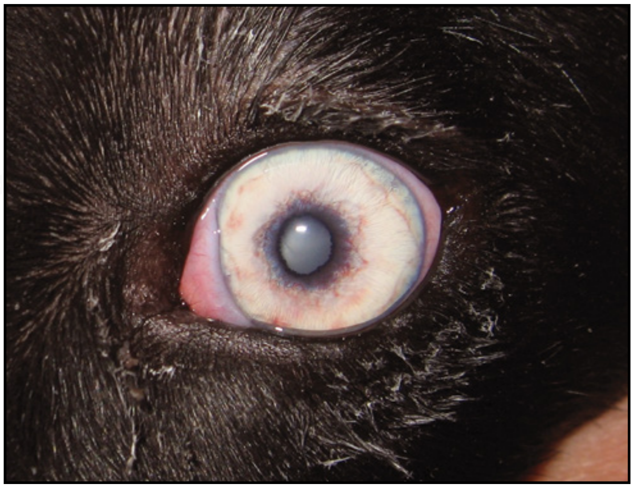


Figure 127.4 Phacolytic uveitis. Note moderate iritis associated with lens-induced uveitis.

CHAPTER 128
PHACOCLASTIC UVEITIS

PRESENTATION

Phacoclastic uveitis describes severe, acute intraocular inflammation as a result of the sudden exposure to lens-associated proteins following lens capsule lacteration or rupture. Rapidly forming "intumescent" cataracts are particularly predisposed to equatorial lens-capsule rupture. Clinically, phacoclastic may manifest as any combination of conjunctival/episcelral hyperemia, corneal edema, endothelial keratic precipitates, aqueous flare, hypopyon, hyphema, iritis, iridal adhesions, pupillary miosis, dyscoria, ectropion uvea, and decreased or elevated intraocular pressure (IOP). Any breed or crossed-breed may be affected.

TREATMENT

The diagnosis of phacoclastic uveitis is made based on history, clinical findings, and the presence of severe intraocular inflammation. Evaluation of lens capsular integrity may be challenging as a result of difficulties associated with examining a painful patient, corneal opacity (notably as a result of corneal edema), intracameral hemorrhage/hypion/fibrin, and/or pupillary miosis. B-mode ultrasonography may contribute to the diagnosis. Treatment typically encompasses aggressive topical and systemic (typically steroidal) anti-flammatory therapy. Lens removal by phacoemulsification is frequently necessary in order to control severe uveitis. A unique response to lenticular trauma in the feline species is the potential for subsequent sarcoma development (also see feline post traumatic sarcoma). Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Bell CM, Pot SA, Dubielzig RR. Septic implantation syndrome in dogs and cats: a distinct pattern of endophthalmitis with lenticular abscess. *Vet Ophthalmol.* 2013;**16**(3):180–185. doi: 10.1111/j.1463-5224.2012.01046.x.

Braus BK, Tichy A, Featherstone HJ, Renwick PW, Rhodes M, Heinrich CL. Outcome of phacoemulsification following corneal and **lens** laceration in cats and **dogs** (2000-2010). *Vet Ophthalmol.* 2017;**20**(1):4–10.

Paulsen ME and Kass PH. Traumatic corneal laceration with associated lens capsule disruption: a retrospective study of 77 clinical cases from 1999 to 2009. *Vet Ophthalmol.* 2012;**15**(6):355–368. doi: 10.1111/j.1463-5224.2011.00990.x.

Wilkie DA, Gemensky-Metzler AJ, Colitz CM, Bras ID, Kuonen VJ, Norris KN, Basham CR. Canine cataracts, diabetes mellitus and spontaneous lens capsule rupture: a retrospective study of 18 dogs. *Vet Ophthalmol.* 2006;**9**(5):328–334.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 128.1 Phacoclastic uveitis. Clinical presentation associated with phacoclastic uveitis secondary to lens capsule rupture.

Figure 128.2 Phacoclastic uveitis. Severe anterior uveitis and hyphema associated with lens capsule rupture.

Figure 128.3 Phacoclastic uveitis. Note corneal vascular "brush border" associated with phacoclastic uveitis.

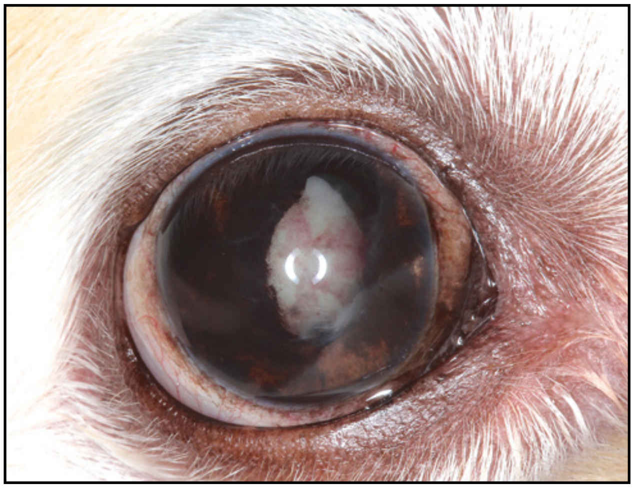


Figure 128.4 Phacoclastic uveitis. Note posterior synechia and pupillary dyscoria.

CHAPTER 129
ANTERIOR LENS LUXATION

PRESENTATION

The canine or feline lens may become dislocated from its normal position within the patellar fossa posterior to the iris. This may be result of hereditary factors (predominantly abnormalities of the suspensory lens zonules and/or vitreous body), chronic intraocular inflammation, vitreous syneresis, glaucoma, and/or trauma. Anterior lenticular luxation may be partial (termed "subluxation") or complete such that the lens becomes displaced into the anterior chamber. Associated changes may include corneal edema, uveitis, varying degrees of cataract formation, the presence of degenerate vitreous material within the pupillary opening and/or anterior chamber and decreased or elevated intraocular pressure (IOP). Changes may be chronic or acute. Commonly affected breeds include multiple terrier breeds (Sealyham, Jack Russell Wire Haired Fox, and miniature bull Terrier) the Australian Cattle Dog, German Shepherd, and Shar Pei.

TREATMENT

The diagnosis of anterior (sub) luxation is made based on clinical examination. Corneal edema and/or significant patient discomfort may render clinical diagnosis challenging, particularly in the case of non-cataractous lenses. Where indicated, diagnostic aides include sedation, anesthesia (topical or general), and/or B-mode ultrasonography. Subluxated lenses may be managed either medically (using anti-inflammatory therapy) or, more commonly, by surgical removal. The long-term use of a miotic agent in the contralateral eye may be indicated in an effort to prevent similar pathology from developing. Appropriate long-term miotic agents include demecarium bromide and the prostaglandin analogues (such as lataoprost). Miotic agents are however generally contraindicated when treating eyes affected by anterior (sub) luxation. Complete anterior lens luxation is most appropriately addressed via surgical intra capsular lens extraction (ICLE) and limited automated vitrectomy. Long-term postoperative anti-inflammatory therapy is typically indicated. Potential complications associated with lenticular (sub)luxation include chronic uveitis, retinal detachment, and/or glaucoma.

Potential complications associated with the use of demecarium bromide include ocular discomfort, and gastrointestinal (GI) distress.

SELECTED REFERENCES

Binder DR, Herring IP, Gerhard T. Outcomes of nonsurgical management and efficacy of demecarium bromide treatment for primary **lens** instability in **dogs**: 34 cases (1990–2004). *J Am Vet Med Assoc*. 2007;**231**(1):89–93.

Curtis R. Lens luxation in the dog and cat. *Vet Clin North Am Small Anim Pract*. 1990;**20**(3):755–773.

Glover TL, Davidson MG, Nasisse MP, Olivero DK. The intracapsular extraction of displaced lenses in **dogs**: a retrospective study of 57 cases (1984–1990). *J Am Anim Hosp Assoc*. 1995;**31**(1):77–81.

Nasisse MP and Glover TL. Surgery for **lens** instability. *Vet Clin North Am Small Anim Pract*. 1997;**27**(5):1175–1192.

Oberbauer AM, Hollingsworth SR, Belanger JM, Regan KR, Famula TR. Inheritance of **cataracts** and primary lens luxation in Jack Russell Terriers. *Am J Vet Res*. 2008;**69**(2):222–227. doi: 10.2460/ajvr.69.2.222.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

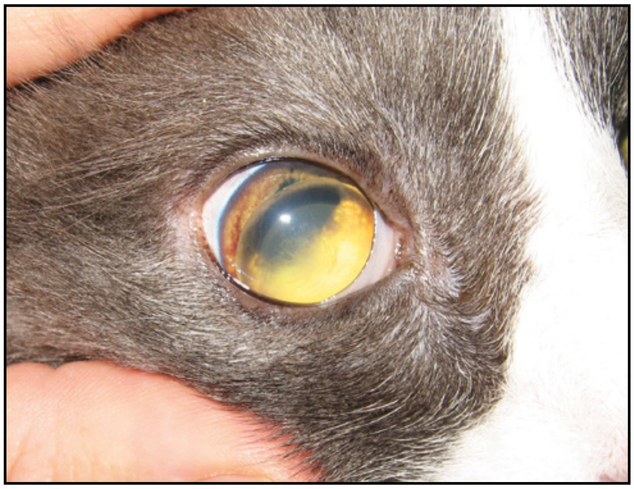


Figure 129.1 Anterior lens luxation. Clinical presentation associated with feline anterior lens luxation.

Figure 129.2 Anterior lens luxation. Note severe episcleral injection associated with secondary glaucoma as a result of anterior lens luxation.

Figure 129.3 Anterior lens luxation. Clinical presentation associated with acute canine anterior lens luxation.

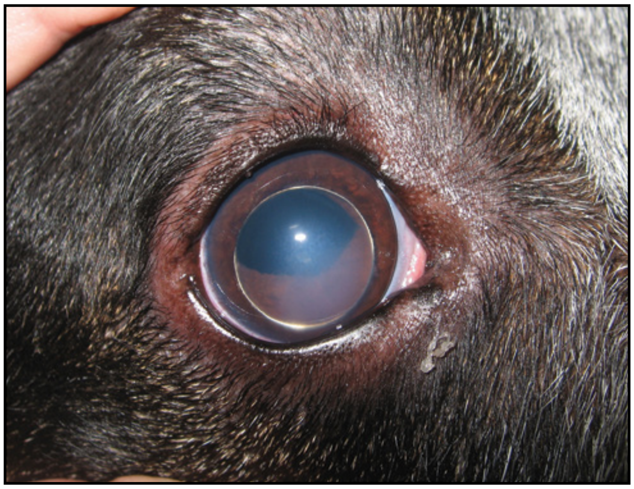


Figure 129.4 Anterior lens luxation. A significant secondary cataract has formed as a result of chronic anterior lens luxation.

CHAPTER 130
POSTERIOR LENS LUXATION

PRESENTATION

The canine or feline lens may become dislocated from its normal position within the patellar fossa posterior to the iris. This may be result of hereditary factors (predominantly abnormalities of the suspensory lens zonules and/or vitreous body), chronic intraocular inflammation, vitreous syneresis, glaucoma and/or trauma. Posterior lenticular luxation may be partial (termed "subluxation") or complete. Associated changes may include uveitis, varying degrees of cataract formation, the presence of degenerate vitreous material within the pupillary opening, and/or anterior chamber and decreased or elevated intraocular pressure (IOP). Changes may be chronic or acute. Commonly affected breeds include multiple terrier breeds (Sealyham, Jack Russell Wire Haired Fox, and miniature bull Terrier) the Australian Cattle Dog, the Chihuahua, Miniature Pincher, and Springer Spaniel.

TREATMENT

The diagnosis of posterior (sub) luxation is made based on clinical examination. Pathological shifting of the lens away from the central visual axis may result in the visualization of a semi-circular region of increased tapetal reflectivity (termed an "aphakic crescent"). B-mode ultrasonography may be used to further define lenticular position if necessary. Subluxated lenses may be managed either medically (using a combination of mitotic and/or anti-inflammatory therapy) or, where indicated, by surgical removal. Appropriate long-term miotic agents include demecarium bromide and the prostaglandin analogs (such as lataoprost). Long-term post-operative anti- inflammatory therapy is typically indicated. Potential complications associated with lenticular (sub)luxation include chronic uveitis, retinal detachment, and/or glaucoma. Potential complications associated with the use of demecarium bromide include ocular discomfort and gastrointestinal (GI) distress.

SELECTED REFERENCES

Binder DR, Herring IP, Gerhard T. Outcomes of nonsurgical management and efficacy of demecarium bromide treatment for primary lens instability in dogs: 34 cases (1990–2004). *J Am Vet Med Assoc*. 2007;**231**(1):89–93.

Curtis R. Lens luxation in the dog and cat. *Vet Clin North Am Small Anim Pract*. 1990;**20**(3):755–773.

Glover TL, Davidson MG, Nasisse MP, Olivero DK. The intracapsular extraction of displaced lenses in dogs: a retrospective study of 57 cases (1984–1990). *J Am Anim Hosp Assoc*. 1995;**31**(1):77–81.

Nasisse MP and Glover TL. Surgery for lens instability. *Vet Clin North Am Small Anim Pract*. 1997;**27**(5):1175–1192.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

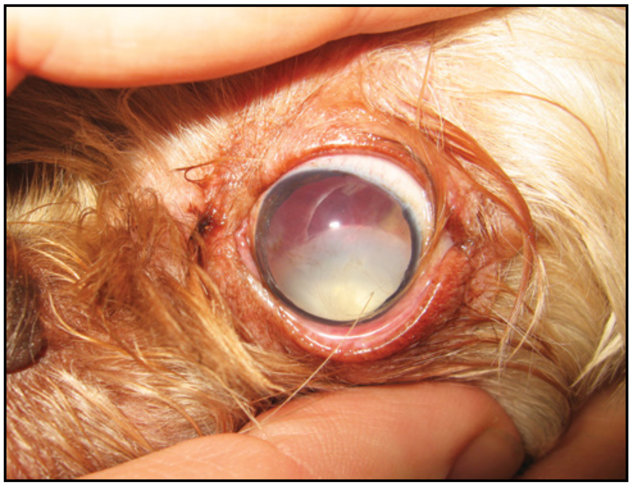


Figure 130.1 Posterior lens luxation. Note the development of a secondary cataract as a result of chronic posterior lens luxation.

Figure 130.2 Posterior lens luxation. Note severe episcleral injection associated with secondary glaucoma as a result of chronic posterior lens luxation.

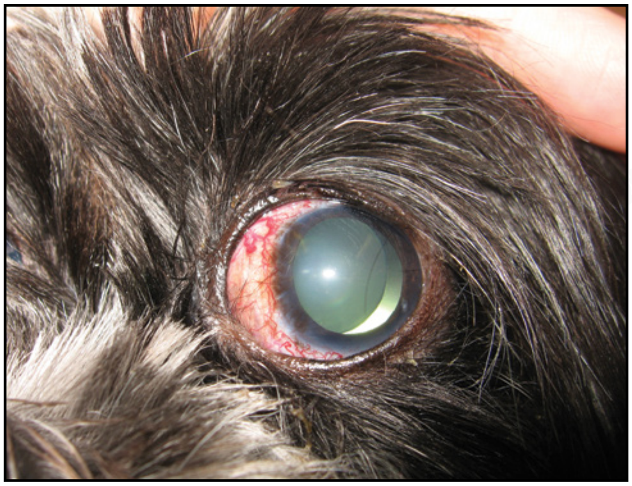


Figure 130.3 Posterior lens luxation. Note the presence of an "aphakic crescent" laterally.

Figure 130.4 Posterior lens luxation. Mild buphthalmos has developed as a result of chronic posterior lens luxation.

CHAPTER 131
FELINE POST TRAUMATIC SARCOMA

PRESENTATION

Feline post-traumatic ocular sarcoma (FPTOS) represents a malignant intraocular neoplasm, which may develop following severe inflammation and/or traumatic injury to the ocular structures (particularly the lens). Neoplastic changes typically take several years to develop. FPTOS encompasses several morphological variants, all of which are associated with the significant potential for extraocular extension and/or distant metastasis. Clinically, changes are typically unilateral and may include corneal edema, keratitis, corneal ulceration, uveitis, iridal thickening and/or dyscoria, the presence of an intraocular mass, hyphema, retinal detachment, glaucoma, and/or exophthalmos.

TREATMENT

The diagnosis of FPTOS is made based on clinical findings in conjunction with history. Soft tissue imaging modalities including B-mode ultrasonography, computed tomography (CT) and/or magnetic resonance imaging (MRI) may contribute to the clinical diagnosis, which is confirmed histologically either by biopsy or following enucleation/exenteration. The treatment of choice is enucleation (or exenteration where extraocular tumor extension is suspected/demonstrated). The evaluation of systemic health and/or potential metastasis via complete blood count (CBC)/chemistry analysis, local lymph node aspiration, and/or three-view radiography and is indicated prior to surgical intervention. In general, chemical ciliary body ablation (CBA), in order to manage glaucoma, is discouraged in cats due to the potential risk for the development of FPTOS.

SELECTED REFERENCES

Dubielzig RR, Everitt J, Shadduck JA, Albert DM. Clinical and morphologic features of post-traumatic ocular sarcomas in cats. *Vet Pathol*. 1990;**27**(1):62–65

Duke FD, Strong TD, Bentley E, Dubielzig RR. Feline ocular tumors following ciliary body ablation with intravitreal gentamicin. *Vet Ophthalmol*. 2013;**16** Suppl 1:188–190. doi: 10.1111/vop.12066.

Wood C and Scott EM. **Feline ocular post-traumatic sarcomas**: current understanding, treatment and monitoring. *J Feline Med Surg*. 2019;**21**(9):835–842.

Zeiss CJ, Johnson EM, Dubielzig RR. Feline intraocular tumors may arise from transformation of lens epithelium. *Vet Pathol*. 2003;**40**(4):355–362.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 131.1 Feline post-traumatic sarcoma. Clinical presentation associated with the development of a feline post-traumatic ocular sarcoma.

Figure 131.2 Feline post-traumatic sarcoma. Note the characteristic unilateral nature of the condition.

Figure 131.3 Feline post-traumatic sarcoma. Note secondary cataract formation.

Figure 131.4 Feline post-traumatic sarcoma. This patient previously suffered a penetrating corneal laceration.

Section 10

Vitreoretinal Disease

CHAPTER 132
THE RETINAL DYSPLASIAS (RDs)

PRESENTATION

The retinal dysplasias (RDs) represent a group of inherited (bilateral) retinal diseases, associated with the anomalous differentiation and proliferation of one of more layers (with or without the involvement of other ocular tissues). The majority of clinical cases occur as a result of hereditary disease; however, external factors such as viral infection (including Canine Adenovirus, Canine Herpes virus, Feline Panleukopenia), radiation exposure and toxicity may induce similar changes. The RDs may involve

- Multifocal linear folds of tissue (notably affecting the Labrador Retriever, American Cocker Spaniel, Beagle, Rottweiler, and Yorkshire Terrier)
- Large irregular or "geographic" areas of tissue (notably affecting the Labrador Retriever, Golden Retriever, English Springer Spaniel, and Cavalier King Charles Spaniel)
- Complete dysplasia with or without retinal detachment (notably affecting the Labrador Retriever, Australian Shepherd, Samoyed, Doberman Pincher, Akita, and Chow Chow)

DIAGNOSIS AND TREATMENT

The diagnosis of retinal dysplasia is made based on funduscopic examination typically possible from 6 to 8 week of age. In some cases, small lesions and folds may disappear by follow-up examination at 6 months to 1 year. Genetic testing is also available for multiple breed-related retinal dysplasias. No medical therapy is likely to be beneficial. Surgical management of retinal detachments may be indicated in selected cases.

SELECTED REFERENCES

Iwabe S, Dufour VL, Guzmán JM, Holle DM, Cohen JA, Beltran WA, Aguirre GD. Focal/multifocal and geographic **retinal dysplasia** in the dog-in vivo **retinal** microanatomy analyses. *Vet Ophthalmol*. 2020;**23**(2):292–304.

MacMillan A. Retinal dysplasia in the dog and cat. *Vet Clin North Am Small Anim Pract*. 1980;**10**(2):411–415.

Petersen-Jones S. Advances in the molecular understanding of canine retinal diseases. *J Small Anim Pract*. 2005;**46**(8):371–380.

Whiteley HE. Dysplastic canine retinal morphogenesis. *Invest Ophthalmol Vis Sci*. 1991;**32**(5):1492–1498.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

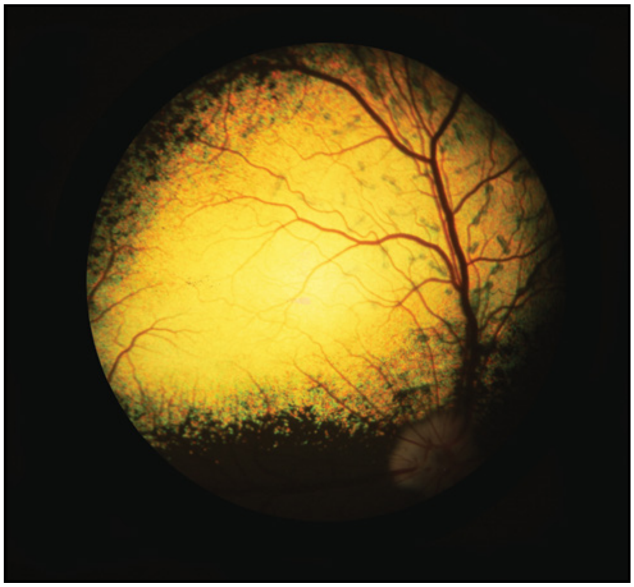


Figure 132.1 The retinal dysplasias. Funduscopic changes consistent with retinal dysplasia. Note multiple areas of linear to vermiform dysplasia.

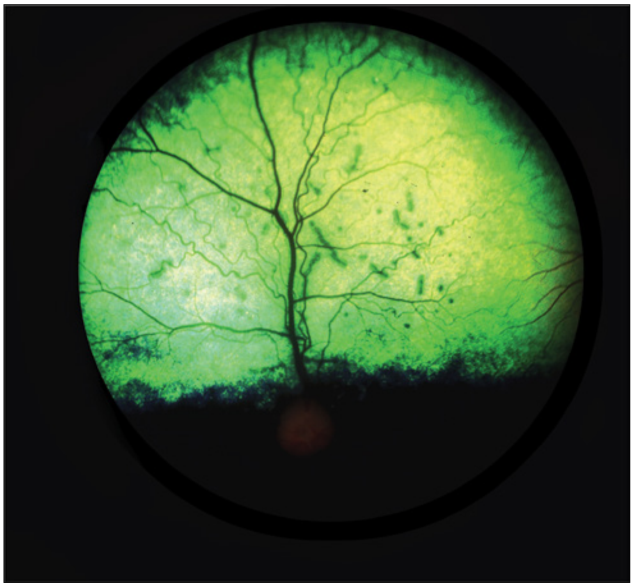


Figure 132.2 The retinal dysplasias. Funduscopic changes consistent with retinal dysplasia. Note multiple areas of linear to vermiform dysplasia.

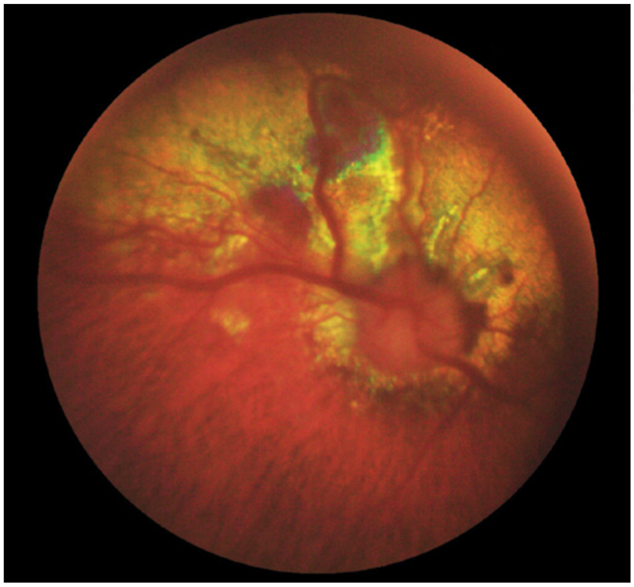


Figure 132.3 The retinal dysplasias. Funduscopic changes consistent with retinal dysplasia. Note large irregular areas of geographic dysplasia.

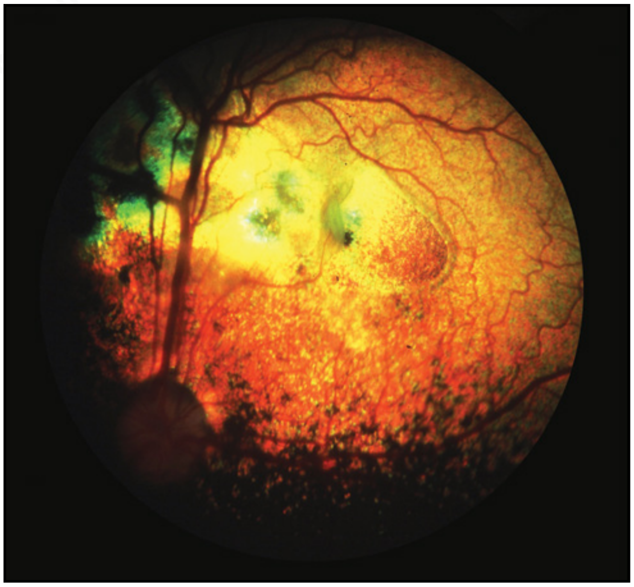


Figure 132.4 The retinal dysplasias. Funduscopic changes consistent with retinal dysplasia. Note large irregular areas of geographic dysplasia and an area of associated retinal detachment.

CHAPTER 133
OCULOSKELETAL DYSPLASIA (OSD)

PRESENTATION

Oculoskeletal dysplasia (OSD) represents a hereditary syndrome comprising varying combinations of osteochondrodysplasia and/or ocular pathology. Skeletal changes typically include shortened limbs (particularly the forelimbs), marked by varus deformities of the elbows and valgus deformities of the carpi, epiphyseal dysplasia, ununited anconeal/coronoid processes, and/or hip dysplasia. Ocular pathology may include any combination of cataracts, retinal folding/dysplasia, retinal detachment (complete or partial), hyaloid remnants, hyphema, and/or glaucoma. Affected breeds include the Labrador Retriever and Samoyed.

DIAGNOSIS AND TREATMENT

The diagnosis of OSD is made based on clinical findings. Ocular ultrasonography and skeletal radiography may support the diagnosis wherever necessary. Genetic testing for affected breeds is also available. Specific treatment is not available; however, secondary ocular complications including cataracts, hyphema, glaucoma, and/or retinal detachment may be managed as individually appropriate.

SELECTED REFERENCES

Carrig CB, Sponenberg DP, Schmidt GM, Tvedten HW. Inheritance of associated ocular and skeletal dysplasia in Labrador retrievers. *J Am Vet Med Assoc*. 1988;**193**(10):1269–1272.

Goldstein O, Guyon R, Kukekova A, Kuznetsova TN, Pearce-Kelling SE, Johnson J, Aguirre GD, Acland GM. COL9A2 and COL9A3 mutations in canine autosomal recessive oculoskeletal dysplasia. *Mamm Genome*. 2010;**21**(7–8):398–408. doi: 10.1007/s00335-010-9276-4.

Sebbag L, Riggs A, Carnevale J. Oculo-skeletal **dysplasia** in five Labrador Retrievers. *Vet Ophthalmol*. 2020;**23**(2):386–393.

Stavinohova R, Hartley C, Burmeister LM, Ricketts SL, Pettitt L, Tetas Pont R, Hitti RJ, Schofield E, Oliver JAC, Mellersh CS. Clinical, histopathological and genetic characterisation of **oculoskeletaldysplasia** in the Northern Inuit Dog. *PLoS One*. 2019;**14**(8):e0220761.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 133.1 Oculoskeletal dysplasia. Clinical presentation associated with oculoskeletal dysplasia, including forelimb varus defects. Inset demonstrates hyphema secondary to retinal detachment.

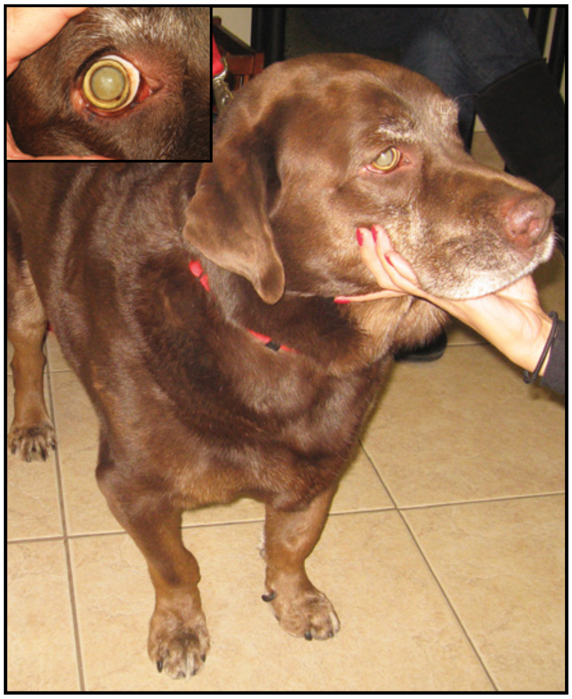


Figure 133.2 Oculoskeletal dysplasia. Inset demonstrates cataractous changes.

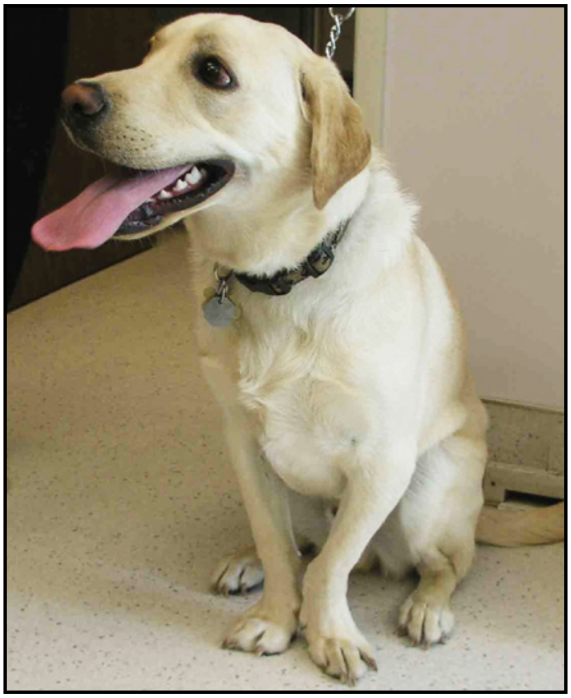


Figure 133.3 Oculoskeletal dysplasia. Characteristic clinical presentation associated with oculoskeletal dysplasia, note ocular abnormalities and forelimb varus defects.

Figure 133.4 Oculoskeletal dysplasia. Inset demonstrates cataractous changes.

CHAPTER 134
COLLIE EYE ANOMALY (CEA)

PRESENTATION

Collie Eye Anomaly (CEA) describes a, congenital, hereditary disease resulting from abnormal embryonic differentiation of ocular tissues. Changes may include any combination of choroidal vascular hypoplasia, retinal pigment epithelial (RPE), and/or tapetal defects, abnormal retinal vasculature, scleral colobomas (typically involving the region surrounding the optic nerve head), retinal dysplasia, and/or retinal detachment (partial or complete). Changes are bilateral but not always symmetrical. Most affected animals are clinically asymptomatic, abnormalities only being detected upon fundic examination; however, patients may present as a result of symptoms associated with retinal detachment and/or hyphema. Commonly affected breeds include the Collie breeds, Australian Shepherd, and Shetland Sheepdog.

DIAGNOSIS AND TREATMENT

The diagnosis of CEA is made based on signalment in combination with clinical findings. Genetic testing is also available for multiple breed-related CEAs. Primary treatment of this disorder is not possible; however, secondary complications, such as hyphema, may be treated symptomatically. Surgical intervention in order to address scleral or retinal pathology may be indicated in select cases. Selective breeding reduces the incidence and severity of disease.

SELECTED REFERENCES

Lowe JK, Kukekova AV, Kirkness EF, Langlois MC, Aguirre GD, Acland GM, Ostrander EA. Linkage mapping of the primary disease locus for collie eye anomaly. *Genomics*. 2003;**82**(1):86–95.

Munyard KA, Sherry CR, Sherry L. A retrospective evaluation of congenital ocular defects in Australian Shepherd dogs in Australia. *Vet Ophthalmol*. 2007;**10**(1):19–22.

Parker HG, Kukekova AV, Akey DT, Goldstein O, Kirkness EF, Baysac KC, Mosher DS, Aguirre GD, Acland GM, Ostrander EA. Breed relationships facilitate fine-mapping studies: a 7.8-kb deletion cosegregates with Collie eye anomaly across multiple dog breeds. *Genome Res*. 2007;**17**(11):1562–1571.

Wallin-Håkanson B, Wallin-Håkanson N, Hedhammar A. Influence of selective breeding on the prevalence of chorioretinal dysplasia and coloboma in the rough collie in Sweden. *J Small Anim Pract*. 2000;**41**(2):56–59.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

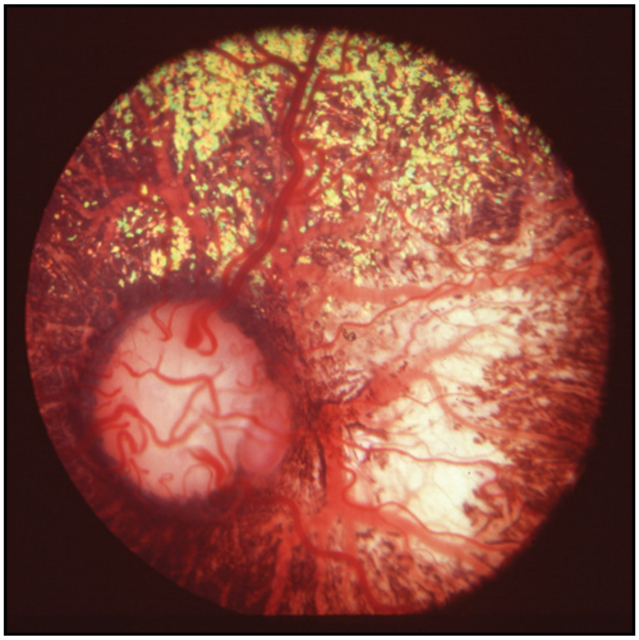


Figure 134.1 Collie aye anomaly. Funduscopic changes consistent with Collie eye anomaly including scleral ectasia, choroidal hypoplasia, abnormal vasculature, and posterior segment colobomas.

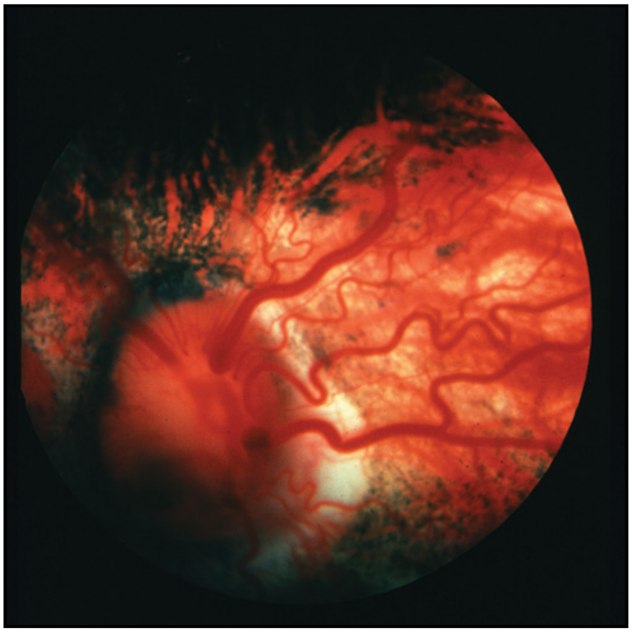


Figure 134.2 Collie aye anomaly. Funduscopic changes consistent with Collie eye anomaly including scleral ectasia, choroidal hypoplasia, abnormal vasculature and posterior segment colobomas.

Figure 134.3 Collie aye anomaly. Funduscopic changes consistent with Collie eye anomaly including scleral ectasia, choroidal hypoplasia, abnormal vasculature, and posterior segment colobomas.

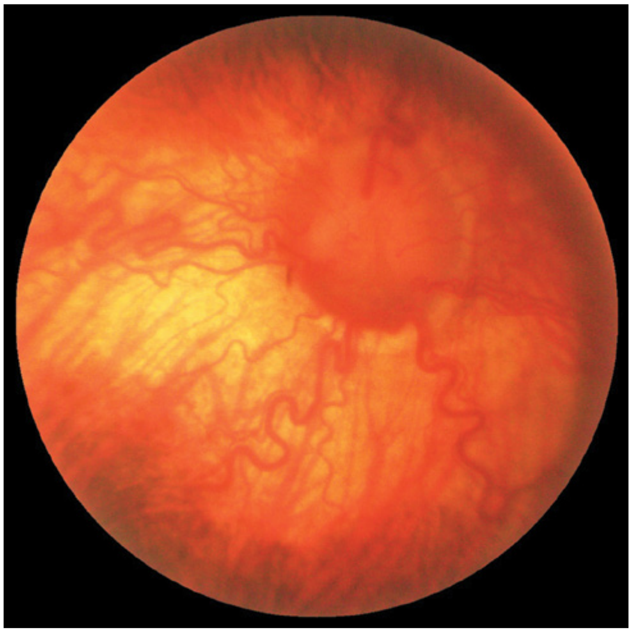


Figure 134.4 Collie aye anomaly. Funduscopic changes consistent with Collie eye anomaly including scleral ectasia, choroidal hypoplasia, abnormal vasculature, and posterior segment colobomas.

CHAPTER 135
THE RETINAL ATROPHIES (RAs)

PRESENTATION

The retinal atrophies (RAs) represent a group of inherited (bilateral) retinal diseases, which result in photoreceptor dysfunction and death, leading to visual impairment and ultimately blindness. Various forms have been described as affecting a large (and increasing) number of dog breeds as well as some cat breeds. The classification of the retinal atrophies is complex, however may be simplified into early and late onset progressive retinal atrophy (PRA), congenital stationary night blindness (CSNB), cone degenration ("day blindness") and the feline RAs.

The early onset (dysplastic) canine RAs include;

- Rod-cone dysplasia (RCD), notably affecting the Irish Setter, Miniature Schnauzer, Norwegian Elkhound, Dachshund, Cardigan Welsh Corgi and Collies
- X-Linked PRA (xlpra), notably affecting the Samoyed and Siberian Huskie
- Early retinal degeneration (ERD), notably affecting the Norwegian Elkhound

The late onset (degenerative) canine RAs include

- Progressive rod cone degeneration (PRCD), notably affecting the Labrador Retriever, Chesapeake Bay Retriever, Nova Scotia Duck-Tolling Retriever, American Cocker Spaniel, American Eskimo, Australian Cattle Dog, Chinese Crested, Miniature Poodle, Toy Poodle, Akita and Finnish Lapphund
- Dominant PRA, notably affecting the Old English and Bull Mastiff
- Retinal Pigment Epithelial Dystrophy (RPED), notably affecting the Labrador Retriever, Golden Retriever, Chesapeake Bay Retriever and Collie breeds

These diseases typically manifest initially as relative mydriasis as well as loss of night-vision (nyctalopia). The onset of symptoms is variable, however, may be noticed in the early onset RAs between 6 weeks and 6 months of age (typically progressing to blindness by 1–5 years of age) and in the late onset RAs between 3 and 5 years of age (typically progressing to complete blindness by 6–8 years of age). CSNB, represents a condition in which affected dogs are congenitally night blind and may additionally develop decreased day vision over the course of several years. The Briard is notably reported as affected by this condition. Cone degeneration (also described as "day blindness" or "hemeralopia"), represents a condition in which affected dogs demonstrate early onset and rapidly progressive loss of cone function so that visual function is lost under daylight conditions. The Alaskan Malamute and German Shorthaired Pointer are notably affected. The feline retinal atrophies are less well described; however largely follow the same natural course with early disease present by 1–2 years of age and advanced disease present by 3–6 years of age. Cat breeds known to be affected by various forms of retinal atrophy include the Abyssinian, Somali, Bengal, and Persian.

DIAGNOSIS AND TREATMENT

The diagnosis of the RAs may be made based on multiple criteria, including clinical findings, funduscopic visualization (varying degrees of tapetal hyperreflectivity, vascular attenuation and ONH degeneration), and/or ERG testing. Genetic testing is also available for multiple breed-related RAs. Accurate gene-based testing facilitates breeding programs designed to help reduce the incidence of disease within a breed. Many patients will develop secondary cataracts over time. The administration of antioxidant containing products has been advocated in effort to slow the progression of disease, however will not prevent blindness.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

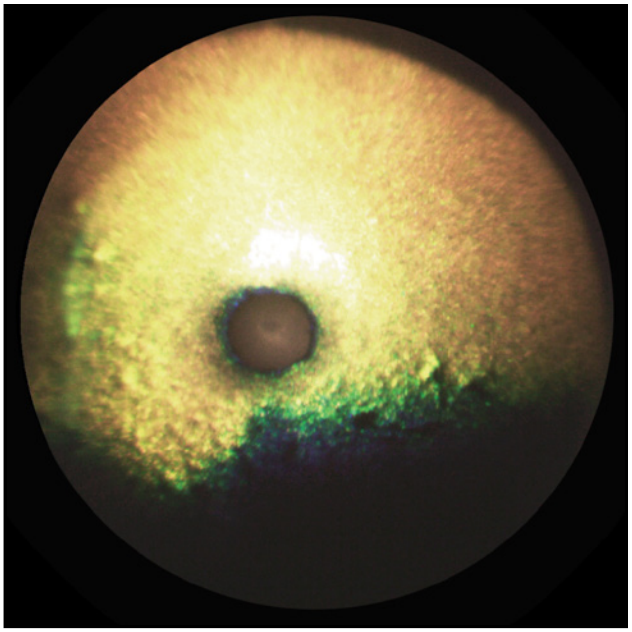


Figure 135.1 The retinal atrophies. Funduscopic findings associated with retinal atrophy, including increased tapetal reflectivity and marked vascular attenuation. Note associated optic nerve head degeneration.

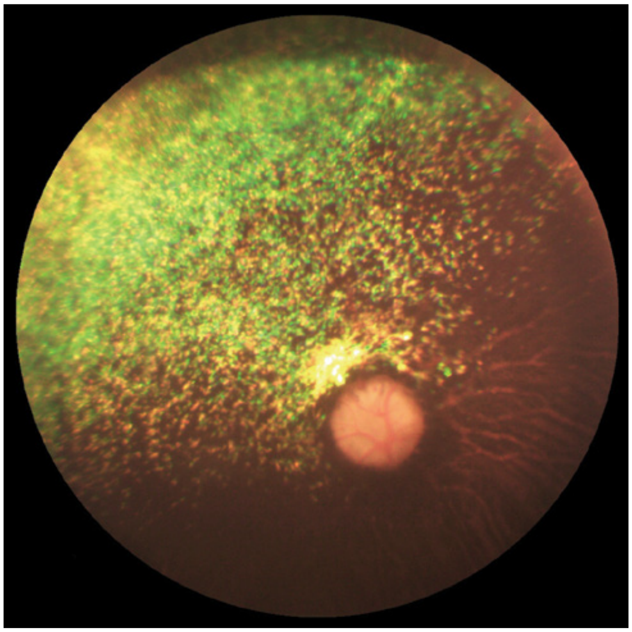


Figure 135.2 The retinal atrophies. Funduscopic findings associated with retinal atrophy, including increased tapetal reflectivity and marked vascular attenuation.

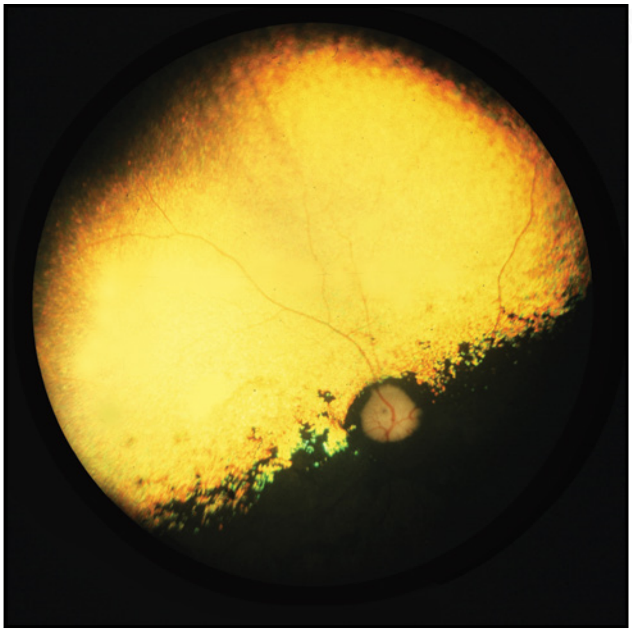


Figure 135.3 The retinal atrophies. Early funduscopic findings associated with retinal atrophy, including moderate vascular attenuation and mildly increased tapetal reflectivity.

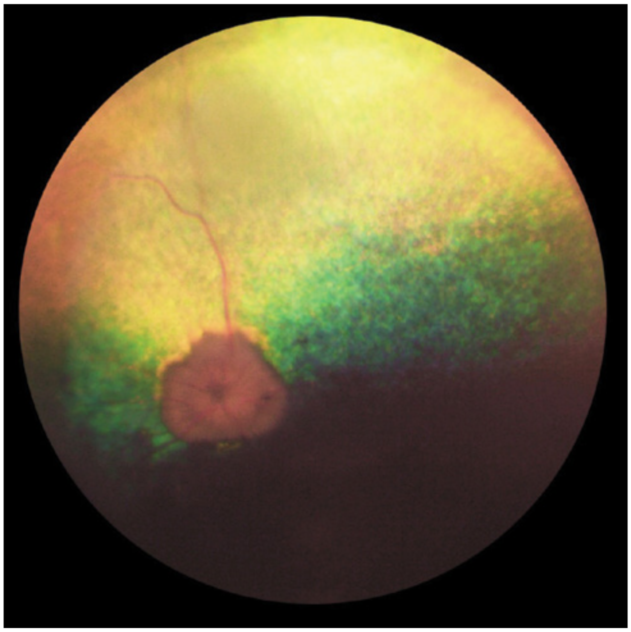


Figure 135.4 The retinal atrophies. Early funduscopic findings associated with retinal atrophy, including moderate vascular attenuation and mildly increased tapetal reflectivity.

SELECTED REFERENCES

Clements PJ, Sargan DR, Gould DJ, Petersen-Jones SM. Recent advances in understanding the spectrum of canine generalised progressive retinal atrophy. *J Small Anim Pract*. 1996;**37**(4):155–162.

Downs LM, Hitti R, Pregnolato S, Mellersh CS. Genetic screening for PRA-associated mutations in multiple dog breeds shows that PRA is heterogeneous within and between breeds. *Vet Ophthalmol*. 2014;**17**(2):126–130. doi: 10.1111/vop.12122.

Narfström K. Hereditary and congenital ocular disease in the cat. *J Feline Med Surg*. 1999;**1**(3):135–141.

Petersen-Jones S. Advances in the molecular understanding of canine retinal diseases. *J Small Anim Pract*. 2005;**46**(8):371–380.

CHAPTER 136
VITREAL DEGENERATION/HERNIATION

PRESENTATION

The vitreous body consists of a transparent hydrogel, which contains collagen and hyaluronic acid and occupies approximately 75% of the volume of the eye. The vitreous body supports both the lens and the neuroretina. Inherited factors as well as age-related degeneration may lead to pathological changes including vitreal syneresis, asteroid hyalosis (calcium/phospholipid bodies), cyst formation, hemorrhage, retinal tears, lenticular instability, and vitreal herniation into the anterior chamber and/or glaucoma. Clinically, degenerate vitreous may be noted to be herniating through the pupil into the anterior chamber, frequently containing fine melanin-rich granules and/or cells (notably in the Italian Greyhound, Boston Terrier, and Jack Russell Terrier) and/or to contain multiple, fine, gravity-dependent crystalline hyaloid bodies – sometimes referred to as "synchysis scintillans" (notably in the Shi Tzu, Minitaure Poodle, and Bichon Frise).

DIAGNOSIS AND TREATMENT

The diagnosis of vitreal degeneration is made based on clinical findings, supported if necessary by B-mode ultrasonography. Treatment may or may not be indicated depending on the severity of changes present and where indicated may comprise topical (steroidal or nonsteroidal) anti-inflammatory therapy, the use of a miotic agent to stabilize subluxted lenses, the removal of luxated lense(s), retinopexy/retinal reattachment, and/or surgical vitrectomy. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration.

SELECTED REFERENCES

Krishnan H, Diehl K, Stefanovski D, Aguirre GD. Vitreous degeneration and associated ocular abnormalities in the **dog**. *Vet Ophthalmol*. 2020;**23**(2):219–224.

Labruyère JJ, Hartley C, Rogers K, Wetherill G, McConnell JF, Dennis R. Ultrasonographic evaluation of vitreous degeneration in normal dogs. *Vet Radiol Ultrasound*. 2008;**49**(2):165–171.

Papaioannou NG and Dubielzig RR. Histopathological and immunohistochemical features of vitreoretinopathy in Shih Tzu dogs. *J Comp Pathol*. 2013;**148**(2–3):230–235. doi: 10.1016/j.jcpa.2012.05.014.

West MC, Sila GH, Aquino SM, Rose MD. Evaluation of **vitreous** degeneration as a potential risk factor for retinal detachment after phacoemulsification in **dogs**. *Vet Ophthalmol*. 2020;**23**(4):721–729.

van der Woerdt A, Wilkie DA, Myer CW. Ultrasonographic abnormalities in the eyes of dogs with cataracts: 147 cases (1986–1992). *J Am Vet Med Assoc*. 1993;**203**(6):838–841.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 136.1 Vitreal degeneration/herniation. Clinical presentation associated with vitreal degeneration and notable asteroid hyalosis.

Figure 136.2 Vitreal degeneration/herniation. Clinical presentation associated with vitreal degeneration and notable asteroid hyalosis.

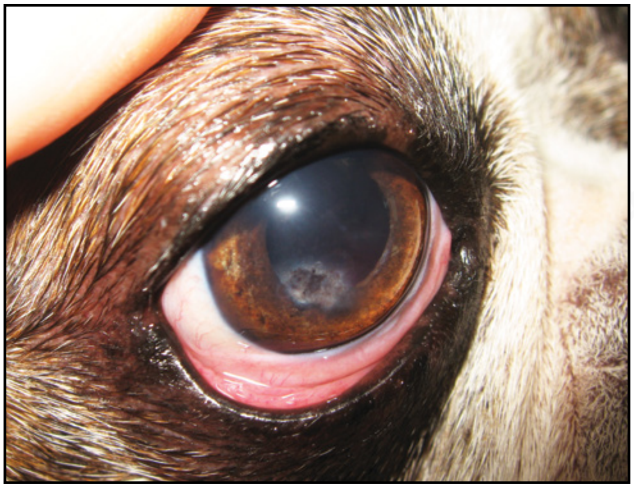


Figure 136.3 Vitreal degeneration/herniation. Herniated, degenerate vitreous is evident in the ventral anterior chamber.

Figure 136.4 Vitreal degeneration/herniation. Pigmented material is visible in the degenerate, herniated vitreous in the ventral anterior chamber.

CHAPTER 137
RETINAL TOXICITY

PRESENTATION

Sensitive neuroretinal tissues may be damaged by exposure to potentially harmful pharmacologic agents; most frequently enrofloxacin in cats and ivermectin in dogs. Toxicity associated with enrofloxacin (and potentially other fluroquinalones) administration in cats may result in varying degrees of visual impairment/blindness. This response appears to be idiosyncratic, however are more commonly encountered in association with significant overdose and/or rapid intravenous administration. Current recommendations are a maximum of 5 mg/kg Q 24 hours. Fundoscopic appearance is initially unremarkable, however visible taperal hyper-reflectivity, vascular attenuation, and ONH degeneration ensue within days to weeks following exposure. Toxicity associated with ivermectin (and potentially other avermectins) may result in varying degrees of visual impairment/blindness as well as neurological defects, altered mentation, and/or thermal dysregulation. This response may be more commonly encountered in association with significant overdose (notably following the inadvertent ingestion of equine anthelminthic products) and/or long-term exposure (notably in association with the management of chronic dermal parasitic infestation). Collie breeds, including the Australian Shepherd and Shetland Sheepdog are particularly susceptible to ivermectin toxicity due to a hereditary defect associated with the MDR1 gene, which regulates molecular transport at the level of the blood brain barrier. Doses as low as 100 μg/kg may be associated with toxicosis in predisposed breeds. Funduscopically, affected dogs may display varying combinations of papilledema and/or irregular to vermiform retinal edema.

DIAGNOSIS AND TREATMENT

The diagnosis of retinal toxicity is made based on clinical findings and history. Electroretinography (ERG) may be used to evaluate photoreceptor function and levels of specific agents (such as ivermectin) may be assessed by laboratory assay. Treatment comprises drug withdrawal and supportive care. Changes associated with enrofloxacin toxicity are generally irreversible. Those associated with ivermectin toxicity are frequently reversible.

SELECTED REFERENCES

Epstein SE and Hollingsworth SR. Ivermectin-induced blindness treated with intravenous lipid therapy in a dog. *J Vet Emerg Crit Care (San Antonio)*. 2013;**23**(1):58–62. doi: 10.1111/vec.12016.

Gelatt KN, van der Woerdt A, Ketring KL, Andrew SE, Brooks DE, Biros DJ, Denis HM, Cutler TJ. Enrofloxacin-associated retinal degeneration in cats. *Vet Ophthalmol*. 2001;**4**(2):99–106.

Kenny PJ, Vernau KM, Puschner B, Maggs DJ. Retinopathy associated with ivermectin toxicosis in two dogs. *J Am Vet Med Assoc*. 2008;**233**(2):279–284. doi: 10.2460/javma.233.2.279.

Wiebe V and Hamilton P. Fluoroquinolone-induced retinal degeneration in cats. *J Am Vet Med Assoc*. 2002;**221**(11):1568–1571.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

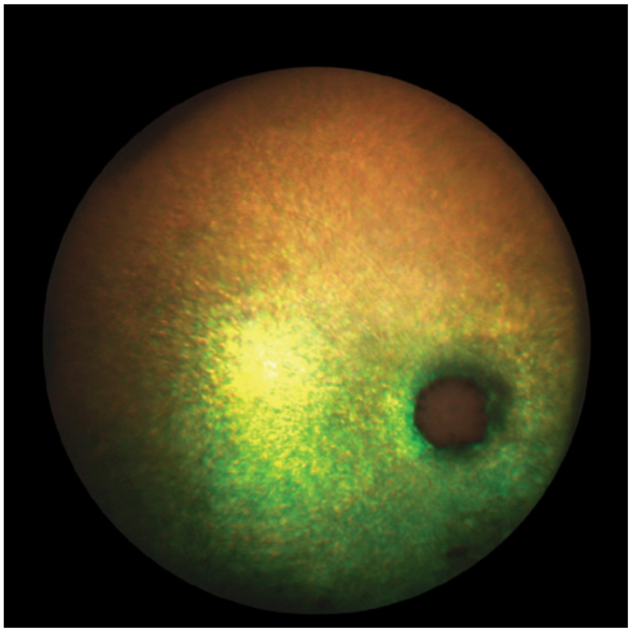


Figure 137.1 Retinal toxicity. Funduscopic changes as a result of enrofloxacin toxicity. Changes include tapetal hyperreflectivity and vascular attenuation.

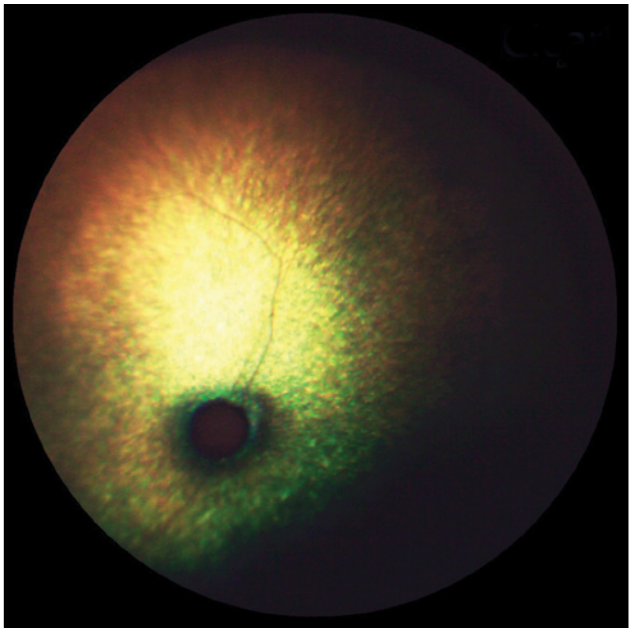


Figure 137.2 Retinal toxicity. Funduscopic changes as a result of enrofloxacin toxicity. Changes include tapetal hyperreflectivity and vascular attenuation.

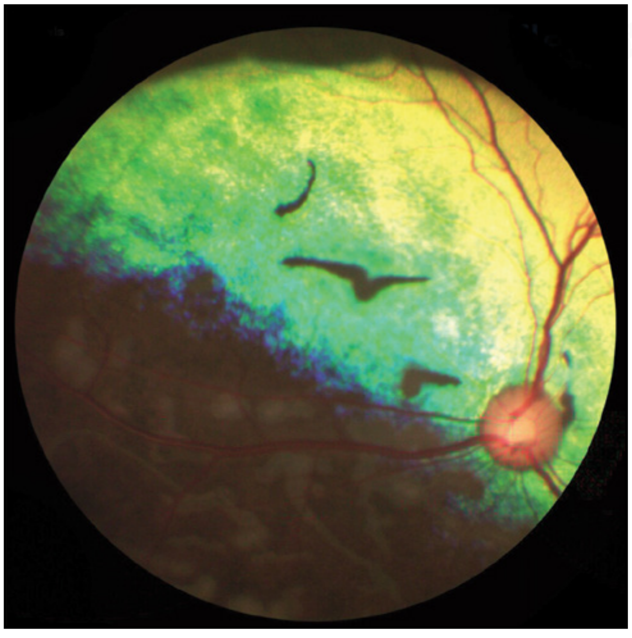


Figure 137.3 Retinal toxicity. Funduscopic changes as a result of ivermectin toxicity. Changes include irregular linear to vermiform areas of retinal edema, notable in the tapetal fundus.

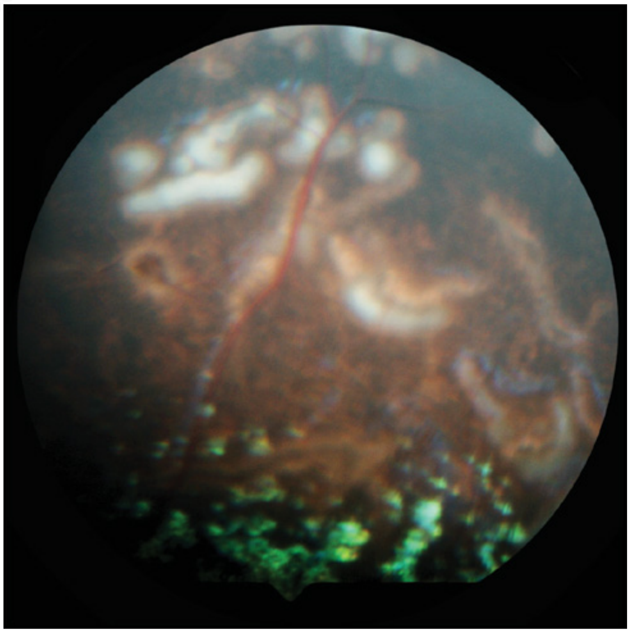


Figure 137.4 Retinal toxicity. Funduscopic changes as a result of ivermectin toxicity. Changes include irregular linear to vermiform areas of retinal edema, notable in the non-tapetal fundus.

CHAPTER 138
SARDS/IMR

PRESENTATION

Sudden acquired retinal degeneration syndrome/immune-mediated retinitis (SARDs/IMR) describes a spectrum of disease resulting in acute onset blindness, which is generally irreversible. The mechanism of photoreceptor death in SARDs/IMR appears to be immune (antibody)-mediated retinopathy. Symptoms generally develop over the course of several days, with affected patients presenting due to severe visual disturbance and disorientation. Clinically, although signs of functional vision are typically absent, sluggish and incomplete pupillary lights reflexes (PLRs) are frequently elicited in the early stages of the disease. Ophthalmic examination (including funduscopic assessment) is otherwise initially unremarkable (although signs of generalized neuroretinal degeneration including tapetal hyper reflectivity and vascular attenuation become evident in the ensuing months). Affected animals are commonly middle-aged, female, small-breed dogs, which may additionally be overweight and/or exhibit symptoms of polyuria/polydipsia/polyphagia. Associations have been made with paraneoplastic syndromes, hyperadrenocorticism, and/or sex hormone imbalances. Commonly affected breeds include the Dachshund and Miniature Schnauzer.

DIAGNOSIS AND TREATMENT

The diagnosis if SARDs/IMR is made based on presentation and clinical findings, supported by electroretinography (ERG), which additionally rules out central causes of blindness (see Central blindess). Routine complete blood count (CBC)/chemistry analysis may indicate the presence of a stress leukogram and/or elevated levels of cholesterol and/or alkaline phosphatase. Attempted treatment of fully extinguished photoreceptor function is fruitless. In rare cases of peracute IMR (associated with a degree of residual visual and/or electroretinographic function), experimental treatment using immunomodulating therapy comprising systemic doxycycline, corticosteroids, and/or immunoglobulin therapy has been described. Potential adverse effects associated with doxycyline, include gastrointestinal (GI) distress, photosensitivity, and hepatic damage. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential side effects associated with the use of immunoglobulins include severe anaphylaxis and/or death.

SELECTED REFERENCES

Grozdanic SD, Harper MM, Kecova H. Antibody-mediated retinopathies in canine patients: mechanism, diagnosis, and treatment modalities. *Vet Clin North Am Small Anim Pract*. 2008;**38**(2):361–387, vii. doi: 10.1016/j.cvsm.2007.12.003.

Keller RL, Kania SA, Hendrix DV, Ward DA, Abrams K. Evaluation of canine serum for the presence of antiretinal autoantibodies in sudden acquired retinal degeneration syndrome. *Vet Ophthalmol*. 2006;**9**(3):195–200.

Komáromy AM, Abrams KL, Heckenlively JR, Lundy SK, Maggs DJ, Leeth CM, MohanKumar PS, Petersen-Jones SM, Serreze DV, van der Woerdt A. Sudden acquired retinal degeneration syndrome (**SARDS**) – a review and proposed strategies toward a better understanding of pathogenesis, early diagnosis, and therapy. *Vet Ophthalmol*. 2016;**19**(4):319–331.

Miller PE, Galbreath EJ, Kehren JC, Steinberg H, Dubielzig RR. Photoreceptor cell death by apoptosis in dogs with sudden acquired retinal degeneration syndrome. *Am J Vet Res*. 1998;**59**(2):149–152.

Montgomery KW, van der Woerdt A, Cottrill NB. Acute blindness in dogs: sudden acquired retinal degeneration syndrome versus neurological disease (140 cases, 2000-2006). *Vet Ophthalmol*. 2008;**11**(5):314–320. doi: 10.1111/j.1463-5224.2008.00652.x.

Stuckey JA, Pearce JW, Giuliano EA, Cohn LA, Bentley E, Rankin AJ, Gilmour MA, Lim CC, Allbaugh RA, Moore CP, Madsen RW. Long-term outcome of sudden acquired retinal degeneration syndrome in dogs. *J Am Vet Med Assoc*. 2013;**243**(10):1425–1431. doi: 10.2460/javma.243.10.1426.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 138.1 SARDs/IMR. Common clinical presentation associated with SARDs in an overweight, middle-aged, female Dachshund.

Figure 138.2 SARDs/IMR. Clinical presentation associated with SARDs/IMR demonstrating acute-onset dilated, poorly responsive pupils and functional blindness in the absence other significant ocular abnormalities.

Figure 138.3 SARDs/IMR. Note the bilateral, symmetric presentation of mydriasis.

Figure 138.4 SARDs/IMR. Note the bilateral, dilated, poorly responsive pupils and functional blindness in the absence other significant ocular abnormalities.

CHAPTER 139
HYPERTENSIVE RETINOPATHY

PRESENTATION

Systemic hypertension may be primary in etiology or may occur secondary to underlying systemic disease (including renal or cardiovascular dysfunction), endocrinopathy (including hyperthyroidism, hyperadrenocorticism, diabetes mellitus), or neoplasia (including lymphoma, multiple myeloma, and pheochromocytoma). Non-hypertensive, hemorrhagic retinopathy may also be associated with inflammatory, infectious, and/or neoplastic disease. Ocular changes are often the presenting symptom of systemic hypertension. Blood-ocular barrier breakdown results in variable combinations of sub-retinal fluid leakage, (pre intra or sub) retinal hemorrhage and/or detachment, hyphema, visual deficits/blindness, and/or secondary glaucoma. Neurological symptoms may additionally be present. Ocular symptoms are usually bilateral, however not necessarily symmetrical. Values indicative of hypertension are variable, however systolic pressures above 160 mmHg in cats and 140 mHg in dogs generally justify therapy, particularly when associated with clinical symptoms.

DIAGNOSIS AND TREATMENT

Treatment comprises addressing underlying disease wherever present. Blood pressure may be directly regulated, typically using amlodipine, and/or an ACE-inhibitor where indicated, with close, regular monitoring of blood pressure being advised. Retinal hemorrhage will typically resolve, and retinal detachment may spontaneously re-attach (notably in cats), depending on the chronicity and severity of the pre-exiting detachment. Surgical reattachment may also be considered in select cases, once underlying pathology has been addressed. Adverse effects potentially associated with the use of amlodipine include lethargy. Adverse effects potentially associated with the use of ACE-inhibitors include lethargy, gastrointestinal (GI) distress, and renal dysfunction.

SELECTED REFERENCES

Jepson RE. Feline systemic hypertension: classification and pathogenesis. *J Feline Med Surg*. 2011;**13**(1):25–34. doi: 10.1016/j.jfms.2010.11.007.

Leblanc NL, Stepien RL, Bentley E. Ocular lesions associated with systemic hypertension in dogs: 65 cases (2005–2007). *J Am Vet Med Assoc*. 2011;**238**(7):915–21.

Maggio F, DeFrancesco TC, Atkins CE, Pizzirani S, Gilger BC, Davidson MG. Ocular lesions associated with systemic hypertension in **cats**: 69 cases (1985–1998). *J Am Vet Med Assoc*. 2000;**217**(5):695–702.

Stepien RL. Feline systemic hypertension: diagnosis and management. *J Feline Med Surg*. 2011;**13**(1):35–43. doi: 10.1016/j.jfms.2010.11.008.

Young WM, Zheng C, Davidson MG, Westermeyer HD. Visual outcome in **cats** with **hypertensive** chorioretinopathy. *Vet Ophthalmol*. 2019;**22**(2):161–167.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 139.1 Hypertensive retinopathy. Clinical presentation associated with feline hypertensive retinopathy. Note typical bilateral presentation.

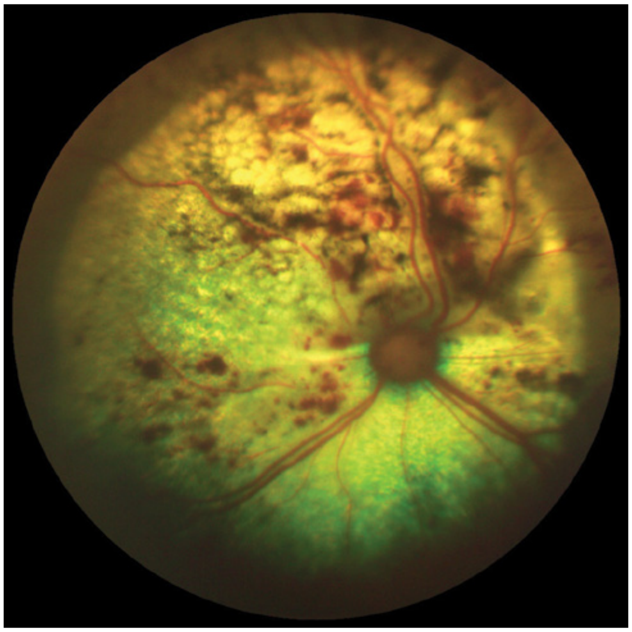


Figure 139.2 Hypertensive retinopathy. Funduscopic findings associated with feline hypertensive retinopathy. Note multifocal retinal hemorrhages.

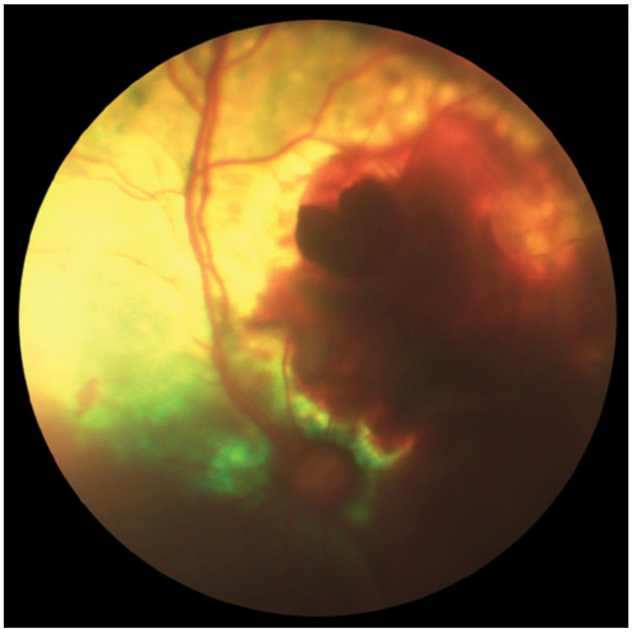


Figure 139.3 Hypertensive retinopathy. Funduscopic findings associated with feline hypertensive retinopathy. Note significant vitreoretinal hemorrhage.

Figure 139.4 Hypertensive retinopathy. Funduscopic findings associated with canine hypertensive retinopathy. Note significant vitreoretinal hemorrhage.

CHAPTER 140
FELINE CHORIORETINITIS

PRESENTATION

The uveal tract is composed of the anterior (iris and ciliary body) and posterior (choroid) tissues. These tissues contain components of the "blood-ocular barrier" which prevents the passage of significant amounts of protein into the aqueous humor. The term uveitis describes inflammation of any of these tissues and is typically associated with variable breakdown of the blood-ocular barrier. The spectrum of clinical symptoms potentially associated with feline chorioretinitis (posterior uveitis) may include a variable degree of anterior uveitis (see also Feline anterior uveitis) vitritis, chorioretinal inflammation (marked by edema, exudation, hemorrhage and/or retinal detachment), visual impairment and/or blindness. Initially, intraocular pressures are typically decreased in patients affected by uveitis; however, secondary glaucoma may occur as a later complication. One or both eyes may be affected. Bilateral uveitis should prompt concern for the presence of systemic disease.

DIAGNOSIS AND TREATMENT

The diagnosis of chorioretinitis is made based on clinical findings. Potential etiologies include trauma, systemic disease, exposure to infectious organisms, the presence of (local or systemic) neoplasia, and/or hereditary factors. Infectious etiologies which may be associated with the development of chorioretinitis in cats include viral (FelV, FIV, FIP, and FHV), protozoal (*Toxoplasma gondii*), bacterial (notably *Bartonealla* spp in cats), and fungal (*cryptococcus, coccidioidomycosis, aspergillus, blastomyces*, and *histoplasmosis*) organisms. Frustratingly, however, the precise etiology of uveitis remains unclear in a significant number of cases. Additionally, clinical uveitis may be recurrent and/or chronic in its behavior. Treatment encompasses addressing underlying systemic, infectious, or neoplastic disease. Additionally, topical and/or systemic anti-inflammatory therapy (nonsteroidal or steroidal) is typically warranted. Long-tern treatment may be indicated in order to minimize the risk of secondary glaucoma. Clindamycin generally represents a suitable empirical/initial antimicrobial choice. Potential side effects associated with the use of Clindamycin include gastrointestinal (GI) distress.

SELECTED REFERENCES

Andrew SE. **Feline** infectious peritonitis. *Vet Clin North Am Small Anim Pract*. 2000;**30**(5):987–1000.
Colitz CM. Feline uveitis: diagnosis and treatment. *Clin Tech Small Anim Pract*. 2005 **20**(2):117–120.
Davidson MG. Toxoplasmosis. *Vet Clin North Am Small Anim Pract*. 2000;**30**(5):1051–1062.
Townsend WM. Canine and feline uveitis. *Vet Clin North Am Small Anim Pract*. 2008;**38**(2):323–346, vii. doi: 10.1016/j.cvsm.2007.12.004.
van der Woerdt A. Management of intraocular inflammatory disease. *Clin Tech Small Anim Pract*. 2001;**16**(1):58–61.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

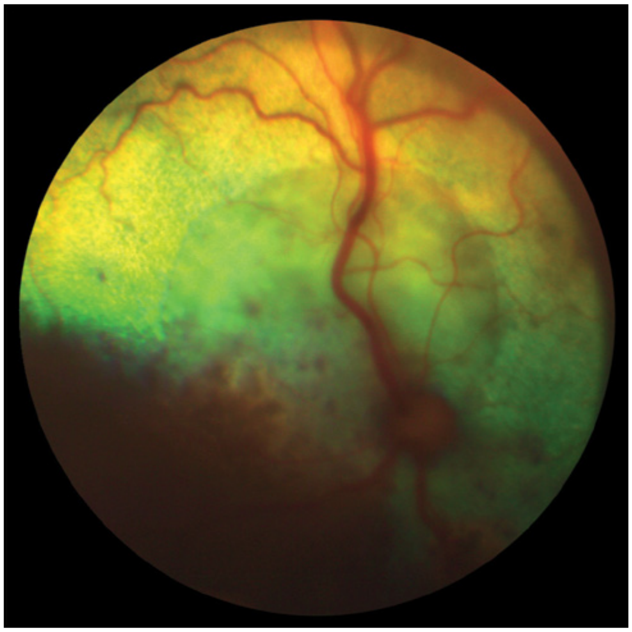


Figure 140.1 Feline chorioretinitis. Funduscopic changes in association with active feline chorioretinitis due to toxoplasmosis.

Figure 140.2 Feline chorioretinitis. Funduscopic changes in association with active feline chorioretinitis due to cryptococcosis.

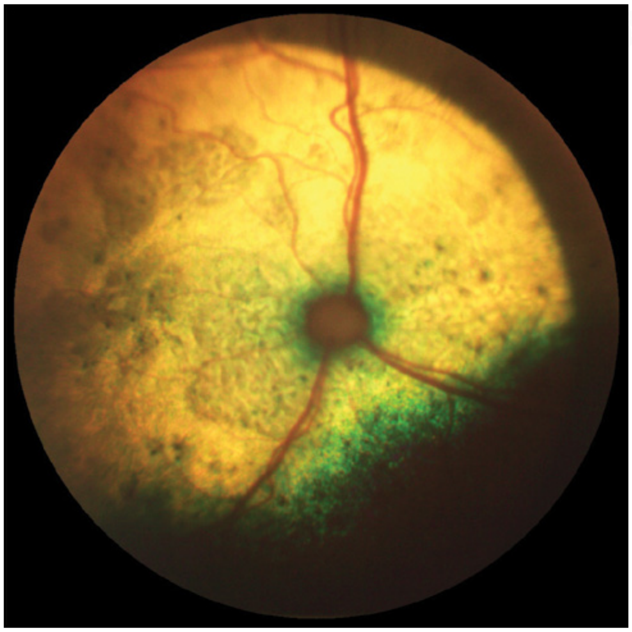


Figure 140.3 Feline chorioretinitis. Funduscopic changes in association with active feline chorioretinitis due to feline leukemia virus.

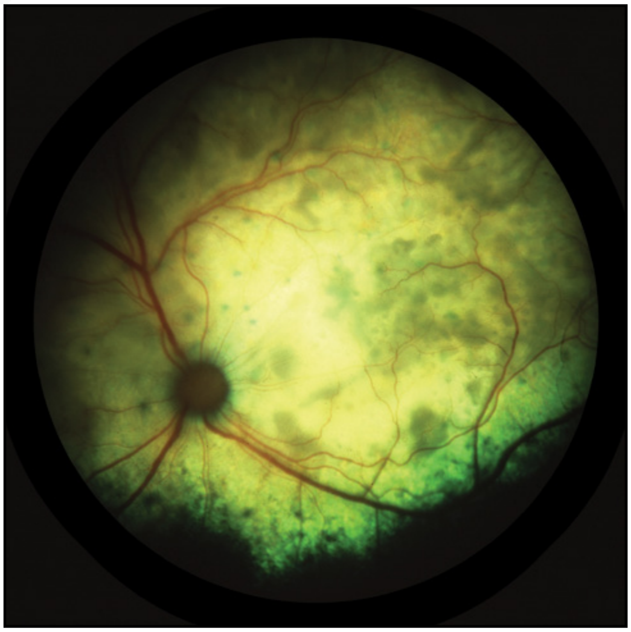


Figure 140.4 Feline chorioretinitis. Funduscopic changes in association with active feline chorioretinitis due to bartonellosis.

CHAPTER 141
CANINE CHORIORETINITIS

PRESENTATION

The uveal tract is composed of the anterior (iris and ciliary body) and posterior (choroid) tissues. These tissues contain components of the "blood-ocular barrier" which prevents the passage of significant amounts of protein into the aqueous humor. The term uveitis describes inflammation of any of these tissues and is typically associated with variable breakdown of the blood-ocular barrier. The spectrum of clinical symptoms potentially associated with canine chorioretinitis (posterior uveitis) may include a variable degree of anterior uveitis (see also Canine anterior uveitis) vitritis, chorioretinal inflammation (marked by edema, exudation, hemorrhage and/or retinal detachment), visual impairment, and/or blindness. Initially, intraocular pressures are typically decreased in patients affected by uveitis, however secondary glaucoma may occur as a complication. One or both eyes may be affected. Bilateral uveitis should prompt concern for the presence of systemic disease.

DIAGNOSIS AND TREATMENT

Diagnosis of chorioretinitis is made based on clinical findings. Potential etiologies include trauma, systemic disease, exposure to infectious organisms, the presence of (local or systemic) neoplasia and/or hereditary factors. Infectious etiologies which may be associated with the development of chorioretinitis in dogs include viral (Canine adenovirus-1 and Canine parvovirus), protozoal (*Toxoplasma gondii*), bacterial (notably *Ehrlichia canis, Rickettsia rickettsia, Leptospira* spp and *Borellia* spp) and fungal (*coccidioidomycosis, aspergillus, blastomyces*, and *histoplasmosis*) organisms. Frustratingly, however, the precise etiology of uveitis remains idiopathic in a significant number of cases. Additionally, clinical uveitis may be recurrent and/or chronic in its behavior. Treatment encompasses addressing underlying systemic, infectious, or neoplastic disease. Additionally, topical and/or systemic anti-inflammatory therapy (nonsteroidal or steroidal) is typically warranted. Long-term treatment may be indicated in order to minimize the risk of secondary glaucoma. Mydriatic agents (Atropine/Tropicamide) should be used with caution where indicated. Doxycycline generally represents a suitable empirical/initial antimicrobial choice. Potential side effects associated with the use of doxycycline include gastrointestinal (GI) distress.

SELECTED REFERENCES

Krohne SG. **Canine** systemic fungal infections. *Vet Clin North Am Small Anim Pract*. 2000;**30**(5):1063–1090.

Massa KL, Gilger BC, Miller TL, Davidson MG. Causes of uveitis in dogs: 102 cases (1989–2000). *Vet Ophthalmol*. 2002;**5**(2):93–98.

Peiffer RL, Cook CS, Möller I. Therapeutic strategies involving antimicrobial treatment of ophthalmic disease in small animals. *J Am Vet Med Assoc*. 1984;**185**(10):1172–1175.

Townsend WM. Canine and feline uveitis. *Vet Clin North Am Small Anim Pract*. 2008;**38**(2):323–346, vii. doi: 10.1016/j.cvsm.2007.12.004.

van der Woerdt A. Management of intraocular inflammatory disease. *Clin Tech Small Anim Pract*. 2001;**16**(1):58–61.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

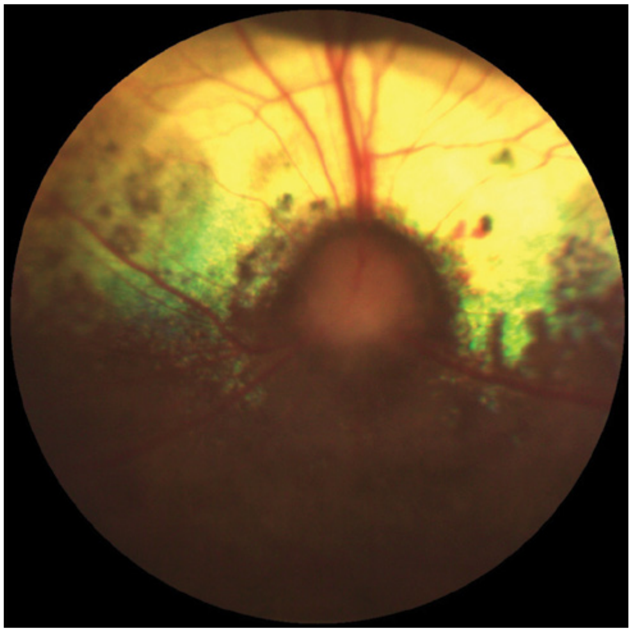


Figure 141.1 Canine chorioretinitis. Funduscopic changes in association with active canine chorioretinitis due to Toxoplasmosis.

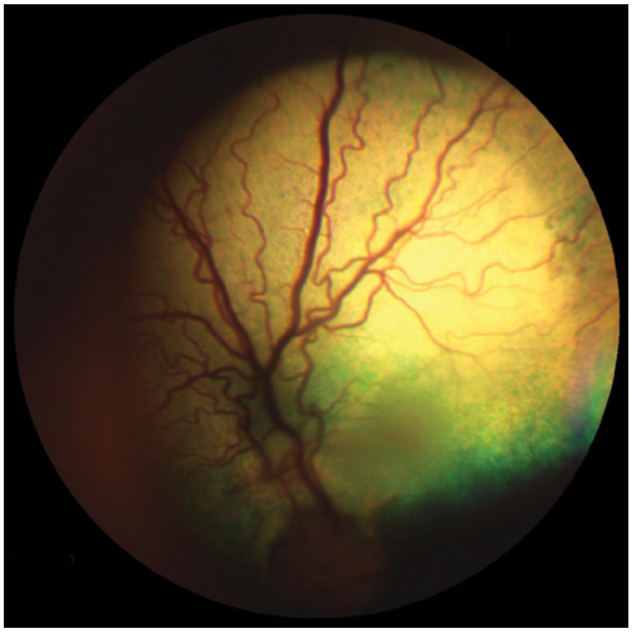


Figure 141.2 Canine chorioretinitis. Funduscopic changes in association with active canine chorioretinitis due to Coccidioidomycosis.

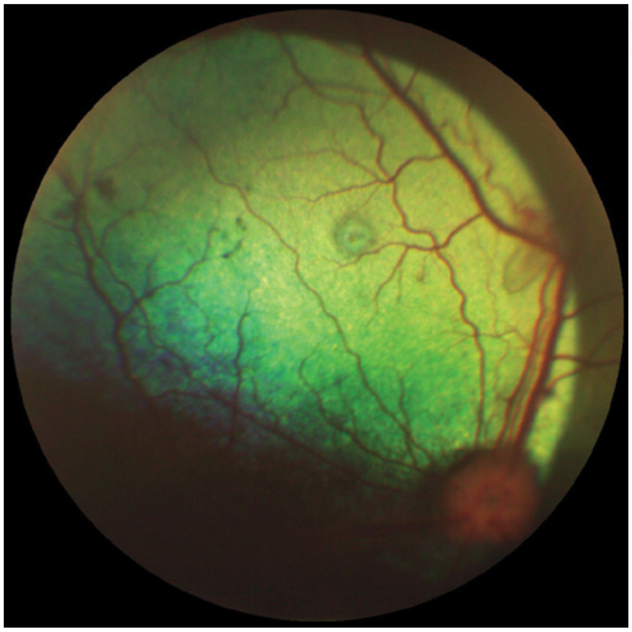


Figure 141.3 Canine chorioretinitis. Funduscopic changes in association with active canine chorioretinitis due to Blastomycosis.

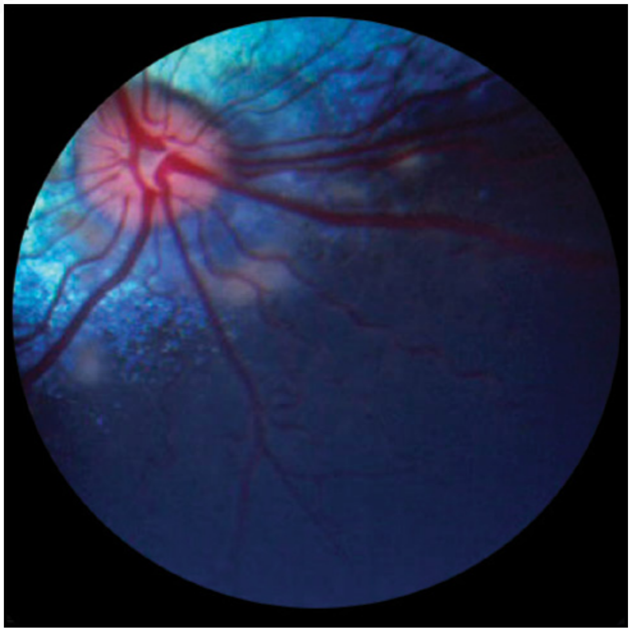


Figure 141.4 Canine chorioretinitis. Funduscopic changes in association with active canine chorioretinitis due to Ehrlichiosis.

CHAPTER 142
RETINAL PIGMENT EPITHELIAL DYSTOPHY (RPED)

PRESENTATION

Retinal pigment epithelial dystrophy (RPED) describes a disease of the retinal pigment epithelium, marked by the pathological accumulation of lipofuscin and associated with more widespread secondary retinal degeneration. This condition has also been referred to as "central progressive retinal atrophy" (CPRA). Clinically, this disease manifests as decreased visual function (which may progress to outright blindness), subtle neurological defects (notably proprioceptive deficits) and/or secondary cataract formation. Fundoscopically, disease presentation is characterized by the (bilateral) presence of scattered tan to brown foci, which may coalesce with time. More generalized secondary retinal degeneration typically ensues. Similarities between this disease and deficiencies in the availability and absorption/metabolism of vitamin E have also been noted. Commonly affected breeds include the Labrador Retriever, Golden Retriever, Cocker Spaniel, Briard, and the collie breeds, notably in European populations.

DIAGNOSIS AND TREATMENT

Diagnosis of RPED is based on clinical findings. The supplementation of large doses of oral vitamin E (600–900iu bid) has been advocated and may help to limit disease progression.

SELECTED REFERENCES

Lightfoot RM, Cabral L, Gooch L, Bedford PG, Boulton ME. Retinal pigment epithelial dystrophy in Briard dogs. *Res Vet Sci*. 1996;**60**(1):17–23.

Lin CT, Gould DJ, Petersen-Jonest SM, Sargan DR. **Canine** inherited **retinal** degenerations: update on molecular genetic research and its clinical application. *J Small Anim Pract*. 2002;**43**(10):426–432.

McLellan GJ and Bedford PG. Oral vitamin E absorption in English Cocker Spaniels with familial vitamin E deficiency and retinal pigment epithelial dystrophy. *Vet Ophthalmol*. 2012;**15** Suppl 2:48–56. doi: 10.1111/j.1463-5224.2012.01049.x.

McLellan GJ, Cappello R, Mayhew IG, Elks R, Lybaert P, Watté C, Bedford PG. Clinical and pathological observations in English cocker spaniels with primary metabolic vitamin E deficiency and retinal pigment epithelial dystrophy. *Vet Rec*. 2003;**153**(10):287–292.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

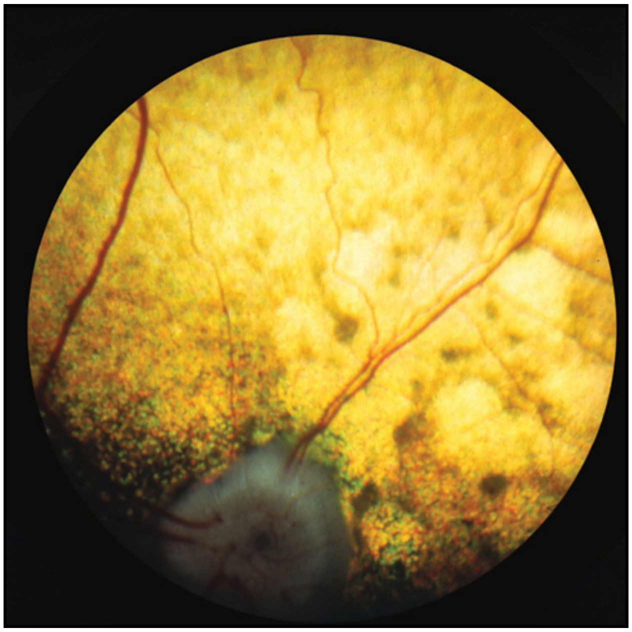


Figure 142.1 Retinal pigment epithelial dystrophy. Funduscopic changes associated with retinal pigment epithelial dystrophy. Multiple discrete areas representing lipofuscin accumulation are visible.

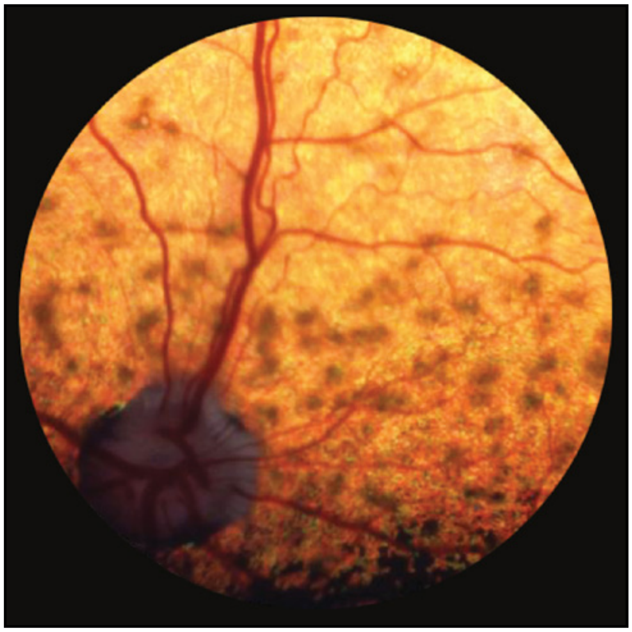


Figure 142.2 Retinal pigment epithelial dystrophy. Funduscopic changes associated with retinal pigment epithelial dystrophy. Multiple discrete areas representing lipofuscin accumulation are visible.

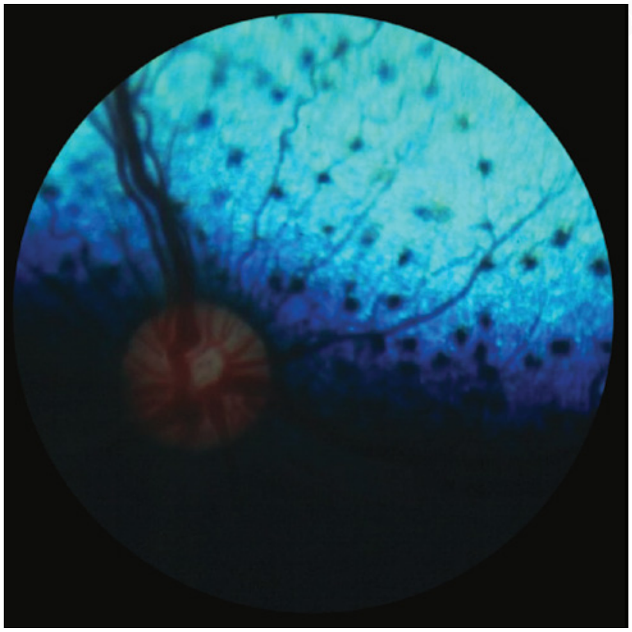


Figure 142.3 Retinal pigment epithelial dystrophy. Funduscopic changes associated with retinal pigment epithelial dystrophy. Multiple discrete areas representing lipofuscin accumulation are visible.

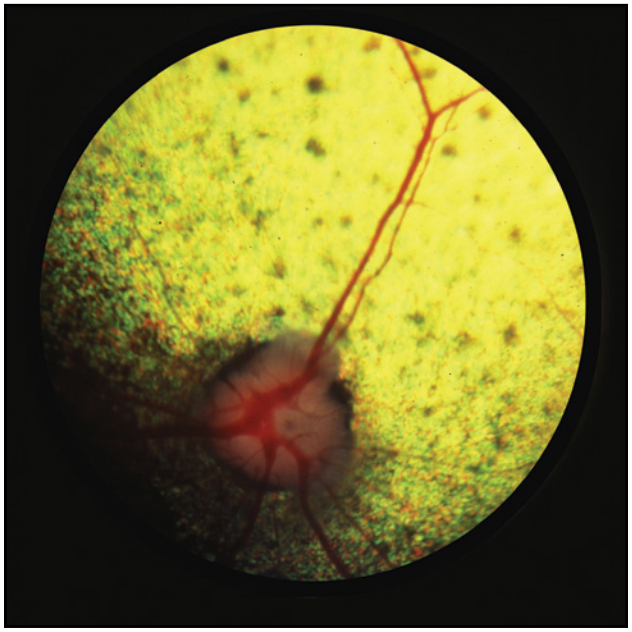


Figure 142.4 Retinal pigment epithelial dystrophy. Funduscopic changes associated with retinal pigment epithelial dystrophy. Multiple discrete areas representing lipofuscin accumulation are visible.

CHAPTER 143
UVEODERMATOLOGIC SYNDROME (UDS) ASSOCIATED CHORIORETINITIS

PRESENTATION

Canine uveodermatologic syndrome (UDS) represents an immune-mediated disease, predominantly affecting melanocytic tissues, likely hereditary in etiology and exhibiting many similarities to Vogt-Koyanagi-Harada (VKH) disease in humans. This condition has consequently also been referred to "VKH-like disease." Ocular symptoms associated with UDS may include any combination of anterior uveitis, chorioretinitis, or panuveitis, frequently marked by pigment loss. Secondary changes may include any combination of cataract formation, retinal detachment, hyphema, and/or glaucoma. Typically, both eyes are affected to a variable degree. Additional symptoms may include any combination of periocular, mucocutaneous oral, and/or nasal vitiligo (loss of pigment), poliosis (whitening of hair), and/or ulcerative dermatitis. Typically, these symptoms are relatively bilaterally symmetrical in appearance (see also Autoimmune blepharitis and uveodermatological associated uveitis). Commonly affected breeds include the Akita, Siberian Husky, Samoyed, Chow Chow, German Shepherd, and Shetland Sheepdog.

DIAGNOSIS AND TREATMENT

The diagnosis of UDS is made based on a combination of signalment and clinical findings. Where representative adnexal or dermal changes are present, the harvesting of a small incisional skin biopsy for histopathological interpretation may support the diagnosis. Treatment usually comprises aggressive, long-term anti-inflammatory, and/or immune-modulating therapy. Typically, this involves the topical and systemic administration of corticosteroids with or without the adjunctive use of additional agents including cyclosporine and/or azathioprine. Potential adverse effects associated with the use of topical corticosteroids include impaired wound healing and corneal degeneration. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of azathioprine include gastrointestinal (GI) distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of cyclosporine include hypersensitivity and GI distress.

SELECTED REFERENCES

Angles JM, Famula TR, Pedersen NC. Uveodermatologic (VKH-like) syndrome in American Akita dogs is associated with an increased frequency of DQA1*00201. *Tissue Antigens*. 2005;**66**(6):656–665.

Carter WJ, Crispin SM, Gould DJ, Day MJ. An immunohistochemical study of uveodermatologic syndrome in two Japanese Akita dogs. *Vet Ophthalmol*. 2005;**8**(1):17–24.

Horikawa T, Vaughan RK, Sargent SJ, Toops EE, Locke EP. Pathology in practice. Uveodermatologic syndrome. *J Am Vet Med Assoc*. 2013;**242**(6):759–761. doi: 10.2460/javma.242.6.759.

Zarfoss MK, Tusler CA, Kass PH, Montgomery K, Lim CC, Mowat F, Thomasy SM. Clinical findings and outcomes for **dogs** with **uveodermatologic syndrome**. *J Am Vet Med Assoc*. 2018;**252**(10):1263–1271.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

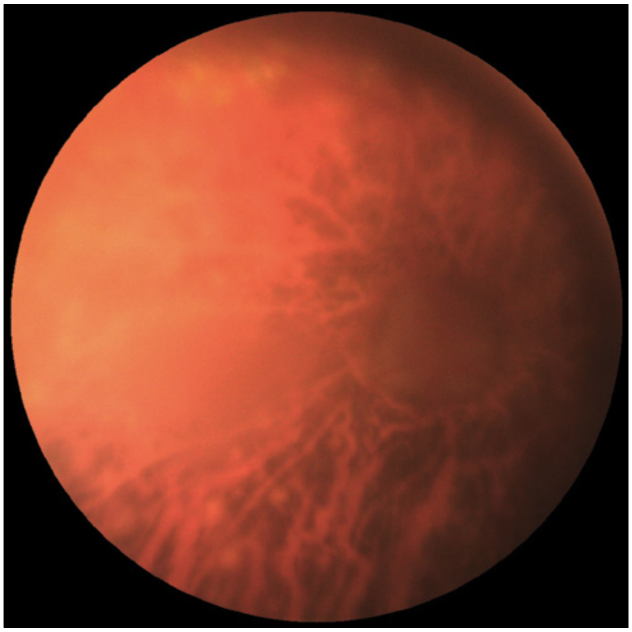


Figure 143.1 Uveodermatologic syndrome (UDS) associated chorioretinitis. Funduscopic changes due to canine uveodermatologic syndrome. Note large coalescing areas of choroidal pigment loss.

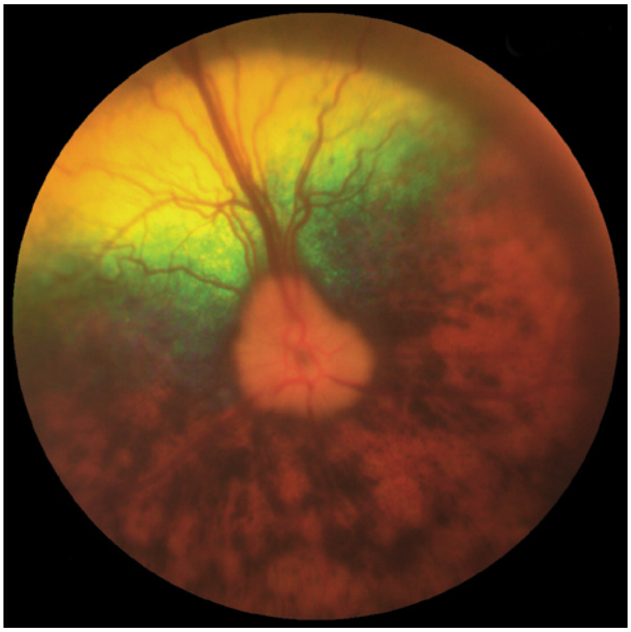


Figure 143.2 Canine Uveodermatologic syndrome (UDS) associated chorioretinitis. Note multifocal to coalescing areas of choroidal pigment loss in non-tapetal fundus.

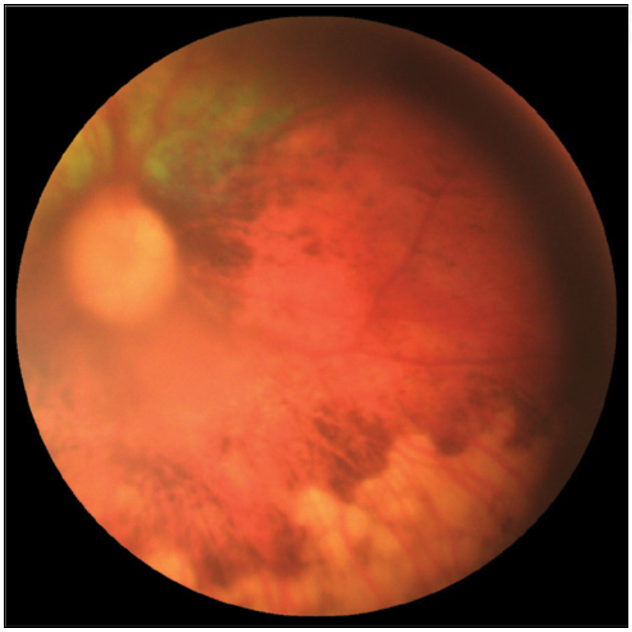


Figure 143.3 Canine Uveodermatologic syndrome (UDS) associated chorioretinitis. Note significant peripheral multifocal to coalescing areas of choroidal pigment loss in non-tapetal fundus.

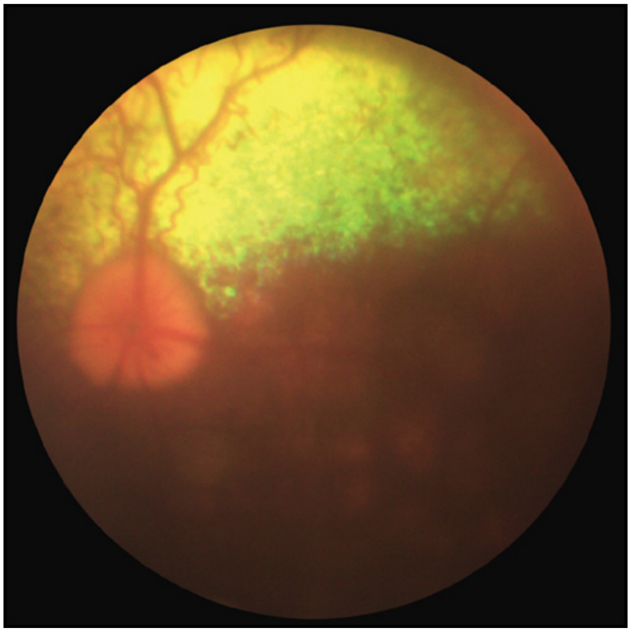


Figure 143.4 Canine Uveodermatologic syndrome (UDS) associated chorioretinitis. Note early multifocal areas of choroidal pigment loss in non-tapetal fundus.

CHAPTER 144
PRIMARY (BULLOUS) RETINAL DETACHMENT

PRESENTATION

Primary, bullous retinal detachment represents a separation of the neurosensory retina from the underlying retinal pigment epithelium (RPE) due to subretinal fluid accumulation. This process is suspected to be immune-mediated in etiology. Clinical symptoms typically comprise bilaterally dilated poorly or nonresponsive pupils in association with acute onset blindness. Retinal tissue may be noted to be "billowing" forward posterior to level of the pupils. Funduscopically, complete or multiple areas of (non-hemorrhagic) bullous detachment are noted. Commonly affected breeds include the German Shepherd, Australian Shepherd, and Labrador Retriever.

DIAGNOSIS AND TREATMENT

The diagnosis of bullous retinal detachment is made based on clinical findings (including funduscopy), supported if necessary by B-mode ultrasonography. Bullous detachments are typically responsive to medical therapy comprising anti-inflammatory to immunosuppressive doses of systemic corticosteroids. The term "steroid-responsive" retinal detachment has also been used to describe this condition. Underlying systemic pathology should be ruled out through routine complete blood count (CBC)/chemistry and infectious titer testing, imaging, and the measurement of systemic blood pressure. Where a positive response to therapy is achieved, the withdrawal of corticosteroids should be pursued cautiously, with some patients requiring long-term treatment. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Andrew SE, Abrams KL, Brooks DE, Kublis PS. Clinical features of steroid responsive retinal detachments in twenty-two dogs. *Vet Comp Ophthalmol*. (1997);**7**, 82–87.

Grahn BH and Wolfer J. Diagnostic ophthalmology. Bilateral multifocal serous retinal detachments. *Can Vet J*. 1997;**38**(4):250–251.

Wolfer J, Grahn B, Arrington K. Diagnostic ophthalmology. Bilateral, bullous retinal detachment. *Can Vet J*. 1998;**39**(1):57–58.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 144.1 Primary (bullous) retinal detachment. Clinical presentation associated with primary (bullous) retinal detachment. Bullous retinal detachment can frequently be visualized by simple external examination, as shown in this figure.

Figure 144.2 Primary (bullous) retinal detachment. Funduscopic findings associated with primary (bullous) retinal detachment. Note the absence of hemorrhage.

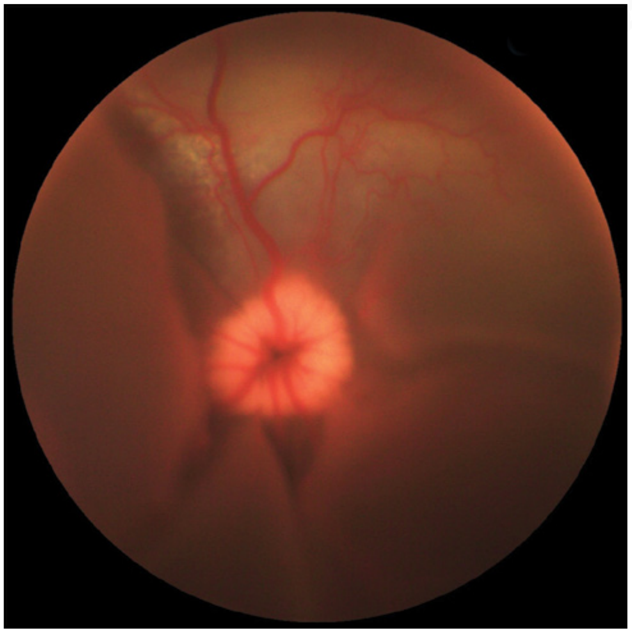


Figure 144.3 Primary (bullous) retinal detachment. Funduscopic findings associated with primary (bullous) retinal detachment. Note the "billowy" appearance of the detached retinal tissue.

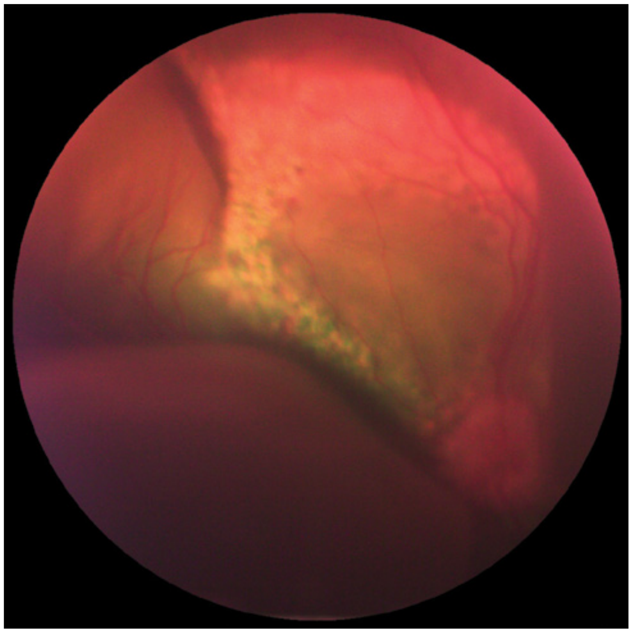


Figure 144.4 Primary (bullous) retinal detachment. Peripheral funduscopic findings associated with primary (bullous) retinal detachment.

CHAPTER 145
RHEGMATOGENOUS RETINAL DETACHMENT (RRD)

PRESENTATION

Rhegmatogenous retinal detachment represents a separation of the neurosensory retina from the underlying retinal pigment epithelium (RPE) stemming from a break in the retinal tissue. Rhegmatogenous detachments may be

- primary (spontaneous) – particularly around the ora ciliaris retina and frequently in association with vitreoretinal degeneration
- secondary – arising as a result of inflammation and/or trauma.

Clinical symptoms may include dilated, poorly responsive pupil(s), vitreous degeneration, herniation, and/or hemorrhage visual deficits and/or blindness. Funduscopically retinal tears and/or folds may be visualized; however, accurate visualization is frequently challenging. Symptoms may be unilateral or bilateral. Frequently affected breeds include the Shi Tzu, Bichon Frise', Italian Greyhound, Yorkshire Terrier, Jack Russell Terrier, and Labrador Retriever.

DIAGNOSIS AND TREATMENT

The diagnosis of rhegmatogenous retinal detachment is made based on clinical findings (including funduscopy), supported if necessary by B-mode ultrasonography. Medical therapy is unlikely to result in resolution of rhegmatogenous retinal detachment. Where pathology is relatively recent (1–3 weeks) and significant secondary complications have not developed, surgical options include

- transpupillary laser "barrier" retinopexy – most suitable for cases involving limited pathology such as small tears or holes or lesions associated with geographic retinal dysplasia
- complete retinal reattachment, typically encompassing, endoscopic retinopexy, vitrectomy, and silicone oil exchange.

SELECTED REFERENCES

Grahn BH, Barnes LD, Breaux CB, Sandmeyer LS. Chronic retinal detachment and giant retinal tears in 34 dogs: outcome comparison of no treatment, topical medical therapy, and retinal reattachment after vitrectomy. *Can Vet J*. 2007;**48**(10):1031–1039.

Itoh Y, Maehara S, Yamasaki A, Tsuzuki K, Izumisawa Y. Investigation of fellow eye of unilateral **retinal detachment** in **Shih-Tzu**. *Vet Ophthalmol*. 2010;**13**(5):289–293.

Papaioannou NG and Dubielzig RR. Histopathological and immunohistochemical features of vitreoretinopathy in Shih Tzu dogs. *J Comp Pathol*. 2013;**148**(2–3):230–235. doi: 10.1016/j.jcpa.2012.05.014.

Steele KA, Sisler S, Gerding PA. Outcome of retinal reattachment surgery in dogs: a retrospective study of 145 cases. *Vet Ophthalmol*. 2012;15 Suppl 2:35–40. doi: 10.1111/j.1463-5224.2012.01009.x.

Vainisi SJ and Wolfer JC. Canine retinal surgery. *Vet Ophthalmol*. 2004;**7**(5):291–306.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

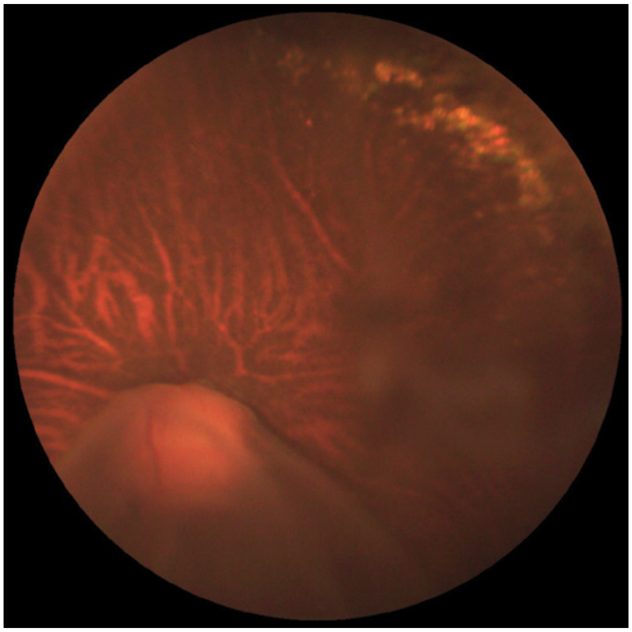


Figure 145.1 Rhegmatogenous retinal detachment. Funduscopic appearance of complete peripheral rhegmatogenous detachment such that the neuroretina remains anchored at the optic nerve head (sometimes described as a "hanging veil" detachment).

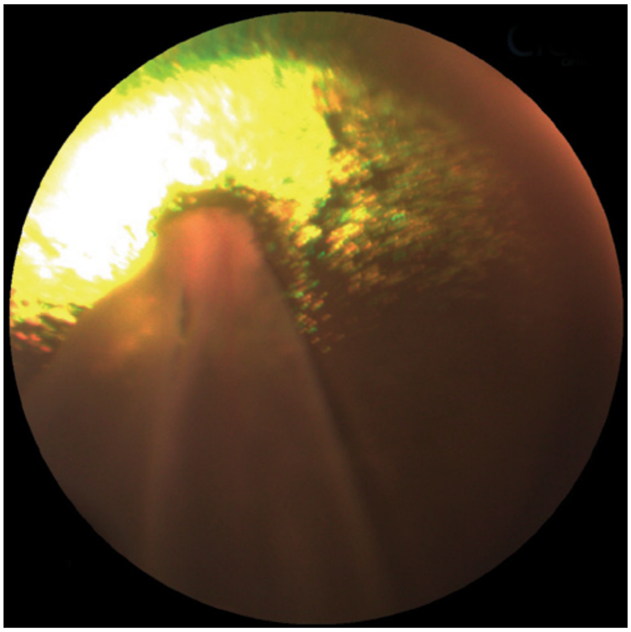


Figure 145.2 Rhegmatogenous retinal detachment. Funduscopic appearance of complete peripheral rhegmatogenous detachment such that the neuroretina remains anchored at the optic nerve head (sometimes described as a "hanging veil" detachment).

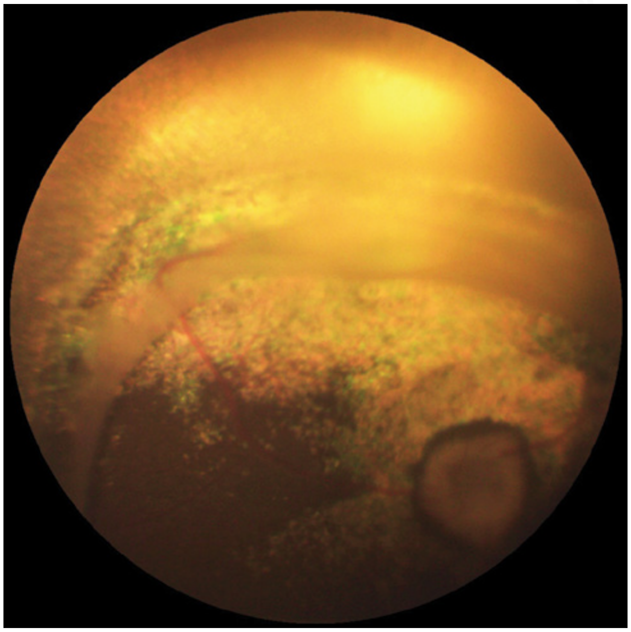


Figure 145.3 Rhegmatogenous retinal detachment. Funduscopic appearance of a rhegmatogenous detachment in association with a giant retinal tear.

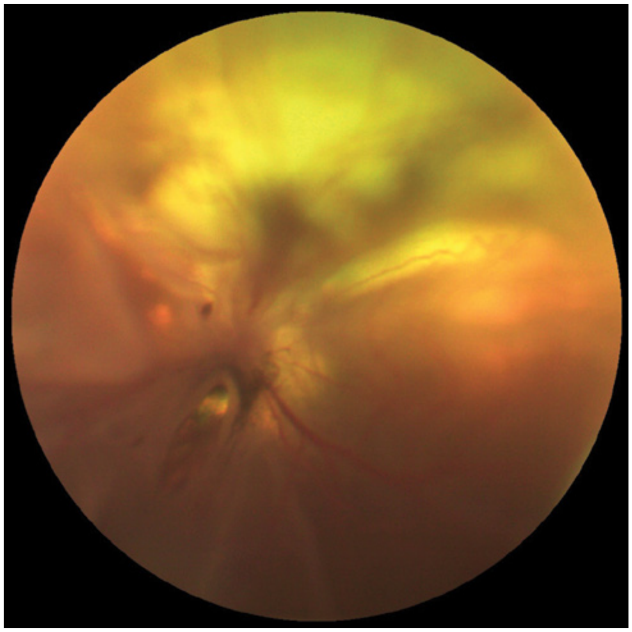


Figure 145.4 Rhegmatogenous retinal detachment. Funduscopic appearance of a rhegmatogenous detachment in association with a small retinal tear.

CHAPTER 146
RETINAL/CHOROIDAL LYMPHOMA

PRESENTATION

Lymphoma affecting the posterior segment occurs with the highest frequency in combination with systemic disease, although in some cases, ocular changes may precede systemic symptoms. One, or more commonly both, eye(s) may be affected and associated ocular findings may include any combination of visual deficits, anterior or panuveitis, retinal hemorrhage and/or detachment and/or glaucoma (see also Feline and canine chorioretintis and uveal lymphoma). Systemic symptoms may include any combination of weight loss, lethargy, inappetence, gastrointestinal (GI) distress, pyrexia, lymphadenopathy, organomegally, hypercalcemia, and/or anemia. Both canine and feline species of any breed or crossed-breed may be affected.

DIAGNOSIS AND TREATMENT

The diagnosis of lymphoma may be made based on clinical findings, ancillary imaging, cytological/histological interpretation, PCR analysis of samples obtained by aqueocentesis as well as lymph node and/or affected organ aspiration/biopsy. Treatment comprises topical anti-inflammatory therapy (typically utilizing a corticosteroid) as well as systemic chemotherapy. Associated ophthalmic changes such as elevated intraocular pressure should be addressed as appropriate. Staging via local lymph node (and/or organ/bone marrow) aspiration, three-view radiography and CBC/chemistry analysis is recommended, prior to initiation of systemic chemotherapy. Diagnostic testing for infectious viruses including FelV, FIV, and FIP is additionally recommended in feline patients. Individual chemotherapeutic protocols are most ideally designed by a veterinary oncologist however typically include a variable combination of prednisone, vincristine, cyclophosphamide, and/or doxyrubicin. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of vincristine include stomatitis, GI distress, neuropathy, hepatopathy, and myelosuppression. Potential adverse effects associated with the use of cyclophosphamide include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of doxorubicin include hypersensitivity, GI distress, cardiac dysfunction, and myelosuppression. The prognosis varies depending on the extent of disease present prior to initiating of therapy.

SELECTED REFERENCES

Cullen CL, Caswell JL, Grahn BH. Intravascular **lymphoma** presenting as bilateral panophthalmitis and retinal detachment in a **dog**. *J Am Anim Hosp Assoc.* 2000;**36**(4):337–342.

Massa KL, Gilger BC, Miller TL, Davidson MG. Causes of uveitis in dogs: 102 cases (1989–2000). *Vet Ophthalmol.* 2002;**5**(2):93–98.

Nerschbach V, Eule JC, Eberle N, Höinghaus R, Betz D. Ocular manifestation of lymphoma in newly diagnosed cats. *Vet Comp Oncol.* 2013. doi: 10.1111/vco.12061.

Ota-Kuroki J, Ragsdale JM, Bawa B, Wakamatsu N, Kuroki K. Intraocular and periocular lymphoma in dogs and cats: a retrospective review of 21 cases (2001–2012). *Vet Ophthalmol.* 2013. doi: 10.1111/vop.12106.

Rutley M, MacDonald V. Managing the canine lymphosarcoma patient in general practice. *Can Vet J.* 2007;**48**(9):977–979.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

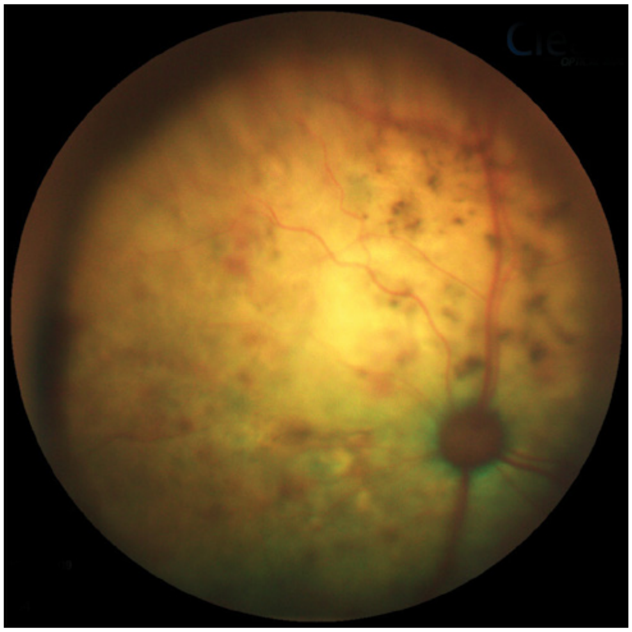


Figure 146.1 Retinal/choroidal lymphoma. Funduscopic findings associated with chorioretinal lymphoma, including chorioretinitis, exudation, subretinal infiltration, and hemorrhage.

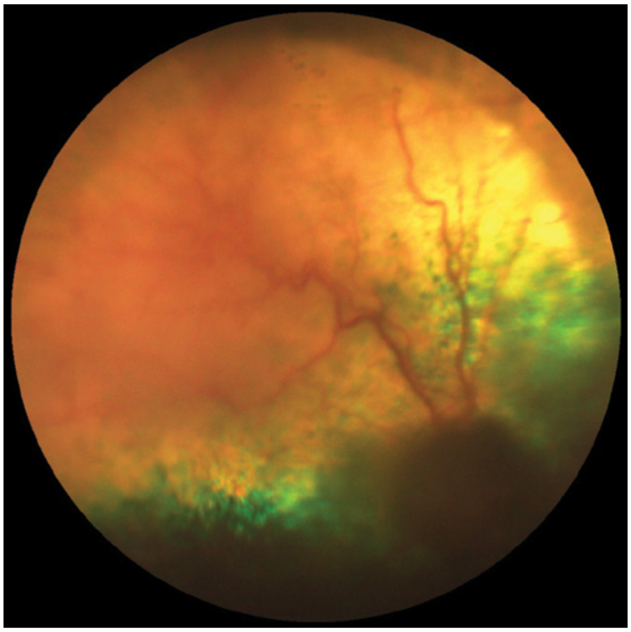


Figure 146.2 Retinal/choroidal lymphoma. Funduscopic findings associated with chorioretinal lymphoma, including chorioretinitis, exudation, and subretinal infiltration.

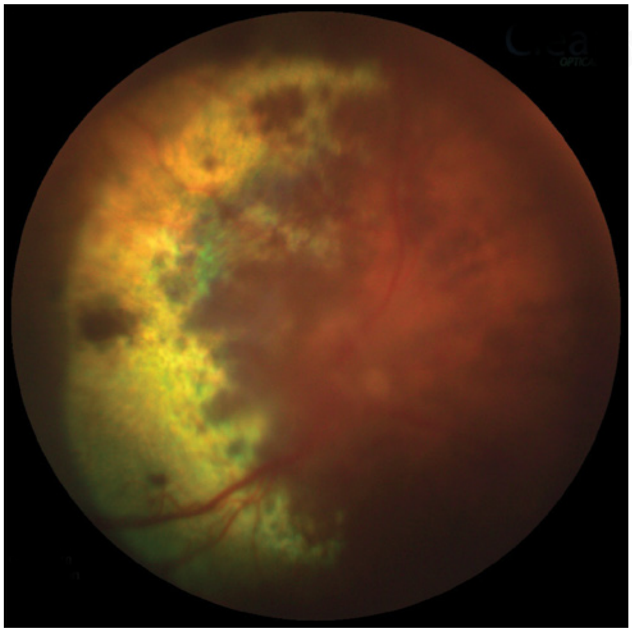


Figure 146.3 Retinal/choroidal lymphoma. Funduscopic findings associated with chorioretinal lymphoma, including chorioretinitis, exudation, and significant subretinal infiltration and hemorrhage.

Figure 146.4 Retinal/choroidal lymphoma. Funduscopic findings associated with chorioretinal lymphoma, including mild chorioretinitis, exudation, subretinal infiltration and multifocal hemorrhage.

CHAPTER 147
MYELOMA

PRESENTATION

The neoplastic proliferation of plasma cells may result in solitary or multiple plasmacytomas (typically affecting osseous and/or soft tissue), as well as multiple myeloma (typically associated with bone marrow as well as generalized symptoms which may include any combination of lethargy, discomfort, osteolysis, pathologic fractures, and/or renal dysfunction). Ancillary laboratory findings may include hyperglobulinemia, anemia, thrombocytopenia, neutropenia, and/or hypercalcemia. Ophthalmic findings may include any combination of visual deficits, anterior uveitis, chorioretinitis, retinal hemorrhage, retinal detachment, and/or secondary glaucoma. Commonly affected breeds include the Cocker Spaniel, the German Shepherd, and the Boxer.

DIAGNOSIS AND TREATMENT

The diagnosis of myeloma is made based on clinical findings as well as serum electrophoresis and/or bone marrow evaluation. Treatment comprises surgical excision wherever possible as well as case-specific chemotherapy and/or radiation therapy. Commonly employed chemotherapeutic agents include prednisone, melphalan, and cyclophosphamide. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of melphalan include gastrointestinal (GI) distress and myelosuppression. Potential adverse effects associated with the use of cyclophosphamide include GI distress, myelosuppression, and hemorrhagic cystitis.

SELECTED REFERENCES

Breuer W, Colbatzky F, Platz S, Hermanns W. Immunoglobulin-producing tumours in dogs and cats. *J Comp Pathol.* 1993;**109**(3):203–216.

Hendrix DV, Gelatt KN, Smith PJ, Brooks DE, Whittaker CJ, Chmielewski NT. Ophthalmic disease as the presenting complaint in five dogs with multiple myeloma. *J Am Anim Hosp Assoc.* 1998;**34**(2):121–128.

Patel RT, Caceres A, French AF, McManus PM. Multiple myeloma in 16 cats: a retrospective study. *Vet Clin Pathol.* 2005;**34**(4):341–352.

Violette NP, Ledbetter EC. Punctate retinal hemorrhage and its relation to ocular and systemic disease in **dogs**: 83 cases. *Vet Ophthalmol.* 2018;**21**(3):233–239.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

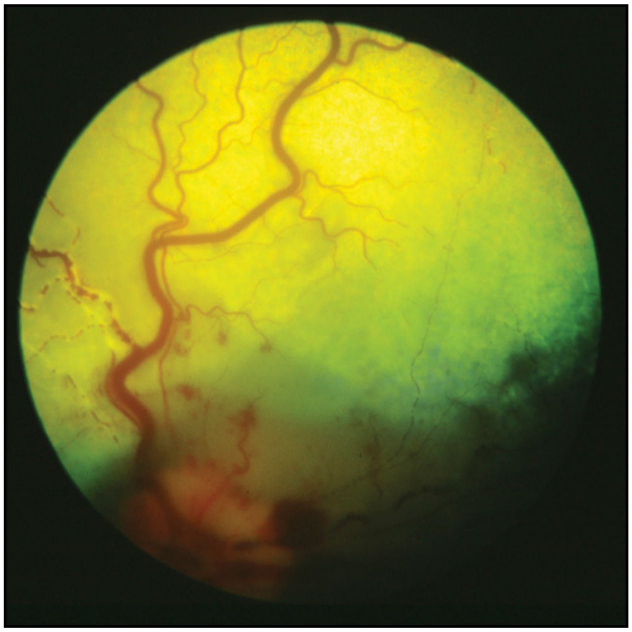


Figure 147.1 Myeloma. Funduscopic changes associated with multiple myeloma, including chorioretinitis, exudation, hemorrhage, and papillitis. Vascular congestion marked by "box-carring" is notable.

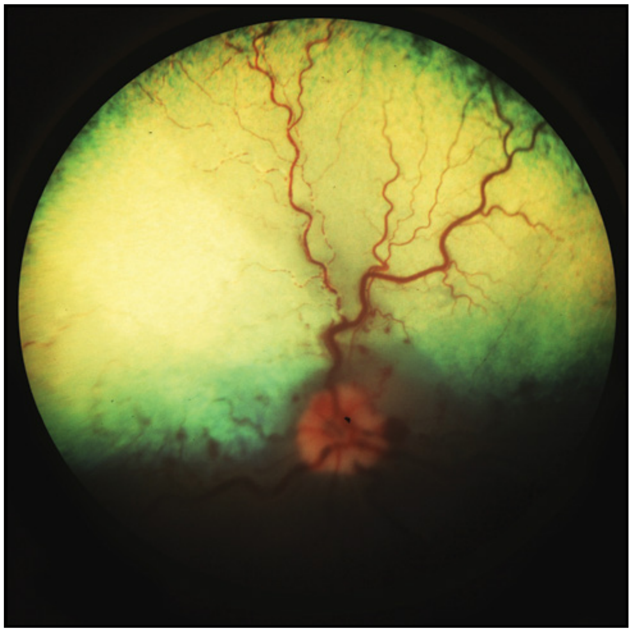


Figure 147.2 Myeloma. Funduscopic changes associated with multiple myeloma, including chorioretinitis, exudation, hemorrhage, and papillitis. Vascular congestion marked by "box-carring" is notable.

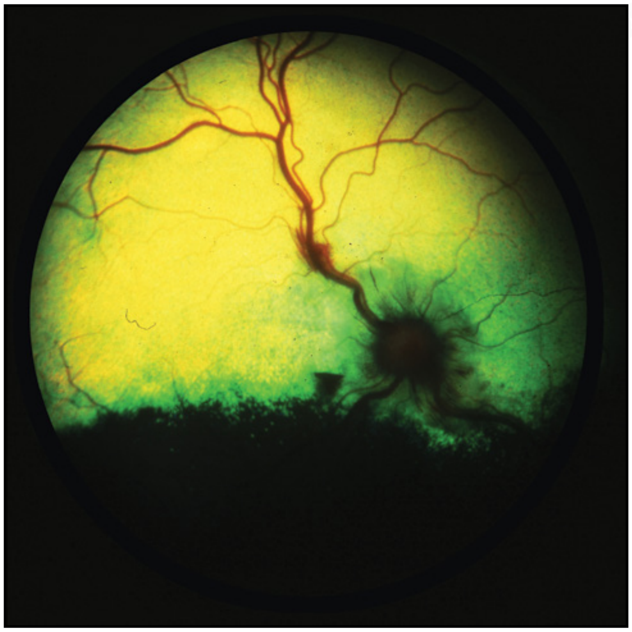


Figure 147.3 Myeloma. Funduscopic changes associated with multiple myeloma, including mild chorioretinitis, exudation, hemorrhage, and papillitis

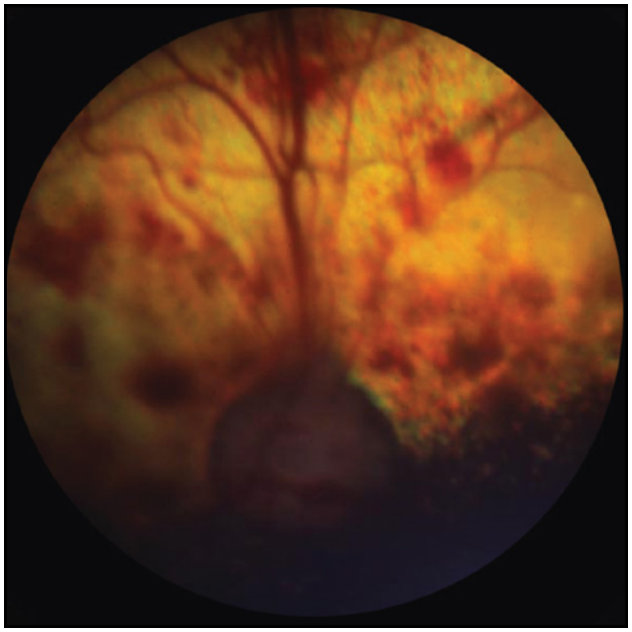


Figure 147.4 Myeloma. Funduscopic changes associated with multiple myeloma, including chorioretinitis, exudation, and marked hemorrhage.

Section 11

Diseases of the Globe and Orbit

CHAPTER 148
MICROPHTHALMIA

PRESENTATION

Congenital microphthalmia describes an abnormally small globe, generally noted in combination with a smaller palpebral fissure and orbit. The degree of microphthalmia varies considerably, extending from only a mild decrease in size of a functional globe, to rare cases of anophthalmos, in which there is a complete absence of ocular tissue. Changes are generally bilateral. Commonly affected breeds include the collie breeds, notably the Rough Collie.

DIAGNOSIS AND TREATMENT

The diagnosis of microphthalmia is made based on clinical findings, supported if necessary by B-mode ultrasonography. The major differential is phthisis bulbus (see also Phthisis bulbus), which unlike congenital microphthalmia, is the result of prior ocular inflammation. There is no treatment for microphthalmia, however in severe cases, secondary conjunctivitis may require either medical management using a topical anti-inflammatory agent or surgical management via the enucleation of residual ocular tissues.

SELECTED REFERENCES

Dell M. Severe bilateral microphthalmos in a Pomeranian pup. *Can Vet J.* 2010;**51**(12):1405–1407.

Gelatt KN, Samuelson DA, Barrie KP, Das ND, Wolf ED, Bauer JE, Andresen TL. Biometry and clinical characteristics of congenital cataracts and microphthalmia in the Miniature Schnauzer. *J Am Vet Med Assoc.* 1983;**183**(1):99–102.

Kern TJ. Persistent hyperplastic primary vitreous and microphthalmia in a dog. *J Am Vet Med Assoc*. 1981;**178**(11):1169–1171. No abstract available.

Priester WA. Congenital ocular defects in cattle, horses, cats, and dogs. *J Am Vet Med Assoc*. 1972;**160**(11):1504–1511. No abstract available.

Shaw GC, Tse MPY, Miller AD. Microphthalmia with multiple anterior segment defects in Portuguese water **dogs**. *Vet Pathol.* 2019;**56**(2):269–273.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 148.1 Microphthalmia. Clinical presentation associated with congenital microphthalmos. Inset demonstrates typical bilateral changes.

Figure 148.2 Microphthalmia. Clinical presentation associated with congenital microphthalmos. Inset demonstrates multiple affected littermates.

Figure 148.3 Microphthalmia. Clinical presentation associated with congenital microphthalmos in a collie breed.

Figure 148.4 Microphthalmia. Clinical presentation associated with congenital microphthalmos, note that the third eyelid covers most of the globe.

CHAPTER 149
PHTHISIS BULBUS

PRESENTATION

Phthisis Bulbus describes a "shrunken" eye, typically as a result of severe and/or chronic intraocular inflammation, infection, aqueous humor leakage, and/or trauma. Clinically, affected globes are blind and may display reduced size, corneal fibrosis, uveal adhesions, cataracts, and/or retinal detachment. Both canine and feline species of any breed or crossed breed may be affected.

DIAGNOSIS AND TREATMENT

The diagnosis of phthisis bulbus is made based on clinical findings, supported if necessary by B-mode ultrasonography. The major differential is congenital microphthalmos (see also Microphthalmos). Treatment of this condition may comprise benign neglect in comfortable patients, the medical management of secondary conjunctivitis if present, or enucleation if severe secondary inflammation, infection, and/or discomfort ensue.

SELECTED REFERENCES

Busse C, Hartley C, Kafarnik C, Pivetta M. Ocular alkaline injury in four dogs – presentation, treatment, and follow-up – a case series. *Vet Ophthalmol.* 2014. doi: 10.1111/vop.12171.

Duke FD, Strong TD, Bentley E, Dubielzig RR. Canine ocular tumors following ciliary body ablation with intravitreal gentamicin. *Vet Ophthalmol.* 2013;**16**(2):159–162. doi: 10.1111/j.1463-5224.2012.01050.x. Epub 2012 Jul 19.

Julien ME, Schechtmann SA, Michau TM, Welihozkiy A, Baldwin TL, Stine JM. Pharmacologic ciliary body ablation for chronic glaucoma in **dogs**: A retrospective review of 108 eyes from 2013 to 2018. *Vet Ophthalmol.* 2021;24 (Suppl 1):125–130.

van der Woerdt A, Nasisse MP, Davidson MG. Lens-induced uveitis in dogs: 151 cases (1985–1990). *J Am Vet Med Assoc.* 1992;**201**(6):921–926.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 149.1 Phthisis bulbus. Clinical presentation associated with phthisis bulbus as a result of previous intraocular inflammation.

Figure 149.2 Phthisis bulbus. Note corneal fibrosis characteristic of phthisis bulbus.

Figure 149.3 Phthisis bulbus. Note significantly reduced size of right globe.

Figure 149.4 Phthisis bulbus. Note secondary conjunctivitis in this patient.

CHAPTER 150
ORBITAL CELLULITIS

PRESENTATION

Orbital cellulitis (and/or abscessation) describes inflammation of any of the periocular tissues. Findings may include blepharospasm, conjunctivitis, chemosis, third eyelid elevation, ocular discharge, deviation of the globe from its normal position, secondary exposure keratitis, periocular swelling/fluctuance, and/or difficulty opening the mouth as a result of pressure placed on inflamed retrobulbar soft tissues by the ramus of the mandible. Symptoms typically develop relatively rapidly (over the course of several days). This process may result from trauma, penetrating wounds, extension of inflammation/infection from adjacent tissues (such as dental disease), the presence of foreign material and/or neoplasia (see also Orbital foreign bodies and retrobulbar neoplasia). Canine and feline species of any breed or crossed breed may be affected.

DIAGNOSIS AND TREATMENT

Evaluating patients affected by orbital cellulitis can be challenging as result of patient discomfort and sedation or anesthesia is frequently required in order to fully assess affected structures. The diagnosis is made based on clinical findings in conjunction (where indicated) with adjective diagnostics including complete blood count (CBC)/chemistry analysis, ultrasonography, radiography, and/or magnetic resonance imaging. Microbial samples may support initial diagnostic finings, including culture and sensitivity testing where the presence of infectious organisms is suspected as well as cytology (FNA) and/or histopathology (biopsy) where appropriate. Therapy comprises the institution of drainage where abscessation has developed as well as the institution of systemic (broad spectrum) antimicrobial and anti-inflammatory therapy. Anti-inflammatory therapy typically comprises the use of corticosteroids where not contraindicated. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Corneal protection in order to avoid exposure keratitis should be considered through the use of topical lubricating agents and/or (where indicated) the placement of temporary tarsorrhaphy sutures. The use of a protective Elizabethan collar will prevent self-trauma until healing is complete.

SELECTED REFERENCES

Armour MD, Broome M, Dell'Anna G, Blades NJ, Esson DW. A review of orbital and intracranial magnetic resonance imaging in 79 canine and 13 feline patients (2004–2010). *Vet Ophthalmol.* 2011;**14**(4):215–226. doi: 10.1111/j.1463-5224.2010.00865.x. Epub 2011 Apr 18.

Betbeze C. Management of **orbital** diseases. *Top Companion Anim Med.* 2015;**30**(3):107–117.

Dennis R. Use of magnetic resonance imaging for the investigation of orbital disease in small animals. *J Small Anim Pract.* 2000;**41**(4):145–155.

Tovar MC, Huguet E, Gomezi MA. Orbital cellulitis and intraocular abscess caused by migrating grass in a cat. *Vet Ophthalmol.* 2005;**8**(5):353–356.

Wang AL, Ledbetter EC, Kern TJ. Orbital abscess bacterial isolates and in vitro antimicrobial susceptibility patterns in dogs and cats. *Vet Ophthalmol.* 2009;**12**(2):91–96. doi: 10.1111/j.1463-5224.2008.00687.x.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 150.1 Orbital cellulitis. Clinical presentation of orbital cellulitis, note significant periocular swelling.

Figure 150.2 Orbital cellulitis. Inset demonstrates associated changes in the adjacent oral cavity.

Figure 150.3 Orbital cellulitis. Note conjunctival chemosis. Inset demonstrates associated changes in the adjacent oral cavity.

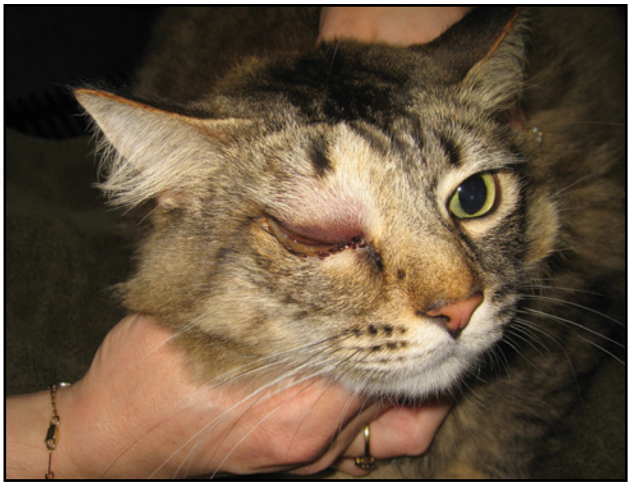


Figure 150.4 Orbital cellulitis. Clinical presentation of orbital cellulitis, note periocular swelling and blepharospasm.

CHAPTER 151
EXTRAOCULAR MYOSITIS (EOM)

PRESENTATION

Extraocular myositis describes a primary (lymphocyte-mediated) inflammatory process affecting the extraocular muscles in dogs. Clinical symptoms comprise acute onset, bilateral, symmetrical exophthalmos. Despite their startling appearance, patients are typically visual, normotensive, and relatively comfortable. Young, large-breed dogs are commonly affected, notably the Golden Retriever.

DIAGNOSIS AND TREATMENT

The diagnosis is made based on clinical findings. Biopsy of affected muscles is only rarely indicated due to the significant risk of serious iatrogenic trauma and subsequent fibrosis. In contrast to orbital myositis, antibodies directed against type 2M fibers are not typically detected. Chronic or untreated cases may develop secondary fibrosis of the extraocular muscles, leading to significant strabismus. Treatment encompasses systemic immunosuppressive therapy, typically comprising corticosteroids, azathioprine, and/or cyclosporine. Therapy can frequently be tapered once symptoms are controlled; however, long-term treatment may be required. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight-gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of azathioprine include gastrointestinal (GI) distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of cyclosporine include hypersensitivity and GI distress.

SELECTED REFERENCES

Allgoewer I, Blair M, Basher T, Davidson M, Hamilton H, Jandeck C, Ward D, Wolfer J, Shelton GD. Extraocular muscle myositis and restrictive strabismus in 10 dogs. *Vet Ophthalmol.* 2000;**3**(1):21–26.

Carpenter JL, Schmidt GM, Moore FM, Albert DM, Abrams KL, Elner VM. Canine bilateral extraocular polymyositis. *Vet Pathol.* 1989;**26**(6):510–512.

Fischer MC, Busse C, Adrian AM. Magnetic resonance imaging findings in **dogs** with orbital inflammation. *J Small Anim Pract.* 2019;**60**(2):107–115.

Williams DL. Extraocular myositis in the dog. *Vet Clin North Am Small Anim Pract.* 2008;**38**(2):347–359, vii. doi: 10.1016/j.cvsm.2007.11.010. Review.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 151.1 Extraocular myositis. Clinical presentation associated with extraocular myositis, note the typical bilaterally symmetric exophthalmos.

Figure 151.2 Extraocular myositis. Common clinical presentation associated with extraocular myositis in a Golden Retriever.

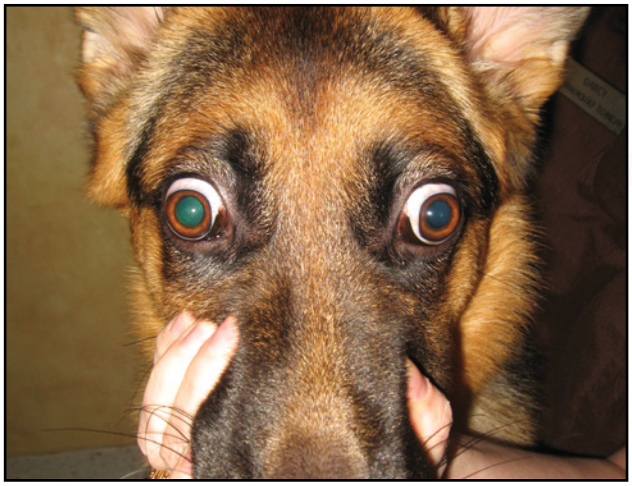


Figure 151.3 Extraocular myositis. Note that the patient is visual and comfortable.

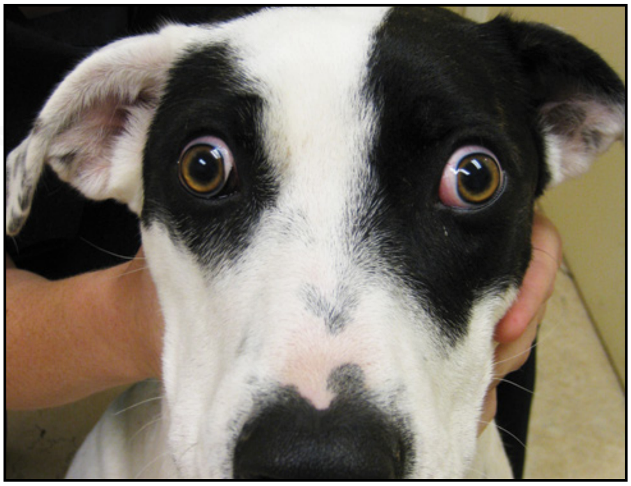


Figure 151.4 Extraocular myositis. Note that this condition typically presents in young, large breed dogs.

CHAPTER 152
ZYGOMATIC SIALOADENITIS

PRESENTATION

Zygomatic sialoadenitis (and/or associated zygomatic salivary mucocoele) describes an inflammatory process affecting the zygomatic salivary gland. Potential causes include bacterial infection, immune-mediated inflammation, and/or trauma. Clinically, affected patients may display any combination of conjunctivitis, ocular discharge, third eyelid elevation, exophthalmos, globe deviation, and/or difficulty opening the mouth. Commonly affected breeds include the Labrador Retriever and Golden Retriever.

DIAGNOSIS AND TREATMENT

The diagnosis of zygomatic sialoadenitis is made based on clinical findings, orbital magnetic resonance imaging, and cytological or histological evaluation of FNA or biopsy samples of affected tissue. Differentials for sialoadenitis represent orbital cellulitis and neoplasia (including neoplasia of the zygomatic salivary gland itself) and (see also Orbital cellulitis and retrobulbar neoplasia). Treatment may comprise medical (systemic antimicrobial and anti-inflammatory therapy) and/or surgical resection of affected tissue.

SELECTED REFERENCES

Bartoe JT, Brightman AH, Davidson HJ. Modified lateral orbitotomy for vision-sparing excision of a zygomatic mucocele in a dog. *Vet Ophthalmol.* 2007;**10**(2):127–131.

Cannon MS, Paglia D, Zwingenberger AL, Boroffka SA, Hollingsworth SR, Wisner ER. Clinical and diagnostic imaging findings in **dogs** with **zygomatic** sialadenitis: 11 cases (1990–2009). *J Am Vet Med Assoc.* 2011;**239**(9):1211–1218.

Lieske DE, Rissi DR. A retrospective study of **salivary gland diseases** in 179 **dogs** (2010–2018). *J Vet Diagn Invest.* 2020;**32**(4):604–610.

McGill S, Lester N, McLachlan A, Mansfield C. Concurrent sialocoele and necrotising sialadenitis in a dog. *J Small Anim Pract.* 2009;**50**(3):151–156. doi: 10.1111/j.1748-5827.2009.00706.x.

Philp HS, Rhodes M, Parry A, Baines SJ. Canine zygomatic salivary mucocoele following suspected oropharyngeal penetrating stick injury. *Vet Rec.* 2012;**171**(16):402. doi: 10.1136/vr.100892. Epub 2012 Aug 18.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 152.1 Zygomatic sialoadenitis. Clinical presentation associated with zygomatic sialoadenitis, note common presentation in a Golden Retriever.

Figure 152.2 Zygomatic sialoadenitis. Note associated conjunctivitis, ocular discharge, third eyelid elevation and discomfort/blepharospasm.

Figure 152.3 Zygomatic sialoadenitis. Note mild periocular swelling.

Figure 152.4 Zygomatic sialoadenitis. Note associated conjunctivitis, third eyelid elevation, and ocular discharge.

CHAPTER 153
ORBITAL FAT PAD PROLAPSE

PRESENTATION

Occasionally, a partial prolapse of the orbital fat pad may be noted in dogs, and less commonly in cats. Fat prolapse results from either congenital or traumatic weakness associated with orbital soft tissues. Clinically, a fat prolapse presents as a smooth, pink to cream-colored, fluctuant, non-painful swelling adjacent to the globe. Manipulation of the globe may exacerbate symptoms. Symptoms are commonly unilateral. Canine and feline species of any breed or crossed-breed may be affected.

DIAGNOSIS AND TREATMENT

A tentative diagnosis of orbital fat pad prolapse is made based on clinical findings. The diagnosis may be confirmed by cytological or histological evaluation of representative fine needle aspirate or biopsy samples, respectively. The major differential for fat pad prolapse is represented by neoplastic tissue, notably benign hibernomas. If indicated, prolapsed tissue may be surgically excised and surrounding soft tissue imbricated. Postoperative care comprises routine systemic and/or topical antimicrobial and/or anti-inflammatory therapy as well as the use of a protective Elizabethan collar to prevent self-trauma.

SELECTED REFERENCES

Boydell P, Paterson S, Pike R. Orbital fat prolapse in the dog. *J Small Anim Pract.* 1996;**37**(2):61–63.

Charnock L, Doran B, Milley E, Preston T. Canine retrobulbar lipoma excision through a ventral transpalpebral anterior orbitotomy. *Can Vet J.* 2020;**61**(3):257–262.

Cho J. Surgery of the globe and orbit. *Top Companion Anim Med.* 2008;**23**(1):23–37. doi: 10.1053/j.ctsap.2007.12.004.

McNab AA. Subconjunctival fat prolapse. *Aust N Z J Ophthalmol.* 1999;**27**(1):33–36.

Ravi M, Schobert CS, Kiupel M, Dubielzig RR. Clinical, morphologic, and immunohistochemical features of canine orbital hibernomas. *Vet Pathol.* 2014;**51**(3):563–568. doi: 10.1177/0300985813493913. Epub 2013 Jun 21.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 153.1 Orbital fat pad prolapse. Clinical presentation associated with prolapsed orbital fat.

Figure 153.2 Orbital fat pad prolapse. Note the lack of inflammation.

Figure 153.3 Orbital fat pad prolapse. Note the common dorsal location and the non-painful presentation.

Figure 153.4 Orbital fat pad prolapse. Note the typical unilateral presentation.

CHAPTER 154
ORBITAL FOREIGN BODIES

PRESENTATION

The orbit comprises the bones of the skull adjacent to the globe, temporal, masseter and pterygoid muscles and the periorbital facia. Foreign material may enter the orbit, including plant-based material (notably retrobulbar "stick injuries" which gain entry to the orbit via the oral cavity and "foxtails" which gain entry to the orbit via the fornix) and metallic objects (notably air gun pellets). Clinical symptoms may include any combination of blepharospasm, conjunctivitis, chemosis, third eyelid elevation, ocular discharge, deviation of the globe from its normal position, secondary exposure keratitis, and/or periocular swelling. Symptoms typically develop relatively rapidly (over the course of hours to days) and are typically unilateral.

DIAGNOSIS AND TREATMENT

Evaluating patients affected by orbital inflammation and/or the presence of foreign material can be challenging as result of patient discomfort and sedation or anesthesia is frequently required in order to fully assess affected structures. The diagnosis is made based on clinical findings in conjunction (where indicated) with adjunctive diagnostics including ultrasonography, radiography, and/or magnetic resonance imaging of affected tissues. Plant-based foreign bodies can be challenging to identify, MRI being most useful in this regard and surgical exploration sometimes being required. Metallic foreign bodies are easily identified using conventional radiography. Differentials include orbital cellulitis and retrobulbar neoplasia (see also orbital cellulitis and retrobulbar neoplasia). Therapy comprises the removal of reactive or organic foreign material (as well as the institution of drainage where abscessation has developed) and the institution of systemic (broad spectrum) antimicrobial and anti-inflammatory therapy. Anti-inflammatory therapy typically comprises the use of corticosteroids wherever possible. Corneoscleral repair and/or lens extraction may be indicated depending on the extent of injury. Severely traumatized globes or those displaying endophthalmitis (see also endophthalmitis), may require enucleation. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Corneal protection in order to avoid exposure keratitis should be considered through the use of topical lubricating agents and/or (where indicated) the placement of temporary tarsorrhaphy sutures.

SELECTED REFERENCES

Cherry RL, Johnson KL, Hespel AM, Tobias KM, Ward DA. Migration of retrobulbar wooden **foreign body** between diagnostic imaging and surgical extraction in a German shepherd **dog**. *Vet Ophthalmol.* 2019;**22**(3):353–359.

Håkansson NW and Håkansson BW. Transfrontal orbitotomy in the dog: an adaptable three-step approach to the orbit. *Vet Ophthalmol.* 2010;**13**(6):377–383. doi: 10.1111/j.1463-5224.2010.00830.x.

Welihozkiy A, Pirie CG, Pizzirani S. Scleral and suprachoroidal foreign body in a dog – a case report. *Vet Ophthalmol.* 2011;**14**(5):345–351. doi: 10.1111/j.1463-5224.2011.00922.x. Epub 2011 Jul 12.

Woolfson JM and Wesley RE. Magnetic resonance imaging and computed tomographic scanning of fresh (green) wood foreign bodies in dog orbits. *Ophthal Plast Reconstr Surg.* 1990;**6**(4):237–240.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 154.1 Orbital foreign bodies. Clinical presentation associated with penetrating orbital and ocular trauma as a result of a metallic air gun pellet.

Figure 154.2 Orbital foreign bodies. Clinical presentation associated with the presence of a grass awn, embedded in the orbital tissues.

Figure 154.3 Orbital foreign bodies. Clinical presentation associated with an oral (plant based) penetrating injury.

Figure 154.4 Orbital foreign bodies. Clinical presentation associated with the presence of a cat claw embedded in scleral and orbital tissues.

CHAPTER 155
ENDOPHTHALMITIS/PANOPHTHALMITIS

PRESENTATION

The term endophthalmitis describes inflammation of the internal tissues of the eye. The term panophthalmitis describes inflammation of the internal tissues as well as external layers of the eye. Panophthalmitis is frequently associated with inflammation of adjacent orbital tissues. Both conditions may result from trauma, infection (external of internal), penetrating wounds, foreign material, post-surgical complication, the extension of neoplasia, or the extension of uncontrolled inflammation from other parts of the eye such as the lens or uvea. Affected patients may display any combination of blepharospasm, conjunctivitis, chemosis, periocular swelling, third eyelid elevation, ocular discharge, keratitis and/or clinically apparent uveitis/chorioretinitis.

DIAGNOSIS AND TREATMENT

Evaluating patients affected by endophthalmitis/panophthalmitis can be challenging as result of patient discomfort and sedation or anesthesia may be required in order to fully assess affected structures. The diagnosis is made based on clinical finings in conjunction (where indicated) with complete blood count (CBC)/chemistry analysis, ultrasonography, radiography, and/or magnetic resonance imaging of affected tissues. Microbial samples may support initial diagnostic findings, including culture and sensitivity testing where the presence of infectious organisms is suspected as well as cytology (FNA) and/or histopathology (biopsy) wherever appropriate. Therapy comprises the institution of drainage where abscessation has developed, as well as the institution of aggressive topical, sub-conjunctival, intraocular, and/or systemic (broad spectrum) antimicrobial therapy. Systemic anti-inflammatory therapy typically comprises the use of corticosteroids wherever possible. Severely and/or chronically inflamed globes frequently require enucleation. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. The use of a protective Elizabethan collar will prevent self-trauma until healing is complete.

SELECTED REFERENCES

Bell CM, Pot SA, Dubielzig RR. Septic implantation syndrome in dogs and cats: a distinct pattern of endophthalmitis with lenticular abscess. *Vet Ophthalmol*. 2013;**16**(3):180–185. doi: 10.1111/j.1463-5224.2012.01046.x. Epub 2012 Jul 19.

Hendrix DV, Rohrbach BW, Bochsler PN, English RV. Comparison of histologic lesions of endophthalmitis induced by Blastomyces dermatitidis in untreated and treated dogs: 36 cases (1986-2001). *J Am Vet Med Assoc*. 2004;**224**(8):1317–1322.

Ledbetter EC, Landry MP, Stokol T, Kern TJ, Messick JB. Brucella canis endophthalmitis in 3 dogs: clinical features, diagnosis, and treatment. *Vet Ophthalmol*. 2009;**12**(3):183–191. doi: 10.1111/j.1463-5224.2009.00690.x.

Scott EM, Esson DW, Fritz KJ, Dubielzig RR. Major breed distribution of canine patients enucleated or eviscerated due to glaucoma following routine cataract surgery as well as common histopathologic findings within enucleated globes. *Vet Ophthalmol*. 2013;**16** Suppl 1:64–72. doi: 10.1111/vop.12034. Epub 2013 Feb 13.

Wooff PJ, Dees DD, Teixeria L. Aspergillus spp. **panophthalmitis** with intralenticular invasion in **dogs**: report of two cases. *Vet Ophthalmol.*. 2018;**21**(2):182–187.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 155.1 Endophthalmitis/panophthalmitis. Clinical presentation associated with endophthalmitis.

Figure 155.2 Endophthalmitis/panophthalmitis. Clinical presentation associated with panophthalmitis, note severe discharge and discomfort.

Figure 155.3 Endophthalmitis/panophthalmitis. Clinical presentation associated with panophthalmitis, note hemorrhagic discharge due to penetrating wound.

Figure 155.4 Endophthalmitis/panophthalmitis. Clinical presentation associated with panophthalmitis, note marked purulent discharge suggestive of severe infection.

CHAPTER 156
PROPTOSIS OF THE GLOBE

PRESENTATION

Proptosis describes partial or complete dislocation of globe from its normal position with the orbit, typically as a result of trauma. Secondary entrapment by severely swollen periorbital tissue is common. Other associated symptoms may include subconjunctival hemorrhage, episcleral hemorrhage, hyphema, and/or exposure keratopathy.

Negative prognostic indicators include

- multiple extraocular muscular avulsions
- corneoscleral rupture
- hyphema

Positive prognostic indicators include

- significantly brachycephalic conformation
- minimal globe displacement
- miotic pupil and/or the presence of a pupillary light reflex (PLR)

Proptosis occurs most commonly in brachycephalic breeds, notably the Shi Tzu, Pug, and Lhasa Apso.

DIAGNOSIS AND TREATMENT

Initial treatment of ocular proptosis encompasses addressing potentially life-threating injuries and stabilizing the patient. Consideration should be given to enucleating severely traumatized globes, which are unlikely to be salvageable. The ocular surface should be moistened with physiologic fluids, prior to globe replacement under general anesthesia. Severe eyelid swelling may necessitate canthotomy in order to facilitate globe replacement. Temporary tarsorrhaphy sutures are typically placed following proptosis reduction and left in place for 10–14 days. Care should be taken to avoid corneal abrasion and to allow for the resolution of eyelid swelling which will occur in the days following repair. Postoperative care comprises routine (systemic) antimicrobial, anti-inflammatory and analgesic therapy. The use of protective Elizabethan collar will prevent self-trauma. Wherever possible, the goal of proptosis reduction is both vision and globe salvage; however, in some cases it may be possible to salvage the globe but not vision. Potential complications following proptosis include permanent strabismus (commonly as a result of medial rectus muscle avulsion), ulcerative keratitis, KCS, neuroretinal degeneration, and/or phthisis bulbi (see also phthisis bulbi).

SELECTED REFERENCES

Ali KM and Mostafa AA. Clinical findings of traumatic **proptosis** in small-breed **dogs** and complications associated with globe replacement surgery. *Open Vet J*. 2019;**9**(3):222–229.

Cho J. Surgery of the globe and orbit. *Top Companion Anim Med*. 2008;**23**(1):23–37. doi: 10.1053/j.ctsap.2007.12.004.

Gilger BC, Hamilton HL, Wilkie DA, van der Woerdt A, McLaughlin SA, Whitley RD. Traumatic ocular proptoses in dogs and cats: 84 cases (1980-1993). *J Am Vet Med Assoc*. 1995;**206**(8):1186–1190.

Mandell DC and Holt E. Ophthalmic emergencies. *Vet Clin North Am Small Anim Pract*. 2005;**35**(2):455–480, vii-viii.

Pe'er O, Oron L, Ofri R. Prognostic indicators and outcome in **dogs** undergoing temporary tarsorrhaphy following traumatic **proptosis**. *Vet Ophthalmol*. 2020;**23**(2):245–251.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 156.1 Proptosis of the globe. Clinical presentation associated with mild to moderate proptosis of the globe.

Figure 156.2 Proptosis of the globe. Clinical presentation associated with severe proptosis of the globe, note brachycephalic patient.

Figure 156.3 Proptosis of the globe. Clinical presentation associated with severe proptosis of the globe, note brachycephalic patient.

Figure 156.4 Proptosis of the globe. Clinical presentation associated with catastrophic proptosis of the globe, enucleation is indicated.

CHAPTER 157
FELINE RESTRICTIVE ORBITAL MYOFIBROBLASTIC SARCOMA (FROMS)

PRESENTATION

Feline restrictive orbital myofibroblastic sarcoma (FROMS) represents a progressive and malignant pathology which may affect the ocular orbital/periorbital, oral and/or cranial structures in cats. Terms including “nodular fasciitis” and “orbital pseudotumor” have also been applied to this condition. Affected animals are typically middle aged to older, with no breed predisposition noted. Clinical symptoms typically include eyelid/periorbital thickening, conjunctivitis, scleral/episcleral infiltration and thickening, ulcerative keratitis secondary to exposure keratopathy, restricted globe movement and/or globe deviation/exophthalmos. Unilateral ocular/orbital disease frequently progresses to become bilateral and/or to involve oral and/or cranial structures, so that affected animals may have difficulty eating. Progression is typically slow (occurring over weeks to months) but relentless.

DIAGNOSIS AND TREATMENT

The diagnosis of FROMS is notably made by a combination of clinical findings, soft tissue imaging (most appropriately MRI) and the histopathological interpretation of affected tissues. Histologic findings typically comprise poorly delineated and infiltrative spindle cell proliferation and the deposition of an associated collagenous matrix and a mild inflammatory component. The diagnosis can be challenging, with more benign inflammatory/reactive processes representing differentials. Affected animals demonstrate infiltration and invasion of spindloid cells, along fascial planes, entrapment and atrophy of normal orbital tissues, secondary keratitis, and minimal associated lymphoplasmacytic inflammation of orbital tissues. Treatment encompasses palliative anti-inflammatory/chemotherapy, usually progressing to enucleation/exenteration. Despite identification and treatment, the prognosis for affected animals is typically poor.

SELECTED REFERENCES

Bell CM, Schwarz T, Dubielzig RR. Diagnostic features of feline restrictive orbital myofibroblastic sarcoma. *Vet Pathol*. 2011;48(3): 742–750.

Isaza D, Robinson NA, Pizzirani S, Pumphrey SA. Evaluation of cytology and histopathology for the diagnosis of feline orbital neoplasia: 81 cases (2004-2019) and review of the literature. *Vet Ophthalmol*. 2020;**23**(4):682–689.

Thomasy SM, Cissell DD, Arzi B, Vilches-Moure JG, Lo WY, Wisner ER, Dubielzig RR, Maggs DJ. Restrictive orbital myofibroblastic sarcoma in a cat – cross-sectional imaging (MRI & CT) appearance, treatment, and outcome. *Vet Ophthalmol*. 2013;16 Suppl 1:123–129.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 157.1 Feline restrictive orbital myofibroblastic sarcoma. Clinical presentation, note significant periocular thickening and discharge. Inset demonstrating oral extension of disease.

Figure 157.2 Feline restrictive orbital myofibroblastic sarcoma. Note firm, painless orbital deformity early in the course of disease.

Figure 157.3 Feline restrictive orbital myofibroblastic sarcoma. Note significant exposure keratopathy associated with decreased eyelid mobility.

Figure 157.4 Feline restrictive orbital myofibroblastic sarcoma. Clinical presentation associated with advanced periocular infiltration.

CHAPTER 158
PERIPHERAL NERVE SHEATH TUMOR (PNST)

PRESENTATION

Peripheral nerve sheath tumors (PNSTs) represent a spindloid neoplastic proliferation of the perineural connective tissues. These tumors include schwannomas, neurogenic sarcomas, neurofibromas, and neurofibrosarcomas and may affect a wide range of soft tissues including the orbital, adnexal, and ocular structures. Clinically, lesions appear as slowly progressive, firm, non-painful swellings of the orbit, fornix, or eyelids. Secondary conjunctivitis and/or deviation of the globe may be associated with their development. Canine and feline species of any breed or crossed breed may be affected.

DIAGNOSIS AND TREATMENT

The diagnosis of PNST is made based on clinical findings in conjunction with histopathological interpretation of biopsy samples. The extent of the lesion and involvement of local tissues may be further assessed by imaging in the form of MRI or CT. Pretreatment staging via thoracic radiography and regional lymph node aspiration is recommended. Treatment comprises wide surgical resection wherever possible, followed by radiation therapy, chemotherapy, and/or metronomic therapy wherever indicated. Individual chemotherapeutic protocols are most ideally designed by a veterinary oncologist however typically include a variable combination of prednisone, vincristine, cyclophosphamide, and/or doxorubicin. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of vincristine include stomatitis, GI distress, neuropathy, hepatopathy, and myelosuppression. Potential adverse effects associated with the use of cyclophosphamide include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of doxorubicin include hypersensitivity, GI distress, cardiac dysfunction, and myelosuppression.

SELECTED REFERENCES

Briffod C, Hélie P, De Lasalle J, de Lorimier LP, Moreau AR, Alves DA, Vanore M. Retrobulbar malignant **peripheral nerve sheath tumor** in a golden retriever **dog**: a challenging diagnosis. *Can Vet J*. 2018;**59**(4):379–384.

Curto E, Clode AB, Durrant J, Montgomery KW, Gilger BC. Retrobulbar pigmented **peripheral nerve sheath tumor** in a **dog**. *Vet Ophthalmol*. 2016;**19**(6):518–524.

Evans PM, Lynch GL, Dubielzig RR. Anterior uveal spindle cell tumor in a cat. *Vet Ophthalmol*. 2010;**13**(6):387–390. doi: 10.1111/j.1463-5224.2010.00837.x.

Hoffman A, Blocker T, Dubielzig R, Ehrhart EJ. Feline periocular peripheral nerve sheath tumor: a case series. *Vet Ophthalmol*. 2005;**8**(3):153–158.

Newkirk KM and Rohrbach BW. A retrospective study of eyelid tumors from 43 cats. *Vet Pathol*. 2009;**46**(5):916–927. doi: 10.1354/vp.08-VP-0205-N-FL. Epub 2009 May 9.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 158.1 Peripheral nerve sheath tumors. Clinical presentation associated with the presence of a peripheral nerve sheath tumor.

Figure 158.2 Peripheral nerve sheath tumors. Note common presentation at or near the medial canthus.

Figure 158.3 Peripheral nerve sheath tumors. Note that this lesion is firm, slowly progressive, and non-painful.

Figure 158.4 Peripheral nerve sheath tumors. Clinical presentation associated with the presence of a peripheral nerve sheath tumor in a feline patient.

CHAPTER 159
RETROBULBAR NEOPLASIA

PRESENTATION

Retrobulbar neoplasia (with or without associated orbital inflammation) is not uncommon, with most orbital neoplasms being both primary and malignant. Prevalent tumor types include carcinoma (squamous cell carcinoma and adenocarcinoma), sarcoma (spindle cell sarcomas, fibrosarcoma, hemangiosarcoma, multilobular orbital sarcoma, osteosarcoma, and chondrosarcoma), meningioma, and lymphoma. Clinical findings may include blepharospasm, conjunctivitis, chemosis, third eyelid elevation, ocular discharge, deviation of the globe from its normal position, secondary exposure keratitis, periocular swelling/fluctuance, and/or difficulty opening the mouth, visual, and/or neurological defects. Symptoms typically develop relatively slowly (over the course of weeks to months), with most patients remaining comfortable until changes are significantly advanced. Affected patients are typically older. Canine and feline species of any breed or crossed breed may be affected.

DIAGNOSIS AND TREATMENT

Diagnostic modalities useful for the investigation of orbital disease include radiography, ultrasonography, computed tomography, and/or magnetic resonance imaging. The diagnosis is made based cytological or histopathological evaluation of samples harvested by FNA or biopsy, typically post-imaging. The major differential represents orbital inflammatory disease (see also orbital cellulitis and zygomatic sialoadenitis). Treatment may include any combination of surgical resection wherever possible (potentially involving enucleation/exenteration), systemic chemotherapy, and/or radiation therapy based on the tumor type present. Anti-inflammatory therapy typically comprises the use of corticosteroids wherever not contraindicated. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Corneal protection in order to avoid exposure keratitis should be considered through the use of topical lubricating agents and/or (where indicated) the placement of temporary tarsorrhaphy sutures.

SELECTED REFERENCES

Armour MD, Broome M, Dell'Anna G, Blades NJ, Esson DW. A review of orbital and intracranial magnetic resonance imaging in 79 canine and 13 feline patients (2004-2010). *Vet Ophthalmol*. 2011;**14**(4):215–126.

Attali-Soussay K, Jegou JP, Clerc B. Retrobulbar tumors in dogs and cats: 25 cases. *Vet Ophthalmol*. 2001;**4**(1):19–27.

Cho J. Surgery of the globe and orbit. *Top Companion Anim Med*. 2008;**23**(1):23–37. doi: 10.1053/j.ctsap.2007.12.004.

Hendrix DV and Gelatt KN. Diagnosis, treatment and outcome of orbital neoplasia in dogs: a retrospective study of 44 cases. *J Small Anim Pract*. 2000;**41**(3):105–108.

Mason DR, Lamb CR, McLellan GJ. Ultrasonographic findings in 50 dogs with retrobulbar disease. *J Am Anim Hosp Assoc*. 2001;**37**(6): 557–562.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 159.1 Retrobulbar neoplasia. Clinical presentation associated with retrobulbar neoplasia (squamous cell carcinoma.)

Figure 159.2 Retrobulbar neoplasia. Clinical presentation associated with retrobulbar neoplasia (adenocarcinoma.)

Figure 159.3 Retrobulbar neoplasia. Clinical presentation associated with retrobulbar neoplasia (fibrosarcoma.)

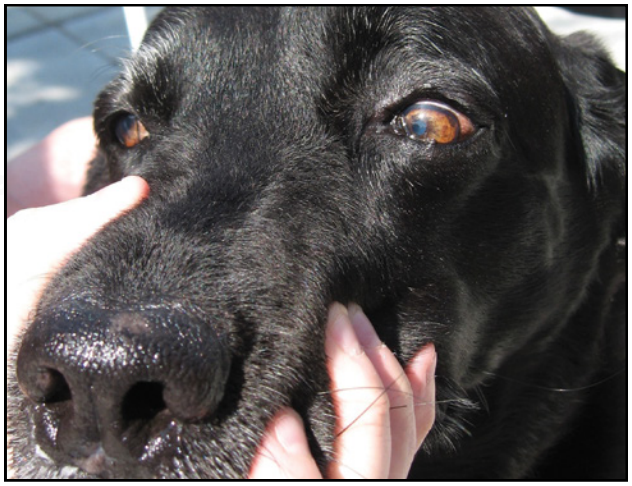


Figure 159.4 Retrobulbar neoplasia. Clinical presentation associated with retrobulbar neoplasia (osteosarcoma.)

CHAPTER 160
ORBITAL MYXOMA/MYXOSARCOMA

PRESENTATION

Myxomas and myxosarcomas are infiltrative connective tissue tumors of fibroblastic origin. Tumors arise from mesenchymal tissues, including muscle, adipose, neurovascular, and fascial tissues with the canine orbit representing a relatively commonly affected site. Tumors may display variable characteristics of malignancy, representing either benign myxomas or malignant myxosarcomas, the differentiation sometimes being challenging. Clinically, affected animals typically exhibit slowly progressive, non-painful localized swelling which may encompass subtle cranial malformation, globe deviation, third eyelid protrusion, and/or exophthalmos. Any breed or sex may be affected however middle-aged large breed dogs are overrepresented.

DIAGNOSIS AND TREATMENT

The diagnosis is made based on a combination of clinical symptoms, soft-tissue imaging, and evaluation of biopsy specimens. Histological changes comprise the presence of neoplastic fibroblasts characterized by abundant mucinous/mucopolysaccharide rich stroma. The treatment of choice generally compromises wide surgical resection wherever possible. Malignant myxosarcomas display the potential for metastasis so that presurgical staging is appropriate. Depending on malignancy, radiation and/or chemotherapy (typically using a combination of cyclophosphamide, chlorambucil, and/or doxorubicin) may be indicated.

SELECTED REFERENCES

Cécile Briffod, Pierre Hélie, Julie De Lasalle, Louis-Philippe de Lorimier, Alicia R Moreau, Derron A Alves, Maria Vanore. Retrobulbar malignant peripheral nerve sheath tumor in a golden retriever dog: a challenging diagnosis. *Can Vet J*. 2018;**59**(4):379–384.

Campos CB, Nunes FC, Gamba CO, Damasceno KA, Souza CM, Campos LC, Cassali GD. Canine low-grade intra-orbital myxosarcoma: case report. *Vet Ophthalmol*. 2015;**18**(3):251–253. doi: 10.1111/vop.12183. Epub 2014 May 19. PMID: 24837165.

Dennis R. Imaging features of orbital myxosarcoma in dogs. *Vet Radiol Ultrasound*. 2008;**49**(3):256–263. doi: 10.1111/j.1740-8261.2008.00361.x. PMID: 18546781.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 160.1 Orbital myxoma/myxosarcoma. Clinical presentation of orbital myxoma, note moderate nonpainful periorbital swelling.

Figure 160.2 Orbital myxoma/myxosarcoma. Note associated inflammation demonstrated by conjunctival hyperemia and discharge.

Figure 160.3 Orbital myxoma/myxosarcoma. Note exophthalmos and deviation of the globe.

Figure 160.4 Orbital myxoma/myxosarcoma. Clinical presentation of orbital myxoma, demonstrating significant ventral orbital infiltration.

CHAPTER 161
MULTILOBULAR TUMOR OF BONE (MTB)

PRESENTATION

Multilobular tumor of bone (MTB) (also described as multilobular osteochondrosarcoma, chondroma rodens and multilobular osteoma) is a relatively uncommon bone tumor which most frequently affects the flat bones of the canine skull (including the zygomatic arch, frontal, occipital and parietal bones, maxilla and mandible) and rarely other sites including the axial skeleton, ribs, and pelvis. Cats may rarely be affected. Affected animals are typically large breed middle aged dogs; however, dogs of any age or breed may be affected dogs with no sex predilection noted. Clinically, patients display variable combinations of a slowly enlarging firm poorly mobile non-painful subcutaneous tissue mass, exophthalmos and/or deviation of the orbit, sinus-obstruction or jaw-pain depending on the location the lesion. Although the most common clinical presentation is that of a solid and firm mass affecting the skull, neurological signs may result from cerebral compression. Lesions are slowly progressive and locally invasive with malignant behavior and metastasis possible, with the lungs most commonly affected.

DIAGNOSIS AND TREATMENT

The diagnosis of MTB is based on a combination of clinical findings, radiography, and evaluation of biopsied/excised tissue. Histologically, lesions are composed of multiple lobules, each centered on a core of cartilaginous or bony matrix and surrounded by variable numbers of spindle cells. Treatment comprises surgical resection of neoplastic tissue wherever possible. Multilobular tumors of bone are locally invasive, and recurrence after surgical resection may occur. As a consequence, adjunctive/follow-up radiation therapy is typically indicated. Chemotherapy may be additionally be recommended, to decrease the risk of metastatic disease.

SELECTED REFERENCES

Leonardi L, Carrano A, Stoppini L, Floris M. Multilobular tumor of the zygomatic bone in a dog. *Open Vet J*. 2014;**4**(1):9–11. Epub 2014 Feb 9. PMID: 26623332.

Dernell WS, Straw RC, Cooper MF, Powers BE, LaRue SM, Withrow SJ. Multilobular osteochondrosarcoma in 39 dogs: 1979-1993. *J Am Anim Hosp Assoc*. 1998;**34**(1):11–18. doi: 10.5326/15473317-34-1-11. PMID: 9527424.

Hathcock JT and Newton JC. Computed tomographic characteristics of multilobular tumor of bone involving the cranium in 7 dogs and zygomatic arch in 2 dogs. *Vet Radiol Ultrasound*. 2000;**41**(3):214–217. doi: 10.1111/j.1740-8261.2000.tb01480.x. PMID: 10850869.

Sweet KA, Nolan MW, Yoshikawa H, Gieger TL. Stereotactic radiation therapy for canine multilobular osteochondrosarcoma: eight cases. *Vet Comp Oncol*. 2020;**18**(1):76–83. doi: 10.1111/vco.12481. Epub 2019 Jun 17. PMID: 30989784.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 161.1 Multilobular tumor of bone. Clinical presentation associated with left-sided orbital deformity.

Figure 161.2 Multilobular tumor of bone. Note significant cranial deformity associated with multilobular tumor of bone.

Figure 161.3 Multilobular tumor of bone. Note left-sided ventral orbital deformity in this patient.

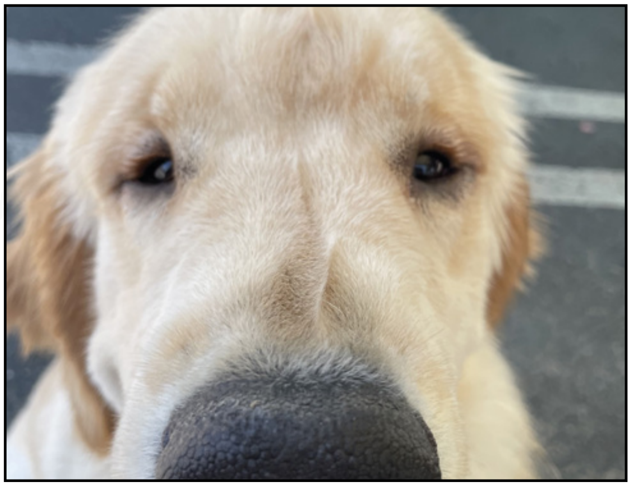


Figure 161.4 Multilobular tumor of bone. Note cranial asymmetry and deformity.

Section 12

The Glaucomas

CHAPTER 162
CONGENITAL GLAUCOMA

PRESENTATION

Congenital glaucoma describes severe malformation of the aqueous drainage tracts, which are both hereditary and congenital (i.e., present at birth). As a result, affected animals display severe, bilateral, ocular pathology, very early in life (generally within weeks to months). Symptoms may include any combination of corneal edema, secondary ulcerative keratitis, episcleral injection, buphthalmos, optic nerve damage, and/or blindness. In some cases, additional ocular abnormalities may be present. Canine and feline species of any breed or crossed breed may be affected; however this condition is uncommon.

DIAGNOSIS AND TREATMENT

Although treatment of affected animals may be attempted, vision salvage is generally not possible and typically, a (bilateral) surgical procedure of comfort is ultimately required (enucleation, cryoablation, intrascleral prosthesis, or chemical ciliary body ablation).

SELECTED REFERENCES

Glaze MB. Congenital and hereditary ocular abnormalities in cats. *Clin Tech Small Anim Pract.* 2005;**20**(2):74–82.

Reinstein S, Rankin A, Allbaugh R. Canine glaucoma: pathophysiology and diagnosis. *Compend Contin Educ Vet.* 2009;**31**(10):450–452; quiz 452–453.

Strom AR, Hässig M, Iburg TM, Spiess BM. Epidemiology of canine glaucoma presented to University of Zurich from 1995 to 2009. Part 1: Congenital and primary glaucoma (4 and 123 cases). *Vet Ophthalmol.* 2011;**14**(2):121–126. doi: 10.1111/j.1463-5224.2010.00855.x.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 162.1 Congenital glaucoma. Clinical presentation associated with congenital glaucoma in a kitten. Note marked buphthalmos, characteristic of young animals affected by glaucoma.

Figure 162.2 Congenital glaucoma. Note common bilateral presentation.

Figure 162.3 Congenital glaucoma. Note significant buphthalmos.

Figure 162.4 Congenital glaucoma. Note characteristic changes, including corneal edema, ulcerative keratitis, and buphthalmos.

CHAPTER 163
PRIMARY GLAUCOMA

PRESENTATION

Primary glaucoma describes pathology associated with a hereditary predisposition for elevated intraocular pressure (IOP) and/or ON degeneration. Although the underlying changes (comprising trabecular meshwork atrophy) generally develop over the course of many years, the presentation of primary glaucoma is typically per acute, frequently developing within just a few hours as a result of ciliary cleft collapse. Symptoms may include any combination of ocular discomfort, blepharospasm, corneal edema, episcleral injection, third eyelid elevation, miosis or mydriasis, visual deficits, and/or blindness. Chronically affected may display buphthalmos with time (see also Buphthalmos). Primary glaucoma has been described in association with numerous canine (as well as a limited number of feline) breeds, notably the Cocker Spaniel, Siberian Husky, Bassett Hound, Boston Terrier, Bouvier des Flanders, Shiba Inu, Chow Chow, and Shar Pei.

DIAGNOSIS AND TREATMENT

The diagnosis of primary glaucoma is made based on clinical findings as well as evaluation of intraocular pressure using a tonometer. Accurate "digital" tonometry is not possible. Adjunctive diagnostics include further evaluation of the external iridocorneal angle structures (via "gonioscopy") and/or the deeper trabecular meshwork and ciliary cleft (via high resolution ultrasonography). Effective treatment depends upon prompt identification of symptoms and typically encompasses the rapid lowering of IOP using intravenous mannitol (with or without the administration of concurrent anti-inflammatory therapy in the form of a corticosteroid). Following the initial stabilization of IOP, more definitive control can be achieved by a combination of medical and/or surgical management using procedures to decease aqueous production and/or maximize its outflow. Long-term adjunctive medical therapy is likely to be required in order to maintain control over IOP as well as to combat subsequent degeneration of optic nerve tissue. Pharmacologic "neuroprotection" (using a variety of potentially beneficial anti-inflammatory, calcium channel blocking, and/or receptor antagonist agents) in order to minimize ongoing optic nerve damage in visual eyes is also indicated. Patients that develop primary glaucoma in one eye are at significant risk for the subsequent development of similar pathology in their contralateral eye. Irreversibly blind, painful, and/or buphthalmic eyes should be addressed through a procedure of comfort (enucleation, cryoablation, intrascleral prosthesis, or chemical ciliary body ablation).

SELECTED REFERENCES

Brooks DE. Glaucoma in the dog and cat. *Vet Clin North Am Small Anim Pract.* 1990;**20**(3):775–797. Review.

Gelatt KN, MacKay EO. Prevalence of the breed-related glaucomas in pure-bred dogs in North America. *Vet Ophthalmol.* 2004;**7**(2): 97–111.

Jacobi S, Dubielzig RR. Feline primary open angle glaucoma. Vet Ophthalmol. 2008;**11**(3):162–165. doi: 10.1111/j.1463-5224.2008.00614.x.

Komáromy AM, Bras D, Esson DW, Fellman RL, Grozdanic SD, Kagemann L, Miller PE, Moroi SE, Plummer CE, Sapienza JS, Storey ES, Teixeira LB, Toris CB, Webb TR. The future of **canine glaucoma** therapy. *Vet Ophthalmol.* 2019;**22**(5):726–740.

Reinstein S, Rankin A, Allbaugh R. Canine glaucoma: pathophysiology and diagnosis. *Compend Contin Educ Vet.* 2009;**31**(10):450–452; quiz 452–453. Review.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 163.1 Primary glaucoma. Clinical presentation associated with primary glaucoma. Episcleral injection is notable.

Figure 163.2 Primary glaucoma. Clinical presentation associated with primary glaucoma. Episcleral injection and mild corneal edema are notable.

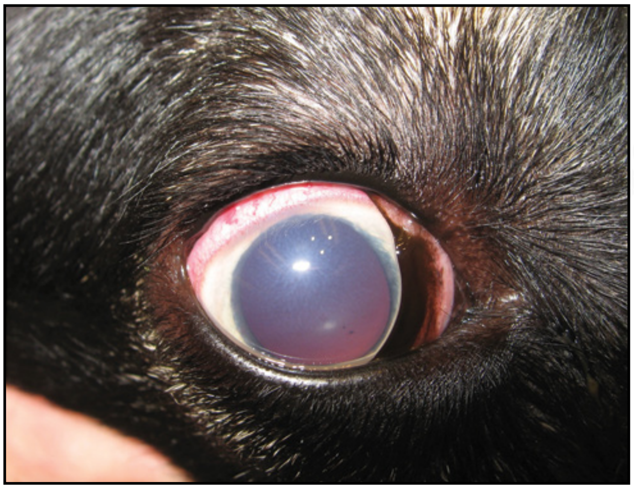


Figure 163.3 Primary glaucoma. Note corneal edema and mydriasis.

Figure 163.4 Primary glaucoma. Note episcleral injection, corneal edema, and mydriasis.

CHAPTER 164
SECONDARY (POST-INFLAMMATORY) GLAUCOMA

PRESENTATION

Secondary glaucoma describes elevated intraocular pressure associated with underlying ocular and/or systemic disease. Common causes of secondary glaucoma include lenticular (sub)luxation (anterior or posterior), ocular and/or systemic neoplasia, severe or chronic uveitis, retinal detachment, lens-induced uveitis, iris bombé, Golden Retriever associated uveitis, uveodermatologic syndrome (see also anterior lens luxation, posterior lens luxation, uveal adenocarcinoma, uveal melanoma, uveal lymphoma, feline and canine anterior uveitis, phacolytic uveitis, phacoclastic uveitis, Golden retriever associated uveitis and glaucoma, and uveodermatological syndrome). Secondary glaucoma may develop acutely, subacutely, or chronically and may affect one or both eyes. Symptoms may include any combination of ocular discomfort, blepharospasm, corneal edema, episcleral injection, third eyelid elevation, miosis or mydriasis, uveitis, visual deficits, and/or blindness. Chronically affected may display buphthalmos with time (see also buphthalmos).

DIAGNOSIS AND TREATMENT

The diagnosis of glaucoma is made based on clinical findings as well as evaluation of intraocular pressure using a tonometer. Accurate "digital" tonometry is not possible. Adjunctive diagnostics may include further evaluation of the intraocular structures using B-mode ultrasonography. Effective treatment depends upon prompt identification of symptoms and typically encompasses the rapid lowering of IOP using intravenous mannitol (with or without the administration of concurrent anti-inflammatory therapy in the form of a corticosteroid). Underlying causes of secondary glaucoma including ocular/systemic inflammation, immune deviation, lenticular stability, and/or neoplasia should specifically be addressed if identified. The use of miotic agents should be avoided when anterior lenticular (sub)luxation is known or suspected as their use may exacerbate symptoms. Following the initial stabilization of intraocular pressure (IOP), more definitive control can be achieved by a combination of medical and/or surgical management using procedures to decease aqueous production and/or maximize its outflow. Pharmacologic "neuroprotection" (using a variety of potentially beneficial anti-inflammatory, calcium channel blocking, and/or receptor antagonist agents) in order to minimize ongoing optic nerve damage in visual eyes is also indicated. Irreversibly blind, painful, and/or buphthalmic eyes should be addressed through a procedure of comfort (enucleation, cryoablation, intrascleral prosthesis, or chemical ciliary body ablation).

SELECTED REFERENCES

Dietrich U. Feline glaucomas. *Clin Tech Small Anim Pract.* 2005;**20**(2):108–116.

Gelatt KN, MacKay EO. Secondary glaucomas in the dog in North America. *Vet Ophthalmol.* 2004;**7**(4):245–259.

Johnsen DA, Maggs DJ, Kass PH. Evaluation of risk factors for development of secondary glaucoma in dogs: 156 cases (1999–2004). *J Am Vet Med Assoc.* 2006;**229**(8):1270–1274.

Pumphrey S. **Canine secondary glaucomas**. *Vet Clin North Am Small Anim Pract.* 2015;**45**(6):1335–1364, vii.

Strom AR, Hässig M, Iburg TM, Spiess BM. Epidemiology of **canine glaucoma** presented to University of Zurich from 1995 to 2009. Part 2: **secondary glaucoma** (217 cases). *Vet Ophthalmol.* 2011;**14**(2):127–132.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 164.1 Secondary (post-inflammatory) glaucoma. Clinical presentation associated with secondary glaucoma as a result of chronic lymphoplasmacytic uveitis.

Figure 164.2 Secondary (post-inflammatory) glaucoma. Clinical presentation associated with secondary glaucoma as a result of intraocular neoplasia.

Figure 164.3 Secondary (post-inflammatory) glaucoma. Clinical presentation associated with secondary glaucoma as a result of hyphema.

Figure 164.4 Secondary (post-inflammatory) glaucoma. Clinical presentation associated with secondary glaucoma as a result of phacoclastic uveitis.

CHAPTER 165
FELINE AQUEOUS HUMOR MISDIRECTION SYNDROME (AHMS)

PRESENTATION

Feline aqueous humor misdirection syndrome (AHMS) represents a unique form of feline glaucoma, which occurs as a result of aqueous accumulation within the vitreous, suspected to arise as a result of abnormalities associated with the anterior vitreous face. This condition has also been termed "malignant glaucoma." Resultant changes include vitreous expansion, anterior displacement of the lens/iris diaphragm, anterior chamber shallowing, ciliary cleft collapse and progressive intraocular pressure (IOP) elevation. Clinical symptoms may include mydriasis, anisocoria, discomfort, visual deficits, and/or blindness. Ultimately, bilateral involvement is the most common. Any breed or crossed breed may be affected.

DIAGNOSIS AND TREATMENT

The diagnosis of AHMS is made based on clinical findings (including tonometry), supported if necessary by B-mode ultrasonography. Management initially comprises medical therapy using topical carbonic anhydrase inhibitors (dorzolamide or brinzolamide) and/or miotic agents (demecarium bromide). With time, progressive IOP elevation, may dictate surgical intervention in the form of lensectomy, vitrectomy, and/or ciliary photocoagulation.

SELECTED REFERENCES

Atkins RM, Armour MD, Hyman JA. Surgical outcome of **cats** treated for **aqueous** humor **misdirection** syndrome: a case series. *Vet Ophthalmol*. 2016;**19** Suppl 1:136–142.

Blocker T and Van Der Woerdt A. The feline glaucomas: 82 cases (1995–1999). *Vet Ophthalmol*. 2001;**4**(2):81–85.

Czederpiltz JM, La Croix NC, van der Woerdt A, Bentley E, Dubielzig RR, Murphy CJ, Miller PE. Putative aqueous humor misdirection syndrome as a cause of glaucoma in cats: 32 cases (1997–2003). *J Am Vet Med Assoc*. 2005;**227**(9):1434–1441.

Wilcock BP, Peiffer RL Jr, Davidson MG. The causes of glaucoma in cats. *Vet Pathol*. 1990;**27**(1):35–40.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

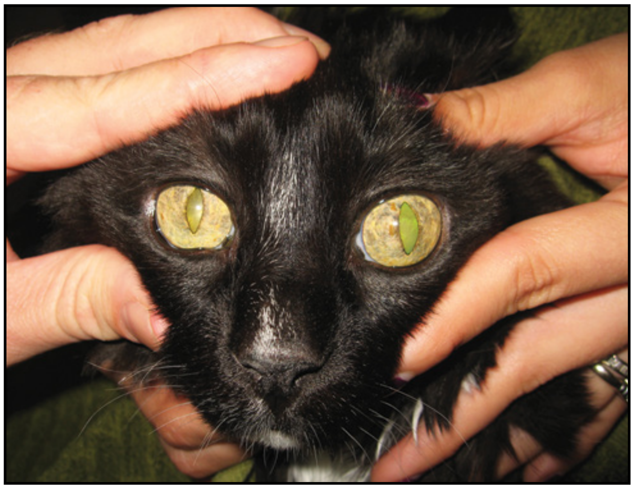


Figure 165.1 Feline aqueous humor misdirection syndrome. Clinical presentation associated with aqueous humor misdirection syndrome, note bilateral presentation.

Figure 165.2 Feline aqueous humor misdirection syndrome. Anisocoria and anterior chamber shallowing are notable.

Figure 165.3 Feline aqueous humor misdirection syndrome. Note significant anisocoria.

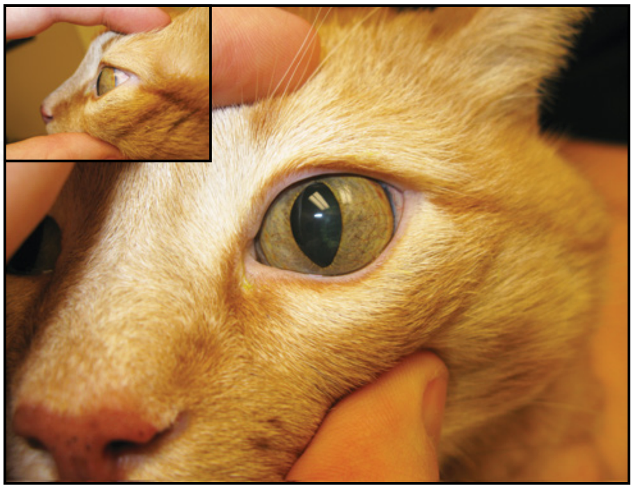


Figure 165.4 Feline aqueous humor misdirection syndrome. Inset demonstrates anterior chamber shallowing.

CHAPTER 166
PIGMENTARY GLAUCOMA

PRESENTATION

Pigmentary glaucoma (also referred to as "ocular melanosis") describes a pathological elevation in intraocular pressure, which occurs as a result of the proliferation and accumulation of cells containing melanin in the aqueous outflow tracts. Studies support a hereditary etiology. Symptoms are generally bilateral, although not always symmetrical and comprise the visible accumulation of pigment within the scleral/episcleral, corneal and/or uveal tissues as well as any combination of corneal edema, blepharospasm, episcleral injection, visual deficits, blindness, and/or buphthalmos. Commonly affected breeds include the Cairn Terrier, Boxer, and Labrador Retriever.

DIAGNOSIS AND TREATMENT

The diagnosis of pigmentary glaucoma is made based on clinical findings as well as evaluation of intraocular pressure using a tonometer. Palliative management of affected patients comprises medical and/or surgical treatment of elevated intraocular pressure (IOP). Unfortunately, however disease progression frequently means that a procedure of comfort is ultimately required (enucleation, cryoablation, intrascleral prosthesis placement, or chemical ciliary body ablation).

SELECTED REFERENCES

Hanselman BA. Melanocytic **glaucoma** in a **cairn terrier**. *Can Vet J*. 2002;**43**(4):296–298.

Petersen-Jones SM, Forcier J, Mentzer AL. Ocular melanosis in the Cairn Terrier: clinical description and investigation of mode of inheritance. *Vet Ophthalmol*. 2007;**10** Suppl 1:63–69.

Petersen-Jones SM, Mentzer AL, Dubielzig RR, Render JA, Steficek BA, Kiupel M. Ocular melanosis in the **Cairn Terrier**: histopathological description of the condition, and immunohistological and ultrastructural characterization of the characteristic pigment-laden cells. *Vet Ophthalmol*. 2008;**11**(4):260–268.

van de Sandt RR, Boevé MH, Stades FC, Kik MJ. Abnormal ocular pigment deposition and glaucoma in the dog. *Vet Ophthalmol*. 2003;**6**(4):273–278.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 166.1 Pigmentary glaucoma. Clinical presentation associated with pigmentary glaucoma. Note corneal edema and visible pigment accumulation in the scleral/episcleral tissues.

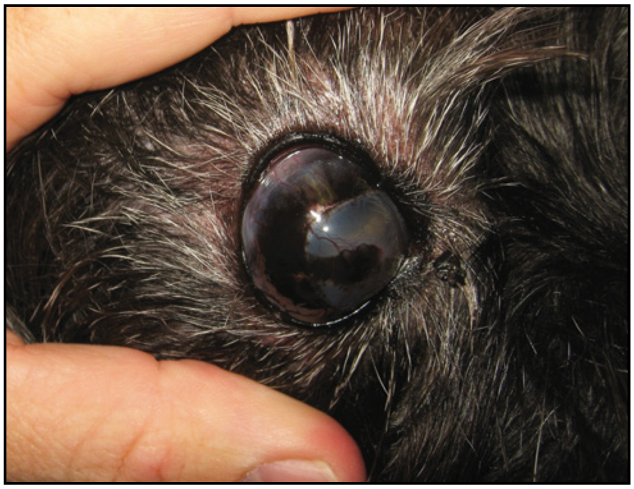


Figure 166.2 Pigmentary glaucoma. Clinical presentation associated with pigmentary glaucoma. Note visible pigment accumulation in the scleral/episcleral and corneal tissues.

Figure 166.3 Pigmentary glaucoma. Clinical presentation associated with pigmentary glaucoma. Note visible pigment accumulation in the scleral/episcleral tissues.

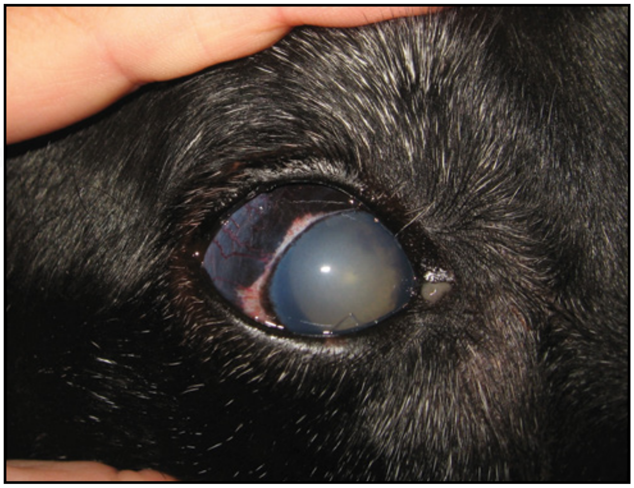


Figure 166.4 Pigmentary glaucoma. Clinical presentation associated with pigmentary glaucoma. Note corneal edema and visible pigment accumulation in the scleral/episcleral tissues and on the anterior lens capsule.

CHAPTER 167
GOLDEN RETRIEVER ASSOCIATED UVEITIS AND GLAUCOMA

PRESENTATION

A syndrome comprising slowly progressive intraocular changes, typically culminating in secondary glaucoma, is well recognized within the Golden Retriever breed. This syndrome has been variably described as "Pigmentary Uveitis," "Golden Retriever Uveitis," and "Pigmentary and Cystic Glaucoma of Golden Retrievers." Findings may include any combination of conjunctival and/or episcleral hyperemia, corneal decompensation, thin-walled uveal cysts within the anterior or posterior chambers, proteinaceous exudation within the anterior chamber, anterior and/or posterior iridal adhesions, pigment dispersion within the anterior chamber and/or onto the anterior lens capsule, cataract formation, hyphema, and/or secondary glaucoma (see also Golden Retriever associated uveitis). Uveal cysts may detach and move into the anterior chamber where they may rupture against corneal endothelial and/or iridal surfaces or collapse and rupture within the iridocorneal angle. The hallmark of this syndrome has been described as the appearance of pigment on the anterior lens capsule, typically distributed in a radial orientation. Initial symptoms are frequently noted in middle age and are usually bilateral, although not necessarily symmetrical. The etiology of this syndrome is poorly understood; however, genetic factors have been proposed based on breed predilection as well as the absence of demonstrable infectious or neoplastic causes.

DIAGNOSIS AND TREATMENT

The diagnosis is made based on a combination of signalment and clinical findings. Treatment is generally empirical, frequently comprising topical and/or systemic anti-inflammatory (either steroidal or nonsteroidal), immune-modulating (azathioprine or cyclosporine), and/or well as anti-glaucoma agents; however, secondary glaucoma is frequently the end point of this disease. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, enteritis, muscular damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of azathioprine include gastrointestinal (GI) distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of cyclosporine include hypersensitivity and GI distress. Patients affected by advanced disease represent high-risk candidates for either cataract or glaucoma surgery based on the frequency and severity of complications encountered. Ultimately, a procedure of comfort (enucleation, cryosurgery, intrascleral prosthesis placement, or chemical ciliary body ablation) may be necessary in order to address blind and/or painful eyes.

SELECTED REFERENCES

Deehr AJ and Dubielzig RR. A histopathological study of iridociliary cysts and glaucoma in Golden Retrievers. *Vet Ophthalmol*. 1998;**1**(2–3):153–158.

Esson D, Armour M, Mundy P, Schobert CS, Dubielzig RR. The histopathological and immunohistochemical characteristics of pigmentary and cystic glaucoma in the Golden Retriever. *Vet Ophthalmol*. 2009;**12**(6):361–368.

Holly VL, Sandmeyer LS, Bauer BS, Verges L, Grahn BH. **Golden retriever** cystic uveal disease: a longitudinal study of iridociliary cysts, **pigmentary uveitis**, and **pigmentary**/cystic glaucoma over a decade in western Canada. *Vet Ophthalmol*. 2016;**19**(3):237–244.

Jost HE, Townsend WM, Moore GE, Liang S. **Golden retriever pigmentary uveitis**: vision loss, risk factors for glaucoma, and effect of treatment on disease progression. *Vet Ophthalmol*. 2020;**23**(6):1001–1008.

Sapienza JS, Simó FJ, Prades-Sapienza A. Golden Retriever uveitis: 75 cases (1994–1999). *Vet Ophthalmol*. 2000;**3**(4):241–246.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 167.1 Golden Retriever associated uveitis and glaucoma. Glaucoma secondary to Golden Retriever-associated pigmentary uveitis. Note pigment on anterior lens capsule.

Figure 167.2 Golden Retriever associated uveitis and glaucoma. Note common bilateral presentation.

Figure 167.3 Golden Retriever associated uveitis and glaucoma. Note characteristic signs of glaucoma including episcleral injection and corneal edema.

Figure 167.4 Golden Retriever associated uveitis and glaucoma. Note pigment on anterior lens capsule as a result of Golden Retriever-associated uveitis.

CHAPTER 168
CYSTIC GLAUCOMA

PRESENTATION

A form of glaucoma associated with uveal cystic formation is noted, particularly in the American Bulldog as well as the Great Dane, Boston Terrier, and Labrador retriever. Affected animals are typically middle-aged with no sex-predilection noted. Clinical findings comprise the presence of multiple thin-walled uveal cysts arising from the posterior iris and ciliary body. Cysts may be attached to uveal tissues, freely mobile within the anterior chamber and/or ruptured on the endothelial surfaces. A degree of pigment dispersion and/or vitreous degeneration may also be present. Changes are typically bilateral however may be asymmetrical. Histologically, affected patients typically display chronic low grade intraocular inflammation, including an inflammatory cell infiltrate and/or the formation pre-iridal fibrovascular membranes. Intraocular pressure (IOP) elevation may result from a combination of iridocorneal angle (ICA) obstruction and trabecular meshwork (TM) degeneration and collapse. Glaucoma is typically gradual (developing across months to years) and vision loss, pain, buphthalmos, and ultimate globe loss may result.

DIAGNOSIS AND TREATMENT

Medical management of affected animals comprises the long-term administration of topical and/or systemic anti-inflammatory agents (steroidal and/or nonsteroidal) as well as the use of IOP lowering agents where indicated. In some cases, surgical intervention comprising cyst ablation/removal and/or lenticular phacoemulsification with or without concurrent endoscopic cyclophotocoagulation (ECP) and/or drainage implant placement may be indicated. The prognosis for successful management is generally positive; however, ongoing going therapy is typically required in order to maintain affected animals in a comfortable and functional state.

SELECTED REFERENCES

Komáromy AM, Bras D, Esson DW, Fellman RL, Grozdanic SD, Kagemann L, Miller PE, Moroi SE, Plummer CE, Sapienza JS, Storey ES, Teixeira LB, Toris CB, Webb TR. The future of canine glaucoma therapy. *Vet Ophthalmol*. 2019;**22**(5):726–740. doi: 10.1111/vop.12678.

Pumphrey SA, Pizzirani S, Pirie CG, Needle DB. Glaucoma associated with uveal cysts and goniodysgenesis in American Bulldogs: a case series. *Vet Ophthalmol*. 2013;**16**(5):377–385. doi: 10.1111/vop.12000.

Spiess BM, Bolliger JO, Guscetti F, Haessig M, Lackner PA, Ruehli MB. Multiple ciliary body cysts and secondary glaucoma in the Great Dane: a report of nine cases. *Vet Ophthalmol*. 1998;**1**(1):41–45. doi: 10.1046/j.1463-5224.1998.00007.x

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

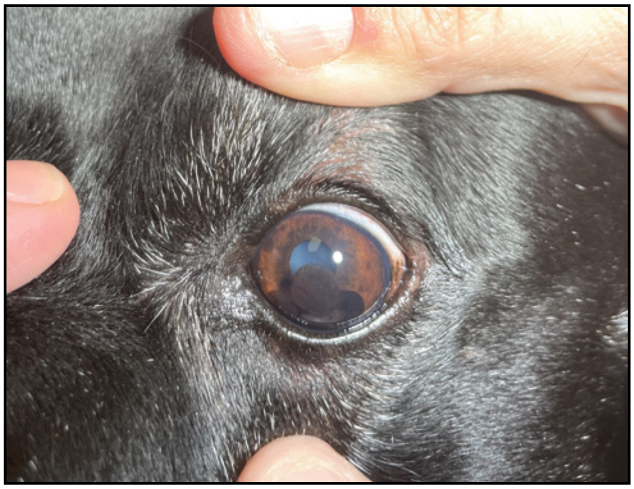


Figure 168.1 Cystic glaucoma. Clinical presentation demonstrating anterior chamber crowding with multiple cysts.

Figure 168.2 Cystic glaucoma. Clinical presentation in a typical breed, the American Bulldog.

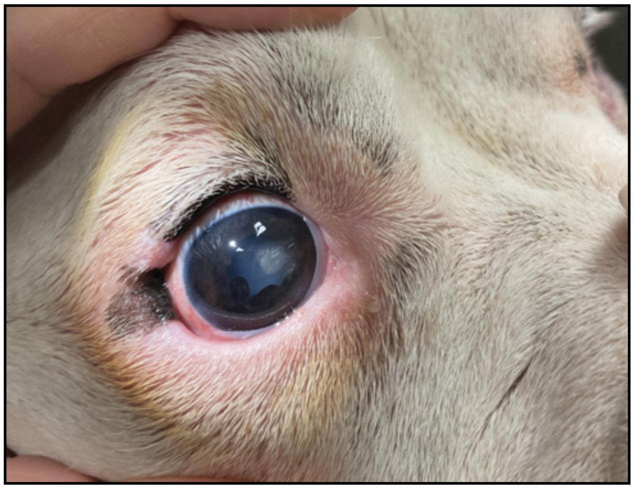


Figure 168.3 Cystic glaucoma. Note presence of multiple cysts in the ventral anterior chamber associated with iridocorneal angle compromise and elevated intraocular pressure.

Figure 168.4 Cystic glaucoma. Note thin-walled iridociliary cysts at pupillary margin and in the ventral anterior chamber.

CHAPTER 169
BUPHTHALMOS

PRESENTATION

Buphthalmos describes physical enlargement of the globe, resulting from severe elevations in intraocular pressure of congenital, primary, or secondary etiology. Buphthalmous should not be confused with

- Exophthalmos (anterior displacement of the globe within the orbit)
- Proptosis (displacement of the globe from the orbit),
- Strabismus (deviation of the globe within the orbit)
- non-bupthalmic glaucoma.

Typically, buphthalmos indicates chronically elevated intraocular pressure (IOP); however, young animals may develop these changes relatively rapidly as a result of juvenile scleral elasticity. Symptoms may include not only globe enlargement but also any combination of conjunctivitis, blepahrospasm, visible tears within the corneal endothelium/basement membrane ("Haab's stria") corneal edema, keratopathy (including ulceration), lenticular (sub)luxation, and/or globe rupture.

DIAGNOSIS AND TREATMENT

Buphthalmos generally implies blindness secondary to chronic ocular changes and associated damage to the optic nerve; however, individuals may retain a degree of functional vision in spite of early buphthalmos – notably feline patients. Generally, surgery comfort (enucleation, evisceration, cryotherapy, and/or chemical ciliary body ablation) of affected globes is indicated. Where possible the underlying cause of glaucoma should be identified (most typically through histopathological interpretation of enucleated tissues), so that any predisposition for similar pathology within the contralateral eye can be addressed.

SELECTED REFERENCES

Bauer BS, Sandmeyer LS, Philibert H, Feng CX, Grahn BH. **Chronic Glaucoma** in **Dogs**: relationships between histologic lesions and the gonioscopic diagnosis of pectinate ligament dysplasia. *Vet Pathol*. 2016;**53**(6):1197–1203.

Brooks DE. Glaucoma in the dog and cat. *Vet Clin North Am Small Anim Pract*. 1990;**20**(3):775–797.

McLaughlin SA, Render JA, Brightman AH 2nd, Whiteley HE, Helper LC, Shadduck JA. Intraocular findings in three dogs and one cat with chronic glaucoma. *J Am Vet Med Assoc*. 1987;**191**(11):1443–1445.

Sandmeyer LS, Bauer BS, Grahn BH. Diagnostic ophthalmology. *Can Vet J*. 2012;**53**(1):96–98.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

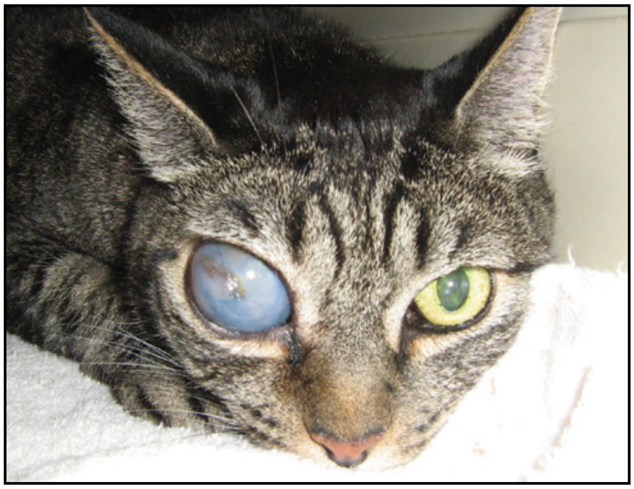


Figure 169.1 Buphthalmos. Clinical presentation associated with severe buphthalmos and chronic feline glaucoma.

Figure 169.2 Buphthalmos. Clinical presentation associated with severe buphthalmos and chronic canine glaucoma.

Figure 169.3 Buphthalmos. Clinical presentation associated with moderate buphthalmos.

Figure 169.4 Buphthalmos. Notable are corneal Haab's striae associated with buphthalmos.

Section 13

Neuro-Ophthalmic Disease

CHAPTER 170
OPTIC NERVE HYPOPLASIA

PRESENTATION

Optic nerve hypoplasia (and in rare cases aplasia) results from a congenitally abnormal differentiation of these tissues. Clinically, affected patients display small, gray-colored, and poorly myelinated optic nerve head tissue. Changes are typically bilateral, however may be unilateral and may be accompanied by other ocular abnormalities including anterior segment dysgenesis, and/or vitreoretinal pathology. Associated visual deficits may vary from mild to complete blindness and are typically noted early in life (weeks to months). Commonly affected breeds include the German Shepherd and Shih Tzu.

DIAGNOSIS AND TREATMENT

The diagnosis of optic nerve hypoplasia is made based on clinical examination including funduscopy, supported if necessary by adjunctive imaging (typically B-mode ultrasonography). No treatment is available for affected animals.

SELECTED REFERENCES

Becker D, Niggel JK, Pearce-Kelling S, Riis RC, Aguirre GD. **Optic nerve hypoplasia** in miniature poodle **dogs**: a preliminary genetic and candidate gene association study. *Vet Ophthalmol*. 2020;**23**(1):67–76.

Negishi H, Hoshiya T, Tsuda Y, Doi K, Kanemaki N. Unilateral optical nerve hypoplasia in a Beagle dog. *Lab Anim*. 2008;**42**(3):383–388. doi: 10.1258/la.2007.007033.

Rampazzo A, D'Angelo A, Capucchio MT, Sereno S, Peruccio C. Collie eye anomaly in a mixed-breed dog. *Vet Ophthalmol*. 2005;**8**(5):357–360.

da Silva EG, Dubielzig R, Zarfoss MK, Anibal A. Distinctive histopathologic features of canine optic nerve hypoplasia and aplasia: a retrospective review of 13 cases. *Vet Ophthalmol*. 2008;**11**(1):23–29. doi: 10.1111/j.1463-5224.2007.00596.x.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

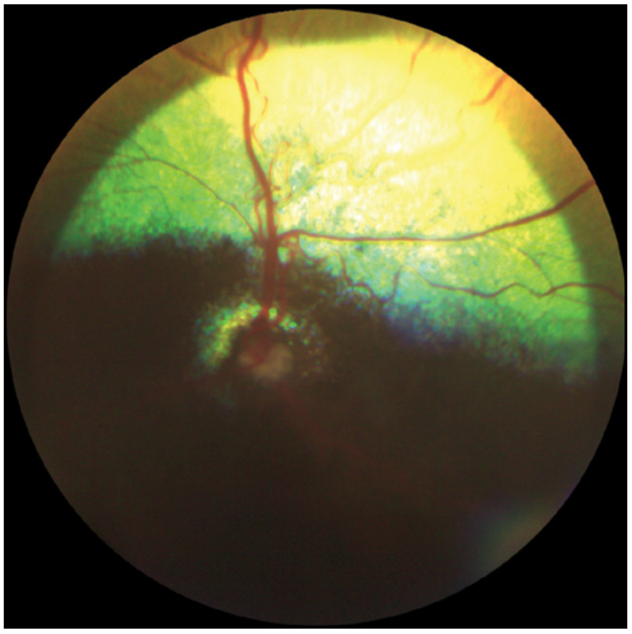


Figure 170.1 Optic nerve hypoplasia. Funduscopic findings associated with optic nerve hypoplasia; notable is the small poorly myelinated optic nerve head.

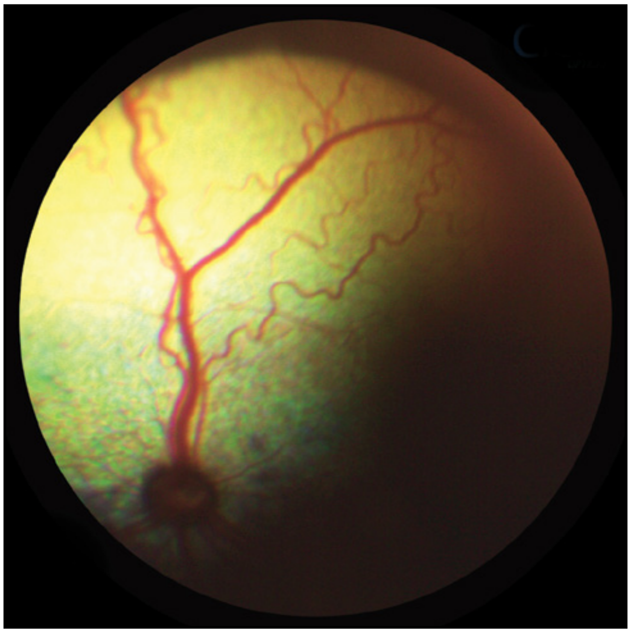


Figure 170.2 Optic nerve hypoplasia. Funduscopic findings associated with optic nerve hypoplasia; notable is the small poorly myelinated optic nerve head.

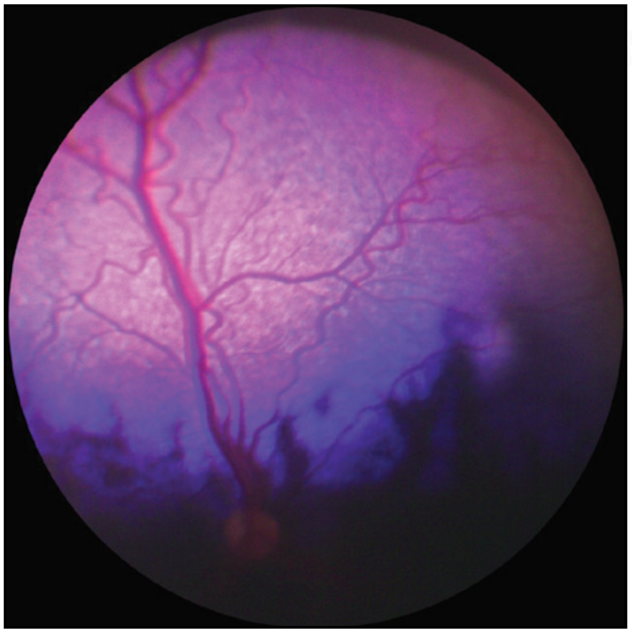


Figure 170.3 Optic nerve hypoplasia. Funduscopic findings associated with optic nerve hypoplasia; notable is the small poorly myelinated optic nerve head.

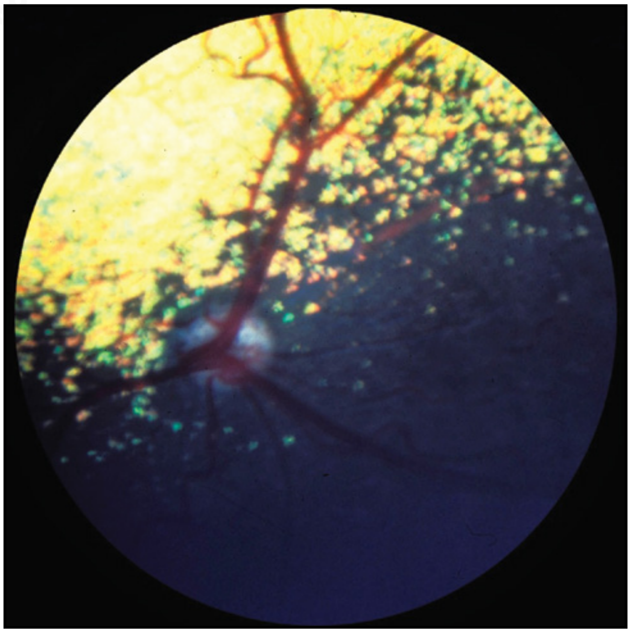


Figure 170.4 Optic nerve hypoplasia. Funduscopic findings associated with optic nerve hypoplasia; notable is the small poorly myelinated optic nerve head.

CHAPTER 171
LYSOSOMAL STORAGE DISEASE (LSD)

PRESENTATION

The lysosomal storage diseases (LSDs) represent a group of relatively uncommon, hereditary, inborn errors of metabolism, which result in the pathological accumulation of metabolic products within cellular lysosomes. Major diseases within this group include

- (the) neuronal ceroid lipofuscinoses
- globoid cell leukodystrophy
- (the) mucolipidoses
- (the) gangliosidoses
- alpha-mannosidosis
- fucosidosis
- (the) mucopolysaccharidoses

Clinical symptoms frequently become apparent early in life, generally by 6–12 months of age, however may vary in severity based both on disease subtype as well as degree of metabolic pathology present. Symptoms may include any combination of lethargy, weakness, dysphagia, "failure to thrive," facial dysmorphism, musculoskeletal malformation, renal/cardiac/hepatic insufficiency, corneal dystrophy, retinopathy, decreased visual function/blindness and/or neurologic dysfunction. Affected dog breeds may include the Beagle, Springer Spaniel, Siberian Husky, German Shorthaired Pointer, Cairn Terrier, West Highland White Terrier, Miniature Pincher, English Setter, Golden Retriever, Dachshund and Miniature Schnauzer. Affected cat breeds may include the Siamese, Persian and Korat.

DIAGNOSIS AND TREATMENT

The diagnosis of LSD can be challenging and is based on clinical findings, serum chemistry values, and specific genetic testing where available. The long-term prognosis for affected animals is generally poor; however, experimental therapy including emerging pharmaceutical agents, enzyme therapy, bone marrow transplantation, and gene therapy may be possible in selected cases.

SELECTED REFERENCES

Haskins ME. Animal models for mucopolysaccharidosis disorders and their clinical relevance. *Acta Paediatr Suppl*. 2007;**96**(455):56–62.

Jolly RD and Walkley SU. Lysosomal storage diseases of animals: an essay in comparative pathology. *Vet Pathol*. 1997;**34**(6):527–548.

Narfström K. Hereditary and congenital ocular disease in the cat. *J Feline Med Surg*. 1999;**1**(3):135–141.

Skelly BJ and Franklin RJ. Recognition and diagnosis of lysosomal storage diseases in the cat and dog. *J Vet Intern Med*. 2002;**16**(2):133–141.

Slutsky J, Raj K, Yuhnke S, Bell J, Fretwell N, Hedhammar A, Wade C, Giger U. A web resource on DNA tests for canine and feline hereditary diseases. *Vet J*. 2013;**197**(2):182–187. doi: 10.1016/j.tvjl.2013.02.021.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 171.1 Lysosomal storage diseases. Clinical presentation associated with mucopolysaccharidoses. Note facial dysmorphism and corneal dystrophy. Inset demonstrates musculoskeletal malformation.

Figure 171.2 Lysosomal storage diseases. Clinical presentation associated with mucopolysaccharidoses. Note facial dysmorphism and corneal dystrophy. Inset demonstrates musculoskeletal malformation.

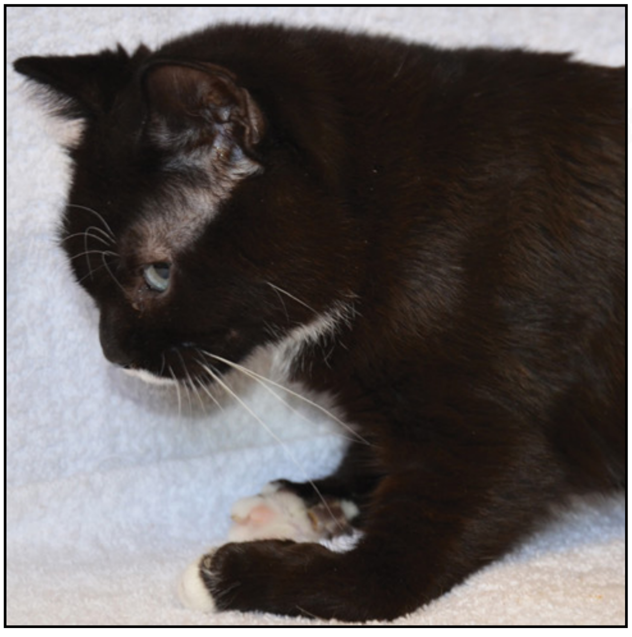


Figure 171.3 Lysosomal storage diseases. Clinical presentation associated with mucolipidosis. Note facial dysmorphism and musculoskeletal malformation.

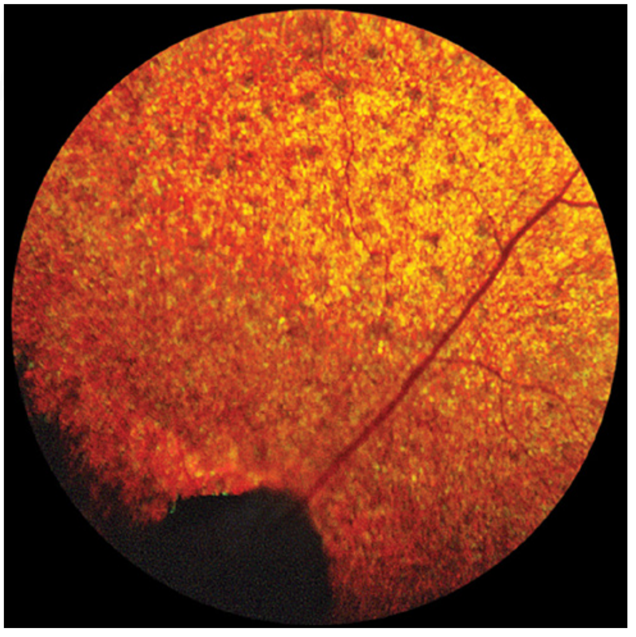


Figure 171.4 Lysosomal storage diseases. Funduscopic changes associated with neuronal ceroid lipofuscinosis. Note multifocal tapetal discoloration as a result of pathological protein accumulation.

CHAPTER 172
SYMPATHETIC DENERVATION (HORNER'S SYNDROME)

PRESENTATION

Sympathetic innervation to the eye mediates the action of the iris dilator and contractor muscles (via "double-reciprocal innervation"), the smooth muscle of the orbit and the Muller's muscles of the upper and lower eyelids. This pathway originates in the hypothalamus, exits the spinal cord with the ventral thoracic roots and synapses in the cranial cervical ganglion, adjacent to the tympanic bullae. Post-ganglionic fibers follow the ophthalmic division of the trigeminal nerve before entering the eye via the long ciliary nerves. Partial or complete interruption of sympathetic innervation to the ocular structures results in a group of symptoms referred to as "Horner's Syndrome," comprising variable combinations of pupillary miosis, third eyelid protrusion, enophthalmos, and/or upper lid ptosis.

DIAGNOSIS AND TREATMENT

The diagnosis of Horner's syndrome is confirmed via pharmacological testing using topical phenylephrine, which results in the resolution of symptoms in a timeframe consistent with the location of the lesion (typically less than 30 minutes). Horner's symptoms may be idiopathic in etiology, or associated with inflammation at any point along the sympathetic tract, notably at the level of the ear canal/tympanic bullae. As a result, underlying inflammatory or neoplastic disease and/or neuropathy should be specifically addressed. Systemic anti-inflammatory therapy using corticosteroids may be beneficial in selected cases; however, most cases of Horner's syndrome display spontaneous resolution of symptoms. Resolution may take weeks to months. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight-gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Boydell P. Idiopathic horner syndrome in the golden retriever. *J Neuroophthalmol.* 2000;**20**(4):288–290.

Kern TJ, Aromando MC, Erb HN. Horner's syndrome in dogs and cats: 100 cases (1975–1985). *J Am Vet Med Assoc.* 1989;195(3):369–373.

Morgan RV, Zanotti SW. **Horner's syndrome** in **dogs** and cats: 49 cases (1980–1986). *J Am Vet Med Assoc.* 1989;194(8):1096–1099.

Simpson KM, Williams DL, Cherubini GB. Neuropharmacological lesion localization in idiopathic Horner's syndrome in golden retrievers and dogs of other breeds. *Vet Ophthalmol.* 2013. doi: 10.1111/vop.12096.

Zwueste DM, Grahn BH. A review of **Horner's syndrome** in small animals. *Can Vet J.* 2019;60(1):81–88.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 172.1 Sympathetic denervation (Horner's Syndrome). Notable characteristics of sympathetic denervation including enophthalmos, upper eyelid ptosis, third eyelid elevation, and miosis are evident.

Figure 172.2 Sympathetic denervation (Horner's Syndrome). Notable characteristics of sympathetic denervation including enophthalmos, upper eyelid ptosis, third eyelid elevation, and miosis are evident.

Figure 172.3 Sympathetic denervation (Horner's Syndrome). Notable characteristics of sympathetic denervation including enophthalmos, upper eyelid ptosis, third eyelid elevation, and miosis are evident.

Figure 172.4 Sympathetic denervation (Horner's Syndrome). Notable characteristics of sympathetic denervation including enophthalmos, upper eyelid ptosis, third eyelid elevation, and miosis are evident.

CHAPTER 173
OPHTHALMOPLEGIA

PRESENTATION

The term ophthalmoplegia describes a syndrome of neurological deficits arising as a result of damage to the oculomotor nerve (CN III) and/or its nucleus. CN III normally provides somatic efferent innervation to the dorsal, medial, & ventral rectus and ventral oblique muscles of the eye, as well as the levator palpebral muscle. This nerve also contains the parasympathetic fibers, which innervate the pupillary muscles (through "double reciprocal innervation"). As a result, lesions affecting CN III may result in

- internal ophthalmoplegia (dilated nonresponsive pupil, without visual deficits)
- external ophthalmoplegia (ventro-lateral strabismus and or upper lid ptosis)
- complete ophthalmoplegia (combined internal and external ophthalmoplegia)

The superficial location of the parasympathetic fibers results in internal ophthalmoplegia being the most common clinical presentation. Ophthalmoplegia frequently results from a lesion affecting CN III at the level of the cavernous venus sinus (so called "cavernous sinus syndrome"). Additional symptoms, depending on site the site of the lesion may include reduced corneal and/or facial sensation as a result of impaired trigeminal nerve function and/or neurogenic keratoconjunctivitis sicca (KCS) (see also neurogenic KCS and xeromycteria).

DIAGNOSIS AND TREATMENT

The diagnosis of ophthalmoplegia is made based on clinical findings as well as pharmacologic testing using topical pilocarpine, which will result in the resolution of mydriasis within 30 minutes. Cranial soft tissue imaging (MRI) is generally indicated in order to visualize affected structures. The treatment of ophthalmoplegia comprises treating the underlying lesion, typically encompassing the management of neoplastic lesions using targeted radiotherapy, and/or chemotherapy. Secondary symptoms including ulcerative keratitis and or KCS should also be managed if present.

SELECTED REFERENCES

Guevar J, Gutierrez-Quintana R, Peplinski G, Helm JR, Penderis J. Cavernous sinus syndrome secondary to intracranial lymphoma in a cat. *J Feline Med Surg.* 2013;**16**(6):513–516. [Epub ahead of print].

Murphy CJ, Koblik P, Bellhorn RW, Pino M, Hacker D, Burling T. Squamous cell carcinoma causing blindness and ophthalmoplegia in a cat. *J Am Vet Med Assoc.* 1989;**195**(7):965–968.

Tetas Pont R, Freeman C, Dennis R, Hartley C, Beltran E. Clinical and magnetic resonance imaging features of idiopathic oculomotor neuropathy in 14 **dogs**. *Vet Radiol Ultrasound.* 2017;**58**(3):334–343.

Theisen SK, Podell M, Schneider T, Wilkie DA, Fenner WR. A retrospective study of cavernous sinus syndrome in 4 dogs and 8 cats. *J Vet Intern Med.* 1996;**10**(2):65–71. Erratum in: J Vet Intern Med 1996 May–Jun;10(3):197.

Webb AA, Cullen CL, Rose P, Eisenbart D, Gabor L, Martinson S. Intracranial meningioma causing internal ophthalmoparesis in a dog. *Vet Ophthalmol.* 2005;**8**(6):421–425.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

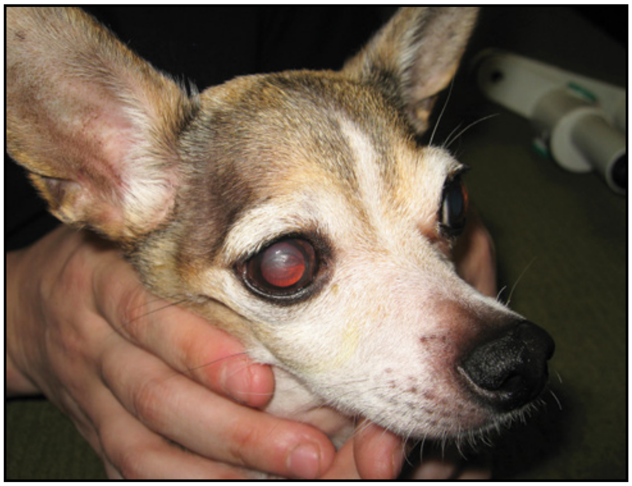


Figure 173.1 Ophthalmoplegia. The characteristic finding is a dilated, non-responsive pupil without visual deficits. This figure displays concurrent corneal disease as a result of trigeminal pathology.

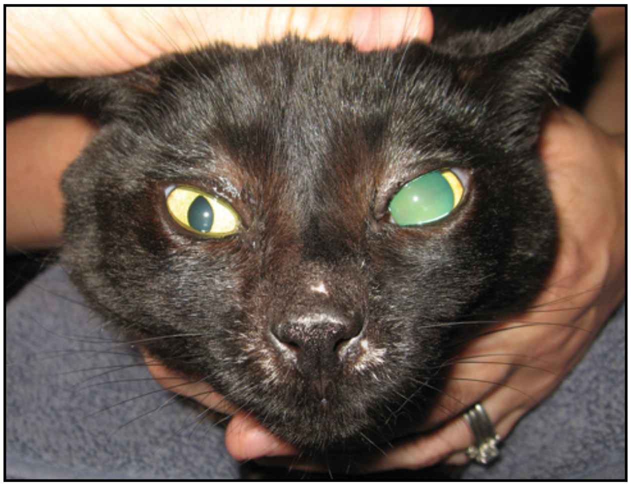


Figure 173.2 Ophthalmoplegia. Note the dilated, nonresponsive pupil in a patient without visual deficits.

Figure 173.3 Ophthalmoplegia. Note the dilated, nonresponsive pupil in a patient without visual deficits.

Figure 173.4 Ophthalmoplegia. Note the dilated, nonresponsive pupil in a patient without visual deficits. This figure displays concurrent ventrolateral strabismus.

CHAPTER 174
NEUROPARALYTIC KERATITIS/ HEMIFACIAL PARALYSIS

PRESENTATION

The facial nerve (CN VII) provides somatic innervation to the facial and periocular muscles, notably to the orbicularis oculi muscle (which mediates eyelid closure) via its auriculopalpebral branch. Interruption of innervation may result in any combination of a widened palpebral fissure, a diminished or absent palpebral reflex, deviation of the nasal planum and/or drooping of the upper and/or lower lips. Symptoms are more commonly unilateral. Inability to effectively close the eyelids results in exposure keratopathy, which may progress to ulcerative and/or perforating keratitis. Associated damage to (parasympathetic) lacrimal innervation may result in concurrent keratoconjunctivitis sicca (KCS) and/or xeromycteria (see also neurogenic KCS and xeromycteria). Commonly affected breeds include the Cocker Spaniel and the Pug.

DIAGNOSIS AND TREATMENT

Cases of facial paralysis are commonly idiopathic in etiology, however potential causes include trauma (notably to the auriculopalpebral branch of the facial nerve where it crosses the zygomatic arch), neoplasia, otitis, vascular infarcts, and/or endocrine-associated neuropathy. Treatment comprises addressing an underlying cause where identified, as well as the administration of systemic anti-inflammatory therapy, typically corticosteroids unless contraindicated. The prognosis for the recovery of neurologic function is variable. Lacrimal function, if impaired/absent can sometimes be re-established using a combination of anti-inflammatory therapy and orally administered pilocarpine. Pilocarpine is dosed in an increasing incremental fashion in order to minimize the risk of unwanted side effects (which comprise gastrointestinal [GI] distress). Frequent corneal lubrication using an ophthalmic visco-gel is recommended. Additionally, partial tarsorrhaphy (temporary or permanent) may be indicated in order to afford improved corneal protection. If lacrimal function cannot be re-established, affected patients may be managed using either topical lubrication or, where indicated, parotid duct transposition surgery. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Chan MK, Toribio JA, Podadera JM, Child G. Incidence, cause, outcome and possible risk factors associated with **facialnerve paralysis** in **dogs** in a Sydney population (2001–2016): a retrospective study. *Aust Vet J.* 2020;**98**(4):140–147.

Garosi LS, Lowrie ML, Swinbourne NF. Neurological manifestations of ear disease in dogs and cats. *Vet Clin North Am Small Anim Pract.* 2012;**42**(6):1143–1160. doi: 10.1016/j.cvsm.2012.08.006. Epub 2012 Oct 10. Review.

Spivack RE, Elkins AD, Moore GE, Lantz GC. Postoperative complications following TECA-LBO in the dog and cat. *J Am Anim Hosp Assoc.* 2013;**49**(3):160–168. doi: 10.5326/JAAHA-MS-5738. Epub 2013 Mar 27.

Varejão AS, Muñoz A, Lorenzo V. Magnetic resonance imaging of the intratemporal facial nerve in idiopathic facial paralysis in the dog. *Vet Radiol Ultrasound.* 2006;**47**(4):328–333.

Wright JA. Ultrastructural findings in idiopathic facial paralysis in the dog. *J Comp Pathol.* 1988;**98**(1):111–115.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 174.1 Neuroparalytic keratitis/hemifacial paralysis. Xeromycteria, marked by a dry, crusted ipsilateral nostril is notable.

Figure 174.2 Neuroparalytic keratitis/hemifacial paralysis. Xeromycteria, marked by a dry, crusted ipsilateral nostril and exposure keratitis are notable.

Figure 174.3 Neuroparalytic keratitis/hemifacial paralysis. Deviation of the nasal planum is notable in this patient.

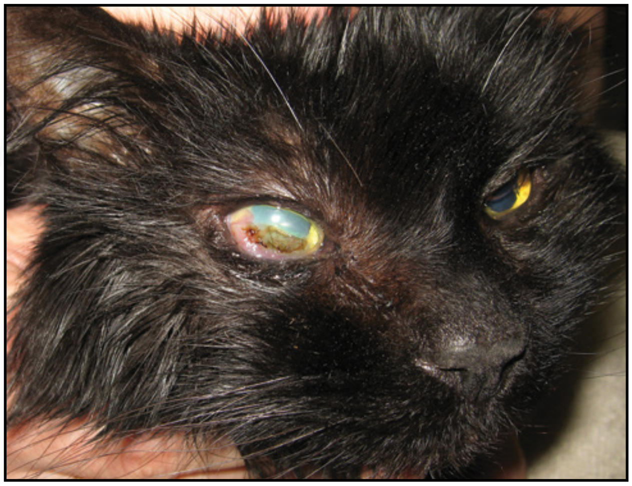


Figure 174.4 Neuroparalytic keratitis/hemifacial paralysis. Note significant associated exposure keratopathy.

CHAPTER 175
NEUROGENIC KCS AND XEROMYCTERIA

PRESENTATION

Parasympathetic innervation to the lacrimal glands originates in the parasympathetic nucleus of the facial nerve. Fibers extend rostrally with the facial nerve and synapse within pterygopalatine ganglion before reaching lacrimal structures via the zygomatic branch of the trigeminal nerve. Damage anywhere along this pathway may result in partial or complete loss of innervation to the lacrimal glands and resultant "neurogenic KCS." Neurogenic keratoconjunctivitis sicca (KCS) is frequently characterized by an associated dry/crusted ipsilateral nostril (termed "xeromcyteria") as a result of impaired innervation of the nasal gland. Potential etiologies include inflammatory, traumatic, and/or neoplastic processes. Additional symptoms, depending on site the site of the lesion may include reduced corneal and/or facial sensation as a result of impaired trigeminal nerve function and/or Horner's syndrome.

DIAGNOSIS AND TREATMENT

The diagnosis of neurogenic KCS is made based on clinical findings. In many cases of neurogenic KCS, lacrimal function is completely absent, marked by Schirmer Tear Test values of 0 mm/wetting per minute (termed "absolute KCS"). Treatment of neurogenic KCS comprises identification and treatment of the underlying lesion where possible. This may encompass cranial imaging (MRI). lacrimal function can sometimes be re-established using a combination of anti-inflammatory therapy and orally administered pilocarpine. Pilocarpine is dosed in an increasing incremental fashion in order to minimize the risk of unwanted side effects (which comprise gastrointestinal [GI] distress). Frequent corneal lubrication using an ophthalmic visco-gel is recommended. Additionally partial (temporary or permanent) tarsorrhaphy may be indicated in order to afford improved corneal protection. If lacrimal function cannot be re-established, effected patients may be managed using either topical lubrication or, where indicated, parotid duct transposition surgery. Potential adverse effects associated with the use of systemic prednisone include polyphagia, polydipsia, polyuria, hair coat changes, weight gain, pancreatitis, GI distress, muscle damage, hepatic impairment, and diabetes.

SELECTED REFERENCES

Foote BC, Michau TM, Welihozkiy A, Stine JM. Retrospective analysis of ocular neuropathies in diabetic **dogs** following cataract surgery. *Vet Ophthalmol.* 2019;**22**(3):284–293.

Matheis FL, Walser-Reinhardt L, Spiess BM. **Canine neurogenic Keratoconjunctivitis sicca**: 11 cases (2006–2010). *Vet Ophthalmol.* 2012;**15**(4):288–290.

Rhodes M, Heinrich C, Featherstone H, Braus B, Manning S, Cripps PJ, Renwick P. Parotid duct transposition in dogs: a retrospective review of 92 eyes from 1999 to 2009. *Vet Ophthalmol.* 2012;**15**(4):213–222. doi: 10.1111/j.1463-5224.2011.00972.x. Epub 2011 Nov 24.

Slatter D and Severin GA. Use of pilocarpine for treatment of keratoconjunctivitis sicca. *J Am Vet Med Assoc.* 1995;**206**(3):287–289.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

Figure 175.1 Neurogenic KCS and xeromycteria. Clinical presentation associated with neurogenic KCS. Xeromycteria, marked by a dry, crusted, ipsilateral nostril is notable.

Figure 175.2 Neurogenic KCS and xeromycteria. Xeromycteria, marked by a dry, crusted, ipsilateral nostril, and significant ocular discharge are notable.

Figure 175.3 Neurogenic KCS and xeromycteria. Xeromycteria, marked by a dry, crusted, ipsilateral nostril, and significant vascular keratitis are notable.

Figure 175.4 Neurogenic KCS and xeromycteria. Xeromycteria, marked by a dry, crusted, ipsilateral nostril, and significant pigmentary keratitis are notable.

CHAPTER 176
OPTIC NEURITIS/MENINGITIS

PRESENTATION

The post-retinal visual pathways comprise the retrobulbar optic nerves, optic chiasm, optic tracts, lateral geniculate nuclei, optic radiations, and visual cortices. Optic neuritis describes inflammation of any portion of the optic nerve(s) with or without concurrent evidence of more generalized CNS inflammation. Presenting signs typically include blindness in association with dilated, non- or poorly responsive pupils. Symptoms may be unilateral or bilateral and are generally acute in onset. Additional ophthalmic findings may include any combination of chorioretinitis, papilledema, and/or hemorrhage of the optic nerve head and surrounding neuroretina. Symptoms of more generalized CNS involvement may include altered mentation, lethargy, ataxia, and/or seizure-like activity. Optic neuritis occurs most commonly in association with granulomatous meningo-encephalitis (GME); however, other potential etiologies include necrotizing leukoencephalitis/meningoencephalitis, infectious encephalomyelitis (associated with viral, bacterial, fungal, rickettsial, protozoal, or parasitic organisms) or neoplasia. The underlying cause of GME frequently remains unclear; however, an auto-immune mechanism is suspected. Canine and feline patients of any breed may develop optic neuritis; however, small breed dogs are more commonly affected, notably the Chihuahua and Dachshund.

DIAGNOSIS AND TREATMENT

The diagnosis of optic neuritis is made based on a combination of clinical findings (including funduscopy) as well as adjunctive diagnostics including complete blood count (CBC)/chemistry analysis, infectious titer testing, CSF evaluation, and/or cranial magnetic resonance imaging. CSF analysis may reveal increased protein levels with or without a leukoctytosis. MR findings typically include subtle, patchy hyperintensity affecting multiple areas of the brain and meninges. Hyperintensity of the ONs may or may not be noted. Electroretinography may additionally be employed in order to rule out primary retinal disease (see SARDs/IMR). Differentials include inherited lysosmal storage diseases (notably the gangliosidoses and neuronal ceroid lipofuscinoses), hydrocephalus, trauma, hypoxia (notably as a result of anesthetic complication), metabolic disease, vascular pathology, or central neoplasia (notably pituitary adenomas, lymphoma, and meningioma). Treatment comprises immuno-modulation using varying combinations of systemic prednisone, azothioprine, and/or cyclosporine. When successful, treatment should be tapered with caution over a significantly extended timeframe (typically months) and long-term therapy may be required in order to prevent recurrence of symptoms. Potential adverse effects associated with the use of systemic corticosteroids include polyphagia, polydipsia, polyuria, hair coat changes, weight-gain, pancreatitis, gastrointestinal (GI) distress, muscle damage, hepatic impairment, and diabetes. Potential adverse effects associated with the use of azathioprine include GI distress, pancreatitis, hepatotoxicity, and myelosuppression. Potential adverse effects associated with the use of cyclophosphamide include GI distress, pancreatitis, hepatotoxicity, and myelosuppression.

SELECTED REFERENCES

Armour MD, Broome M, Dell'Anna G, Blades NJ, Esson DW. A review of orbital and intracranial magnetic resonance imaging in 79 canine and 13 feline patients (2004–2010). *Vet Ophthalmol.* 2011;**14**(4):215–226. doi: 10.1111/j.1463-5224.2010.00865.x. Epub 2011 Apr 18.

Nell B. Optic neuritis in dogs and cats. *Vet Clin North Am Small Anim Pract.* 2008;38(2):403–415, viii. doi: 10.1016/j.cvsm.2007.11.005. Review.

Bedos L, Tetas R, Crespo V, Shea A. Presumed **optic neuritis** of non-infectious origin in **dogs** treated with immunosuppressive medication: 28 **dogs** (2000–2015). *J Small Anim Pract.* 2020;**61**(11):676–683.

Smith SM, Westermeyer HD, Mariani CL, Gilger BC, Davidson MG. **Optic neuritis** in **dogs**: 96 cases (1983–2016). *Vet Ophthalmol.* 2018;**21**(5):442–451.

Posporis C, Beltran E, Dunning M, Espadas I, Gillespie S, Barry AT, Wessmann A. Prognostic factors for recovery of vision in **Canine Optic Neuritis** of unknown etiology: 26 **dogs** (2003–2018). *Front Vet Sci.* 2019;**22**;6:415.

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.

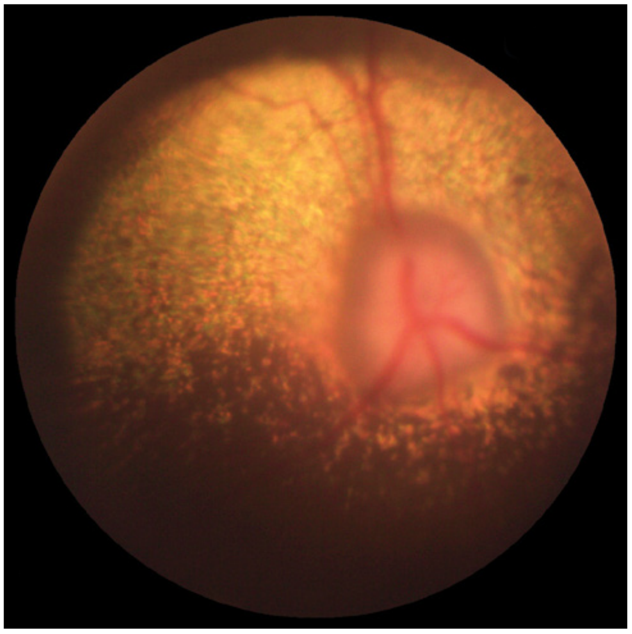


Figure 176.1 Optic neuritis/meningitis. Funduscopic findings associated with optic neuritis. Note optic nerve swelling/papilledema.

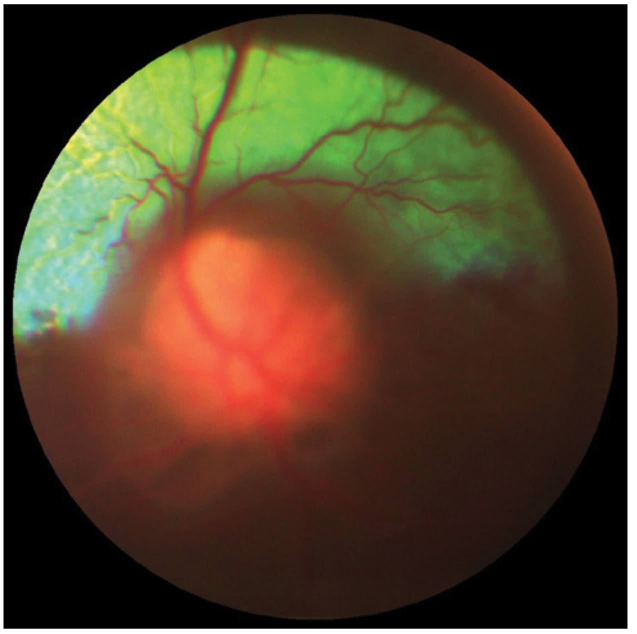


Figure 176.2 Optic neuritis/meningitis. Funduscopic findings associated with optic neuritis. Note optic nerve swelling/papilledema.

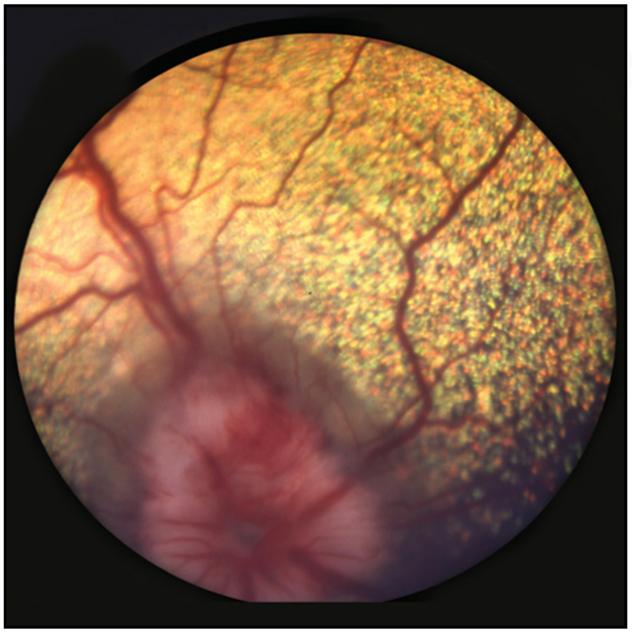


Figure 176.3 Optic neuritis/meningitis. Funduscopic findings associated with optic neuritis. Note peripapillary edema and hemorrhage.

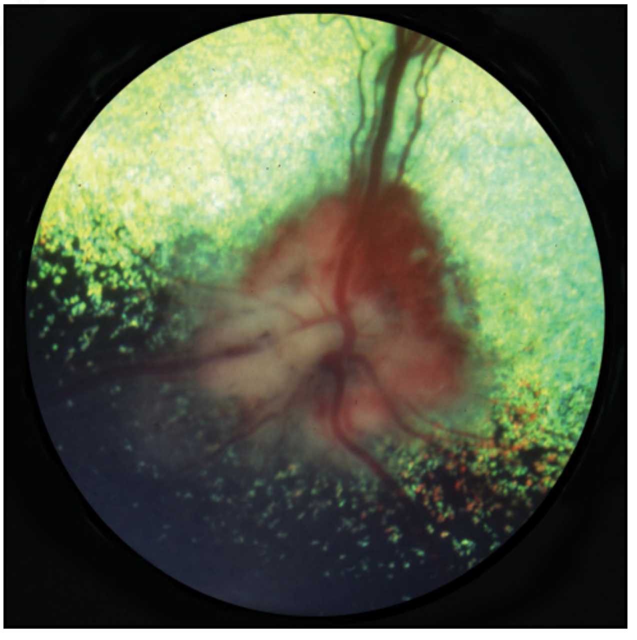


Figure 176.4 Optic neuritis/meningitis. Funduscopic findings associated with optic neuritis. Note peripapillary edema and hemorrhage.

INDEX

Page numbers in *italics* refer to illustrations

Clinical Atlas of Canine and Feline Ophthalmic Disease, Second Edition.
Edited by Douglas W. Esson and Sara Calvarese.