Color Atlas of Veterinary Ophthalmology

Second Edition



Kirk N. Gelatt and Caryn E. Plummer



WILEY Blackwell

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Kirk N. Gelatt
Distinguished Professor of Comparative Ophthalmology Emeritus,
Department of Small Animal Sciences,
College of Veterinary Medicine, University of Florida,
Gainesville, FL, USA

and

Caryn E. Plummer
Associate Professor of Comparative Ophthalmology and Service Chief,
Veterinary Ophthalmology Service,
Department of Small Animal Clinical Sciences,
College of Veterinary Medicine, University of Florida,
Gainesville, FL, USA

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Preface

Based on the success of the publication of the first edition of *Veterinary Ophthalmology* in 1981, subsequent editions were released in 1991, 1999, 2007, and 2013. The continued expansion and more rapid development of veterinary ophthalmology worldwide resulted in the current fifth edition having 35 chapters, 64 authors, and a text of more than 2100 pages. This second edition of the color atlas presents diseases based on their clinical appearances, and provides introductory information to complement the photos to further understand the characteristics of each disease. The most common eye diseases are emphasized, but to be inclusive, the less frequent diseases (and species) have been added.

Ophthalmology is heavily based on direct clinical examination and diagnostics, and hence often photographed. This heavily pictorial text introduces the veterinary medical student and veterinary practitioner to clinical veterinary ophthalmology based on the clinical appearances of the diseases that one would encounter in small and large animal practice. When possible, multiple photographs of selected ophthalmic diseases are included to demonstrate the different stages of these diseases as presented to the clinician, and when medical and/or surgical therapies alter their appearance. In contrast to most color atlases, we have provide a comprehensive text describing each ophthalmic disease (history, clinical findings, diagnosis, recommended therapy, and prognosis) as well as many species, including dog, cat, horse, cattle, and exotic animals. As a result, this color atlas has the largest collection of clinical photographs currently available in a single book, and for many readers a reasonably complete ophthalmic reference for your veterinary medical library and clinic.

This second edition has added chapters and more than doubled the color clinical photographs from the first edition. The first two new chapters are divided into: clinical anatomy with emphasis on the gross morphology and ophthalmic structures the clinician encounters during his/her clinical examination, and ophthalmic diagnostics most useful in general practice. Chapter 3 illustrates the different ophthalmic tissue responses to diseases common all animals, followed with chapters on canine ophthalmology (Chapters 4–13), feline ophthalmology (Chapter 14), equine ophthalmology (Chapter 15); food and fiber animal ophthalmology (Chapter 16); pet exotic animal ophthalmology (Chapter 17); systemic diseases with ophthalmic manifestations in the dog, cat, horse, and food animals (Chapter 18); and neuro-ophthalmology with emphasis on clinical syndromes (Chapter 19). Appendix 1 is a glossary or condensed selection of ophthalmic words, to assist the reader with sometimes confusing nomenclature (derived from the Greek rather than Latin).

Within each chapter, the diseases are divided into sections on: (1) congenital or developmental; (2) inflammatory; (3) traumatic; (4) degenerative; and (5) neoplasia. Often the text for a color atlas is a single sentence noting the disease. However, for this color atlas, additional clinical information has been included. The text for the color illustrations usually includes: (1) the clinical history; (2) the clinical signs and findings associated with the disease; (3) the rule outs or differential diagnoses; (4) the recommended treatment; and (5) prognosis. If a disease changes its appearance significantly over time or during therapy, multiple illustrations are used.

In diagnostic ophthalmology the clinician relies heavily on direct observations of the ophthalmic tissues and interpretations of these lesions. Only in ophthalmology can the examiner directly observe 2–3 cm into a complex organ, and directly observe the body's vasculature, and part of the central nervous system. There is no substitute or shortcut for a complete ophthalmic examination. The majority of treatment failures are not based on the drug choices or surgical procedures, but because of an incorrect initial diagnosis. The goals of this color atlas are to expand your clinical proficiency and result in improved patient care.

A book of this magnitude and number of photographs has many contributors; the majority from the ophthalmology faculty members, residents, and graduate

students at the University of Florida, College of Veterinary Medicine, during nearly 40 years, and personal veterinary ophthalmology libraries of nearly 60,000 color photographs. Early photographs were recorded by 35 mm color film, and later digitized. Since about 2005, all photographs were digitalized. Additional photographs were provided over the years by other veterinary ophthalmologists, including the late Keith C. Barnett, Paul M. Barrett, Cheryl L. Cullen, Andras Komaromy, Charles L. Martin, Reuben Merideth, Alain Regnier, the late Glenn A. Severin, Ron L. Sigler, Aubrey A. Webb, and many others.

Kirk N. Gelatt, VMD, Diplomate Emeritus ACVO
Distinguished Professor of Comparative Ophthalmology Emeritus

Caryn E. Plummer, DVM, Diplomate ACVO
Associate Professor of Comparative Ophthalmology and Service Chief, Veterinary Ophthalmology Service

1

Ocular Anatomy

The Globe

The eye is a very elegant organ, and a wonderful example of the intimate relationship of structure to function. Each part of the eye is designed to achieve or contribute to the special sense of sight. The globe is composed of three basic layers or coats. The outer coat is the fibrous tunic composed of the cornea, the sclera, and the juncture of the two called the limbus. The fibrous tunic gives the eye a constant shape and form which is imperative for a functional visual system. In addition, the anterior portion of the fibrous tunic, the cornea, is transparent, enabling light to pass through, and shaped in a manner that makes it a powerful lens which refracts light rays centrally towards the visual axis of the eye.

The middle layer, or vascular tunic, is the uvea which consists of the iris, the ciliary body, and the choroid. The most anterior portion of the vascular tunic, the iris, extends from the ciliary body centrally just anterior to the surface of the lens. The iris is heavily pigmented and contains muscles which change the shape and size of the iris and the pupillary aperture to control the amount of light that enters the posterior segment to stimulate the retina. The ciliary body is involved in both the production and outflow of aqueous humor, a fluid which flows through the anterior segment. Aqueous humor is secreted from ciliary body processes, which are heavily pigmented central extensions of the ciliary body. Aqueous humor leaves the eye through the iridocorneal angle, a portion of which (the uveal meshwork sinus) is of ciliary body origin. The ciliary body and its processes provide a base on which lenticular zonules are attached. These zonules are fine fibrous bands which attach to the outer portions of the lens and hold it in place. Contractions of ciliary body muscle alter the tension of these zonules and are able to change the shape or position of the lens. This process, called accommodation, alters the degree to which light is refracted. Thus, the lens acts as a fine focusing mechanism, while the cornea serves as the most powerful fixed "lens" of the visual system. The choroid, located in the posterior half of the eye, is found between the outer sclera and the retina. It functions to provide nourishment to the highly metabolic retina and to modify internal light reflection and scatter, as it is either heavily pigmented or reflective. In some species, a special reflective structure, called the tapetum, is located within the choroid and acts to improve photoreceptor stimulation in dim illumination.

The third layer of the eye is the nervous coat which is made up of the retina and associated optic nerve. Briefly, the retina contains light sensitive cells (photoreceptors) which, after a series of intermediate modifying processes, transmit impulses to the brain via the optic nerve.

In addition to the three tunics, additional ocular components fill the interior of the globe: (i) the intraocular fluids (aqueous humor and vitreous humor) and (ii) the crystalline lens.

Aqueous humor is continuously produced by ciliary body processes at a slow rate and fills the anterior and posterior chambers of the eye (between the cornea anteriorly and the lens posteriorly), then drains out of the eye into the bloodstream through the iridocorneal angle to regulate the intraocular pressure of the normal eye. Aqueous humor provides vital nutrients to the avascular lens and cornea and also assists in removing metabolic waste products.

Vitreous humor, a gelatinous fluid, occupies the large chamber in the back of the eye. The vitreous humor helps support and distend the globe and also provides an optically clear medium through which light can pass essentially unaltered.

The crystalline lens is a transparent, avascular, nonpigmented, flattened spheroidal structure lying behind the iris held in place by lenticular zonules. The lens is responsible for focusing light that has entered the eye onto the retina (Figure 1.1).

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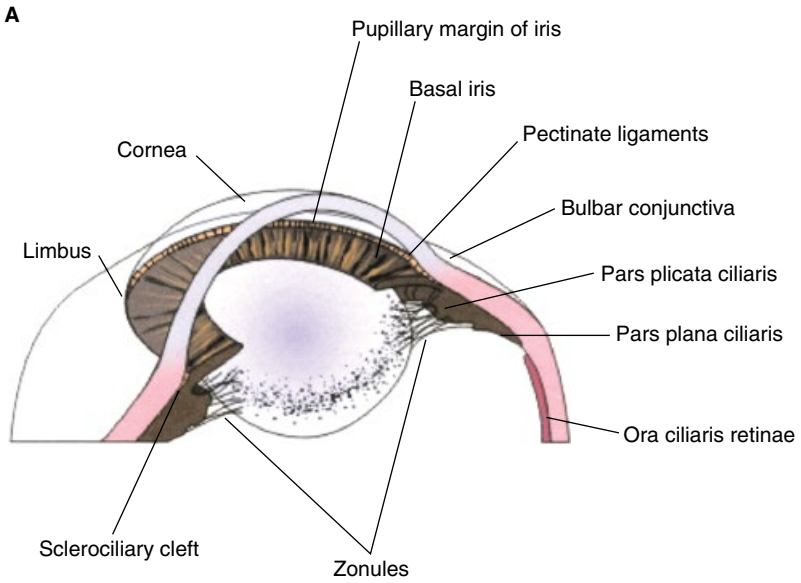
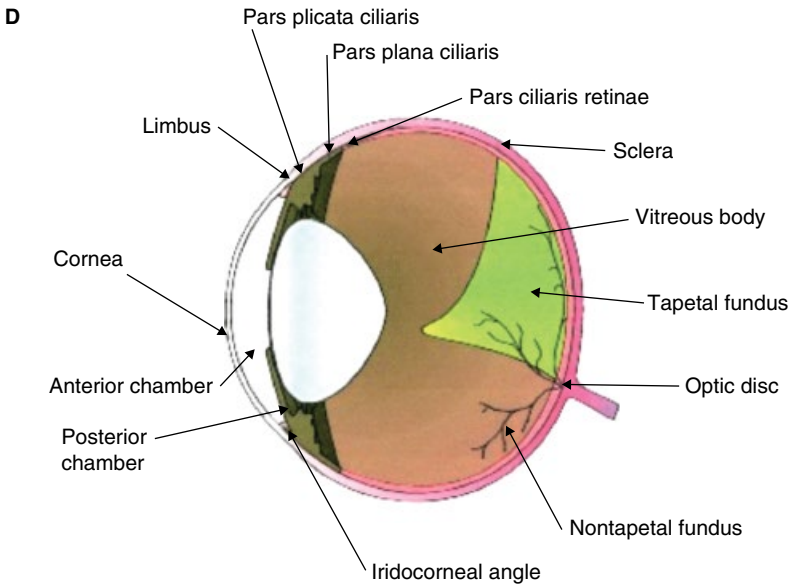




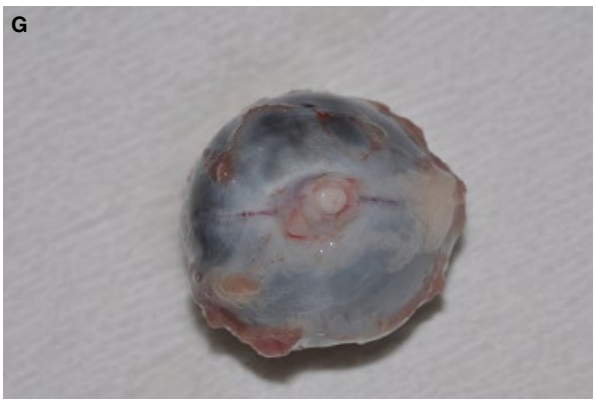


Figure 1.1 (A) The anterior eye showing the cornea, limbus, iris, ciliary body (pars plicata and pars plana), and the zonules that suspend the lens from the ciliary processes. The anterior chamber is the space between the interior cornea and the anterior lens and iris which is filled with aqueous humor. The bulbar conjunctiva covers the sclera which is the posterior continuation of the fibrous tunic (the cornea is the anterior portion). The pupil is the aperture in the center of the iris. (B) A normal horse eye. The iris in this animal is blue in color. There is a pigmented extension of the posterior pigmented epithelium of the iris along the dorsal pupil margin which is called the corpora nigrum or granula iridica. (C) A freshly enucleated canine globe from the front. (D) The globe from the side showing the cornea, limbus, anterior chamber, iridocorneal angle, posterior chamber, ciliary body (pars plicata, pars plana, and the ciliaris retinae), vitreal chamber, sclera, and optic nerve. The fundus is divided into tapetal and nontapetal sections. The tapetum is located within the dorsal choroid. The choroid, or the posterior aspect of the vascular tunic, lies interior to the sclera (the anterior extension of the vascular tunic is the ciliary body and the iris) and the retina lies interior to the choroid and adjacent to the vitreous body. (E) A normal horse eye in profile. (F) A freshly enucleated canine globe in profile. The optic nerve is observed extending from the posterior aspect of the globe. (G) Posterior aspect of a freshly enucleated canine globe. Running along the sclera anteriorly at the 3 and 9 o'clock positions are the long posterior ciliary arteries. These are important landmarks for surgical approaches to the eye. The insertions of the extraocular muscles (the muscles themselves have been removed) which move the globe are appreciable. (H) In this prosection, the cornea and a sector of the iris have been removed to reveal the lens equator and the ciliary body behind. (I) In this prosection, the cornea, and the anterior uvea have been removed revealing the lens sitting within the patellar fossa of the vitreous. (J) The lens has been removed from the globe and placed upon a page of type. Note the clarity and the magnification. (K) In this prosection, the anterior segment and lens have been removed, revealing the retina (artifactually detached in areas), the retinal vasculature, the tapetum in the dorsal choroid, the pigmented nontapetal fundus, and the optic disc. (L) Prosection of the posterior globe. In this example, the globe has been cut in order to flatten it. Source: (A, D) Gelatt KN and Gelatt JP 2011. Reproduced with permission of Elsevier.









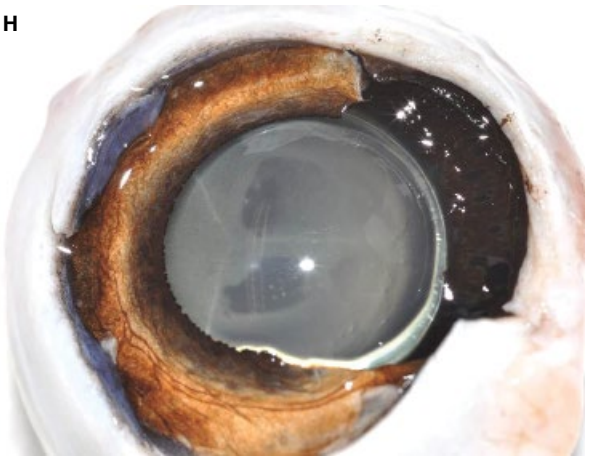
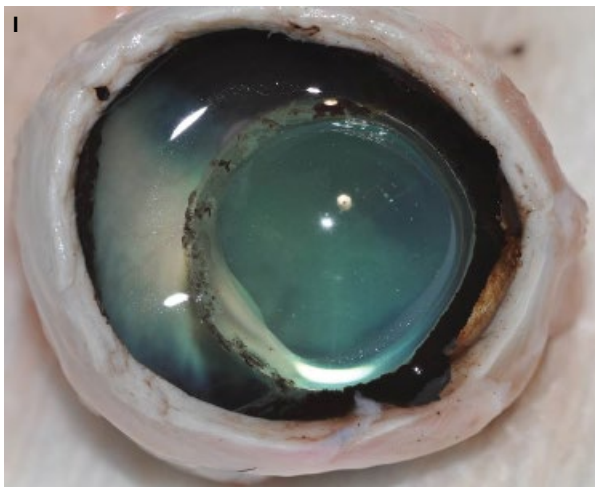


Figure 1.1 (*Continued*)





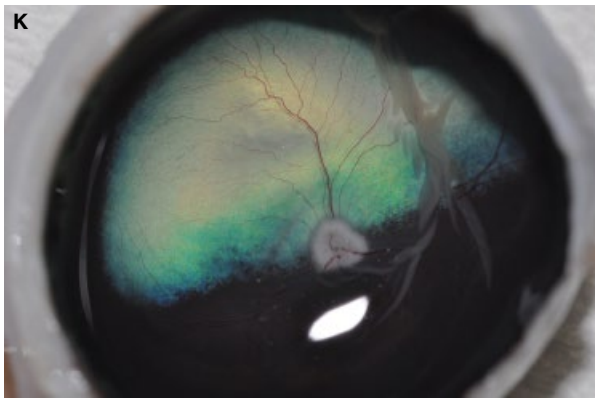




Figure 1.1 (*Continued*)

The Adnexa

The orbit is a bony fossa that separates the eye from the cranial cavity, surrounds and protects it, and provides several pathways through foramina for the various blood vessels and nerves involved in the function of the eye. The orbit in the dog and cat is an incomplete bony orbit composed of five, sometimes six bones, the supraorbital ligament, and the periosteum. The ruminant large animals usually have enclosed orbits. Closure of the temporal side of the orbit is accomplished by the union of the zygomatic bone and the frontal bone. The enclosed orbit is essential for protective purposes.

The eyelids are dorsal and ventral folds of thin skin continuous with the facial skin. The free edges of the dorsal and ventral lids meet to form the lateral and medial canthi. The opening formed by the free edges is the palpebral fissure. The fissure is prevented from assuming a circular shape by medial (nasal) and lateral (temporal) palpebral ligaments which attach the canthi to the orbital wall. The medial ligament inserts into periosteum of the nasal bones whereas laterally it inserts into temporal fascia. The lateral ligament is absent or rudimentary in the dog and is replaced by the retractory anguli oculi muscle. Closure of the eyelids is achieved by the contraction of the orbicularis oculi muscle located deep in the lids around the palpebral fissure. Opening or parting of the lids is by relaxation of the orbicularis oculi and contraction of the levator palpebrae superioris which inserts on the orbicularis oculi muscle.

The free margin of the eyelid can contain a row of cilia or lashes. These lashes are directed away from the anterior surface of the cornea. The inner surface of the lids is lined with a mucous membrane, the (palpebral) conjunctiva. The conjunctiva is reflected onto the globe (bulbar conjunctiva). The junction between the palpebral and

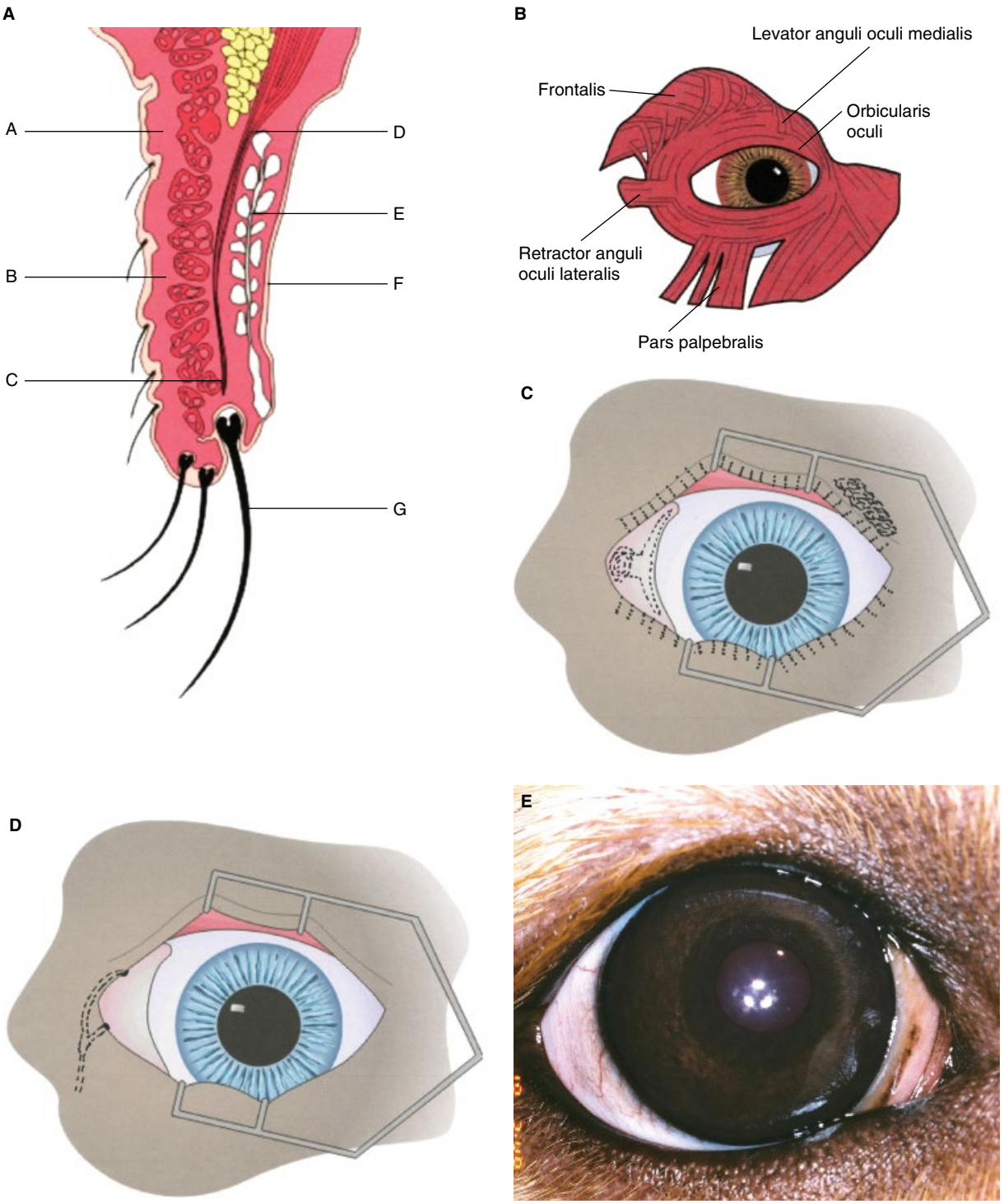


Figure 1.2 (A) The eyelids. A. Haired skin. B. Orbicularis oculi (eyelid musculature responsible for closure of the palpebral fissure). C. Tarsal plate. D. Insertion of the levator palpebrae superioris (responsible for elevation of the upper eyelid). E. Meibomian glands. F. Palpebral conjunctiva. G. Cilia (eyelash). (B) The eyelid musculature showing the muscles of facial expression and eyelid movement. The levator palpebrae superioris and the Müller's muscles, responsible for eyelid opening, are not shown. (C) The location of the lacrimal glands (orbital and gland of the third eyelid) and the meibomian glands lining the upper and lower eyelids along their margins. (D) The location of the lacrimal puncta of the nasolacrimal apparatus in the medial canthus and the subcutaneous pathway into the nasolacrimal duct. (E) A normal dog eye. Note the apposition of the eyelids to the globe, the smooth, regular margins of the lids and the third eyelid, and the normal appearance of the conjunctiva and the nictitans. Source: (A-D) Gelatt KN and Gelatt JP 2011. Reproduced with permission of Elsevier.

bulbar conjunctiva is the fornix. The conjunctiva is the most exposed of all mucous membranes. Its primary functions are preventing dessication of the cornea, increasing mobility of the eyelids and globe, and providing a barrier against microorganisms and foreign bodies. Ventrally, an additional fold is formed by the reflection of the conjunctiva over the nictitans. The nictitans (third eyelid) is a large, semilunar fold of conjunctiva that protrudes from the medial canthus over the anterior surface of the globe (from the dorsomedial orbit in birds). It contains a cartilaginous plate which is T-shaped, the horizontal part of it being parallel with the free edge of the membrane. The nictitans gland surrounds the caudal end of the shaft of the cartilaginous plate with the majority of the gland on the bulbar surface. It produces approximately 30% of the tears. The largest lacrimal gland, which is responsible for producing the majority of the aqueous tears, is located in the dorsal orbit.

Visible through the conjunctiva on the posterior surface of the eyelid margin are the meibomian glands. These form parallel rows of lobules which have their ducts opening close to the lid margins. The glands in the distal eyelid stroma are sebaceous in nature and contribute to the oily component of tear film. Each gland is made of a number of holocrine acini which are arranged in vertical columns and open into a central duct.

The tear film is considered an anatomic structure as well. This fluid covering the partially exposed anterior segment of the globe is necessary for maintaining an optically uniform corneal surface, removing foreign material and debris from the cornea and conjunctival sac, providing oxygen and other nutritional requirements to the cornea, and preventing the development of ocular surface infections. It normally consists of an aqueous component, a lipid component, and a mucous component. Aqueous tear fluid, once it has fulfilled its duties, drains through lacrimal puncta in the upper and/or lower eyelids at the medial canthus into the nasolacrimal sac and duct which subsequently drain into the nasal passages or the oropharynx (Figure 1.2).

2

The Ophthalmic Examination and Diagnostics

A thorough ophthalmic examination can provide a rapid and accurate diagnosis for many ophthalmic diseases, because most ocular structures can be visualized either directly or indirectly. Furthermore, the eye lends itself to numerous simple and efficient diagnostic procedures, many of which can be performed during a routine examination. This chapter demonstrates examination and diagnostic techniques. Most of these procedures are noninvasive, and a thorough understanding of them can facilitate the identification and diagnosis of many ocular disorders.

The basic equipment necessary to perform a proper ocular examination includes a bright, focal light source (a Finoff transilluminator is ideal), Schirmer tear test strips, ocular stains (vital dyes), topical anesthetic, mydriatic agent, eyewash, sterile culture swabs, forceps, surgical blades, glass slides, cannulas for nasolacrimal duct irrigations, and an ophthalmoscope for examination of the fundus. Magnification is very helpful to identify small or subtle lesions.

The eye examination must be complete, organized, and strategic. Certain tests or observations must precede others to avoid interference or spurious conclusions. If indications for microbiologic sampling are present, samples are taken on moistened sterile swabs before instillation of any diagnostic drugs (stains, mydriatics, local anesthetics) as they can contain preservatives that prevent microbial growth. The Schirmer tear test must be performed before excessive ocular manipulation and instillation of any ophthalmic solutions or ointments, otherwise the result will be an inadequate reflection of tear production. The pupillary light reflexes should be evaluated before mydriatics or miotics are used. Similarly, measurement of intraocular pressure (IOP) should precede instillation of mydriatics. A thorough examination assesses all ocular and periocular structures, outside to inside and front to back. Accurate recording of examination will permit assessment of progress (Figure 2.1).

External Examination

The ophthalmic examination begins with an indirect assessment of vision (menace response, visual placing, maze testing) and comfort, and should be performed prior to sedation and nerve blocks, if these are necessary. The distant evaluation assesses the size, position, and direction of the globe and its movements. Any ocular discharge and asymmetry should be noted. It is important to examine each eye successively and to assess ocular and adnexal structures for symmetry.

The distant examination should be performed from different angles when facing the patient as subtle changes in globe position can become apparent when viewing, say, from above the animal's head. Evaluation of ocular movements can be achieved by turning the animal's head from side to side. Normal saccadic and optokinetic movements are noted as the eyes move back and forth in synchronicity, with the fast phase occurring in the direction of head movement. Complete external examination should include palpation of the orbit and retropulsion of both globes (Figure 2.2).

Nerve Blocks

Akinesia of the palpebral or auriculopalpebral (branch of palpebral) nerves facilitates examination of the eye in large animals, particularly equids. There are three main points at which the auriculopalpebral or palpebral nerves can be blocked in the horse. The first is just anterior to the base of the ear where the auriculopalpebral nerve emerges from the parotid salivary gland and becomes subcutaneous on the lateral aspect of the coronoid process. Here local anesthetic can be injected into the depression just caudal to the ramus of the mandible at the ventral edge of the temporal portion of the zygomatic arch. The rostral auricular artery and vein should be

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avoided. The second is just lateral to the highest point of the caudal zygomatic arch where the palpebral nerve can be "strummed" under the skin over the dorsal border of the bone. The third is where the palpebral nerve lies on the zygomatic arch caudal to the bony process of the frontal bone (Figure 2.3).

Tear Film Assessment

The nasolacrimal system has both secretory and drainage functions. Evaluation of this apparatus should note any tearing or hypofunction, as well as the endpoint of drainage. Excessive tearing can be caused by partial or complete obstruction of the drainage apparatus and increased lacrimation by ocular irritation and uveitis. The quality and quantity of the tear secretion is indirectly assessed by observation of the normal preocular tear film, which consists of three main components. The middle, or aqueous, layer is produced primarily by the lacrimal and nictitans glands in mammals, and deficiencies can be identified via the Schirmer tear test (Figure 2.4). The folded end of the Schirmer tear test strip is inserted in the lower conjunctival fornix, in contact with the cornea, near the junction of the middle and temporal thirds of the eyelids where it should remain for 1 minute. Tears are measured from the fold of the strip in millimeters per minute, immediately following removal. In common domestic species, tear production greater than 15 mm/minute in the absence of disease is considered adequate. The inner, mucin layer of the tear film is produced primarily by conjunctival goblet cells and the outer, lipid layer is produced by the meibomian glands in the eyelids. Deficiencies of the outer and inner layers result in qualitative dry eye. These can be assessed by tear film break up time (rapid evaporation) which utilizes topical fluorescein, rose Bengal, or lissamine green stains to delineate foci of mucin absence.

The passage of tears through the nasolacrimal duct can be indirectly observed with fluorescein passage (Jones' test). If fluorescein is not observed at the nares, active flushing, or the injection of normal saline solution through the nasolacrimal drainage apparatus either orthograde (i.e., from the lacrimal puncta) or retrograde (i.e., from the distal nare's opening), can be performed.

Corneoconjunctival Culture

Culture and sensitivity testing provide useful information for the diagnosis and determination of appropriate antimicrobial therapy in many corneal and conjunctival diseases. Cultures should be obtained very carefully from any deep or progressive corneal ulcers, or those that fail to heal in a reasonable amount of time. Cultures can also be taken from purulent or granulomatous conjunctival lesions or from animals with chronic conjunctivitis that does not respond to therapy. Both corneal and conjunctival cultures should be obtained early in the ophthalmic examination, before administration of topical solutions or ointments (Figure 2.5).

Corneoconjunctival Cytology

Corneal or conjunctival cytology is extremely useful in the diagnosis of certain forms of inflammatory or neoplastic conditions and is very helpful for the planning of

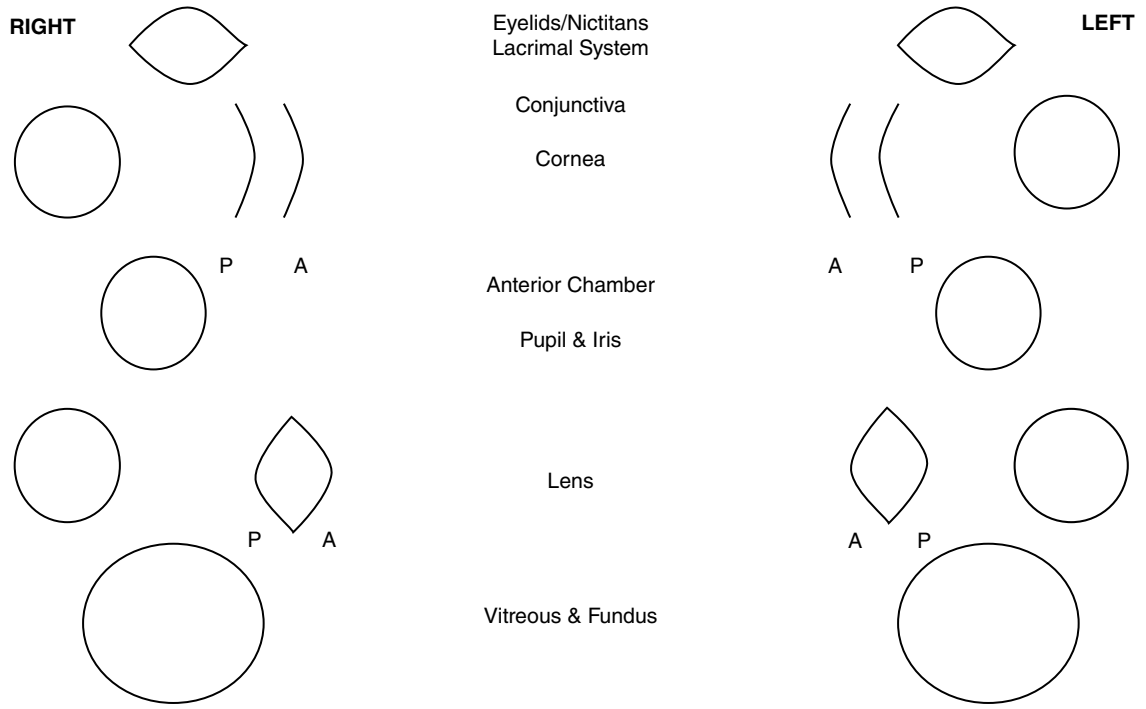


Figure 2.1 (A) Essential equipment for ophthalmic examination.

B

Patient Label Here	Faculty/Resident/Student:	Date:
	Diagnosis:	
Present Treatment:	Recommended Treatment:	

Reflexes	**Right**	**Left**	**Tests**	**Right**	**Left**
Direct PLR	+ / –	+ / –	Schirmer Tear Test	mm/min	mm/min
Consensual PLR	+ / –	+ / –	IOP	mmHg	mmHg
Menace	+ / –	+ / –	Fluorescein	+ / –	+ / –
Dazzle	+ / –	+ / –	Jones Test	+ / –	+ / –
Palpebral	+ / –	+ / –	Rose Bengal	+ / –	+ / –



Special Procedures:		Sedation: _____
ERG: ☐	Culture: ☐	General Comments:
Gonioscopy: ☐	Cytology: ☐	
N-L Flush: ☐	Ultrasound ☐	
Photography: ☐	Other: ☐	
_____		

Figure 2.1 (*Continued*) (B) An example of an examination form for recording findings.







Figure 2.2 (A) Pupillary light reflexes should be assessed with a bright light (here, a Finoff transilluminator is being used) in both bright and dim lighting conditions. (B) Retropulsion of the globes should be equal on each side and nonpainful to the patient. (C) Assessing the patient for asymmetry is an important part of the ophthalmic examination. In this case, a retrobulbar mass has resulted in relative exophthalmos and strabismus. Radiation therapy has resulted in cataract formation and whitening of the hair coat.



Figure 2.2 (*Continued*) (D) Characterization of any ocular discharge can help with diagnosis and staging of severity and chronicity of ocular conditions.



Figure 2.3 Landmarks for the auriculopalpebral motor block to facilitate a complete ophthalmic examination in a horse.

empiric therapy for corneal ulcers. Instruments for collecting cytologic samples include cotton or Dacron swabs, cytobrushes, spatulas (Kimura platinum spatula), and the blunt end of a scalpel blade (Figure 2.6). Impression cytology can also be used for sample collection in some instances. Topical anesthesia (i.e., 0.5% tetracaine or 0.5% proparacaine) should be applied to the ocular surface prior to sample acquisition, and care must be taken to avoid ocular trauma.

Ophthalmic Stains

Fluorescein dye is used to detect corneal and conjunctival defects, aqueous humor leakage (Seidel's test), precorneal tear film (PTF) deficiencies (tear film break-up time; TFBUT), and to assess nasolacrimal duct patency (Figure 2.7). TFBUT is a measure of the stability of the PTF which involves recording the time it takes for fluorescein applied to the ocular surface to dissipate, or the PTF to dissociate (dark spots in a diffuse film of fluorescein will develop and indicate break-up). Patency of the nasolacrimal apparatus is tested by applying sodium fluorescein to the eye and timing the passage of fluorescein through the system to the external nares (i.e., Jones' test). This dye is hydrophilic, binding readily to exposed corneal stroma when ulcers are present. The dye will not bind to intact healthy corneal epithelium, however, or to the endothelium and Descemet's membrane.

Rose Bengal is used to assist in the diagnosis of PTF disorders (mucin deficiency) and superficial corneal epithelial abnormalities. Besides being used to assess the integrity of the PTF, rose Bengal can also be used to demonstrate very small superficial ulcers and erosions such as the punctate corneal lesions often present in early stages of keratomycosis in the







Figure 2.4 (A) Schirmer tear test 1 being performed in a dog. The notched end of the strip is inserted behind the eyelid margin along the lateral lower eyelid so that it comes into contact with the globe. (B) Nasolacrimal patency can be assessed indirectly with the application of topical fluorescing stain to the ocular surface. After several minutes, the dye should be seen exiting the nares (Jones' test positive). Note the green colored nasal discharge from the left nare. (C) Cats may not have fluorescein exit from their nares. Some individuals drain tears into their oral pharynx, so assessment of nasolacrimal patency includes opening the mouth and observing the base of the tongue for fluorescein.

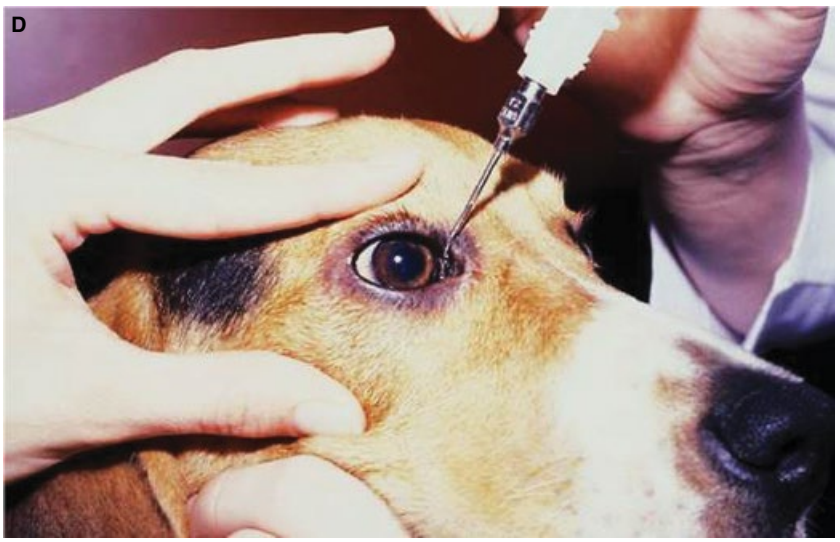




Figure 2.4 (*Continued*) (D) If the Jones' test is negative, the next step in assessing patency of the nasolacrimal apparatus is to flush the nasolacrimal duct manually. After application of a topical anesthetic, a small cannula is introduced into one of the lacrimal puncta. Mild pressure is applied to force fluid through the duct. If it does not flow appropriately, dacryorhinocystography should be considered. (E) Flushing of the nasolacrimal duct in horses is usually performed retrograde from the nasal punctum.

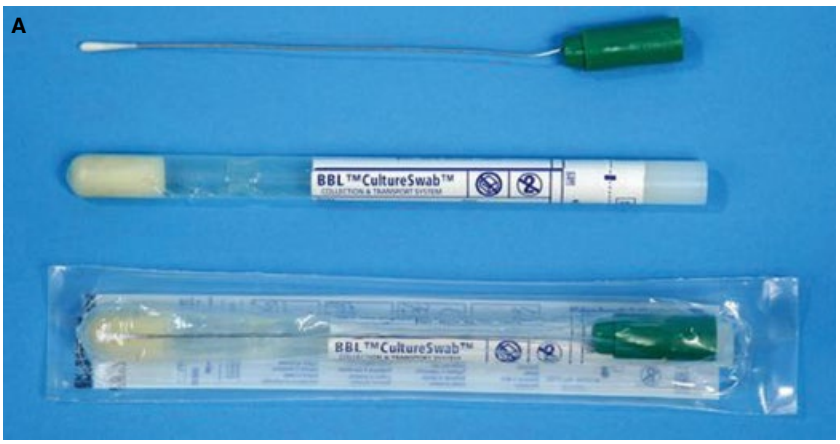


Figure 2.5 (A) Equipment employed in sample acquisition for microbial culture and susceptibility testing.



Figure 2.5 (*Continued*) (B) Taking a sample for microbiologic culture and susceptibility testing.



Figure 2.6 (A) The basic equipment necessary to perform cytology. (B) Taking a corneal sample from a melting corneal ulcer with a cytology brush.





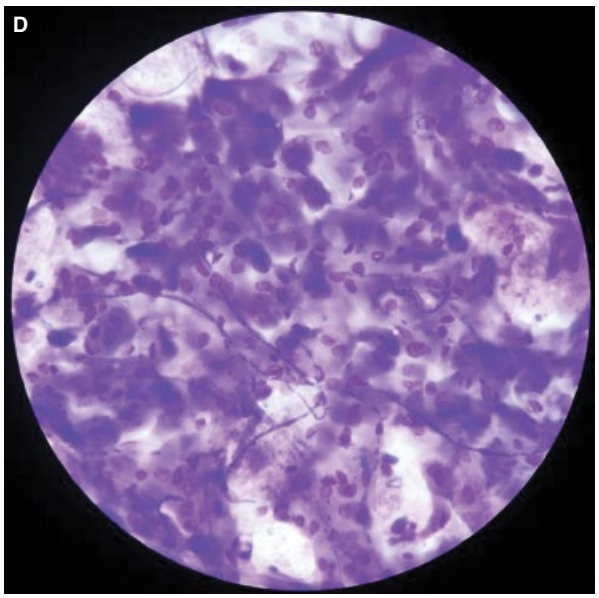
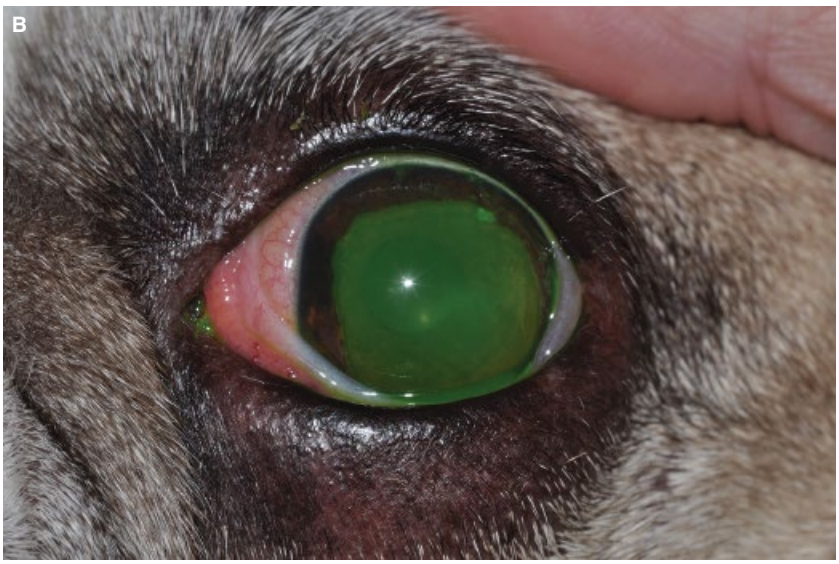


Figure 2.6 (*Continued*) (C) Taking a corneal sample with the dull side of a scalpel blade. (D) A stained example of corneal cytology from a case of fungal keratitis. There are numerous epithelial cells. The linear septate structures are hyphal elements.



Figure 2.7 (A) Topical ophthalmic stains can be applied to the ocular surface directly from the impregnated strip or applied to the eye after dilution with saline in a syringe.



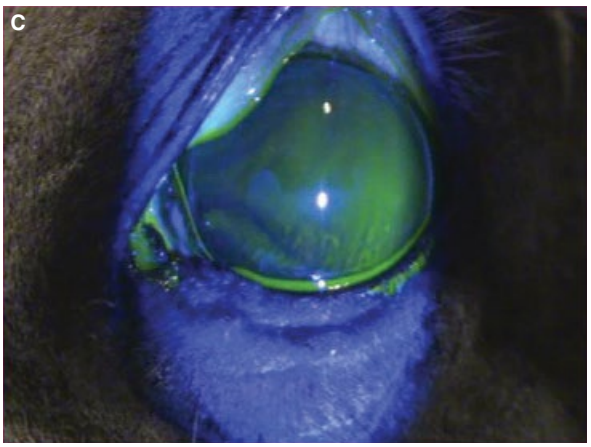


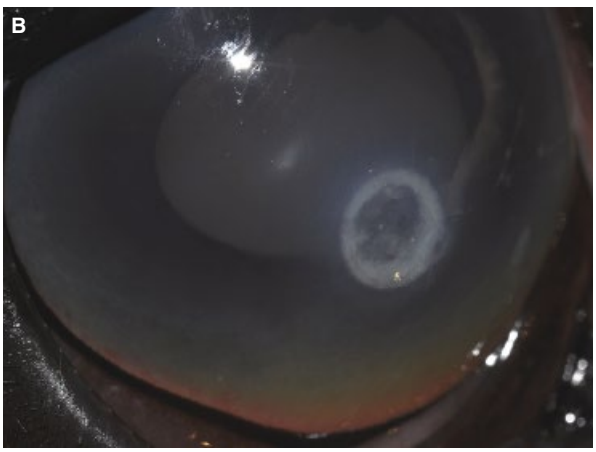


Figure 2.7 (*Continued*) (B) Fluorescein delineating the margins of a superficial corneal ulcer. (C) Fluorescein employed in determining tear film break-up time. (D) Rose Bengal staining a qualitative dry eye.



Figure 2.7 (*Continued*) (E) Rose Bengal highlighting a dendritic ulcer in a cat with feline herpesvirus type 1 keratitis.





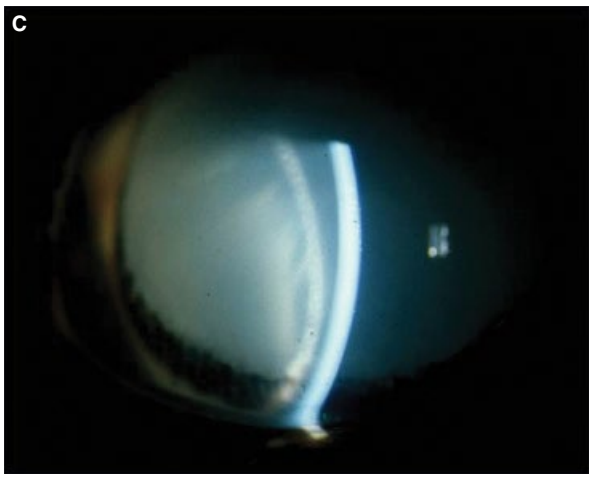


Figure 2.8 (A) Performing slit lamp biomicroscopy. (B) All corneal ulcers should be evaluated for depth or degree of stromal loss. Lesions or deposits in the cornea can be localized by depth as well. This infected corneal ulcer in a horse is approximately 40% depth in the center and the cellular infiltrate in the periphery is in the anterior stroma. (C) Evaluation of the anterior segment should include assessment of the anterior chamber depth. In this eye, the cataractous lens has moved anteriorly at its ventral aspect. Note the narrowing of the space between the cornea and the lens in the ventral anterior chamber. Aqueous flare is visible in this image as well.

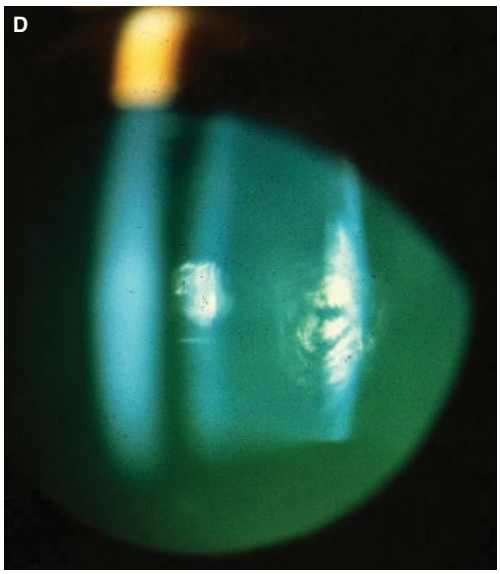


Figure 2.8 (*Continued*) (D) Slit lamp biomicroscopy can help with cataract localization within the lens. In this dog there are two small cataracts, one in the anterior cortex and the other in the posterior aspect of the lens.





Figure 2.9 (A) Estimating intraocular pressure with a TonoVet rebound tonometer. (B) Estimating intraocular pressure with a TonoPen applanation tonometer.



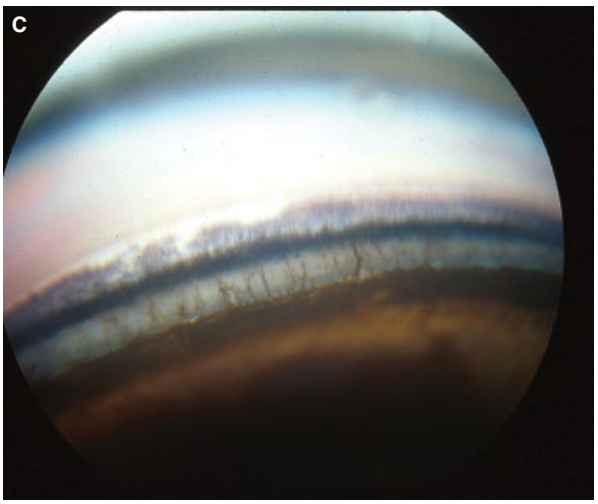




Figure 2.10 (A) A variety of types of goniolenses are available for examination of the iridocorneal angle. (B) Performing gonioscopy with a Franklin goniolens. (C) The view of the normal iridocorneal angle of a dog as seen with gonioscopy.



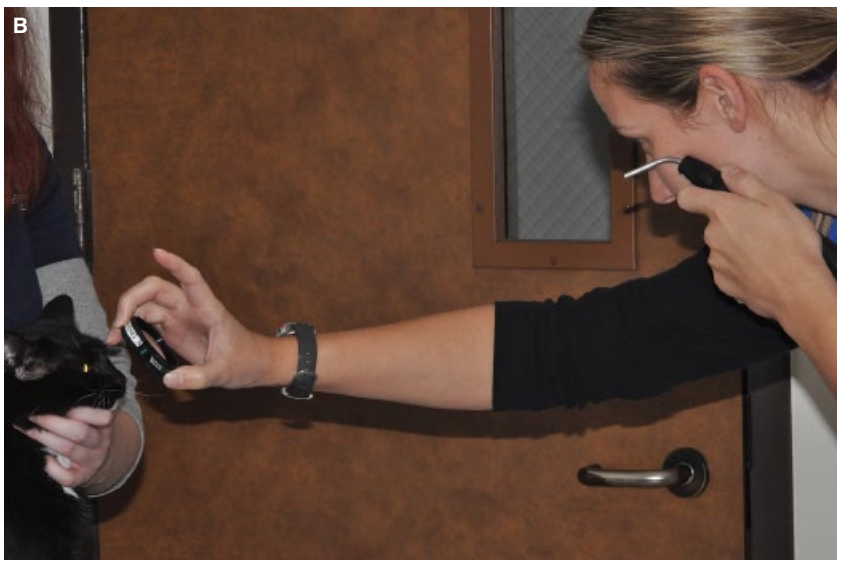




Figure 2.11 (A) Performing direct ophthalmoscopy. (B) Performing monocular indirect ophthalmoscopy. (C) Performing binocular indirect ophthalmoscopy.







Figure 2.11 (*Continued*) (D) The view of the fundus of a dog as seen with indirect ophthalmoscopy. (E) The view of the fundus of a cat as seen with indirect ophthalmoscopy. (F) Performing ophthalmoscopy with the PanRetinal ophthalmoscope, which is a hybrid of direct and indirect ophthalmoscopy.



Figure 2.11 (*Continued*) (G) This canine patient has been appropriately dilated prior to ophthalmoscopy.

horse, punctate keratitis in the dog, and dendritic lesions caused by feline herpesvirus type 1 infection in the cat.

Anterior Segment

The cornea is normally avascular, nonpigmented, nonkeratinized, transparent, smooth, and shiny. The presence of surface irregularities (growth or defects), blood vessels, pigment, or other opacities indicates disease. The anterior chamber is observed for its transparency and its depth. The iris is evaluated for color, consistency, pupillary membrane strands, pupil size, shape, and stability. The pupil can be abnormally dilated with iris atrophy, glaucoma, retinal disease, or optic nerve disease, or it can be abnormally small with Horner's syndrome or anterior uveitis. The lens is evaluated for its position, size, shape, surface irregularities, and its transparency, with both direct illumination and retroillumination (Figure 2.8).

Intraocular Pressure

Before administration of mydriatics, all animals presented for ophthalmic examination should have the IOP evaluated by instrumental tonometry. Tonometry is the indirect measurement or estimation of IOP. It is an essential diagnostic procedure during eye examination in animals, particularly of any eye that is red or painful, has focal or diffuse corneal edema, mydriasis, orbital trauma, a lens luxation, or a history of glaucoma, either of the eye of interest or the fellow eye. Eyes with IOP elevations to 25–40 mmHg may not demonstrate overt signs of glaucoma. Applanation and rebound tonometry are the most common methods employed in veterinary ophthalmology and these techniques are relatively quick and simple and provide crucial information (Figure 2.9).

Gonioscopy

Gonioscopy involves the use of special lenses that mitigate the interference of the corneal curvature which impairs direct visualization of the iridocorneal angle (ICA; Figure 2.10). It is very useful in small animals. Large animals, particularly horses, have areas of their ICA that are visible on direct observation and do not require goniolenses.

Posterior Segment

The vitreous humor is normally a clear and homogenous gel that fills the space between the posterior axial lens capsule, posterior chamber, and ocular fundus. The ocular fundus which posteriorly borders the vitreal cavity includes the optic nerve head (which should be assessed for shape, color, and topography), the retina and its retinal vasculature (evaluate position of retina and number, size, and degree of perfusion of its vessels), and the tapetal (in species with a tapetum) and nontapetal regions (evaluate reflectivity, pigmentation, depigmentation, hemorrhage, exudates). Ophthalmoscopy is a difficult, but very important, procedure for the clinician to master. With diligence and practice, this technique can provide the veterinarian with yet another tool with

which to examine and investigate patients and their problems.

Following adequate mydriasis, the examiner places the direct ophthalmoscope snuggly against his/her brow and identifies the patient's fundic reflex from a distance of approximately 0.5–0.75 m. Once the fundic reflex is identified, the examiner moves toward the patient to a point approximately 2–3 cm from the eye (Figure 2.11). The advantages of direct over indirect ophthalmoscopy include greater magnification, availability of options such as the slit and cobalt blue filters, and ability to alter the dioptric power of the ophthalmoscope. Disadvantages include a small field of view which often results in lesions being missed, particularly in the periphery (which is nearly impossible to visualize with this technique), short working distance between examiner and patient, lack of stereopsis, and greater distortion when the visual axis is not completely transparent.

Indirect ophthalmoscopy allows the clinician a larger field of view of the fundus which can be viewed from a larger and safer working distance from the patient than with direct ophthalmoscopy. The image generated is inverted and reversed (upside down and backwards). To perform the fundic exam following mydriasis, the examiner grasps the patient's muzzle with one hand and stabilizes the head. The other hand is used to hold the eyelids open and to position the lens 2–4 cm in front of the patient's cornea. Successful examination of the ocular fundus requires intimate knowledge of the normal variations within each species.

3

Clinical Signs and Their Interpretations

As clinicians for animal patients, we must gather most of our evidence by what we can observe, feel, smell, and hear. Laboratory and imaging methods then either support our working diagnosis or assist in the development of a list of differential diagnoses which it is hoped will allow us to isolate and ascertain the abnormality, develop strategies for treatment, and provide an accurate prognosis. In this introductory clinical chapter, some of the common clinical signs and observations are summarized, and their pathogenesis and significance presented. In the subsequent chapters additional information of some of these abnormal findings are expanded.

Blepharospasm and Ophthalmic Pain

Animals with ophthalmic diseases can show pain through a number of clinical signs. Blepharospasm is mediated by reflex involving branches of the trigeminal nerve within the cornea and conjunctiva (sensation), and the facial nerve (motor) which innervates the orbicularis oculi muscle, which is responsible for closure of the palpebral fissure. The source of the pain can be within the orbit, eyelids, conjunctiva, cornea, iris, and ciliary body. Pain receptors within the lens, vitreous, and retina-choroid apparently do not exist.

There is also an axonal reflex by which corneal pain is directly transmitted to the anterior uvea, resulting in the release of prostaglandins, histamine, and acetylcholine, a breakdown of the blood–aqueous barrier, iridocyclitis, and aqueous humor flare. This pathway is completely local and does not involve any sensory or motor neural pathway or the brain. Blepharospasm usually signals pain and possible inflammation, therefore the clinician should recognize the need to examine and determine the cause (Figure 3.1). Essential or primary blepharospasm appears rare in animals.

Ocular Discharge

Conjunctival discharges are divided into: serous or catarrhal; mucus (mucoid); and mucopurulent. Often conjunctivitis in animals is secondary to eyelids, nasolacrimal and tear, and corneal diseases. The character of these conjunctival discharges can vary throughout the disease as well as their quantity. Following an insult, the conjunctival flora in secondary conjunctivitis proliferates resulting in mucopurulent exudates that can mask the original insult. As conjunctivitis becomes chronic, secondary thickening, pigmentation, and follicle formation develop (Figure 3.2).

Globe Position

The position of the globe within the orbit varies by species and, especially in the dog, by breed. Changes within the orbit tissues can also influence the position of the globe within the palpebral fissure. Loss of fatty tissues or fibrosis after trauma or orbital surgery can result in reduced orbital tissue (enophthalmia) and restricted globe mobility. In contrast, increase in the orbital mass associated with cellulitis, mucocele formation, and neoplasia can force the globe forward into the palpebral fissure (exophthalmia) and cause strabismus. The direction of the strabismus can often assist in the localization of the mass (Figure 3.3).

Globe Size

Globe size varies widely among the animal species, and for many of the domestic species direct and ultrasonic measurements are available. Globes smaller than normal are termed microphthalmia and those larger than normal are macrophthalmia (also termed megalophthalmia) (Figure 3.4). Corneal and globe measurements

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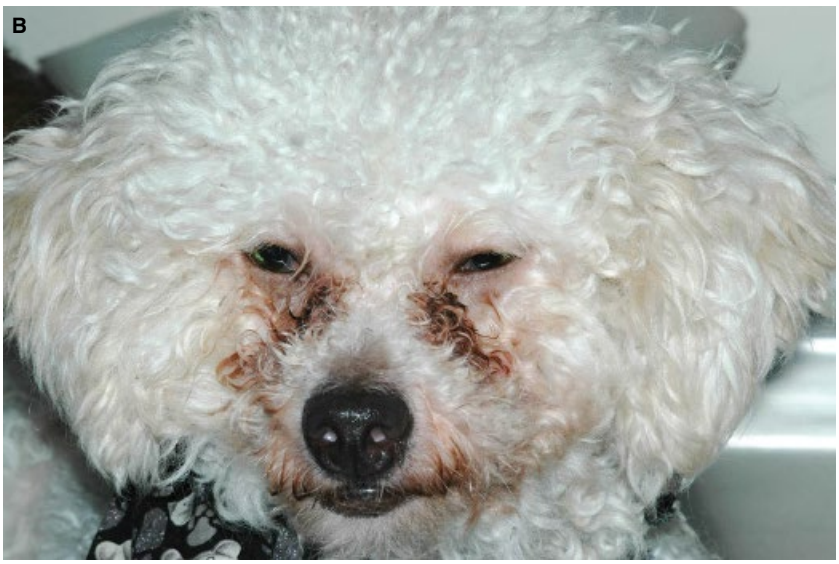






Figure 3.1 (A) Pain/blepharospasm in a cat secondary to entropion. The eyelid outer margin and hair are directly contacting the conjunctival and corneal surfaces. (B) Pain/blepharospasm secondary to distichiasis and a corneal ulcer in a young Bichon Frise. (C) Blepharospasm in a horse with severe anterior uveitis and infected corneal ulcer and blepharitis. There is an abundance of mucoid ocular discharge. (D) Subtle pain in a horse may be demonstrated not by overt blepharospasm but instead by a downward deviation of the cilia (eyelashes).

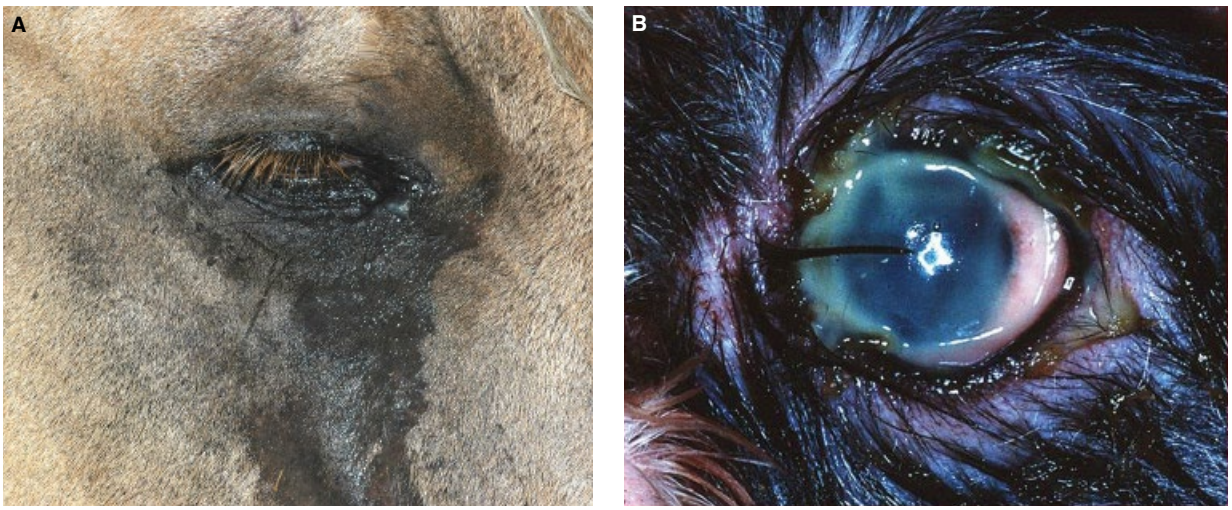


Figure 3.2 (A) Epiphora can be the result of overproduction of tears, usually a reflex manifestation of pain, or because there is an obstruction of outflow of tears through the nasolacrimal apparatus. This horse has severe anterior uveitis and pain. (B) Mucopurulent discharge usually indicates chronicity or severity. It is common in keratoconjunctivitis sicca as a compensatory mechanism for the lack of aqueous tears.

Figure 3.3 (A) Exophthalmos is the anterior displacement of the globe. In this cat, a retrobulbar squamous cell carcinoma has pushed the globe forward. There is conjunctival hyperemia and chemosis and serosanguinous ocular discharge. (B) Same cat as in part A from a view looking down. If exophthalmos is subtle, the dorsal view can make the difference in globe position more appreciable.

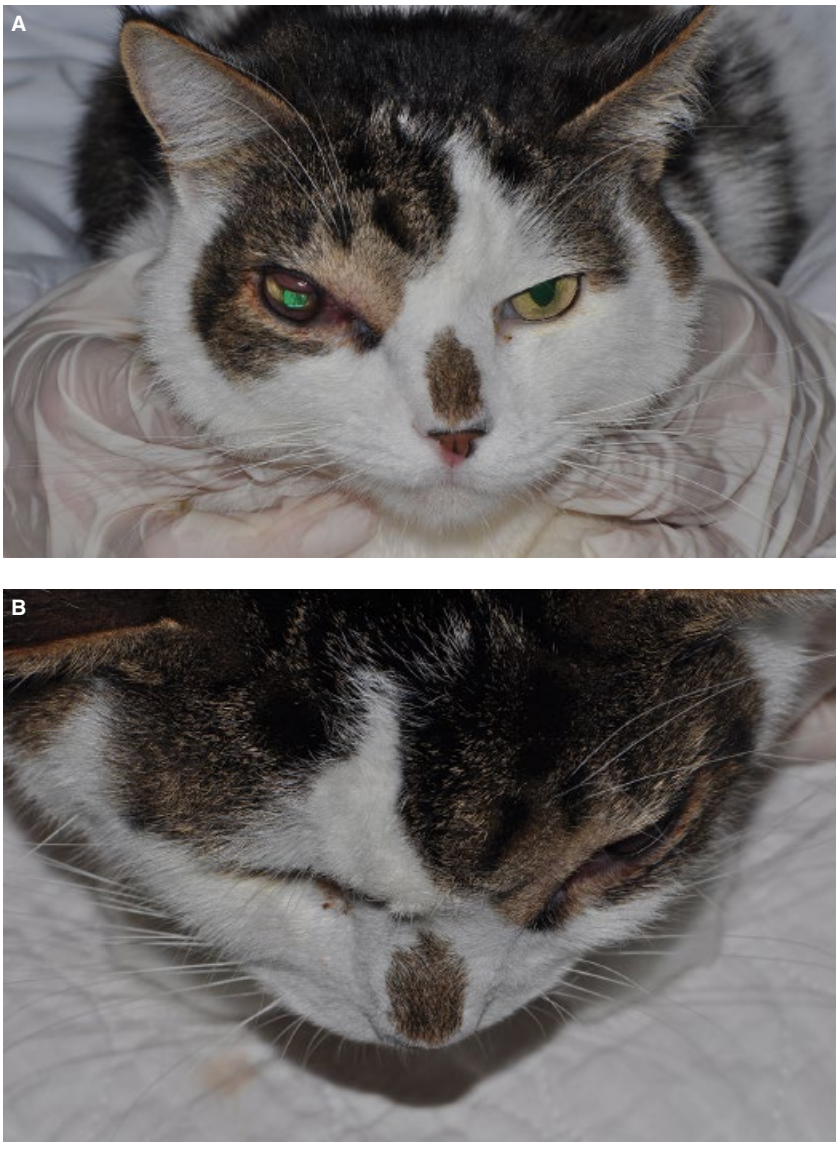








Figure 3.3 (*Continued*) (C) When the globe is farther back into the orbit than usual, this is referred to as enophthalmos. This occurs as the result of pain, loss of orbital contents, or sympathetic denervation to the periocular structures. (D) Strabismus is the deviation of the globe from its normal directional axis. This dog has a dorsolateral strabismus of the right globe. (E) Dorsal strabismus in a Chihuahua following trauma.







Figure 3.4 (A) Microphthalmia is a congenitally small globe. (B) Phthisis bulbus is an acquired reduction in size of the globe, usually the result of chronic inflammation or chronically elevated intraocular pressure that damages the ciliary body over time and reduces its ability to produce aqueous humor in quantities sufficient to keep the globe turgid. (C) Buphthalmos is an enlargement of the globe resulting from elevation in intraocular pressure with glaucoma.

are closely related. The differences in globe size in dogs is also related to breed and has not been documented to date.

Vascular Changes

The conjunctiva, consisting of the palpebral, fornix, and bulbar components, accommodates eyelid movements as well as movements of the globe. The conjunctiva is also important in tear dynamics and immunologic protection of the external ocular surfaces. Its superficial layer contains minute lymphoid follicles which provide the different immunologic components.

Conjunctival Vascular Response

As part of the conjunctival inflammatory response, local vasodilation of conjunctival vessels occurs. These conjunctival vessels have a small diameter and branching pattern, blanch quickly to topical 1–2% epinephrine, and are mobile and move when the conjunctival surface is manipulated (Figure 3.5).

Ciliary Flush

Ciliary flush or a diffuse hyperemia of both the bulbar conjunctiva and episcleral blood vessels is associated with inflammation of the iris and ciliary body (Figure 3.6). Ciliary flush should be distinguished clinically from



Figure 3.5 (A) Conjunctival hyperemia, especially of the ventral conjunctiva, in a dog, associated with conjunctivitis and ectropion. Note the enlarged lymphoid follicles which indicate some degree of chronicity. (B) Severe conjunctival hyperemia in a cat affecting the upper and lower palpebral conjunctiva and the conjunctiva of the nictitans.







Figure 3.5 (*Continued*) (C) Conjunctival hyperemia in an English Bulldog with keratoconjunctivitis sicca. (D) Chemosis is edema of the conjunctiva. It can be quite pronounced, especially in allergic conditions. (E) Conjunctival hyperemia in a horse secondary to an allergy.



Figure 3.6 Iridocyclitis and associated ciliary flush secondary to corneal ulceration and uveitis in a dog.

conjunctival hyperemia because it signals intraocular involvement and inflammation. As the iridal and ciliary vasculature are supplied primarily by branches of the anterior ciliary arteries and veins that traverse the anterior sclera, it is not surprising that these same vessels are involved with the hyperemia and exudation that occurs with irritated anterior uveal vasculature.

Chemical mediators for these vascular responses include histamine, serotonin, plasmin, kinins, complement, and the eicosanoids (prostaglandins and leukotrienes). Knowledge of the exact substances involved in these inflammatory responses facilitates the development of specific or targeted therapy.

Episcleral Vascular Response

With acute and chronic ocular hypertension, the episcleral veins become enlarged. It is important to distinguish the deeper episcleral vessels from the more superficial conjunctival blood vessels (Figure 3.7). Episcleral blood vessels have a larger diameter, do not branch, are not mobile with conjunctival movements, and do not rapidly blanch to topical 1–2% epinephrine (they will vasoconstrict, however, in a few minutes rather than seconds for the conjunctival vessels).

Corneal Changes

Edema

To remain clear, the cornea must be slightly dehydrated (detumescence). This state is normally maintained by an energy dependent Na^{2+}/K^{+} pump within the corneal endothelium. With damage to the endothelium, such as with surgery or iridocyclitis, this endothelial pump is impaired and edema develops within the corneal stroma. The edema interrupts the orderly arrangement of the corneal stromal fibers and glycosaminoglycans causing the cornea to appear cloudy or bluish in color. As this corneal opacity is made up of extracellular fluids for the most part, topical applications of hyperosmotic agents (such as 2–5% NaCl or glucose) can temporarily reduce this edema, and permit improved visualization of the inner ocular structures. When significant amounts of fluid accumulate in the cornea, it can coalesce into superficial microbullae, which often rupture and create superficial corneal ulcers (Figure 3.8).

Corneal Vascularization: Different Types and Their Significance

The cornea in health appears perfectly clear. Nearly any pathologic process adversely affects corneal transparency. Loss of the epithelium, invasion of the stroma with inflammatory cells and blood vessels, and edema cause the cornea to loss its transparency and appear cloudy to nearly opaque. The cornea is normally devoid of blood vessels. However, with corneal diseases and anterior uveitis (iridocyclitis), vascularization of the cornea can occur starting from its periphery. The more serious or prolonged the insult, the more extensive the corneal vascularization. As these corneal vessels are directed toward the area of the insult, localization of the corneal vessels within the cornea can assist in determining the site and depth of the insult.

Corneal Vascularization

Corneal vessels are divided into superficial and deep, depending on their source and their depth within the cornea. Superficial corneal blood vessels arise from the limbal and conjunctival blood vessels, and extend into

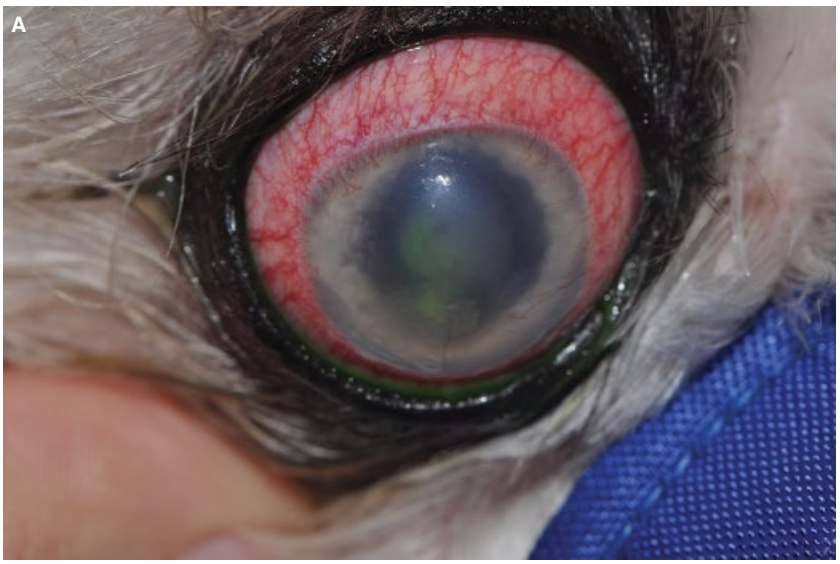






Figure 3.7 (A) Combination of episcleral venous congestion (associated with elevated intraocular pressure) and ciliary flush (associated with iridocyclitis) in a Shih Tzu with uveitis, secondary glaucoma, and a corneal ulcer. (B) Combination of episcleral venous congestion (associated with elevated intraocular pressure) and ciliary flush (associated with iridocyclitis) in a Beagle with lymphoma. (C) Episcleral injection in an American Cocker Spaniel with primary glaucoma and a subluxated lens.



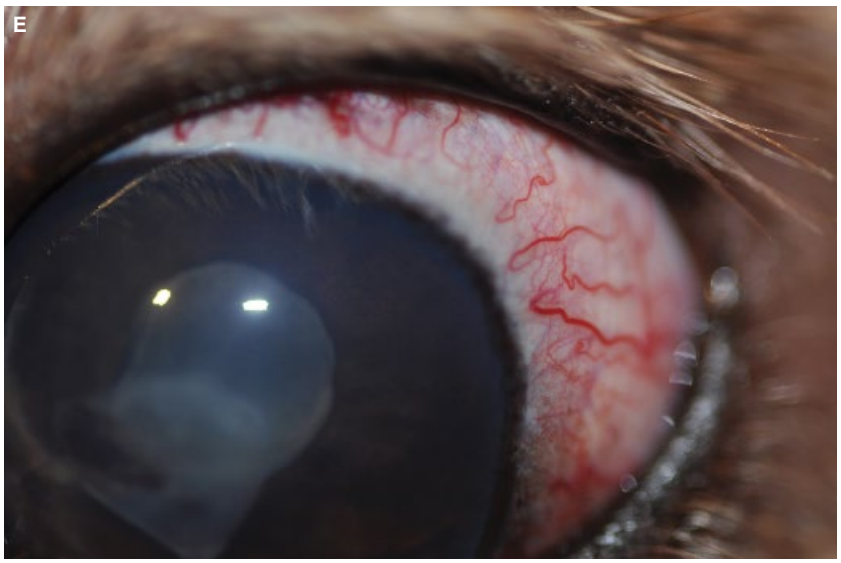
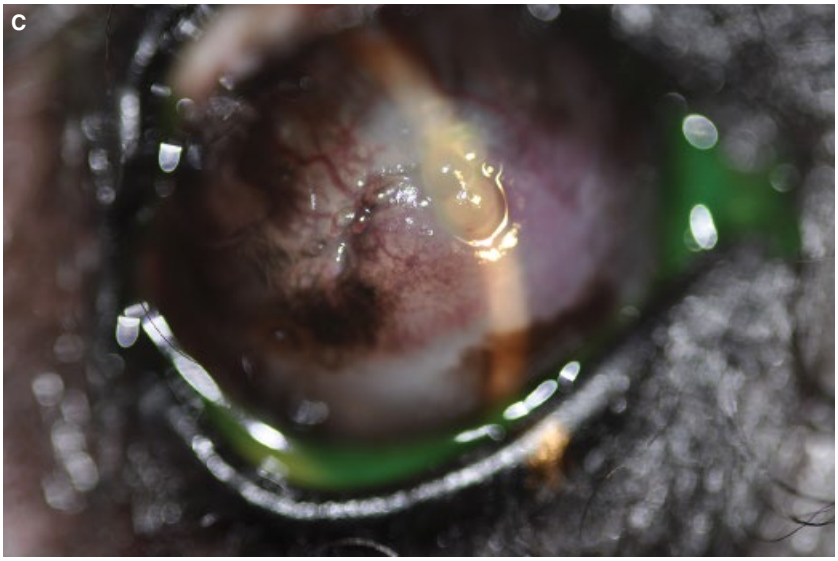


Figure 3.7 (*Continued*) (D) Episcleral injection in a Bassett Hound with primary glaucoma and a subluxated lens. (E) Episcleral injection in a Golden Retriever with pigmentary uveitis and secondary glaucoma. Note the diffuse corneal edema and the fibrin clot in the pupil. There is posterior synechiae as well.



Figure 3.8 (A) Corneal edema accompanies glaucoma and iridocyclitis in a horse.





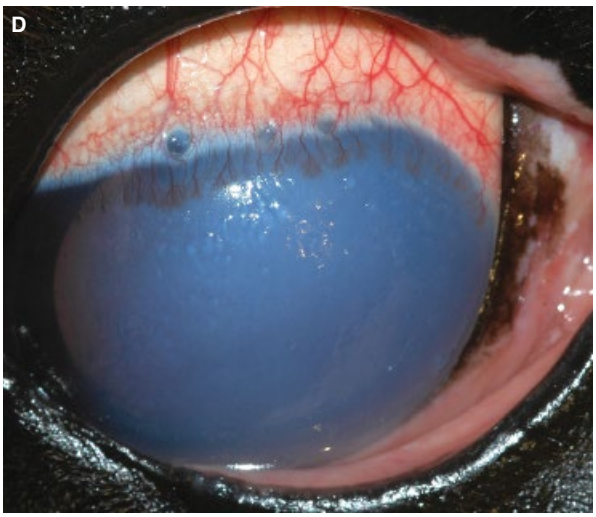


Figure 3.8 (*Continued*) (B) Generalized corneal edema can also occur when the "endothelial pump" is impaired, and fluid accumulates within the stroma as in endothelial dystrophy in this old dog. (C) Corneal edema accompanied by bullae (vesicle or blister) in a horse. This cornea has been chronically irritated and inflamed. Note the vascularization and pigmentation. (D) Diffuse severe corneal edema in a horse with multiple small bullae. There is also vascularization entering the cornea along its dorsal margin.

the cornea within the superficial stroma. Their presence signals surface or superficial corneal disease. They appear as large vessels with many variable sized, often dichotomous, branches (tree-like). Once the corneal disease has resolved, these vessels may remain as "ghost vessels" capability of re-canalization rapidly if another insult occurs. These nonperfused vessels can be visualized with high magnification (biomicroscopy).

Deep corneal blood vessels develop from the anterior uveal vasculature and are much smaller than superficial corneal blood vessels. These signal concurrent inflammation of the anterior uvea or iris and ciliary body. These vessels are very fine ("paintbrush"), and can invade the cornea for several millimeters (Figure 3.9).

As the presence of corneal vasculature causes some reduction in corneal transparency, the medical control of corneal blood vessels, either to stimulate their development (angiogenesis) or to cause their regression, is a major aim of pharmacological research.

Corneal Pigmentation

Of our animals species, the dog's cornea is the most apt to develop pigmentation in response to low-grade chronic insults, such as chronic keratoconjunctivitis sicca (Figure 3.10). There are various sources and sites of corneal pigmentation: (i) superficial (within the epithelium arising from corneal epithelium and/or wandering melanocytes); (ii) superficial to mid stroma (often associated with superficial corneal blood vessels from the limbus and corneal periphery); and (iii) corneal endothelial (migrate from iridal tissue following iris prolapse or anterior synechia). Both the cat and horse also demonstrate corneal and conjunctival pigmentation, but the extent of the pigmentation is less than in the dog.

Corneal Cellular Infiltrate

Cellular infiltrate within the cornea stroma is a significant sign of inflammation and often indicates the presence of infection. White blood cells migrate through the corneal collagen lamellae and coalesce at the site of disease (Figure 3.11).

Corneal Sequestration

Corneal sequestration or foci of corneal stromal degeneration and necrosis occurs primarily in cats (Figure 3.12). The origin of the pigmentation is still unresolved; iron and melanin have been proposed. There seems to be two types of sequestra in the different animal species. In cats, corneal degeneration has been associated with chronic irritation, entropion, and corneal disease associated with feline herpesvirus (FHV-1). Observed in its earliest stage, it appears as faint brown pigment beneath an intact

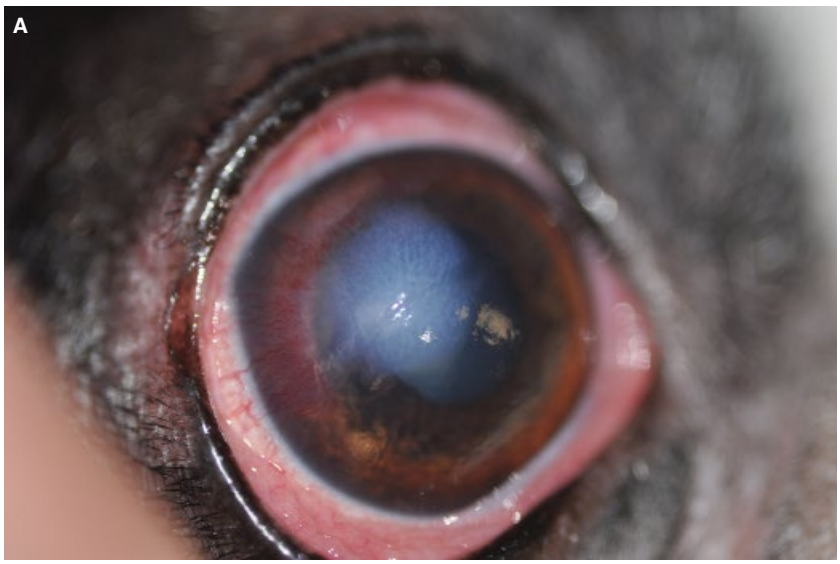
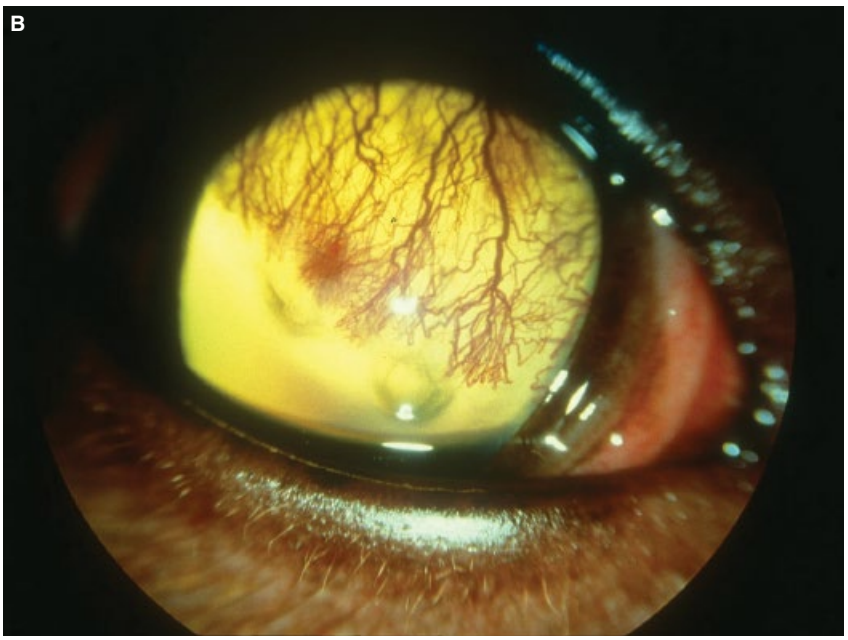


Figure 3.9 (A) Superficial corneal ulceration in a Boxer dog with edema and superficial blood vessels approaching the ulcer. These superficial corneal vessels invade the corneal stroma at the rate of about 0.6–1.0 mm/day and this rate can be influenced by the intensity of the insult and the effects of medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs).



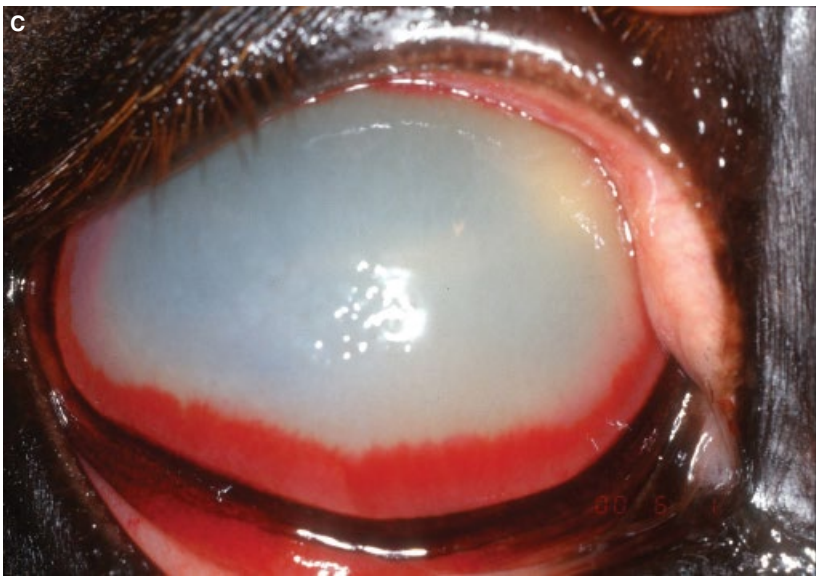
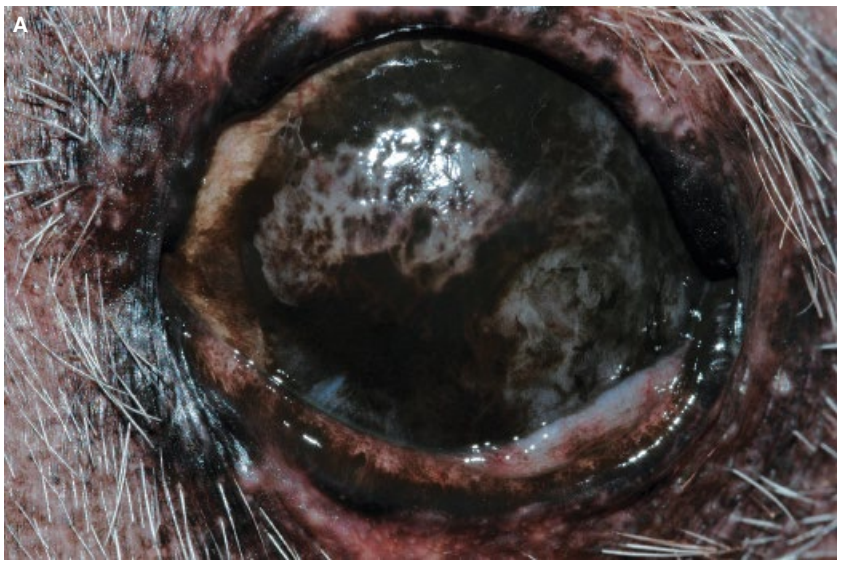


Figure 3.9 (*Continued*) (B) Superficial corneal vascularization in a dog associated with chronic keratoconjunctivitis sicca and a ventral paraxial corneal ulcer. (C) Deep corneal vascularization associated with corneal stromal abscess (at the 2 o'clock position) and secondary iridocyclitis in a horse. The deep corneal vascularization is located primarily in the deep corneal stroma and signals disease of the iris and ciliary body. These vessels are very fine and shorter.

corneal epithelium which gradually enlarges and becomes more dense brown–black, dislodges the overlying corneal epithelium (i.e., corneal ulceration), and stimulates superficial corneal vascularization and inflammation. Some superficial corneal sequestra can spontaneously slough; others are deep and extend to Descemet's membrane and require excision and often some type of reconstructive corneal graft. The second type of sequestra can occur in other species (i.e., horse and cow) and appear as a large area of the exposed and dried corneal stroma following chronic corneal ulceration and/or loss of eyelid function (with facial nerve paralysis).



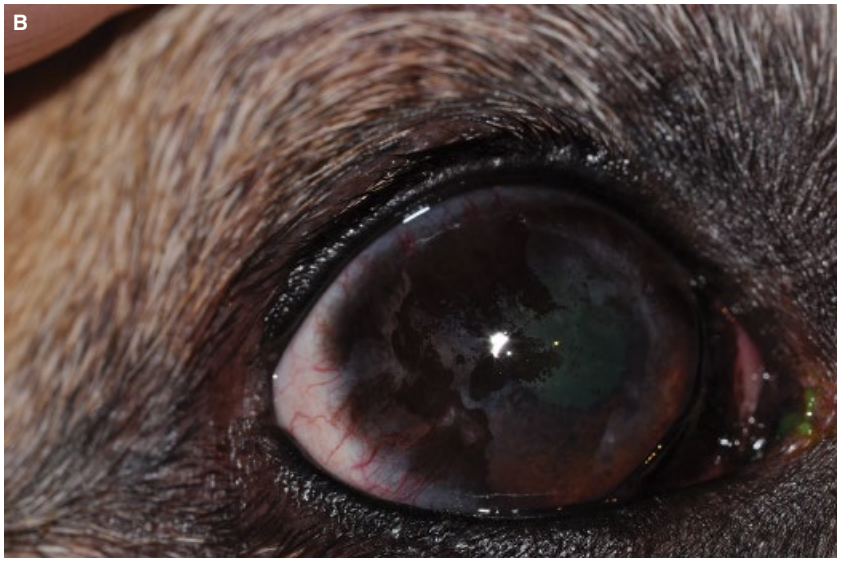


Figure 3.10 (A) Corneal pigmentation that is progressing in a Pekingese secondary to lagophthalmia. New pigmentation is developing in the more central cornea with corneal vascularization, and "older" pigmentation is in the peripheral cornea. Pigmentary keratitis signals low grade chronic irritation as with distichiasis, trichiasis, entropion, ectropion, ectopic cilia, and other insults. (B) Corneal pigmentation with invading vasculature in chronic superficial keratitis (pannus) in a German Shepherd dog.

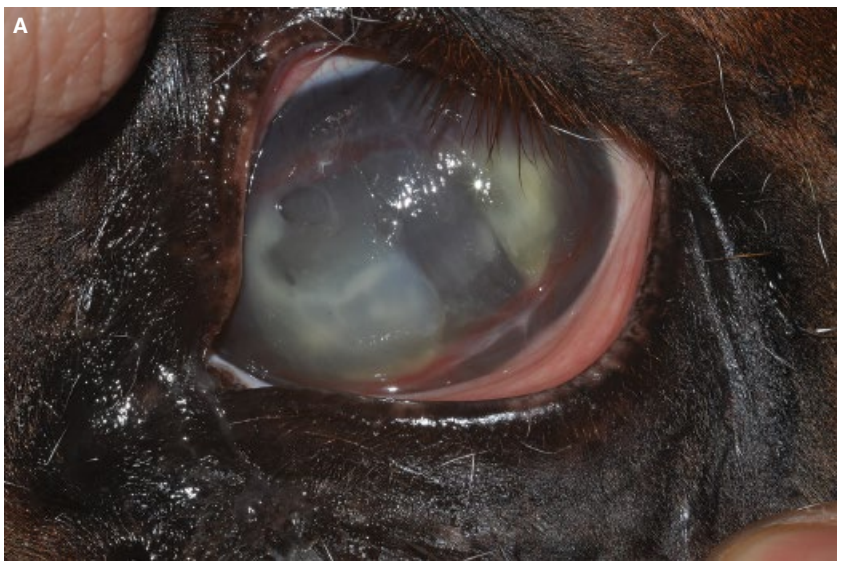


Figure 3.11 (A) Cellular infiltrate within the cornea appears white to yellow in color in an infected corneal ulcer in a horse.

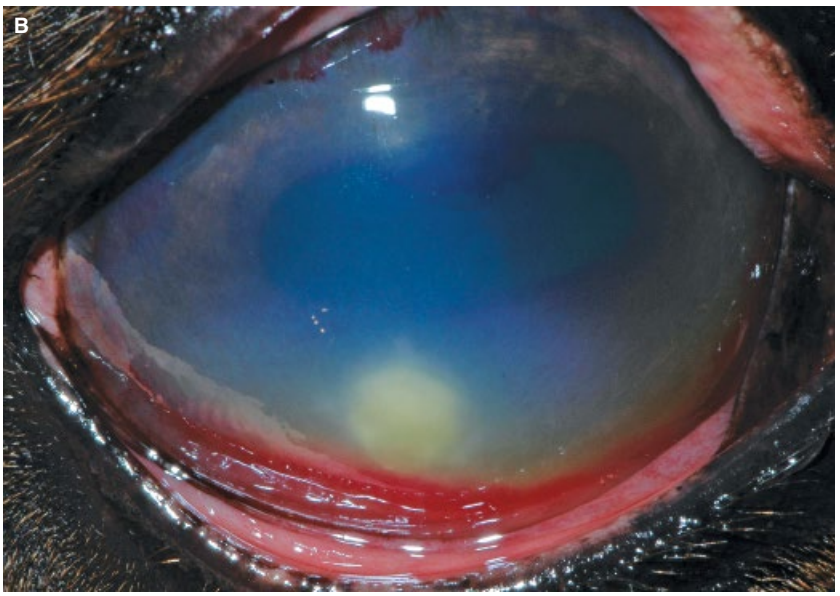




Figure 3.11 (*Continued*) (B) In this example of a stromal abscess in a horse, the infiltrate surrounds a fungal pathogen. Note the deep corneal vessels, the surrounding edema, and the miosis which signals concurrent uveitis. (C) Cellular infiltrate in a cat with stromal keratitis, an immune-mediated form of corneal inflammation.

Corneal Scarring

Once the orderly arrangement and the fixed distances between corneal stromal fibers and extracellular glycosaminoglycans are disturbed, corneal scarring results (Figure 3.13). In contrast to the fairly homogenous edema with fuzzy and indistinct margins, the white corneal scarring has sharp edges and can occur at any depth within the cornea. Often, corneal blood vessels are interspersed, suggesting the disease process is still active and possibly responsive to medical and/or surgical therapy. Pigmentation, calcification, and lipid deposits may also be eventually deposited in corneal scars.

Corneal Dystrophy and Degenerations

Corneal degenerations are unilateral or bilateral and are not inherited. They have associated corneal vasculature and often some other primary corneal or ocular disease is present. In contrast, corneal dystrophies

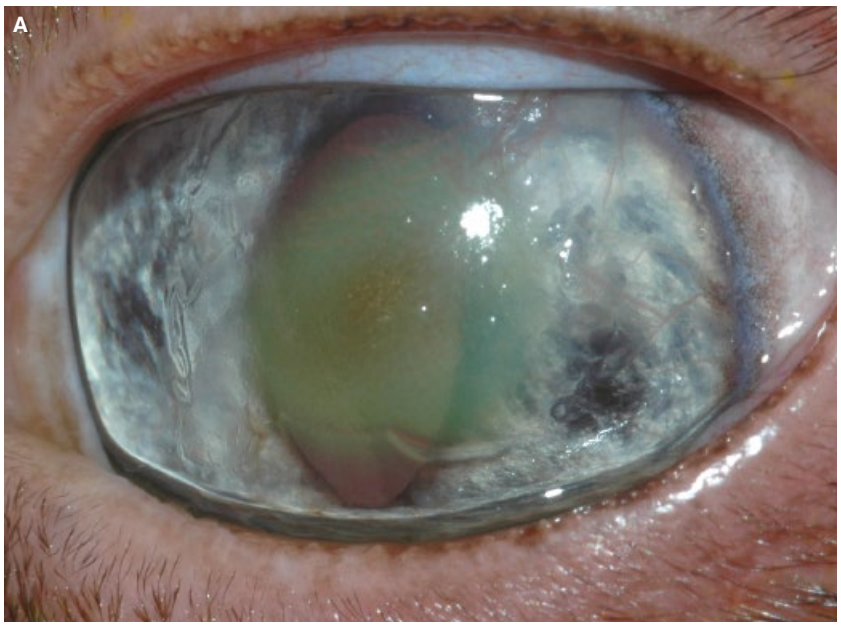






Figure 3.12 (A) The first suggestion of the development of a sequestrum in the cat is a very faint light brown lesion within the anterior stroma. (B) As the sequestrum increases in size and density, superficial corneal vascularization begins to surround the lesion, often with signs of superficial keratitis. The epithelium overlying the sequestrum is eroded. Fluorescein stain is not usually retained by the sequestrum but it may stain the edges of the sequestrum and the surrounding inflamed cornea. (C) This sequestrum extends to nearly Descemet's membrane and has incited an intense keratitis. Surgical removal with some type of reconstructive graft (conjunctival flap or corneoconjunctival transposition) is indicated.

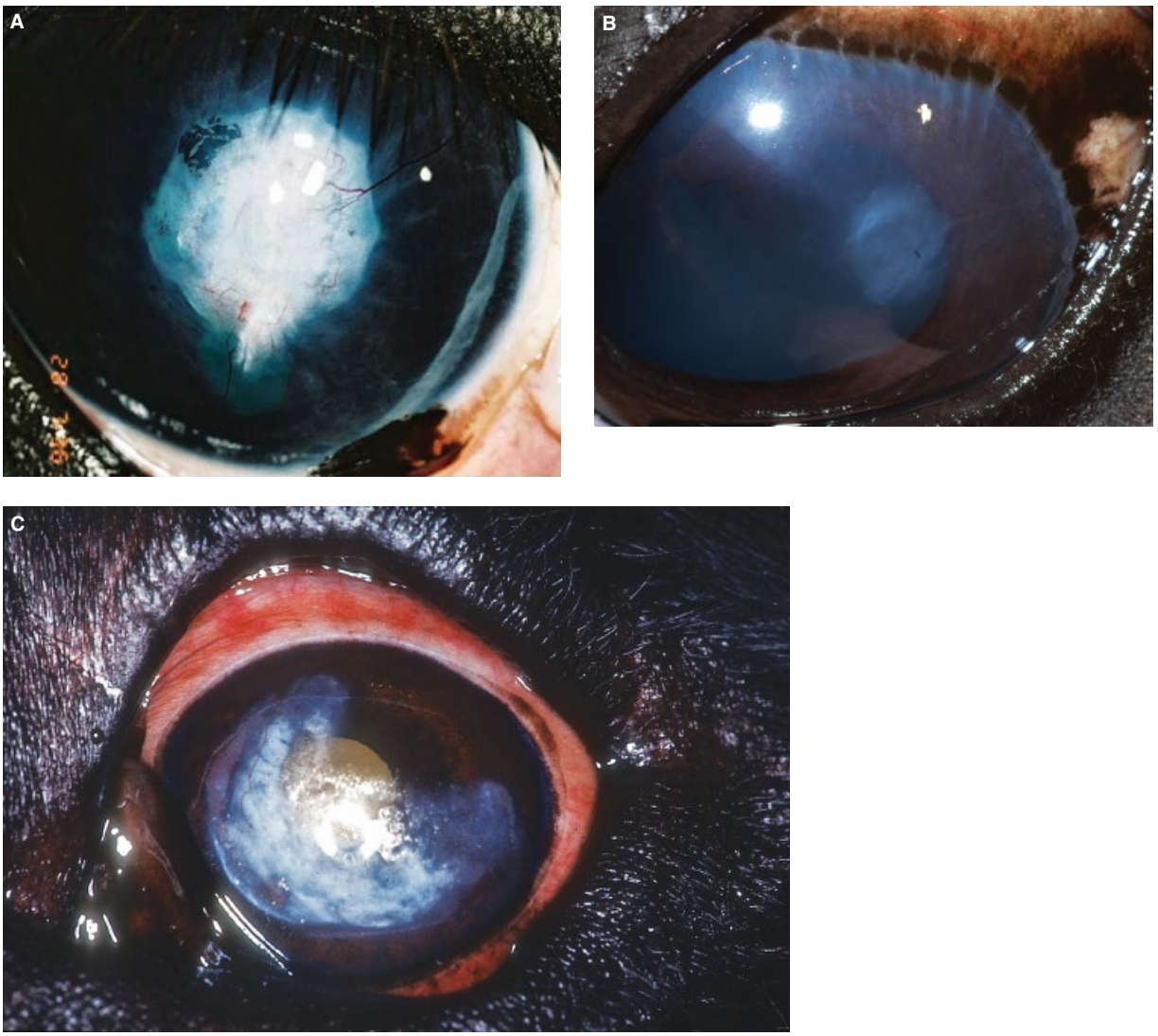


Figure 3.13 (A) Corneal scarring (fibrosis) occurs when inflammation, neoplasia, or other causes disrupt the normal organization of the corneal lamellae preventing light from penetrating the corneal tissue. New and active scars are usually vascularized but those completely healed usually have no obviously perfused blood vessels and are level or even slightly depressed from the surrounding corneal surface. (B) Corneal fibrosis following corneal transplantation to remove a deep stromal abscess in a horse. The dense linear foci of fibrosis adjacent and perpendicular to the limbus are scars from suture tracks. The paraxial area of fibrosis in the temporal cornea is the original site of the abscess and subsequently the donor corneal graft which has incorporated into the host tissue. (C) Deep corneal scarring and lipid degeneration in a puppy following a full-thickness corneal and anterior lens capsular laceration secondary to a cat's claw.

occur in both eyes, can affect the corneal epithelium, stroma, or endothelium, are usually devoid of vasculature, and can be breed-related. In the dog, a large number of primary, usually stromal, corneal dystrophies occur, and these deposits usually consist of triglycerides and cholesterol. These deposits appear as white focal crystalline refractive opacities. The actual depth of the area of cornea affected is often breed-specific (i.e., epithelial; anterior stroma, mid-stroma, and deep stroma; and endothelial).

Corneal degenerations are generally secondary to other corneal or other systemic diseases, and often unilateral or asymmetric if bilateral. Histologically, degeneration appears different from the dystrophies, showing evidence of previous inflammation or trauma, prior to the deposition of fatty or mineral substances.

Calcification is much less common than corneal lipidosis/cholesterosis, and seems more frequent in microphthalmic globes, such the Collie eye anomaly or the homozygous merle dogs with microphthalmia, scleral colobomas, cataracts, retinal dysplasia, and retinal detachments. The opacities that cause calcification appear much finer, gray rather than white, and often situated in the central superficial corneal stroma.

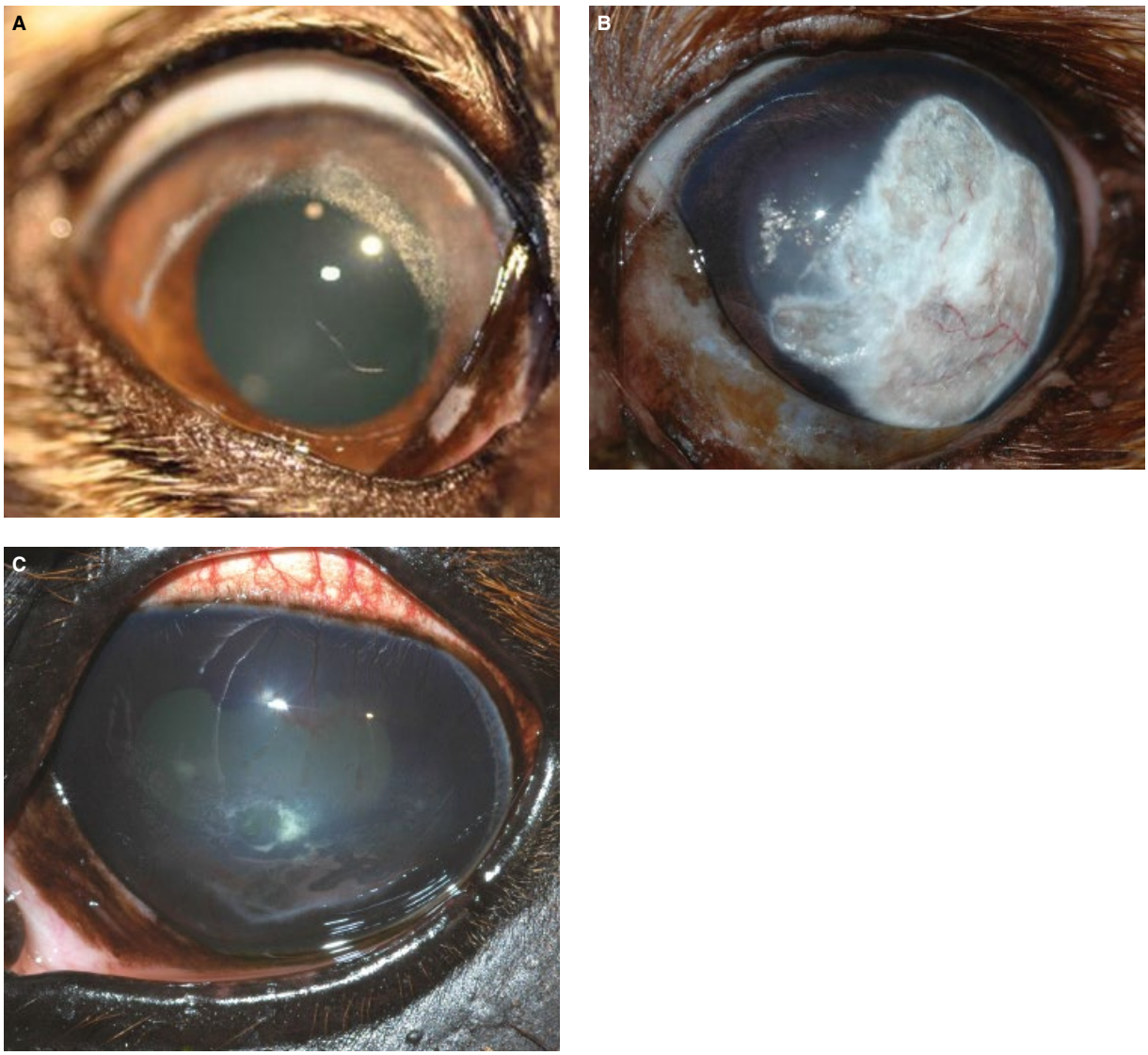


Figure 3.14 (A) Corneal lipidosis is associated with many inherited primary corneal stromal dystrophies in dogs as well as degenerations secondary to other corneal and intraocular diseases. The bilateral lipid deposits appear as discrete white opacities usually within the anterior corneal stroma. Vascularization may or may not be visible. (B) A severe case of corneal lipidosis in a dog. (C) Calcification of the cornea is much less frequent that lipidosis, and can occur simultaneously. Calcification appears as minute white to gray deposits within the corneal stroma. It can occur as the result of chronic inflammation or chronic medication administration. This horse has equine recurrent uveitis and the mineral is forming a band in the axial cornea where the palpebral fissure opens. Blood vessels are approaching the mineral deposits.

Corneal endothelial degeneration and loss can result in progressive corneal edema, usually first detected in the central or temporal cornea. Affected dogs are usually over 10 years of age. The endothelium naturally loses viable cells during aging, but excessive cell loss, either from damage (degeneration as the result of inflammation or trauma) or programmed cell death (endothelial dystrophy is an inherited condition), which causes cell densities <1000 cells/mm^2, will result in corneal edema and thickening (Figure 3.14).

Hemorrhage and the Eye

Hemorrhage in the ophthalmic structures often indicates serious disease: trauma; inflammations (often secondary to systemic disease); neoplasms of the uveal tract; retinal inflammations and detachments; and systemic diseases (i.e., neoplasms, bleeding diatheses, systemic hypertension). Determination of the exact site of the hemorrhage(s) is critical in developing prognosis and therapy. Hemorrhage can occur in any tissue of the eye

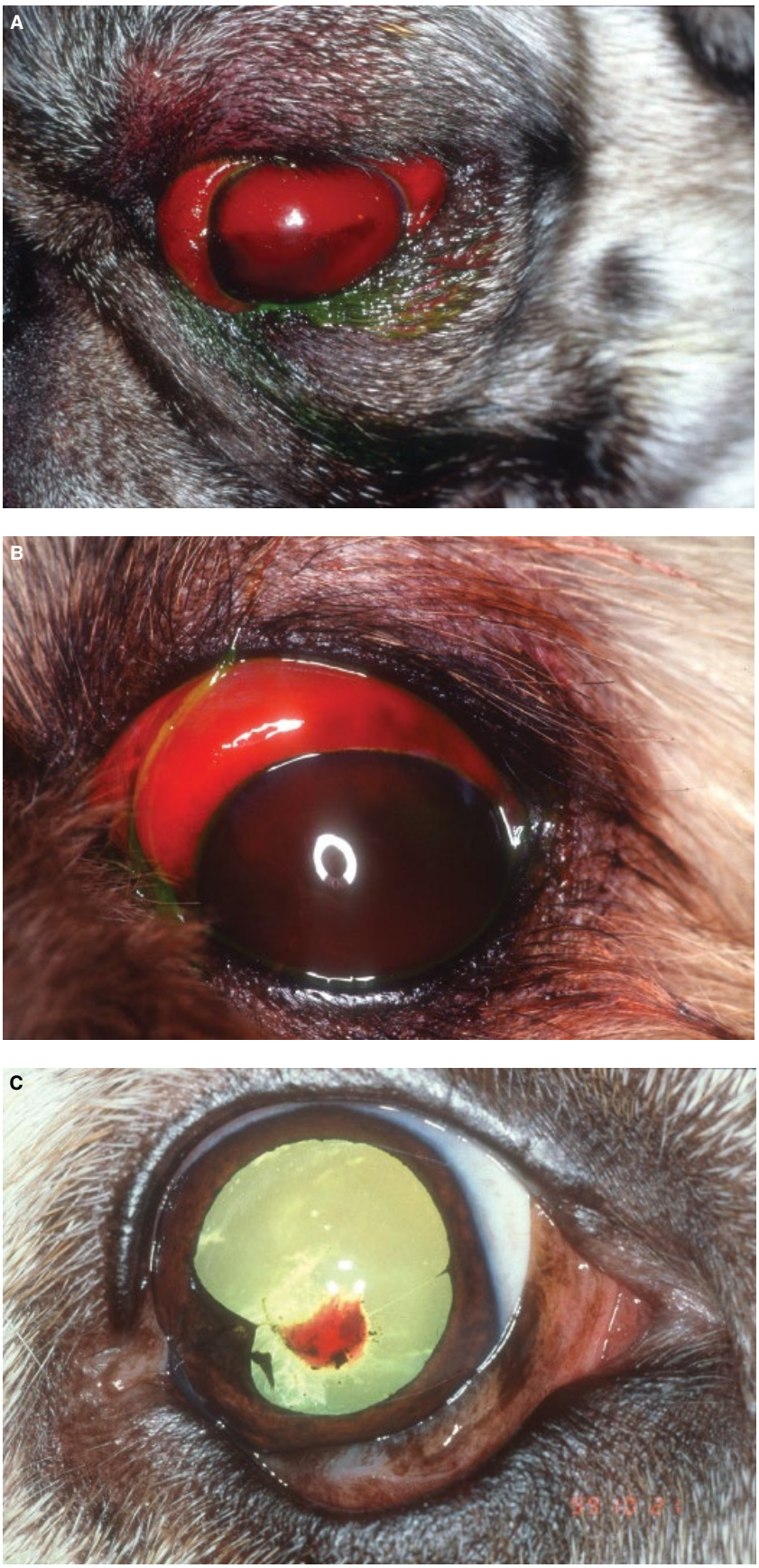
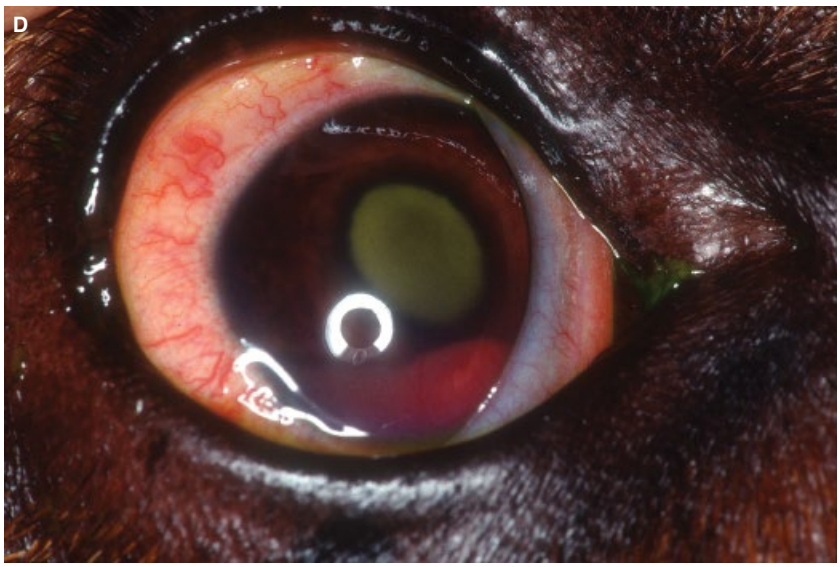
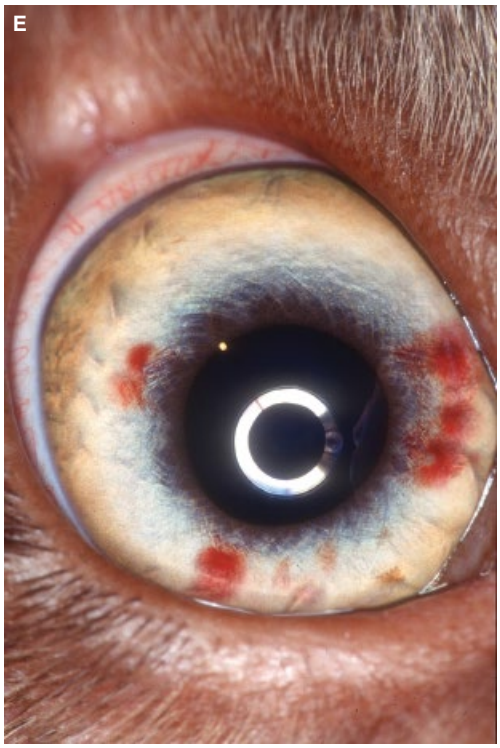


Figure 3.15 (A) Orbital hemorrhage secondary to trauma in a dog. The combination of blood in the orbit, subconjunctival tissues, and anterior chamber (hyphema) usually indicates considerable damage to the entire eye. (B) Subconjunctival hemorrhage can occur secondary to several blood clotting disorders, and simple trauma to the face in a dog. Hyphema is present concurrently. (C) Hyphema following laser photocoagulation for an iridal melanoma in a dog.





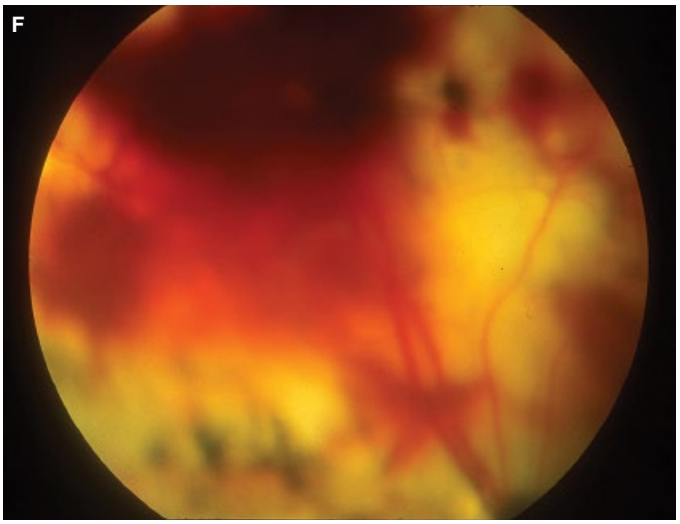


Figure 3.15 (*Continued*) (D) Hyphema associated with systemic lymphoma in a dog. (E) Iridal hemorrhages associated with systemic disease (Rocky Mountain spotted fever) in a dog. (F) Vitreal and intraretinal hemorrhages in systemic hypertension in an aged cat.

(Figure 3.15). Important considerations when hemorrhage is detected include: ocular tissue involved; relative volume; color (red indicates fresh hemorrhage while purple to brown indicates old hemorrhage); displacement and impact on ocular tissue(s); and clot formation (suggests hemorrhage has stopped).

Opacity in the Anterior Chamber (Flare, Hypopyon, and Fibrin)

The eye, like the brain, has highly selective barriers to block most substances, proteins, inflammatory and neoplastic cells, and erythrocytes from entering the ocular

tissues. The major ocular barrier is the blood–aqueous barrier, present in the iris and ciliary body (anterior uvea) which consists of highly specialized cell gap junctions (puncta adherentes) between the pigmented and nonpigmented ciliary body epithelia. In contrast, the posterior uvea or choroid vessels are quite "leaky," and are the major site for escape of red blood cells and plasma in systemic hypertension contributing directly to retinal hemorrhages and detachment. The second major ocular barrier is the blood–retina barrier consisting of the retinal blood vessel walls, including the retinal capillaries. Both of these barriers, blood–aqueous and blood–retina, as well as the transcorneal barrier, can be significant impediments for antibiotics and most other ophthalmic drugs to reach intraocular tissues in therapeutic concentrations (Figure 3.16).

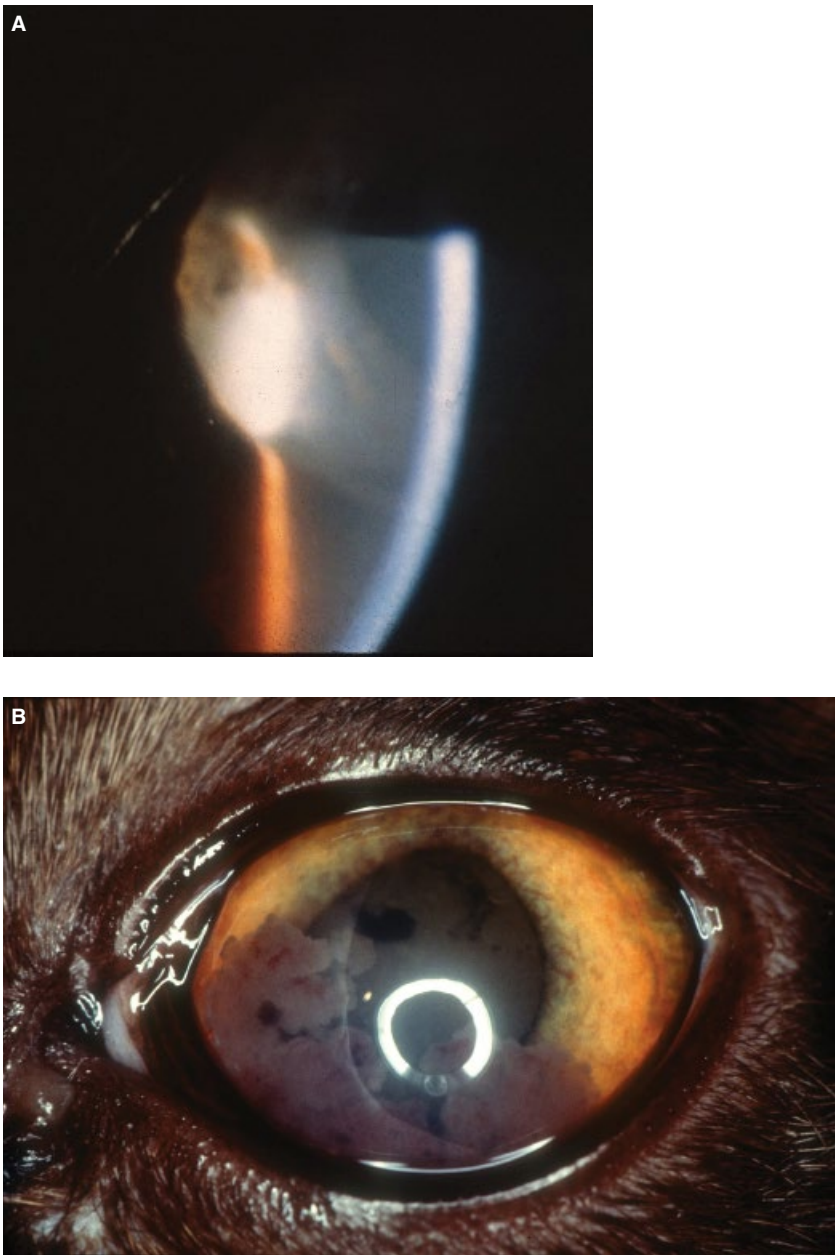
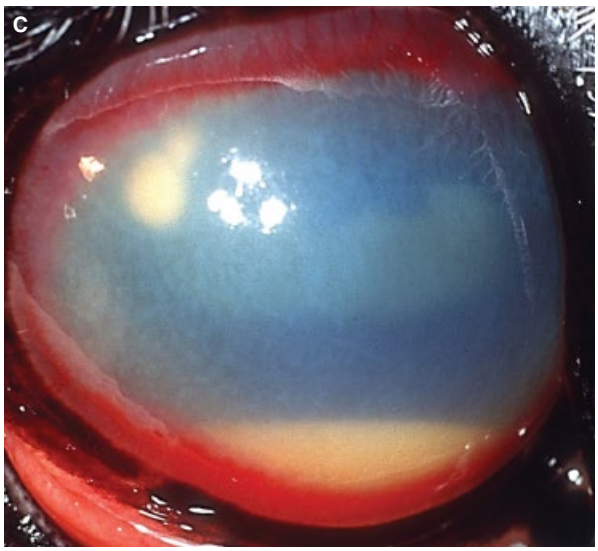


Figure 3.16 (A) A slit beam image demonstrating the Tyndall effect, which occurs when a light beam reflects from proteins and cells in the aqueous humor, resulting in a translucent to opaque media. (B) Feline infectious peritonitis is a good example of anterior chamber infiltration by large numbers of inflammatory cells including plasma cells, macrophages, and lymphocytes which "clump" together and adhere to the posterior cornea and anterior lens capsule. Sometimes red blood cells are also incorporated in these keratitic precipitates rendering them pink or red.



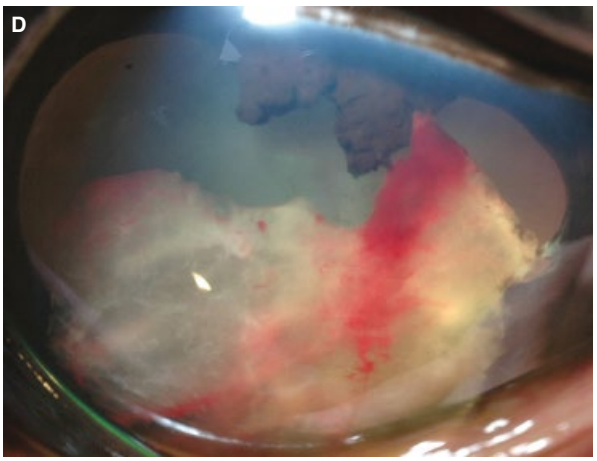




Figure 3.16 (*Continued*) (C) Hypopyon in the horse and an intense iridocyclitis secondary to a paraxial stromal abscess. (D) Fibrin in the anterior chamber of a horse with uveitis. Some hemorrhage is also present. (E) Fibrin in the anterior chamber of a horse with a corneal perforation.

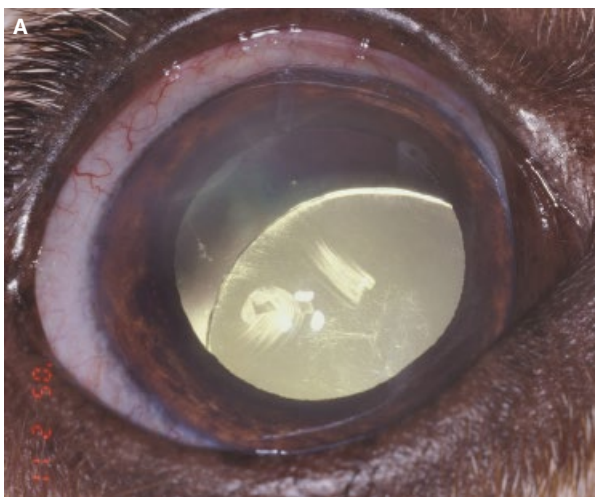


Figure 3.17 (A) Pupil size is usually increased (mydriasis) in glaucoma when intraocular pressure is elevated. This Bassett Hound has primary glaucoma and lens subluxation.

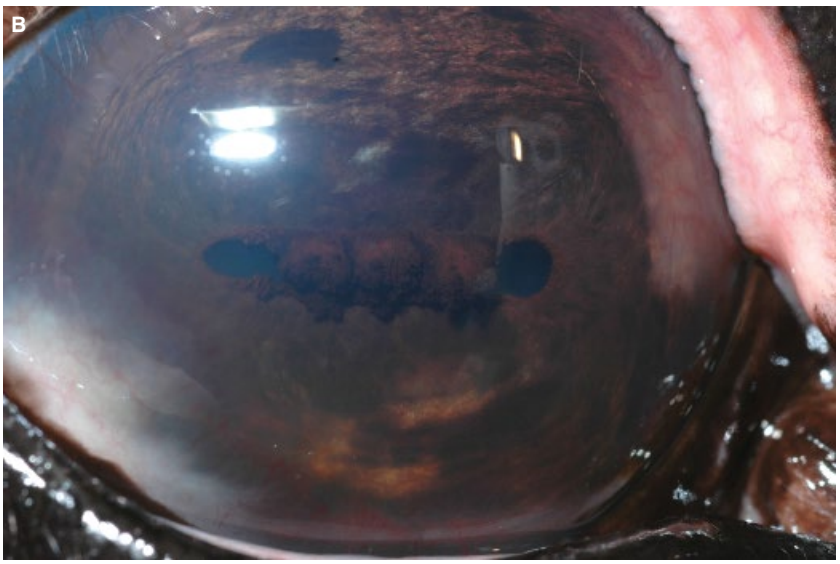


Figure 3.17 (*Continued*) (B) Pupil size is reduced (miosis) with anterior uveitis or iridocyclitis. This horse has anterior uveitis secondary to a corneal abscess (ventrolateral paralimbal cornea at 8 o'clock). Miosis is the hallmark sign of uveitis.

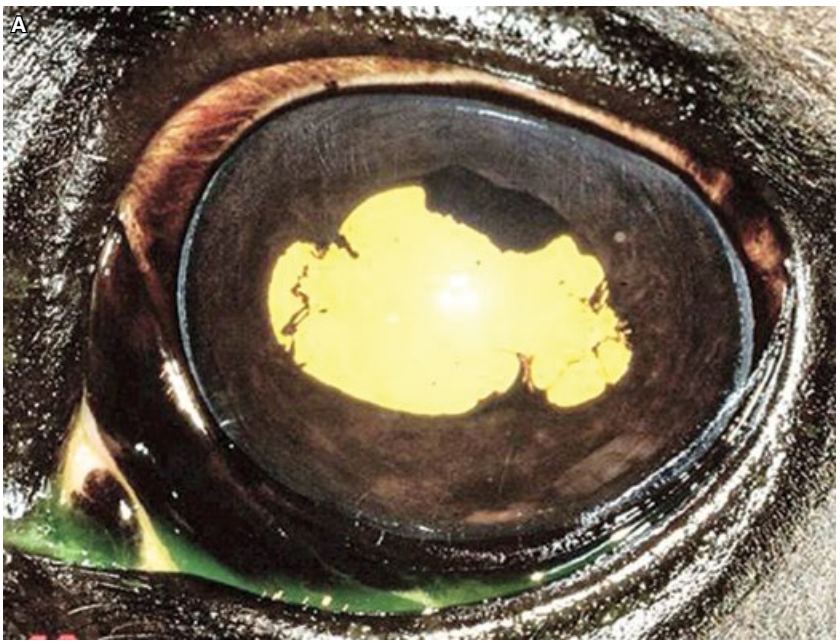
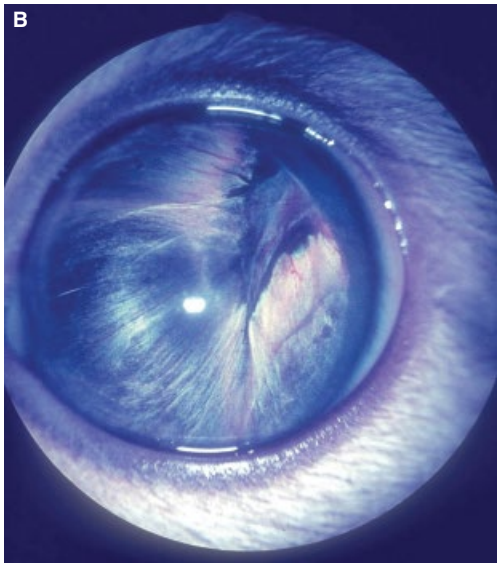


Figure 3.18 (A) Pupil shape can be very irregular with adhesions (posterior synechiae) between the posterior iris and anterior lens capsule as with chronic anterior uveitis in a horse.

Changes in the Pupil

Pupil size varies markedly, depending on background illumination, temperament of the animal, intensity of illumination used to stimulate the pupil, and presence of eye disease. Fortunately, in the normal state, most patients have relative equal pupil size, and the observer can compare one pupil size with the opposite (fellow) eye. A small pupil (miosis), combined with other findings, can signal the irritation of the iridal sphincter muscle that occurs with iridocyclitis. A large pupil can suggest problems with the iridal sphincter musculature, iris atrophy, or ocular hypertension (glaucoma) (Figure 3.17). The combination of glaucoma and iridocyclitis usually results in a mid-range pupil.



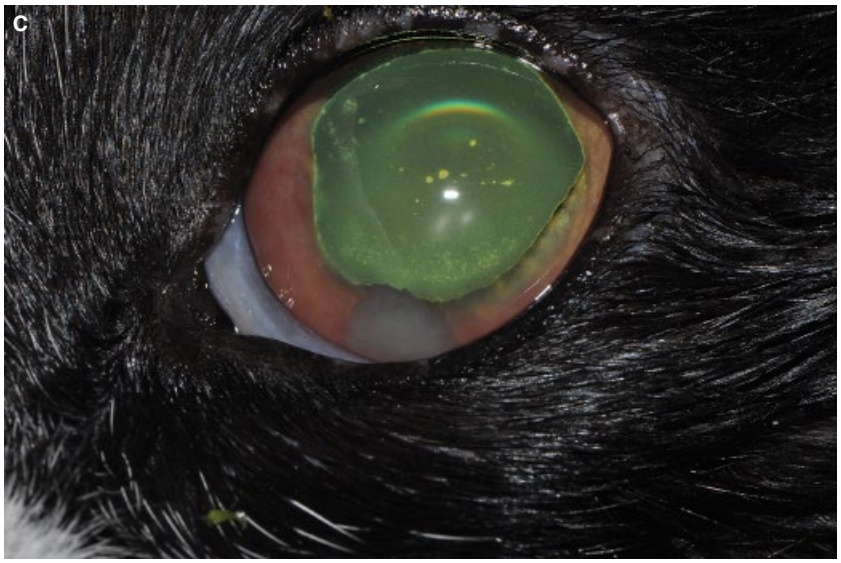


Figure 3.18 (*Continued*) (B) The pupil margins of the iris can adhere together (annular posterior synechiae) and completely obstruct the pupillary flow of aqueous humor as in the cat with anterior uveitis. This condition is termed iris bombé. (C) In this cat with anterior uveitis, the dyscoria is the result of posterior synechiae. The cellular infiltrate in the ventral anterior chamber are neoplastic cells.



Figure 3.19 (A) Rubeosis irides is the term given to the proliferation of new vessels or neovascularization on the iris surface. In this Siberian Husky with uveitis, the normally blue iris has turned red because of new vessels and congestion of existing vessels. Corneal vessels are present in this example as well.

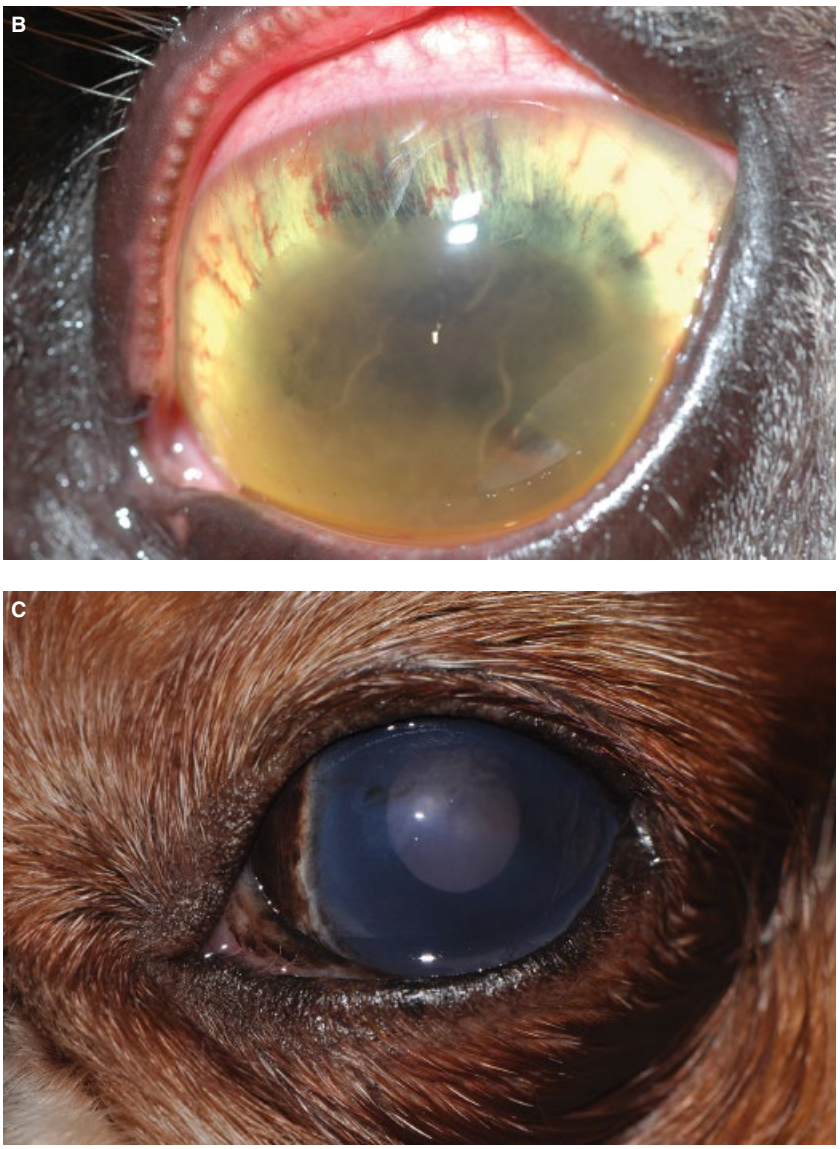


Figure 3.19 (*Continued*) (B) Uveitis can result in iris color change. In this horse with a normally blue iris, the stroma has turned yellow because of the leakage of proteins. Note, too, the new vessels on the iris surface and the fibrin clot in the anterior chamber which is obscuring visualization of the pupil. (C) Hyperpigmentation of the iris usually develops with chronic uveitis. In this dog, the iris is several shades darker brown than normal as the result of pigment proliferation during chronic uveitis. There is also diffuse corneal edema.

The veterinary clinician will encounter a variety of pupil shapes (round, oval, slit, and even square – four-sided), depending on the animal species. Abnormal pupil shapes can signal congenital or developmental anomalies, or posterior (iris to lens) or anterior (iris to posterior cornea) adhesions (usually post-inflammatory) (Figure 3.18). Pupil anomalies include corectopia (off-center pupil), corectasia (dilation), coloboma (notch in pupil), and polycoria (multiple pupils), which are frequently encountered in microphthalmic eyes.

With iridal swelling, the iris thickens and becomes sticky. In its normal position or when contricted (miosis) with iridal swelling, temporary to permanent attachments can develop on the central lens (which it contacts normally when constricted). Use of mydriatics to keep the iris mobile and to dilate the pupil so it does not adhere to the central lens (which it does not contact normally as the peripheral lens is too distant) are part of the therapy used for iridocyclitis.

Iris Color Change

This iris can change color in the presence of uveitis or a neoplastic proliferation within its stroma (Figure 3.19).

Inflammations of the Retina and/or Choroid

Inflammations of the retina and choroid are termed retinochoroiditis (assumes the retinal inflammation is first and then spreads to the deeper choroid; e.g., canine distemper and cryptococcosis) or chorioretinitis (initial

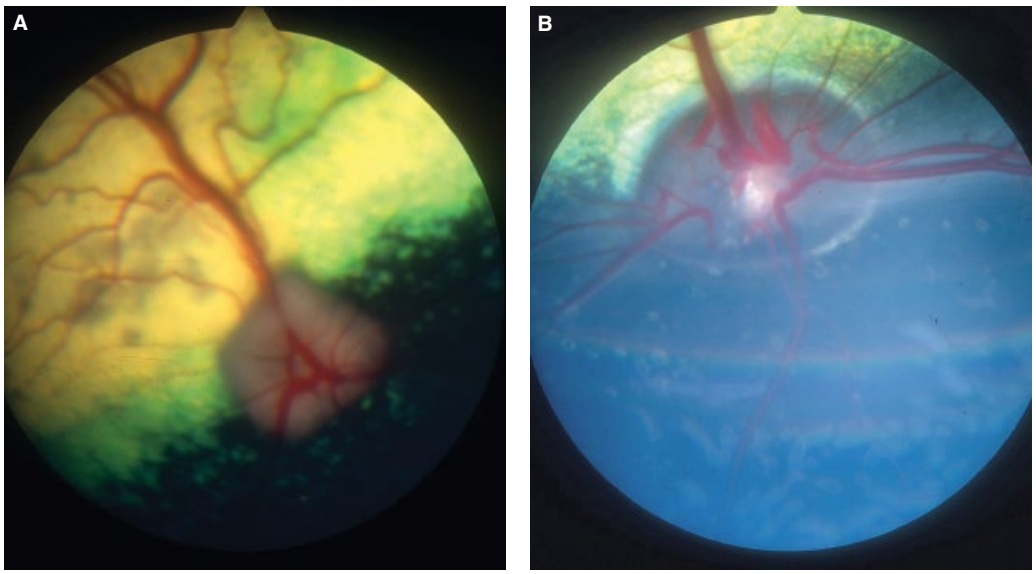


Figure 3.20 (A) Acute chorioretinal inflammations of the tapetal fundus appear as raised translucent to white opacities with irregular or fuzzy margins, often near retinal blood vessels as in this young dog with canine distemper. (B) Acute chorioretinal inflammation involving the nontapetal fundus is easier to detect, because of the dark brown to black underlying retinal pigment epithelium. The inflamed areas appear white raised areas with irregular margins as in this young bull with pneumonia.

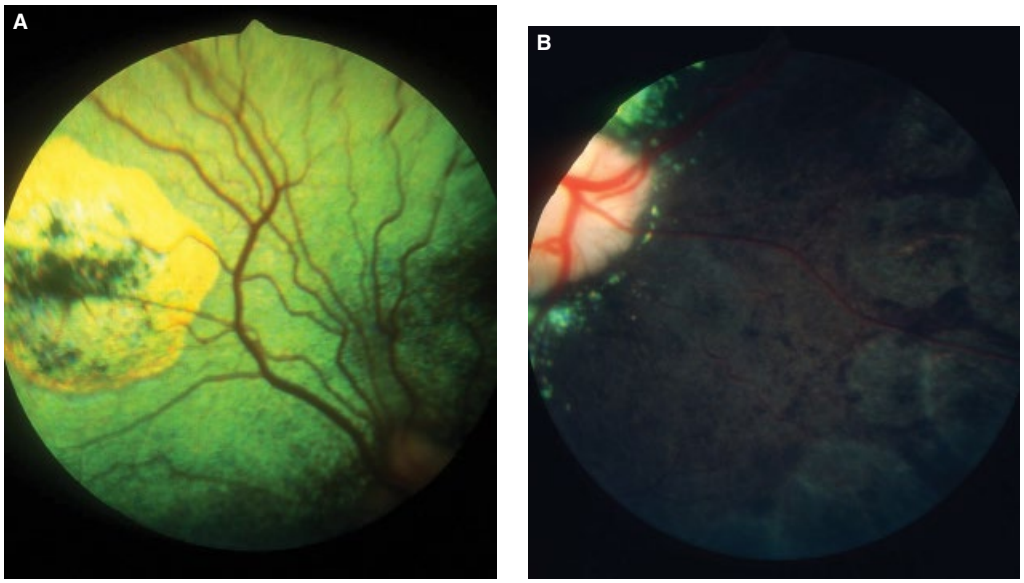


Figure 3.21 (A) Chronic chorioretinal inflammation (retinopathy/chorioretinopathy/retinal–chorioretinal scars) of the tapetal fundus can involve partial to full-thickness focal destruction of the retina and tapetal lucidum, and the resultant proliferation and migration of the retinal pigment epithelium. They appear as focal to diffuse hyperreflective areas with variable pigmentation, and sharp or smooth margins such as in this young dog with distemper. (B) Chronic chorioretinal inflammation of the nontapetal fundus appears as diffuse to focal gray to white areas with sharp margins, variable proliferation and migration of the retinal pigment epithelium, and changes in the retina blood vessels such as in this young dog with distemper.

inflammation is in the choroid and extends to the retina; e.g., feline infectious peritonitis and malignant catarrhal fever in cattle). Posterior ocular inflammations affecting both eyes are important manifestations of systemic diseases for which ophthalmoscopy can often provide diagnostic and prognostic information. As viewed ophthalmoscopically, the neurosensory retina appears as transparent tissue with blood vessels traversing it. It lies atop the choroid and is intimately associated with it. The "eye shine" (seen best at night in many animal species) is usually yellow–green to blue and represents reflected light from the dorsal tapetal fundus (the tapetum is beneath the retinal pigment epithelium in the anterior choroid). The nontapetal fundus, generally the ventral aspect of posterior eye, lacks a reflective tapetum and appears brown to black because of the presence of pigment in the retinal epithelium. When edema, inflammation, detachment, or hemorrhages occur within the retina, these changes can be observed directly as the retina's transparency is altered. Retinal edema or chorioretinitis in the tapetal fundus appear as slightly gray and raised translucent areas while these same changes in the nontapetal fundus appear gray, yellow, or white, and are easier to detect. Active foci, edema, or inflammation usually have fuzzy and indistinct borders (Figure 3.20).

Healed or chronic chorioretinitis lesions have sharp margins, pigmentary changes by the retinal pigment epithelium and choroid (either pigment proliferation, migration, or loss), and damage to the reflectivity of the retinal blood vessels and tapetal layer (Figure 3.21). Retinal degenerations (thinning) in the tapetal fundus results in hyperreflective lesions.

4

Canine Orbit

The orbit consists of the bones that surround the globe, the orbital fat and fascial planes, the extraocular muscles, and the afferent and efferent nerves that supply the orbital contents or simply pass through the orbit to serve the nose or other fascial structures. The orbit of the dog is a not infrequent site for congenital, traumatic, inflammatory and neoplastic diseases. Variations in orbital conformation influence the types of disorders that affect dogs. Breeds with shallow orbits have very prominent globes that are subject to trauma and exposure (brachycephalic ocular syndrome, see Appendix B), while breeds with very deep orbits and enophthalmia have greater protection. Age is another consideration for etiology of disease. Young dogs tend to get into harm's way and various forms of trauma; older dogs, in general, represent the majority of patients with orbital neoplasia. Orbital disease may affect the orbital tissues within the extraocular muscle cone, the orbital tissues surrounding the extraocular muscles, and the periorbita (periosteum) and bones that delineate the orbit.

Congenital Orbital Anomalies

Microphthalmia is an eye that is abnormally small and may contain normal but reduced intraocular tissues (nanophthalmia), or more commonly true microphthalmia in which additional ocular abnormalities occur, especially cataracts and retinal dysplasia. The condition is the most common orbital anomaly in the dog and is observed most often in the Beagle, Akita, Chow Chow, Cavalier King Charles Spaniel, and Irish Wolfhound breeds.

The condition can usually be detected after the eyelids open (10–14 days postnatal) and is unilateral or bilateral. The palpebral fissure is smaller than normal, and the nictitating membrane protrudes to a variable extent. The cornea is smaller than normal. The pupillary light reflexes can be present or absent. Cataracts may be present, as in the Miniature Schnauzer. Retinal detachments/dysplasia can be present in the Bedlington Terrier, Yorkshire Terrier, Labrador Retriever, and other breeds. In the merle-colored breeds (merle ocular dysgenesis), such as the Australian Shepherd, Rough Collie, Shetland Sheepdog, and Dachshund, microphthalmia, iridal defects and heterochromia, equatorial staphylomas, cataracts, retinal dysplasia and retinal detachments frequently occur in the homozygous merle dogs with excessive white coats (Figure 4.1, see also Figure 10.2 and 18.1).

Severe microphthalmia usually causes vision loss, but mild or moderate microphthalmia may still permit reasonable functional vision. Progression of cataracts or retinal abnormalities can eventually impair vision. There is no treatment, and the possibility of inheritance is high in selected breeds of dogs.

Acute and Chronic Orbital Cellulitis

Orbital cellulitis is common in dogs, and tends to occur in younger animals (<5 years). Acute disease is usually diffuse without specific pockets of exudate (Figure 4.2). Occasionally, orbital cellulitis becomes subacute to chronic and inflammatory exudate will accumulate, and is termed, orbital abscessation, which is more resistant to antibiosis.

The history is usually an acute onset of eyelid and orbital swelling, pain upon opening and complete closing of the mouth, difficulty with mastication, and general malaise. Common clinical signs include unilateral orbital swelling, exophthalmia, strabismus (if the infection is more isolated or focal within the orbit), protrusion and hyperemia of the nictitating membrane, conjunctival chemosis and hyperemia and mucopurulent discharge. Corneal edema, miosis, and aqueous flare occur with severe disease. Palpation of the orbit is usually painful and attempts to fully open the mouth generate pain and sometimes aggression. An elevated leukocyte count (20,000–30,000) with neutrophilia (80–90%) and pyrexia are usually present.

Treatment requires systemic broad spectrum antibiotics and, if a discrete pocket of exudate is present, surgical establishment of drainage (usually behind the last upper molar tooth). Fine needle aspiration of the inflamed

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orbital tissue (ultrasound guidance is helpful) for cytology and microbial culture and susceptibility testing is recommended. Topical treatment includes broad spectrum antibiotics and mydriatics, if corneal disease and uveitis are present. If an impaired blink and exposure keratitis occur, a temporary complete tarsorrhaphy is recommended. Prognosis is generally good provided the infection responds rapidly to the systemic antibiotics. Enophthalmia, impaired ocular mobility (due to fibrosis), and optic nerve atrophy are infrequent sequelae.

The main differential diagnoses for acquired orbital disease include orbital neoplasia (usually slow onset; biopsy), eosinophilic/masticatory myositis and extraocular polymyositis (both conditions usually bilateral; muscle biopsy), zygomatic sialoadenitis and cysts, as well as sinus and upper tooth root infections.



Figure 4.1 (A) Bilateral but not symmetrical microphthalmia in a homozygous blue merle Australian Shepherd puppy. (B) Close up of microphthalmia with multiple ocular defects including heterochromia, corectopia (off center pupil), and basal iridal coloboma (atypical equatorial coloboma). (C) Australian Shepherd litter of puppies with 3 normal eyed blue merles and 3 excessively white blue merles with colobomatous microphthalmia (merle ocular dysgenesis). (D) Bilateral microphthalmia in a mixed breed puppy.



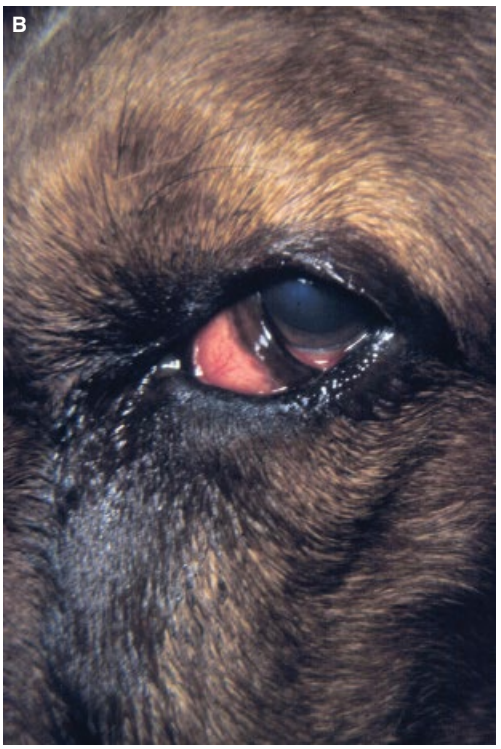






Figure 4.2 (A) Acute orbital cellulitis in a Basset Hound of three days duration. (B) Resolution of the condition several days later after therapy with high levels of systemic antibiotics, and topical antibiotics and 1% atropine. (C) German Pointer with unilateral retrobulbar abscess. (D) Oral view on a dog revealing swelling and hyperemia of the palatal tissue posterior to the last maxillary molar. This is an access point for sample collection and drainage of a retrobulbar abscess.

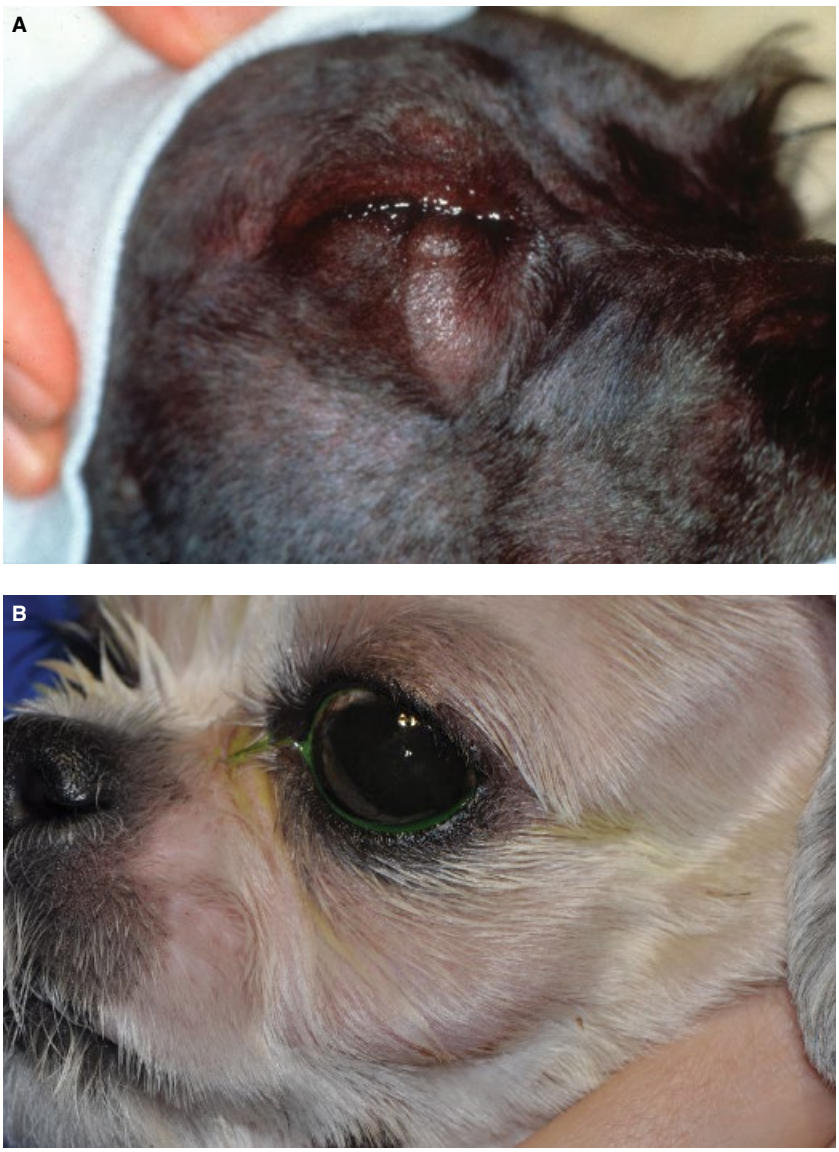


Figure 4.3 (A) Zygomatic salivary mucocele in large dog. The condition had progressed over several weeks. (B) Zygomatic salivary mucocele in a small breed dog. Note the swelling ventral to the globe. The eye was mildly exophthalmic and resistant to retropulsion.

Zygomatic Salivary Inflammations, Cysts & Mucoceles

Zygomatic salivary inflammations, cysts or mucoceles, and neoplasms affect the orbit because the gland is located in the ventroanterior orbit. With mucoceles, slow onset exophthalmia and protrusion of the nictitating membrane are the typical presenting signs (Figure 4.3). A fluctuating swelling can occur in the dorsolateral, ventromedial or ventral conjunctiva, or in the mouth, and attempts to open and examine the mouth do not cause discomfort. Diagnosis is confirmed with ultrasonography and aspiration of the contents of the mucocele. Recommended treatment is the surgical excision of the mucocele and the zygomatic gland.

Zygomatic adenitis and neoplasia appear less frequently. Both adenomas and adencarcinomas produce clinical signs similar to the mucoceles, but can be differentiated with ultrasonography. Surgical excision is recommended but borders of the mass are frequently difficult to identify during surgery. Exenteration with removal of all orbital tissues probably offers the best prognosis.

Masticatory or Eosinophilic Myositis

Masticatory or eosinophilic myositis appears as an acute disease that progresses to chronicity with inflamed muscles undergoing atrophy and fibrosis. Affected muscles typically include the temporalis, masseter, and pterygoid muscles. The disease appears to be immune-mediated and targets these specific muscles.

The acute disease appears with rapid onset of exophthalmia, protrusion of the nictitans, and swelling and pain of the temporalis muscles (Figure 4.4). Difficulty eating and completely closing the mouth occur early. As the disease progresses the inflammatory changes become more subtle, and marked atrophy of the temporalis muscles, enophthalmia, impaired ocular mobility occur, and the protuberances of the dorsal skull become prominent. Loss of body weight eventually results because the dog's mastication is impaired and food intake reduced. Eventually the ability to completely open the jaw is markedly reduced.

Diagnosis requires biopsy of the affected muscles. Leukocytosis with marked peripheral eosinophilia as well as increased levels of serum phosphokinase are usually present. Treatment of the acute condition includes immunosuppressive levels of systemic corticosteroids or azathioprine. Recurrences are common, and long-term therapy may be necessary.



Figure 4.4 Acute masticatory myositis in a German Shepherd dog. The swelling of the temporal muscles cause the bilateral exophthalmia and protrusion of the nictitating membranes.

Extraocular Polymyositis

Extraocular polymyositis tends to affect Golden Retrievers and Labrador Retrievers, although it can affect any individual (Figure 4.5). Females are more commonly affected. Presenting signs include bilateral exophthalmia, anterior globe displacement, very limited globe mobility resulting from involvement of all of the extraocular muscles, retraction of the upper eyelids, and congestion of the episcleral blood vessels. Histopathology of cases indicates predominate CD3 lymphocytic infiltrate in the muscles; in chronic cases some fibrosis, enophthalmia and strabismus of these muscles occurs. Systemic immunosuppressive agents are used to effect and are based on response.

Strabismus

Strabismus is the deviation of the globe from its normal orientation. It can result from a variety of causes, including congenital anomalies, traumatic injuries and immune-mediated conditions. Lateral strabismus post-proptosis is very common following injury or rupture of the medial rectus muscle. Restrictive fibrosis, a disease that affects one or both eyes, occurs most frequently in the large breeds, and is presented as progressive esotropia (globe is rotated medially). The esotropia can become so severe that the cornea cannot be visualized and vision is lost (Figure 4.6). Histopathology of affected muscles confirms variable lymphocytic infiltration and fibrosis. Systemic immunosuppressive therapy can be effective, but relapses do occur.

Traumatic Proptosis

Proptosis or an acute anterior displacement of the globe through the palpebral fissure can follow significant trauma in any breed of dog, or rather minor trauma (restraint) in the brachycephalic breeds. On presentation, the globe protrudes through eyelids that cannot cover the cornea, the nictitating membrane protrudes, and variable degrees of chemosis, conjunctival hyperemia, and corneal desiccation are present (Figure 4.7). Pupil size is variable. The globe displacement can progress as orbital hemorrhage and edema continue. Traumatic proptosis is an ophthalmic emergency and rapid globe replacement is essential for best results.

As major trauma is often involved, the entire patient must be examined, and the feasibility for general anesthesia ascertained. In the meantime, protection of the cornea with home products (such as maple syrup, corn syrup or honey) or artificial tears minimizes corneal



Figure 4.5 (A) Golden Retriever with bilateral polymyositis. The eyes are relatively fixed in position. (B) Same dog (A) after immunosuppressive corticosteroid therapy.

damage. The globe is repositioned following lateral canthotomy and maintained within the orbit by a complete temporary tarsorrhaphy with 3 to 4 horizontal mattress sutures (with stents) placed partial-thickness in the eyelids. Medical therapy consists of topical broad spectrum antibiotics and 1% atropine and systemic antibiotics and sometimes corticosteroids.

Sometimes several extraocular muscles insertions are torn and/or the optic nerve is transected. In these patients enucleation is recommended. Prognosis for return of vision varies with the severity of the insult, but is generally 20–50%. Lateral strabismus associated with either tearing or palsy of the medial rectus muscle, keratoconjunctivitis sicca, and optic nerve atrophy are common sequelae.

Orbital Trauma and Hemorrhage (Traumatic Exophthalmia/ Incomplete Proptosis)

Orbital trauma can be divided into intraorbital hemorrhage, emphysema, and fractures. Orbital hemorrhages are perhaps more frequent than the other two conditions. Hemorrhage within the orbit produces variable exophthalmos, lagophthalmos, and can be concurrent with subconjunctival and intraocular hemorrhages (Figure 4.8). Skull radiography and ultrasonography are usually the initial diagnostic imaging methods performed. If fractures are a concern, computed tomography (CT) is the modality of choice for imaging the orbit.



Figure 4.6 (A) Esotropia accompanied by microphthalmos in a young Samoyed. Often these eyes are not visual. (B) Lateral strabismus following rupture of the medial rectus resulting from traumatic proptosis. Note the hemorrhage at the insertion site. (C) Restrictive strabismus in a Shar Pei dog. The globe is rotated ventromedial and the cornea hidden totally and vision lost.

Superimposition of bones in skull radiography can make interpretation and identification of fractures difficult. Traumatic orbital hemorrhage can resemble proptosis, but the globe is only exophthalmic and the eyelid margins remain in their normal anatomic orientation.

With orbital hemorrhage, ocular mobility as well as an impaired blink response occur. With loss of lid function, lagophthalmia, corneal drying and subsequent damage result. Scleral rupture, retinal detachment, and other intraocular sequelae occur infrequently. Topical and systemic antibiotics and corticosteroids are usually indicated. With lagophthalmia, a temporary complete tarsorrhaphy is recommended until a normal blink reflex returns. Prognosis is usually guarded for return of vision, especially for those cases complicated with intraocular hemorrhage.

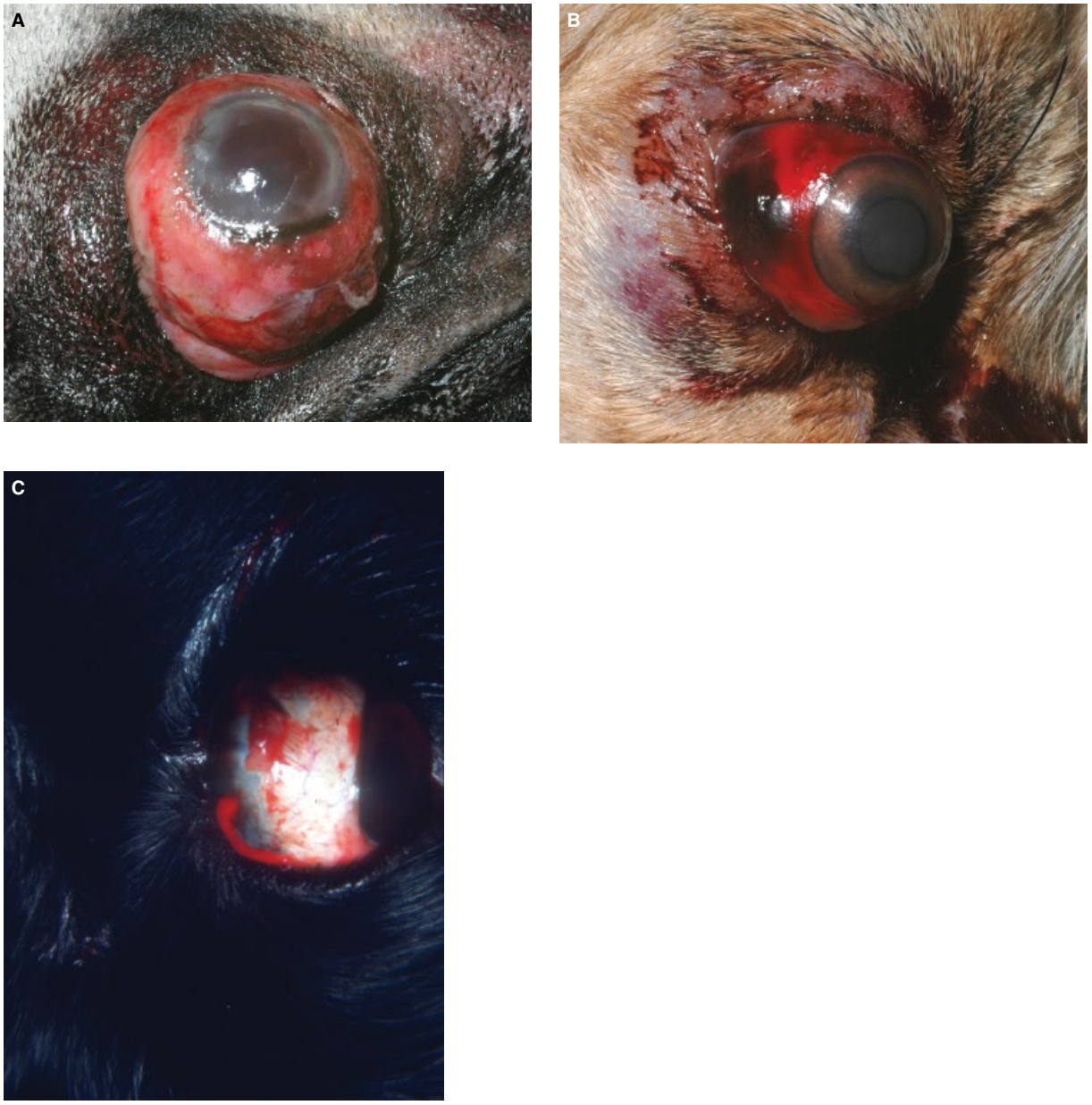


Figure 4.7 (A) Traumatic proptosis in a mixed breed dog after fighting with a larger dog. The globe became trapped beyond the palpebral fissure, causing immediate drying of the corneal surface. Replacement is recommended. (B) Severe traumatic proptosis in an American Cocker Spaniel after being hit by a car. Retrobulbar and subconjunctival hemorrhage can exacerbate the displacement. (C) Traumatic proptosis in an America Cocker Spaniel with the globe nearly totally exposed and tearing of several of the extraocular muscles. Enucleation is recommended.

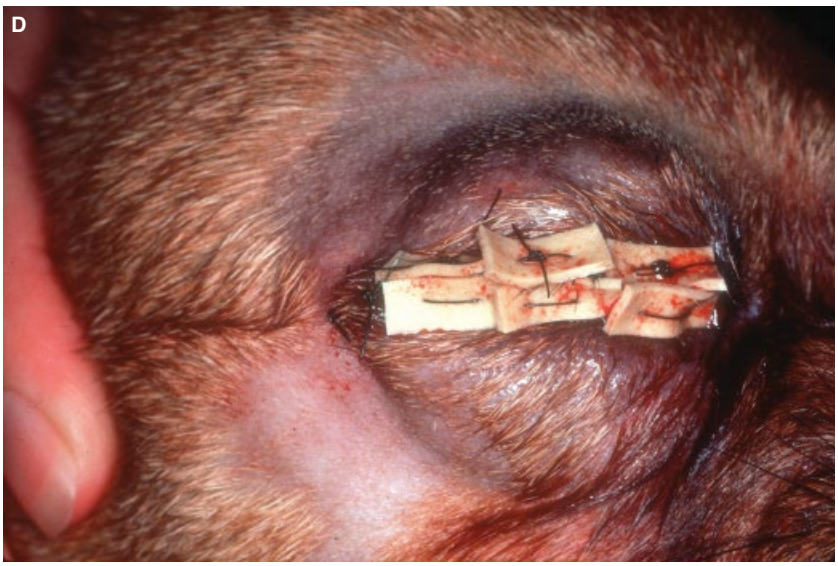




Figure 4.7 (*Continued*) (D) Surgical therapy consists of a complete temporary tarsorrhaphy. Rubber band stents help distribute the suture pressure along the entire eyelid length, prevent necrosis when the sutures are very tight or the orbital swelling is excessive, and can be adjusted during the 10 to 20 days that the sutures are needed before a vigorous protective blink returns. (E) Exotropia in a Pug 3 weeks after traumatic proptosis and replacement of the globe. As the *medial rectus m* is the shortest of the extraocular muscles, it is the most susceptible to tearing and transection during proptosis.

Craniomandibular Osteopathy

Craniomandibular osteopathy is a non-neoplastic proliferative bone disease that primarily affects the Scottish and White West Highland Terrier breeds (Figure 4.9). Ophthalmic signs consist of variable exophthalmia and sometimes impaired ocular mobility. Diagnosis by radiography reveals typical periosteal abnormalities of the temporal and mandibular bones. This disease can affect mastication because of involvement of the temporomandibular joint. The eyes do not usually require therapy.

Orbital Neoplasia

Orbital neoplasms in dogs generally occur in older animals (average 7 years), and can originate from any of the orbital tissues (60–70%) as well as the adjacent sites (usually nasal and sinus cavities) and systemic metastases (about 30–40%). Because these patients often present with advanced stages of neoplasia, prognosis is usually guarded, and survival times are limited (usually months).

Orbital neoplasms generally present as slow growing masses that produce a non-painful exophthalmia,





Figure 4.8 (A) Moderate orbital trauma after a dog fight in a Maltese. Both subconjunctival and retrobulbar hemorrhage are present. The globe is fixed and cannot move. (B) A more severe case of orbital trauma in a Pug hit by a baseball. Bright red blood is present within the entire anterior chamber.

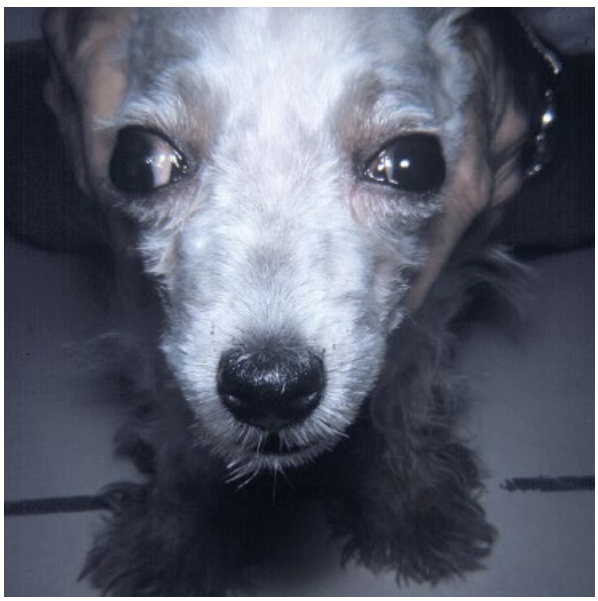


Figure 4.9 Craniomandibular osteopathy in a White West Highland Terrier puppy. The bony orbital abnormalities have displaced the eyes forward and lateral (exotropia).

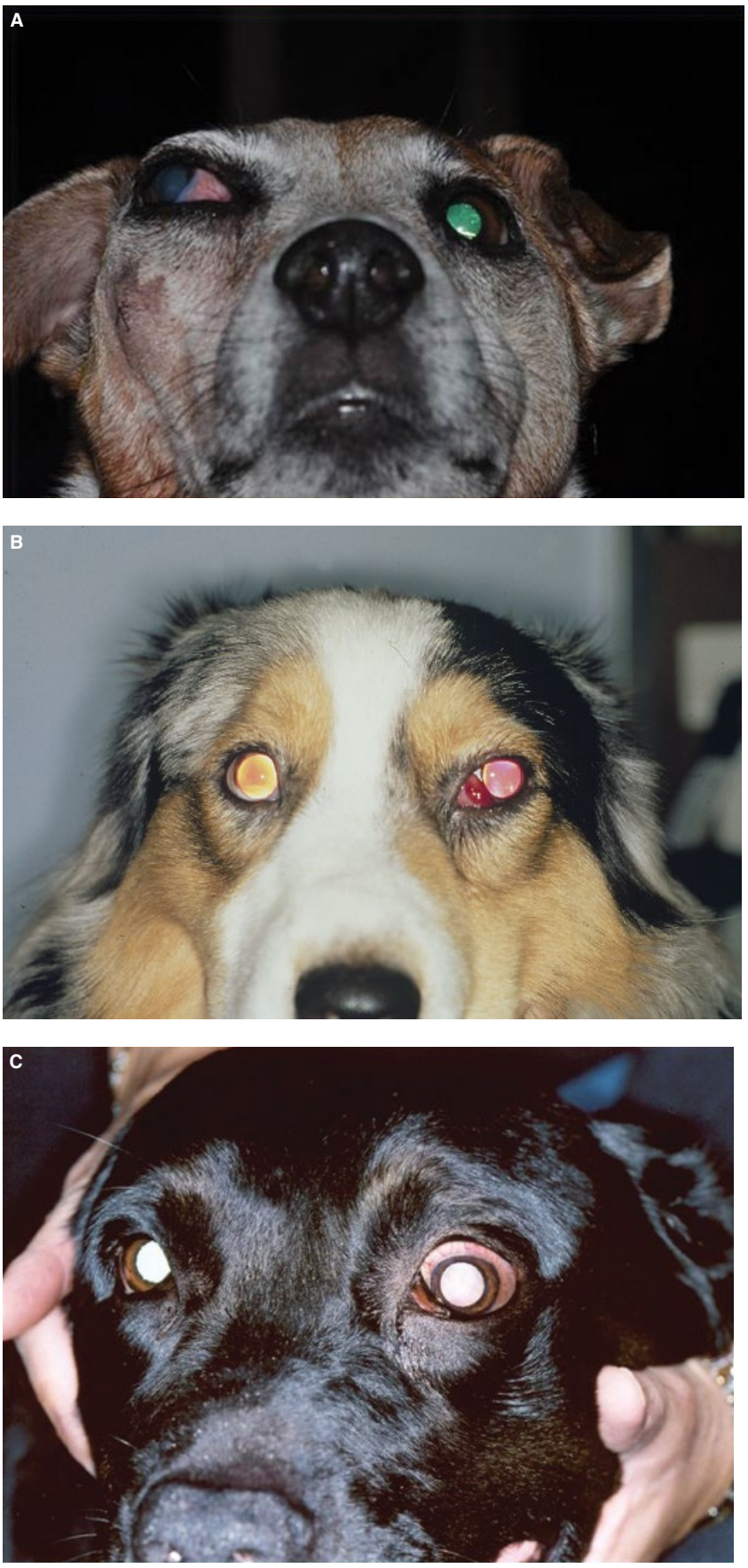


Figure 4.10 (A) Orbital osteosarcoma in a mixed breed dog. The globe is exophthalmic and dorsally and laterally deviated. Note the significant disfigurement of the face. (B) Orbital lymphosarcoma in an Australian Shepherd. The tumor has produced unilateral exophthalmia and protrusion of the nictitating membrane. (C) Labrador mix had adenocarcinoma which resulted in exophthalmos and impingement of the optic nerve (note the mydriasis).

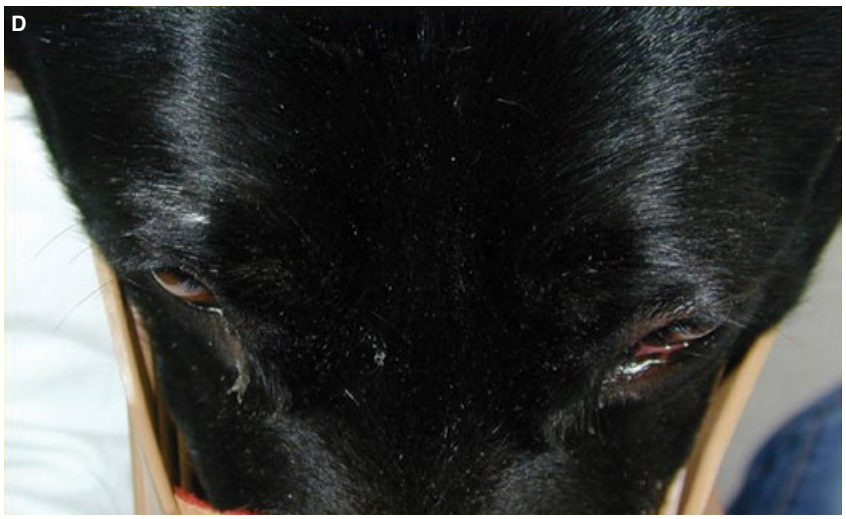


Figure 4.10 (*Continued*) (D) Retrobulbar tumor in this Labrador Retriever causing anterior displacement of the globe, as viewed from above.





Figure 4.11 (A) Clients must be advised of the post-operative appearance of the orbit after enucleation. In dogs with short-hair coats the concave permanent complete tarsorrhaphy is more disfiguring. (B) In dogs with longer hair coats and less prominent eyes, the adverse effects of enucleation are less obvious. The enucleation procedure was performed in this dog five months earlier.

protrusion of the nictitating membrane, chemosis and hyperemia of the conjunctiva, and occasional indentation of the posterior segment (as viewed by ophthalmoscopy). Orbital masses within the extraocular muscle cone tend only to result in exophthalmia, while masses outside the cone result in exophthalmia, strabismus, and impaired ocular mobility (Figure 4.10). Adenoma and adenocarcinoma, especially from the nasal cavity, can develop multiple mucoceles.

Diagnosis is made through a combination of imaging of the orbit (radiography, ultrasonography, CT, or MRI) and sampling of affected tissues for cytology and/or histopathology. Prognosis for benign and localized masses is good; but most orbital neoplasms are



Figure 4.11 (*Continued*) (C) Bilateral enucleations are sometimes necessary as in this older American Cocker Spaniel with blindness from advanced primary glaucoma. The dog had already adapted to blindness, and adjusted to the absence of pain, and became a very good house pet within a protective environment.

Figure 4.12 A: (A) Intraocular silicone prosthesis (ISP) can be used to treat painful eyes without removal of the eye as in this Beagle with end-stage glaucoma. The globe size is maintained by the intraocular silicone sphere, and placement within the corneoscleral tunic avoids daily removal and cleaning. Corneal pigmentation and fibrosis almost always occur. (B) ISP one year after placement. Note the corneal edema, fibrosis and vascularization.







Figure 4.13 A. Phthisis bulbus in a Schnauzer mix after aphakic glaucoma of several months duration. Note the corneal edema (from very low intraocular pressure) and pigmentation.

malignant and infiltrative. Exenteration and orbitectomy can be successful, but many clients may not be able to accept the disfigurements that follow these radical surgeries.

Orbital Surgical Results

Postoperative Enucleation

The enucleation procedure is reserved for blind painful eyes affected with end-stage glaucoma, primary intraocular neoplasms, and painful phthisis bulbus (atrophy of the globe). In the enucleation surgery, the globe is removed leaving the remaining orbital tissues behind a complete permanent tarsorrhaphy. Postoperative orbital pitting can be more cosmetically acceptable in a long-haired dog than in the short-haired breeds. A silicone or methyl methacrylate sphere can be inserted during the enucleation surgery to minimize this disfigurement (Figure 4.11).

Intraocular Prosthesis

The intraocular prosthesis consists of a silicone sphere positioned after evisceration of the globe (removal of all of the intraocular tissues, leaving only the sclera and cornea). This procedure permits the treatment of painful eyes (usually after blinding glaucoma), while still permitting retention of a mobile globe, and usually eliminates the need for local and systemic medications for the eye following the immediate postoperative period. Eyes with intraocular tumors and persistent intraocular infections are generally not candidates for this surgery. As aqueous humor is no longer present, corneal vascularization and eventual complete pigmentation occur (Figure 4.12).

Phthisis Bulbus - Sequelae of Intraocular Diseases

Phthisis bulbus or atrophy of the eye occurs after considerable damage occurs to the ciliary body resulting in marked reduction in the production of aqueous humor (Figure 4.13). The result is a hypotonic globe (intraocular pressure less than 5 mm Hg) with persistent corneal edema, cataract formation, and retinal detachment and degeneration. The most common causes include primary and secondary glaucomas, intractable iridocyclitis, and trauma.

5

Canine Eyelids

The eyelid functions to protect the cornea and globe, regulate light entering the eye, aid in the distribution of the tears across the cornea and to the nasolacrimal drainage system, and contribute part of the precorneal (preocular) tear film (from meibomian glands).

Congenital and Developmental Disease

Neonatal ophthalmia is an infection that occurs beneath closed eyelids in young animals with physiologic ankyloblepharon (Figure 5.1). It can be associated with delayed or incomplete opening of the pup's eyelids at 12–14 days postnatal. Bacterial conjunctivitis is common and can be exacerbated in cats if there is a concurrent infection with feline herpesvirus type 1 (FHV-1). The conjunctival sac distends and purulent material may begin to extrude through an opening between the eyelid margins if one exists. Early treatment with topical antibiotics and surgical opening of the palpebral fissure is recommended before corneal ulceration develops.

Agenesis of the eyelids occurs most commonly in cats; however, it is occasionally present in dogs and other species (Figure 5.2). Most often the coloboma affects the lateral aspects of the upper eyelids. Lack of lid margin often results in trichiasis, superficial keratitis, and persistent irritation. Surgical correction is recommended if these signs persist. Surgical procedures to treat lid agenesis involve filling the missing lid with sliding or pedicle eyelid skins flaps from the lower ventral lateral eyelid or the lateral commissure of the mouth. Other extra- and intraocular defects are often present concurrently, and can impact the overall prognosis.

Dermoids or choristomas affect the lids infrequently, and more often involve the bulbar conjunctiva, limbus, or even the nictitating membrane (Figure 5.3). They can be inherited in the German Shepherd, St. Bernard, and Dalmatian breeds. Lid dermoids are characterized as a focal lid mass covered with long coarse hair. Histologically, they consist of connective and adipose tissues as well as normal skin tissues. Surgical correction requires excision of the entire dermoid and reconstruction of the defect.

Breed-Associated Eyelid Disorders

Structural abnormalities of the eyelids represent the largest group of lid diseases in the dog. Blepharophimosis, blepharostenosis, or micropalpebral fissure occur in certain breeds, such as the Chow Chow, English Bull Terrier, Shetland Sheepdog, and Kerry Blue Terrier (Figure 5.4). The globe is usually normal, but sometimes microphthalmia is concurrent. Entropion often develops with micropalpebral fissures, and can result in corneal damage or persistent irritation, so surgical correction is recommended. Often, the area of the lids affected is breed-specific and bilateral.

Euryblepharon or macropalpebral fissure is divided clinically into two types.

1) In brachycephalic breeds, macropalpebral fissures are associated with shallow orbits and a short maxilla covered with nasal skin folds. This predisposes the eye to proptosis, exposure, and recurrent corneal ulcerations. Keratitis is exacerbated by lagophthalmia, infrequent and inadequate blinking, reduced central corneal sensitivity, impaired precorneal film dynamics, and accelerated central corneal epithelial turnover (Figure 5.5).
2) The second type of euryblepharon occurs in the large and giant breeds of dogs, and is caused by excessively long eyelids that usually result in combined entropion–ectropion. Unfortunately, this condition is often a requirement of certain breed standards. Breeds commonly affected are the St. Bernard, Bloodhound, and Clumber Spaniel (Figure 5.6).

Color Atlas of Veterinary Ophthalmology, Second Edition. Kirk N. Gelatt and Caryn E. Plummer.
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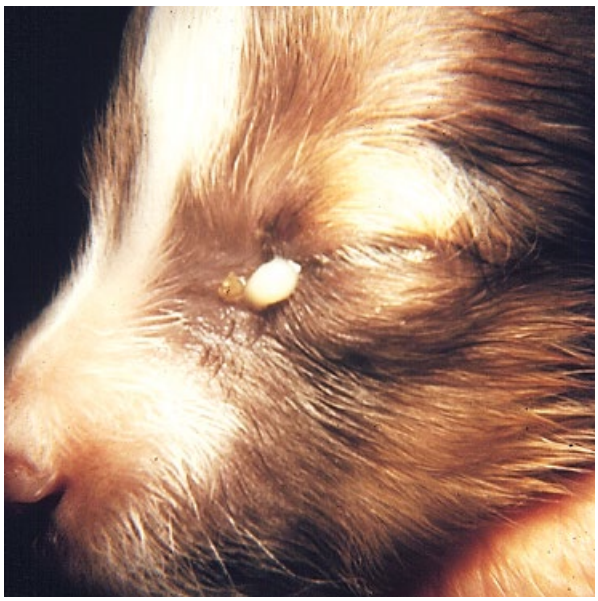


Figure 5.1 Ankyloblepharon and neonatal ophthalmia in a Shetland Sheepdog puppy. Note the marked swelling of the eyelids. Purulent exudate is escaping at an opening in the medial ankyloblepharon.

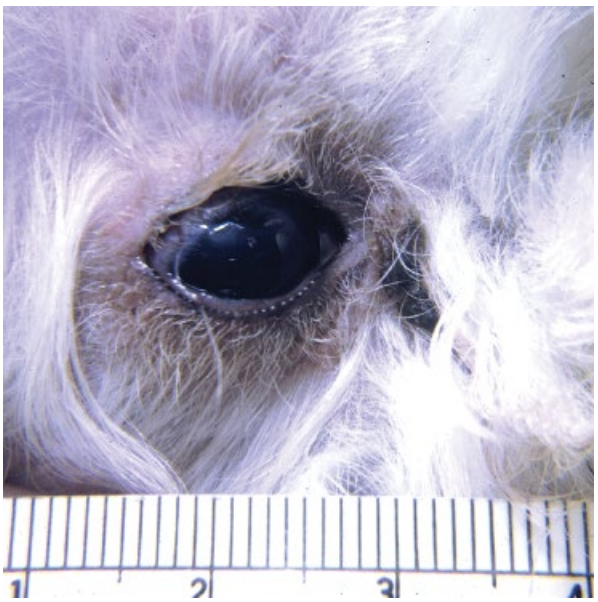


Figure 5.2 Agenesis of the lateral upper eyelid in a Miniature Poodle puppy. Lack of eyelid margin in the affected area has resulted in trichiasis and local keratoconjunctivitis.



Figure 5.3 (A) Dermoid of the palpebral conjunctiva and lower eyelids in a Cairn Terrier. Note the long and coarse hairs which cause considerable discomfort. (B) Dermoid of the upper eyelid in a young Golden Retriever.

Treatment of euryblepharon is surgical; however, the choice of surgical procedure depends on the breed and specific anatomy of the individual. Most cases will benefit from some sort of lid shortening or stabilizing procedure.

Entropion is a very common lid disease in the dog wherein the eyelid margin inverts to cause contact of haired skin with the bulbar conjunctiva and cornea. Entropion usually requires surgical correction. The sometimes profound discomfort (blepharospasm) that results aggravates further the original entropion and results in spastic entropion. Pain generated by the eyelid hairs touching the cornea and conjunctiva can often account for more than 50% of the entropion. The real

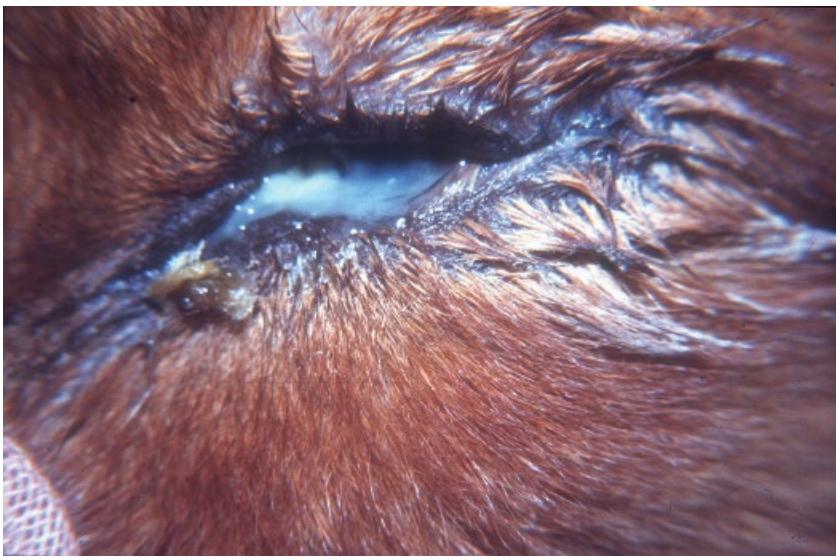


Figure 5.4 Blepharophimosis or a reduced palpebral fissure in a Chow Chow puppy. The shortened eyelids usually develop entropion and require surgical correction (lateral canthoplasty). The entropion has resulted in corneal and conjunctival irritation and a mucopurulent discharge.





Figure 5.5 (A) Pekingese dog has euryblepharon or an enlarged palpebral fissure which results when the eyelids are longer than normal. The result is greater exposure of the cornea (note the central scar from a previous ulcer) and conjunctiva (which is visible nearly 360° around the cornea). (B) Euryblepharon in a Shih Tzu. Note the dramatic amount of scleral show. This animal is at risk for proptosis.



Figure 5.6 Clumber Spaniel with very prominent "V" notch in the central lower eyelid.

danger with entropion is corneal disease, which manifests with ulcerations and scarring. Entropion is often exhibited in young and growing puppies (such as the Chinese Shar Pei) but can self-correct as the animal grows. Permanent surgical correction should be minimal or postponed until the puppy has approximated its adult size lest over-correction occur. As entropion can affect the upper lid, lower lid, and lateral canthus, surgical correction varies according to the amount and position of the defect.

Entropion is generally believed to be inherited as an autosomal dominant, but breeding studies are lacking. Entropion is often breed-related and occurs in the Chow Chow, Shar Pei, St. Bernard, English Bulldog, English and American Cocker Spaniels, English Springer Spaniel, Labrador Retriever, Bull Mastiff, Great Dane, Irish Setter, Norwegian Elkhound, and Toy and Miniature Poodles, among others (Figure 5.7).

Ectropion is less common than entropion in the dog, and results when the eyelid margins are everted from the eye, exposing the ventral conjunctiva. Ectropion can be developmental or secondary to inflammation, trauma, surgery, or aging. Exposure of the conjunctiva and cornea results in persistent inflammation and irritation of the conjunctiva, impaired tears dynamics, epiphora, and exposure keratitis (often seen as corneal vascularization and pigmentation). Surgical correction of ectropion is recommended if the secondary corneal and conjunctival disease cannot be resolved medically or if the abnormality increases in severity. Most lower eyelids with ectropion are longer than normal, and most corrective surgeries involve some shortening of the lower lid (Figure 5.8).

The combination of entropion and ectropion can occur in the larger breeds of dog. Certain breed standards require a "diamond-shaped" palpebral fissure, a prominent nictitating membrane, and/or a drooping low eyelid. These standards result in persistent eye disease throughout life, and sometimes corrective surgery is required. The breed standards should be re-evaluated and updated to modern societal, ethical, and medical levels to eliminate these unnecessary eyelid abnormalities and animal discomfort. Breeds with combined entropion–ectropion include Bloodhound, St. Bernard, Newfoundland, Clumber Spaniel, and Bull Mastiff (Figure 5.9).

In combined entropion–ectropion, the lids are longer than normal, and the lateral canthus is unstable. Also in many of these breeds, the excessive forehead skin above the eyes is distorting and intensifies the eyelid defects; as a result, facial and/or forehead skin lifts or rhytidectomy are often combined with lid surgeries. In some of the large and giant breeds, especially in the males, enophthalmia accompanies the eyelid abnormalities.

Distichiasis

In distichiasis, additional cilia or eyelashes are present; they usually emerge from the opening of the meibomian or tarsal glands and result from metaplasia of these glands. These cilia can be short to long, fine to coarse, and may or may not contact the conjunctiva and/or cornea. They can grow through the dog's life and produce no clinical signs or they can be very irritating, inducing lacrimation and epiphora, persistent

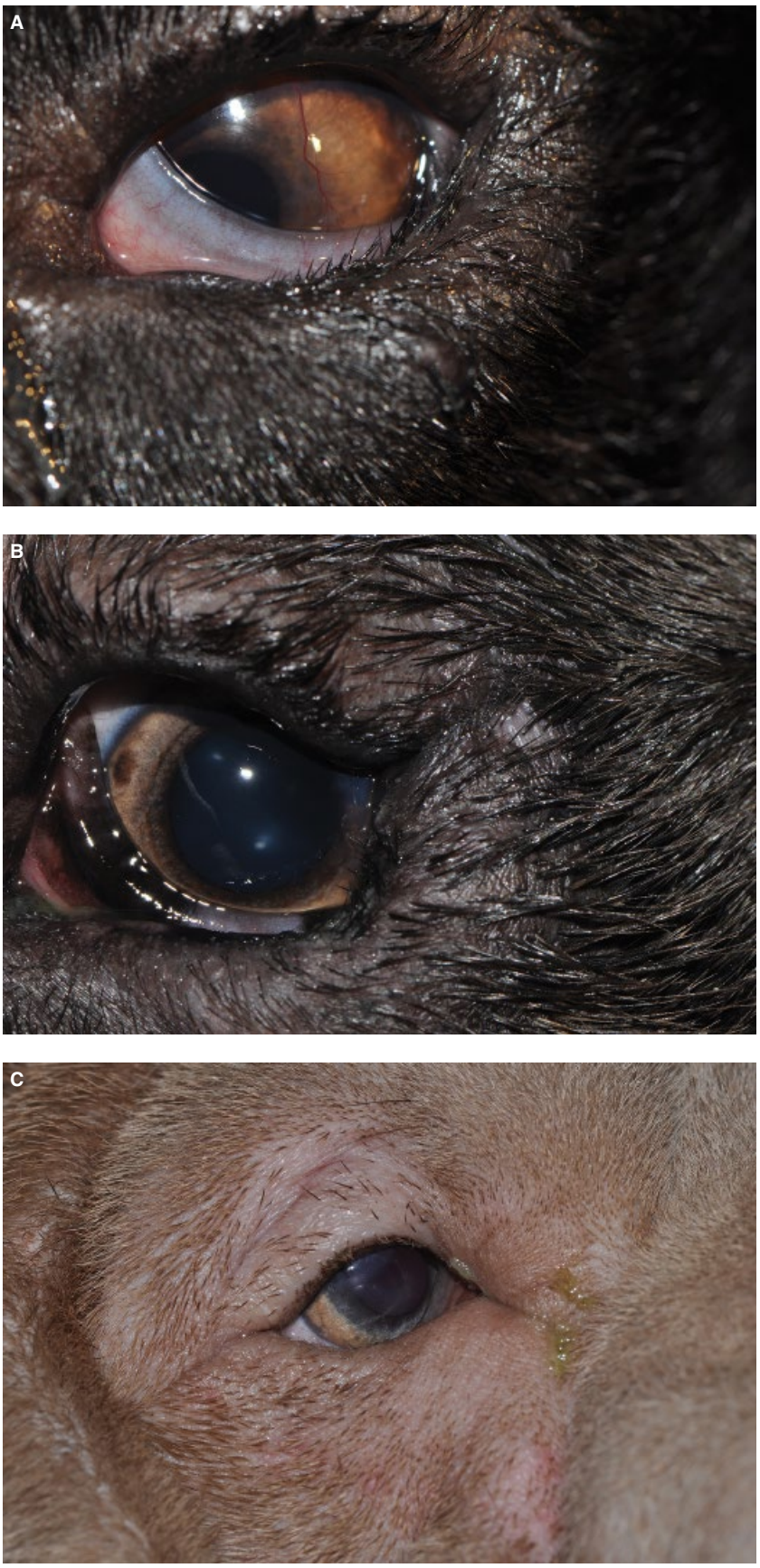


Figure 5.7 (A) Entropion appears to be inherited in many breeds; in the Norwegian Elkhound the entropion is usually confined to the lateral and lower eyelid, and may self-correct in young dogs. (B) Lateral canthal and lower entropion in a young Labrador Retriever. (C) Lateral lower eyelid entropion in a Shar Pei. Note the temporal keratitis.





Figure 5.7 (*Continued*) (D) Chinese Shar Pei puppy with nearly 360° entropion. At this age, maintaining the eyelids open using tacking sutures is recommended. (E) Entropion affecting upper and lower eyelids of a mature Chinese Shar Pei. Surgical correction may also include a face lift (removal of one or more of the facial skin folds).

conjunctival hyperemia and conjunctivitis, and superficial corneal vascularization, ulceration, and pigmentation (Figure 5.10).

Distichia are common in the American Cocker Spaniel (probably more than 90% of dogs are affected), English Cocker Spaniel, Longhaired Dachshund, Bulldog, Pekingese, Flat-coated Retriever, Shetland Sheepdogs, and Toy and Miniature Poodles. Distichia that induce ocular irritation and disease should be treated by removal or destruction of their follicles. Many methods have been recommended: simple epilation, electrolysis, several surgical procedures, and cryothermy.

Ectopic Cilia

Similar clinical signs can occur with ectopic cilia, abnormal cilia that emerge from the palpebral conjunctiva, usually in the middle of the upper eyelid (Figure 5.11 and front cover). As these cilia directly contact the cornea, they can cause considerable pain, blepharospasm, eyelid swelling, and ulceration (which signals the position of the ectopic cilia). Treatment is surgical excision (en bloc) or cryotherapy of the cilia's follicle(s).

Trichomegaly

In trichomegaly, abnormally long eyelashes occur. This condition is common in the upper eyelids of the American Cocker Spaniel in which these lashes exceed several centimeters in length (Figure 5.12). They are not associated with eye disease, but can require periodic trimming.



Figure 5.8 (A) Severe bilateral ectropion in Bloodhound with chronic discomfort and secondary bacterial conjunctivitis. (B) Severe cicatricial ectropion in a Bracco Italiano following surgery to correct entropion.

Trichiasis

Trichiasis occurs when the eyelids margin is inverted, as with entropion, or when the nasal fold or its hairs contact the conjunctiva and/or cornea, typically in the brachycephalic breeds. The irritation produces lacrimation, epiphora, blepharospasm, and, in the acute phase, corneal ulceration, or, more commonly, chronic keratitis, vascularization, and pigmentation.

When trichiasis is associated with entropion, surgical correction is recommended and usually resolves the condition. For facial folds causing trichiasis in breeds such as the Pekingese, temporary relief can be obtained with flattening the hairs with petroleum jelly or permanently by partial resection of the nasal folds (Figure 5.13).

Eyelid Trauma

Eyelid lacerations are infrequent in dogs except where leash laws are absent or not enforced. Dog and cat fights as well as automobile accidents are the usual causes. Eyelid lacerations are usually presented soon after the accident, and involve the eyelids (both upper and lower) as partial-thickness, pedicle-shaped, horizontal or vertical tears involving the eyelid margins (Figure 5.14). The upper lid is more apt to be lacerated than the lower lid.

A complete eye examination is indicated for all eyelid lacerations, as the globe can also be involved. Hyphema and vitreal hemorrhage signal corneal, limbal, or scleral rupture which can require additional correction, and can adversely affect overall prognosis. Partially torn eyelids

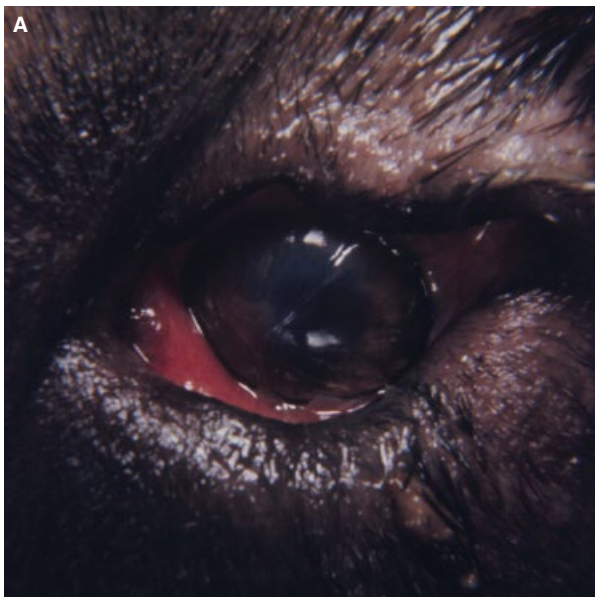




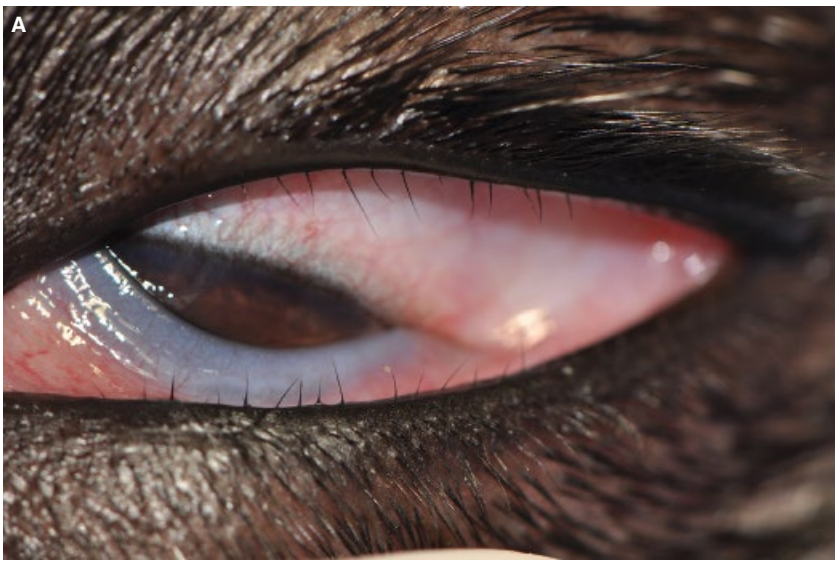
Figure 5.9 (A) Combined entropion–ectropion in a young St. Bernard dog with elongated eyelids. Both corneal and conjunctival diseases result from the exposure as well as direct lid contact with the cornea. Lateral canthal entropion and instability often compound the surgical correction. (B) Combined entropion–ectropion in an English Bulldog. Note the central notches in both the upper and lower eyelids.

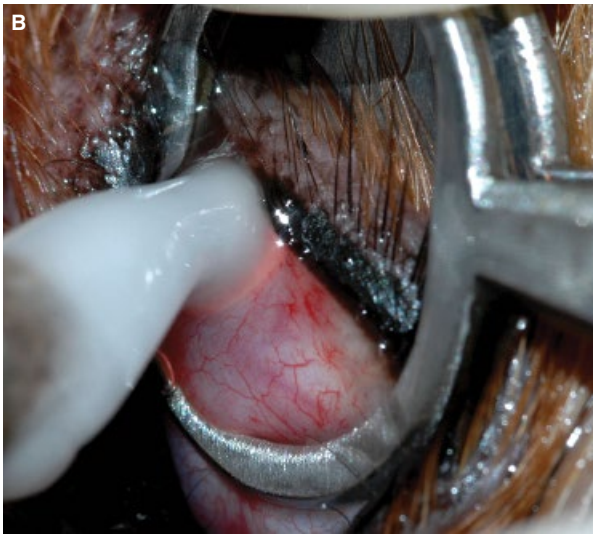
should be minimally debrided, without excision, and the cut surfaces should be re-apposed as precisely as possible. Apposition of the lid margin is usually the starting point, and, if possible, two-layer closure (skin-orbicularis muscle and tarsoconjunctival layers) should be attempted in most cases. A figure-of-eight suture is useful for the lid margin as it positions the knot away from the cornea.

Diffuse and Focal Blepharitis

Blepharitis is not uncommon in the dog, and is associated with a number of pathogens (bacteria, fungi, parasites, leishmaniae). It can be acute or chronic, diffuse or localized, and affects young dogs most often. Blepharitis in young dogs frequently accompanies puppy pyoderma or puppy strangles, with lesions often concentrated about the face (Figure 5.15). Variably sized pustules to frank abscesses affect the eyelids and facial skin. These focal lesions are often sterile. Pruritus and self-trauma may require an Elizabethan collar. Topical and systemic steroids, and infrequently antibiotics, are indicated.

Blepharomycosis (*Microsporum* and *Trichophyton* spp.) can affect young dogs who present with expanding alopecia, scaling, and hyperemia. The condition can affect only the eyelids or be generalized. Skin scrapings or culture on Sabouraud agar are useful diagnostic tests. Both demodectic and sarcoptic mange can also affect the





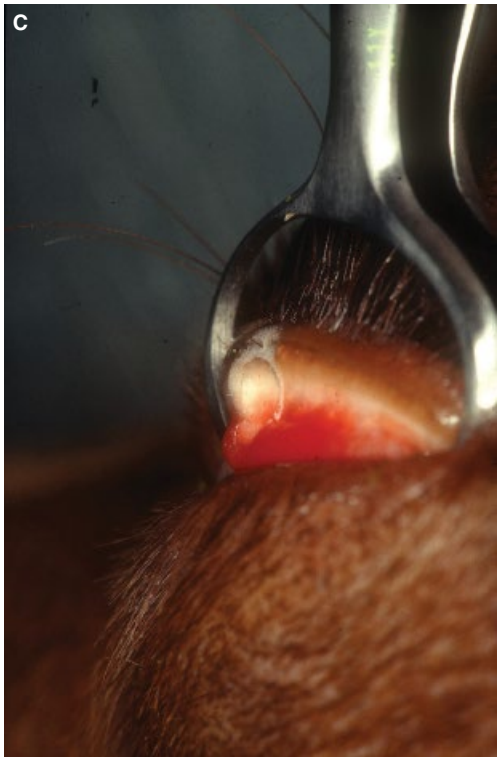


Figure 5.10 (A) Multiple distichia are emerging from the lateral lower eyelid margin in a mixed-breed dog. Very fine distichia are frequently non-irritating. (B) Cryotherapy is the most often performed procedure as it can be repeated and tends not to affect the eyelid margins (unlike many of the surgical procedures). Application of a cryoprobe to the palpebral conjunctiva near the eyelid margin targets the follicles of distichia. (C) Immediate appearance after cryotherapy. Over-freezing can cause necrosis of the eyelid tissues and under-freezing does not destroy the distichia follicles. Recurrence of cilia after these surgeries is in the range of 10–30%, so multiple treatments may be necessary.

eyelids, manifesting with hyperemia, alopecia, pruritus, and, often, secondary bacterial infection and self-trauma (Figure 5.16). For current therapy, consult a dermatology reference.

Several types of immune-mediated blepharitis can result in focal or generalized disease. Blepharitis can occur in dogs, such as the German Shepherd Dog, that suffer chronic superficial keratitis (pannus) (Figure 5.17). Variable sized inflamed skin ulcers appear in one or both medial canthi. The overlying hair is usually lost, but pruritus is infrequent. Treatment consists of intermittent or chronic topical corticosteroids or immunomodulating

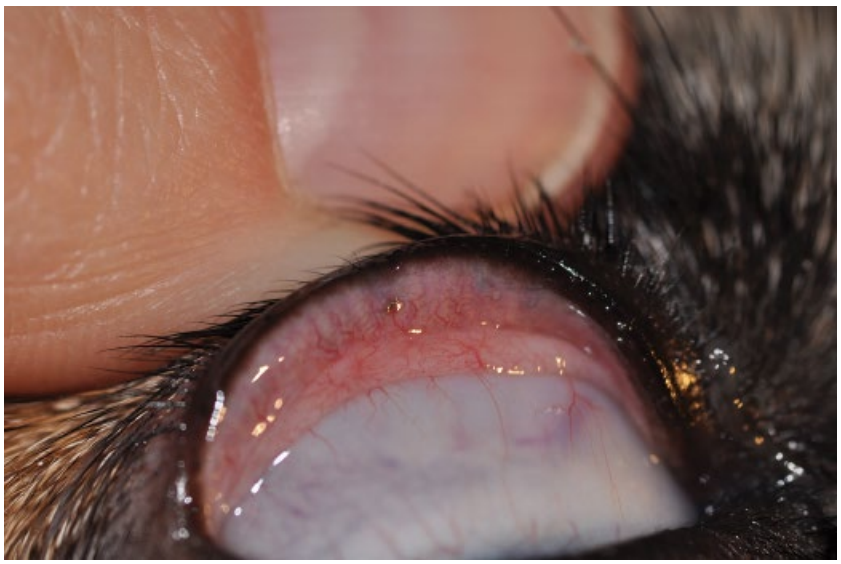


Figure 5.11 An ectopic cilia is emerging from the central palpebral conjunctiva beyond the lid margin. Cilia such as this are very irritating and can contribute to ulceration of the cornea. There are several hairs visible beneath the palpebral conjunctiva in this individual which have not yet emerged.



Figure 5.12 Trichomegaly affecting both upper eyelids in an American Cocker Spaniel. These extremely long eye lashes do not produce eye disease and are usually periodically trimmed.



Figure 5.13 (A) Trichiasis secondary to nasal folds in a Pekingese dog. The hairs from the upper aspects of both nasal folds contact the medial corneas causing irritation and medial pigmentary keratitis.

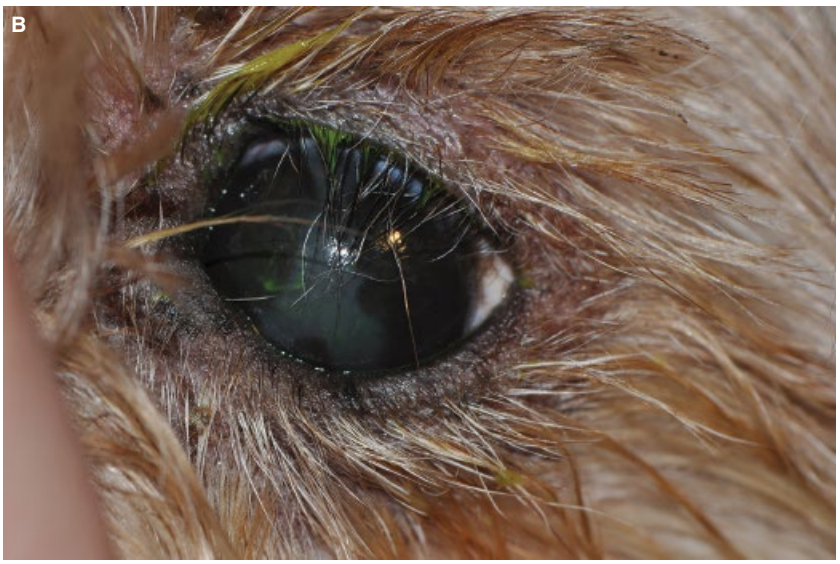


Figure 5.13 (*Continued*) (B) Upper eyelid trichiasis in a Shih Tzu.





Figure 5.14 (A) Eyelid laceration affecting the lower lateral eyelid and canthus in a Labrador Retriever. The torn eyelid margin should be carefully apposed by sutures (rather than excised) to the remaining eyelid. (B) Previous eyelid laceration that was not corrected. Fibrosis of the wound edges often results in persistent irritation.

drugs (cyclosporine, tacrolimus). The goal of topical therapy is to determine the minimum frequency of therapy necessary to keep the disease in check for the rest of the animal's life.

Pyogranulomatous blepharitis is characterized by a generalized chronic inflammation of the upper lid, lower lid, or both. The lids can markedly increase in thickness, impairing their blink function (Figure 5.18). Biopsy reveals microscopic pyogranulomas. *Staphylococcus* sp. are usually incriminated, and are often resistant to topical and systemic antibiotics. Intralesional injections of antibiotics as well as use of autogenous bacterins are most effective.

Blepharitis can accompany systemic immune-mediated conditions such as pemphigus or lupoid complex diseases or other immune-mediated ophthalmic disease,



Figure 5.15 Puppy pyoderma blepharitis in a young Rottweiler puppy. Note the multiple focal abscesses affecting the skin about the mouth and eyelids.

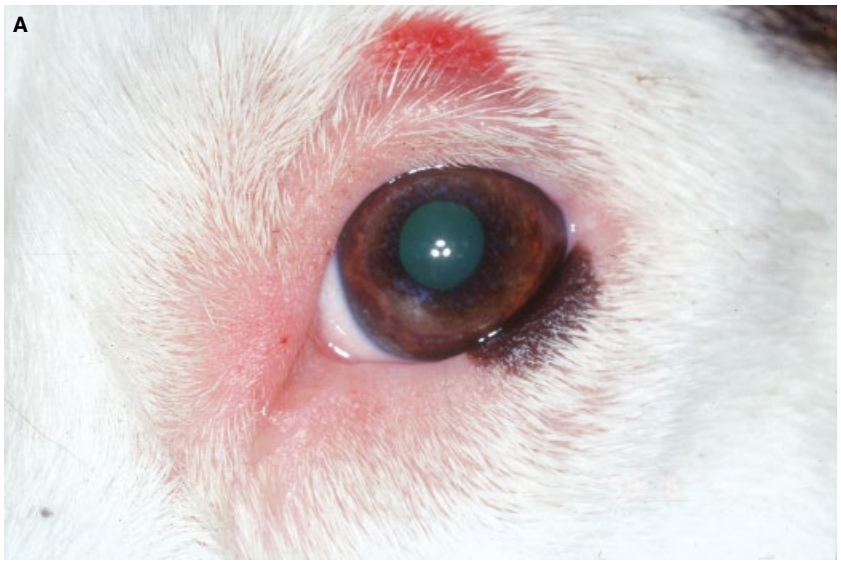




Figure 5.16 (A) Sarcoptic mange in a young dog. The skin has hyperemia, alopecia, pruritus, and often the effects of self-trauma. (B) Demodex in a young dog; the disease is often confined to the eyelids and face.



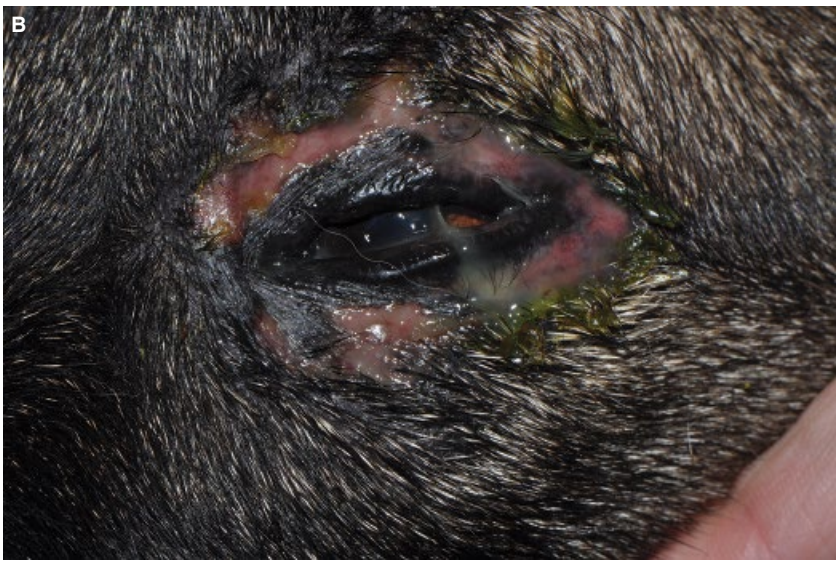


Figure 5.17 (A) Immune-mediated blepharitis affecting the medial canthus in a dog. Note the localized lid inflammation. In the German Shepherd this condition is often coupled with chronic superficial keratitis (pannus). (B) Severe ulcerative immune-mediated blepharitis in a German Shepherd Dog.

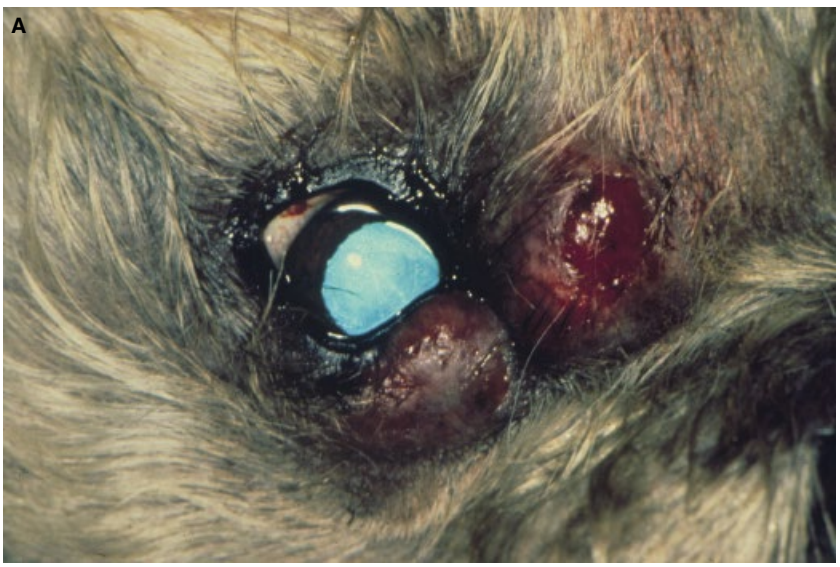


Figure 5.18 (A) Pyogranulomatous blepharitis affecting the upper and lower eyelid in a dog. Biopsy of the inflamed lids is recommended to confirm the diagnosis and exclude neoplasia.



Figure 5.18 (*Continued*)
(B) Pyogranulomatous blepharitis in a chocolate Labrador Retriever affecting both the upper and lower eyelids. The condition was bilateral.



Figure 5.19 Uveodermatologic syndrome typically results in severe panuveitis, which can result in secondary glaucoma and cataract formation, in addition to depigmented or ulcerative lesions of the skin, particularly in highly pigmented areas.

such as uveodermatologic syndrome, also known as Vogt–Koyanagi–Harada syndrome (Figure 5.19; see also Figures 5.19, 10.7, 18.26, and 18.27). The diagnosis of these conditions requires histopathology, and treatment usually involves immunosuppressive therapy.

Focal to diffuse inflammation of the meibomian glands (meibomianitis) occurs most often in adults. *Staphylococcus* and *Streptococcus* spp. organisms are most commonly involved. Meibomianitis can be acute or chronic, but usually affects both upper and lower eyelids bilaterally. The enlarged and often impacted glands appear as multiple focal lid swellings. Eversion of the lid margin usually reveals distinct areas of conjunctival hyperemia and swelling, and focal microabscesses within the tarsal glands (Figure 5.20). Chronic meibomianitis can result in lid fibrosis, and either entropion or ectropion, and requires surgical correction once the primary condition has been resolved.

Treatment of acute meibomianitis consists of topical hot packing, topical broad spectrum antibiotics, and often corticosteroids. If the inflammation is severe, systemic broad spectrum antibiotics may be indicated. The abscessed material can be gently expressed from the tarsal glands out their ducts, taking care not to rupture the gland and distribute the infection into the adjacent lid tissues.



Figure 5.20 (A) Acute meibomianitis affecting the lower eyelids in an American Cocker Spaniel. The blepharitis is localized to the palpebral surface of the lids. (B) Chronic meibomianitis with numerous impacted glands. These chronic cases are often quite stubborn to treat, and cause long-term lid fibrosis and entropion. (C) Severe meibomianitis affecting both upper and lower eyelids in a Shih Tzu.

Chronic meibomianitis is more difficult to resolve, and culture and bacterial sensitivity of the tarsal gland discharge is indicated to ensure proper topical and often systemic antibiotic choice.

Focal inflammations of the glands of Zeis and Moll, and tarsal or meibomian glands, are termed "stye" or hordeolum along the external lid or eyelid margin; on the inside of the eyelid and beneath the palpebral conjunctiva they are termed chalazion. In both conditions, the meibomian secretions are often trapped within the gland or its ducts and appear as swollen inflamed masses (Figure 5.21).

Chalazia accompany acute to chronic meibomianitis, and glandular secretions become trapped within the tarsus and beneath the palpebral conjunctiva. As a result of the bacteria and trapped glandular secretions, a granulomatous inflammation develops with formation of palpebral subconjunctival abscesses comprised of caseous inflammatory material. Resolution often requires incision over the affected

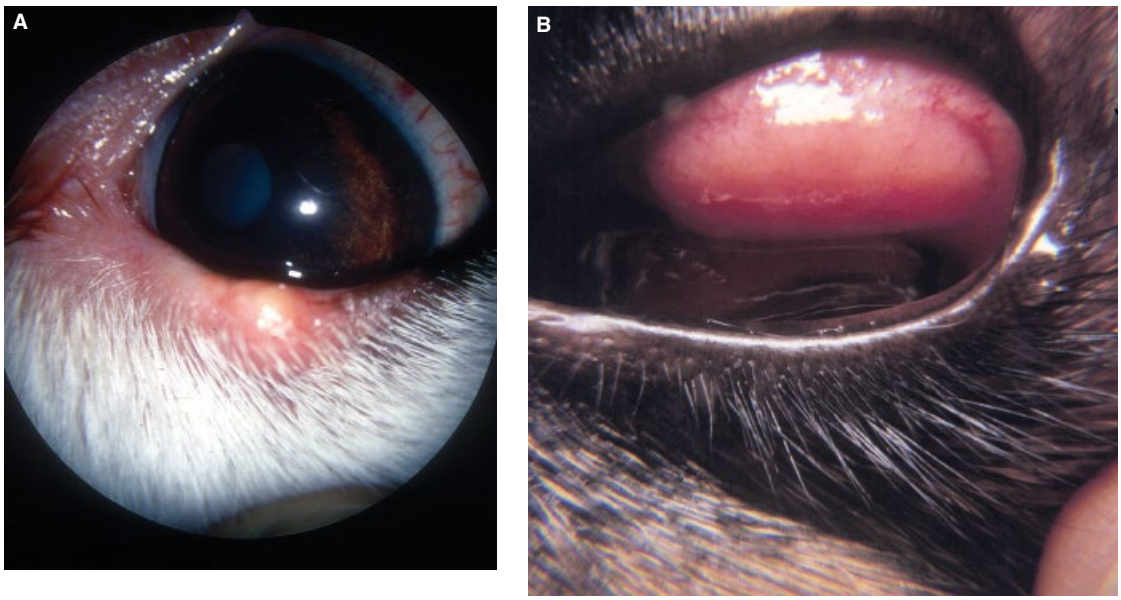


Figure 5.21 (A) Stye or hordeolum in a young dog. The inflamed glands are located on the eyelid margin, and can be quite irritating. (B) Large chalazion affecting the upper eyelid in a young Collie-mix female. There is marked blepharitis and swelling of the upper palpebral aspects of the lid.



Figure 5.22 Proliferative keratoconjunctivitis in a young Rough Collie. Eyelids, conjunctiva, and cornea are affected. The condition was bilateral.

palpebral conjunctiva and curettage of the caseous exudates. Topical antibiotics are administered for several days.

Inflammatory Lid Masses

Although a variety of inflammatory eyelid masses, including nodular granulomatous episclerokeratitis (especially in the American Cocker Spaniel breed), fibrous histiocytoma, and recurrent proliferative keratoconjunctivitis (especially in the Collie breeds), can have very similar histopathology, these diseases have distinct clinical signs. Often appearing as focal or nodular masses, they can involve the eyelids, conjunctiva (and nictitans), and the cornea (Figure 5.22). Diagnosis should be established by biopsy. Therapy includes topical and systemic corticosteroids or immunomodulating drugs. Recurrence is common.

Eyelid Neoplasia

Eyelid neoplasms are common in dogs, especially those over 7–10 years of age. In contrast to the cat, most canine eyelid tumors are clinically benign. The most common types in the dog are meibomian adenomas or epitheliomas (Figure 5.23). About 70–80% of all canine eyelids tumors

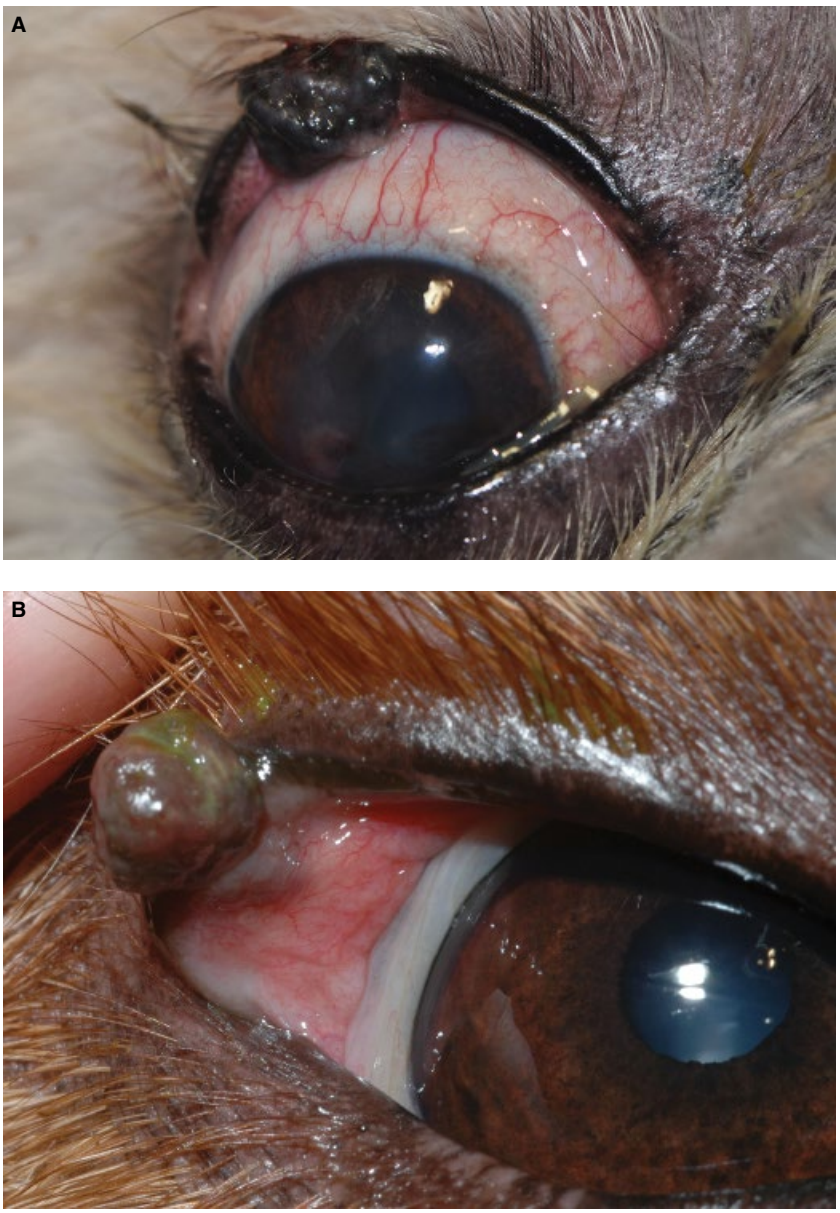


Figure 5.23 (A) Adenoma of the meibomian gland of the upper eyelid in an aged mixed-breed dog. (B) The full extent and origin of these tumors can be appreciated by everting the upper lid and inspecting the lid margin and the palpebral conjunctiva overlying the base of the tumor.

arise from the tarsal or meibomian glands, and occur in older dogs. Often nodular, they can irritate the eye by direct contact of the mass with the cornea and/or conjunctiva. Fortunately, these tumors are not very invasive and are usually slow growing. They are not known to metastasize. Early excision provides for the most limited eyelid tissue loss and best cosmetic and functional results. Any mass that comes into contact with the cornea should be excised promptly to prevent or treat untoward consequences and irritation for the cornea. Other tumors of decreasing frequency include papillomas, benign and malignant melanomas, histiocytomas, mastocytomas, and basal cell and squamous cell carcinomas.

Melanomas can appear at the eyelid margin or lid skin distant to the margin as irregular, raised, firm masses (Figure 5.24). They can be histologically benign or malignant, but tend not to be as highly aggressive or apt to spread as malignant melanomas at other locations.

Eyelid neoplasms not associated with the tarsal glands include squamous cell carcinomas (SCC), cutaneous epitheliotropic lymphoma (CEL), fibrosarcomas, and mast cell tumors (Figure 5.25). Biopsy is recommended for these masses to determine the most appropriate form of therapy. SCC of the canine eyelid can present as either persistent ulcerations or fleshy pink masses. Therapy is by surgical excision, often combined with an adjunctive treatment modality. Mastocytomas typically require large resections to achieve clean margins which may necessitate reconstructive blepharoplasty.



Figure 5.24 Melanoma of the lower eyelid margin in a mixed-breed dog. These tumors typically arise from and spread along the eyelid margin. Because the lid margin is infiltrated (usually in both directions), tumor excision is more difficult and some lid scarring can occur.



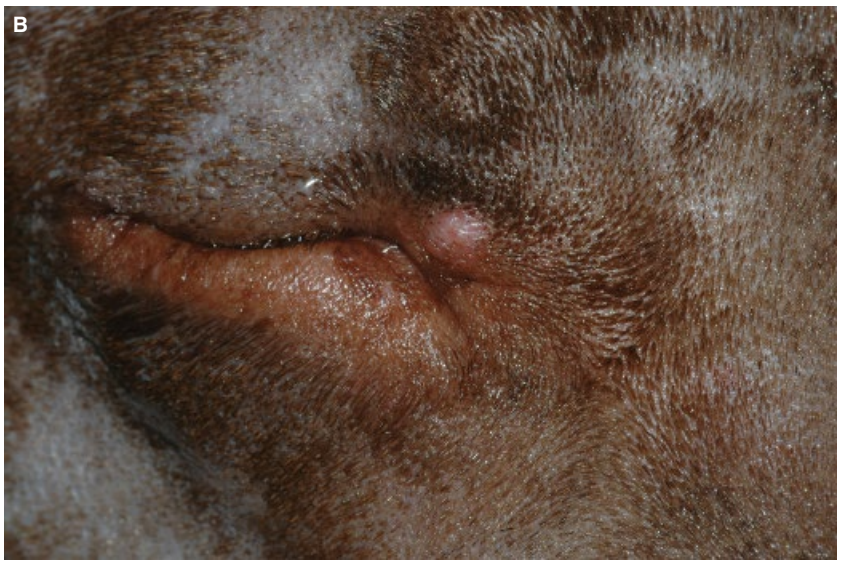


Figure 5.25 (A) Squamous cell carcinoma (SCC) of the eyelid in terrier-Boxer mix. The history was one of persistent nonresponsive ulceration of both eyelids and the third eyelid. Histopathology confirmed SCC. Tumor was excised by surgery. (B) Mast cell tumors in a dog. These tumors present as firm raised pink masses with or without ulcerations, or can appear as roughened areas of alopecia and erythema.

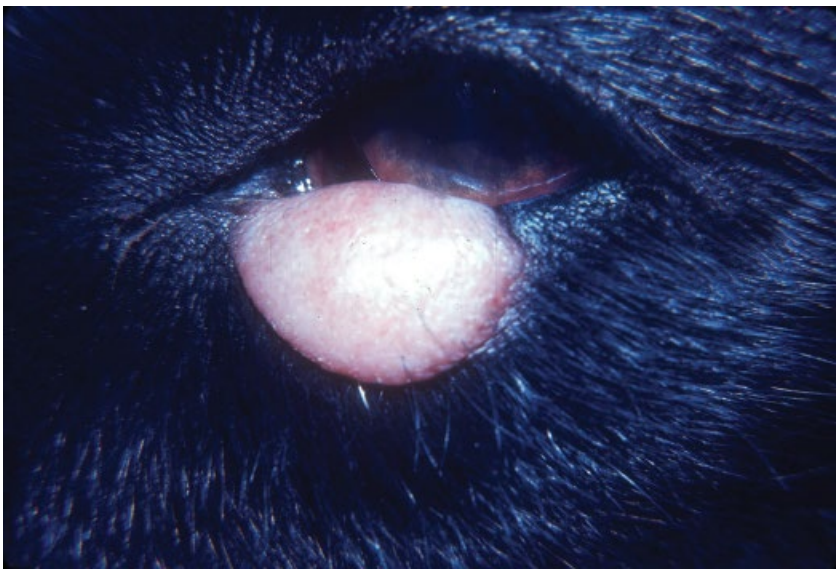


Figure 5.26 Histiocytoma in a 1-year-old Afghan Hound. This mass rapidly developed over 2 months, and just as rapidly regressed spontaneously over the next 2 months. In young dogs, eyelid histiocytomas can expand rapidly only to regress spontaneously a few weeks later. This tumor type in older dogs, however, can be quite malignant and should be removed with wide margins. Biopsy of these masses guides therapy.



Figure 5.27 Oral papillomatosis in puppies and young dogs, a viral-induced tumor, can also affect the eyelids. They also regress spontaneously in a few weeks, and removal is considered necessary only when corneal contact and ocular irritation develop.

Surgical removal of lid tumors should be performed when the tumor contacts the cornea or conjunctiva and causes irritation, blepharospasm, and lacrimation. Surgery is considerably easier when these tumors are small, and before extensive blepharoplastic procedures are necessary to repair the surgical defects.

Eyelid Tumors in Young Dogs

The eyelid masses in young dogs differ considerably from those most commonly encountered in aged dogs. Papillomas and histiocytomas are most commonly encountered in the young dog (Figures 5.26 and 5.27).

6

Canine Tear and Nasolacrimal Systems

Tear and nasolacrimal systems diseases are common in the dog and, for the most part, can be successfully treated medically. Deficiency of aqueous tears, or keratoconjunctivitis sicca (KCS), is the most frequent form of chronic conjunctivitis in the dog and the most common disease of the tear production and drainage system. The incorporation of the Schirmer's tear test into the routine ophthalmic examination has markedly increased the early clinical detection of this disease. Early KCS, when some tear production is still present, is the most responsive to lacrimomimetic drugs (topical cyclosporine or tacrolimus). Chronic KCS patients need to be examined periodically long-term and often for life. Forms of qualitative tear film deficiencies also occur wherein the amount of liquid tears is normal but the quality of the tears is impaired, which results in rapid evaporation or uneven distribution of the tear film and clinical signs consistent with KCS.

Nasolacrimal drainage disorders are characterized by epiphora, or the spillage of normal levels of tears onto the skin at the medial canthus. Blockage of the nasolacrimal system causes epiphora (normal production, inadequate drainage) and a chronic and medically resistant mucopurulent conjunctivitis. In contrast, lacrimation is an increased level of tear production, usually secondary to pain, which can also overwhelm the nasolacrimal drainage system. The two most useful diagnostic procedures for evaluating the tear drainage system are topical fluorescein passage from the eye to the nose (Jones' test) and the nasolacrimal flush (cannulating the upper or lower lacrimal punctum, flushing with sterile saline, and examining for exit from the nostrils). For chronic nasolacrimal drainage disorders and those conditions in which the blockage cannot be resolved by flushing and/or catheterization, dacryocystorhinography and other imaging techniques can be used to localize the blockage site. Surgical bypass procedures are available (conjunctivorhinostomy and conjunctivobuccostomy) to address these problems.

Diseases of the Tear Producing System

The primary tear producing glands in the dog are the lacrimal gland (located in the anterior dorsolateral orbit) and the superficial gland of the nictitating membrane. The tears are a composite of serous tears produced by the lacrimal gland, mucoserous tears produced by the superficial gland of the nictitans, mucin from the conjunctival goblet cells, and lipid from the meibomian glands.When this tear complex is on the cornea it is referred to as the precorneal or preocular film. The different types of artificial tear preparations are designed to mimic natural tear composition, and specific types are available when particular parts of the tears are missing.

Quantitative Changes in Tears

Acute KCS occurs infrequently, and is often overshadowed by the signs of corneal ulceration. As corneal ulcers are painful and cause lacrimation, any corneal ulcer with normal to low Schirmer's tear test levels should also be considered a candidate for KCS. In acute KCS, a rapidly progressive central corneal ulcer develops with malacia that – without surgical intervention – can advance to descemetocele and iris prolapse within 12–24 hours. Acute KCS is also not infrequent in the toy and small breeds of dogs treated with sulfonamide-related drugs and certain nonsteroidal anti-inflammatory drugs in the treatment of arthritis (Figure 6.1).

Treatment consists of topical artificial tears, broad spectrum antibiotics, 1% tropicamide (for the iritis; atropine is avoided as it decreases tear production bilaterally), and sometimes topical serum, cyclosporine or tacrolimus, and, if necessary, bulbar conjunctival grafts for the often rapidly progressive corneal ulcer. Increases in tear production produced by cyclosporine or tacolimus often

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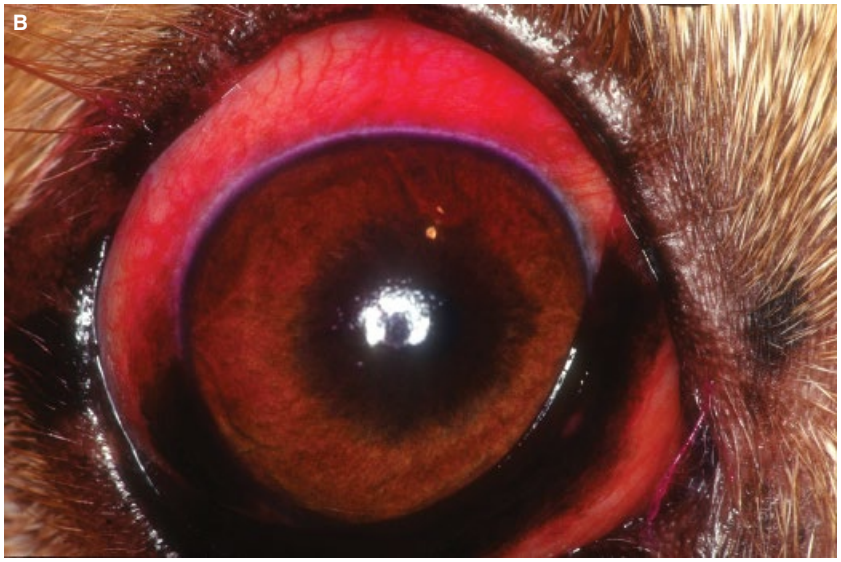




Figure 6.1 Acute keratoconjunctivitis sicca (KCS) in dogs. (A) Acute keratoconjunctivitis (KCS) in a puppy secondary to canine distemper. Often distemper-associated KCS is bilateral and acute. (B) Acute KCS in a terrier-mix dog. The eye has been stained with rose Bengal. Note the lack of corneal luster and diffuse stain retention by both the cornea and bulbar conjunctiva. (C) Acute KCS in a Shih Tzu. Note the superficial ulcerations of both corneas. Profound blepharospasm is often present with acute onset KCS. This patient has had a topical anesthetic applied to facilitate examination.

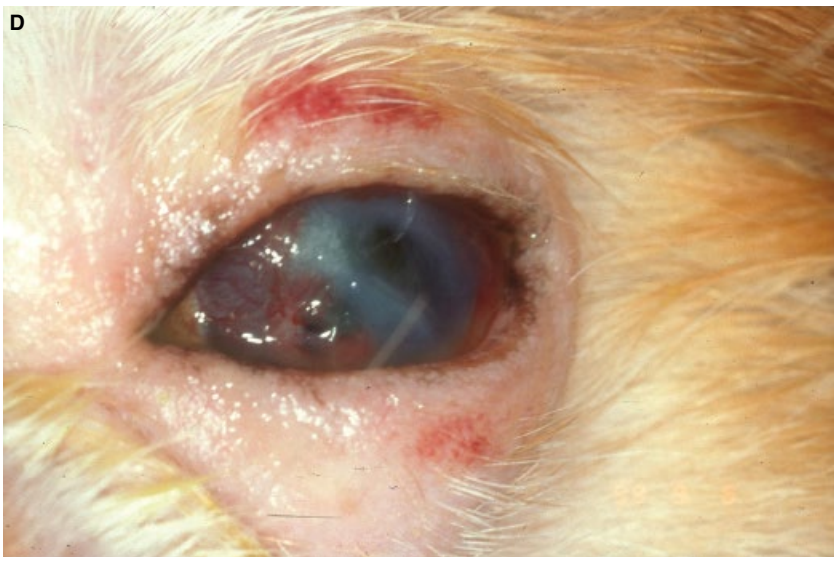


Figure 6.1 (*Continued*) (D) Acute KCS in a young dog with corneal ulceration and iris prolapse.

require several weeks and can be quite gradual. The Schirmer's tear test can detect these gradual increases while visual inspection may not.

Chronic Keratoconjunctivitis Sicca

Chronic KCS occurs frequently in dogs, and more often in certain breeds, such as Beagle, Cavalier King Charles Spaniel, English Bulldog, Lhasa Apso, Shih Tzu, West Highland White Terrier, Pug, Bloodhound, and the American Cocker Spaniel (Figure 6.2). The most common cause of chronic KCS is an immune-mediated dacryoadenitis. Other etiologies include drug toxicity (especially the sulfonamides), irradiation, neurogenic insults, surgical trauma (ear and facial surgery), trauma, previous prolapse of the nictitan gland, and systemic diseases.

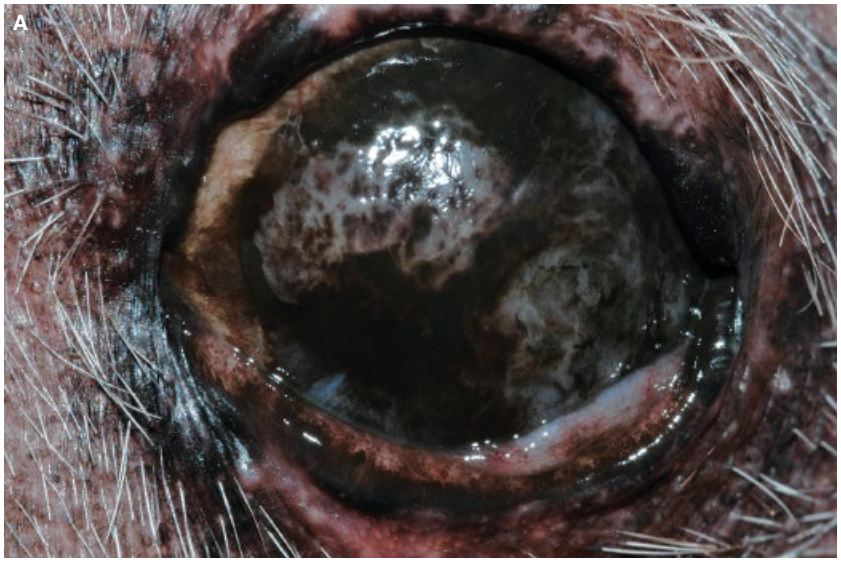
In its early stages, chronic KCS appears as conjunctivitis that responds temporarily to topical antibiotics, only to recur after a few weeks following discontinuation of therapy. Routine use of Schirmer's tear tests for these patients is essential to diagnose the early stage of this disease, when it also has the highest possibility of responding to topical cyclosporine. With chronically low levels of tears, progressive changes occur in the conjunctiva and cornea. The conjunctiva remains hyperemic, edematous, thickened, and often variably pigmented. A persistent thick and tenacious mucopurulent exudate occurs. The cornea eventually becomes vascularized, pigmented, and causes progressive vision impairment. Movement of the eyelids across the cornea and conjunctiva is difficult and painful due to the dry surfaces and abrasion.

Fortunately, over 80% of chronic KCS patients respond to cyclosporine, but often this topical drug must be administered long-term, if not for the life of the animal. Antibiotics and corticosteroids are also employed topically to eliminate intermittent secondary bacterial conjunctivitis. Higher frequencies or the more potent topical corticosteroids (1% prednisolone or 0.1% dexamethasone) must be used with caution in the brachycephalic breeds, as their "dry" corneas are more susceptible to ulceration. If tear levels are not improving, oral pilocarpine (one drop of 2% pilocarpine per 5 kg; dosage adjusted to the individual dog) placed in the pet's food can be used to supplement topical cyclosporine or as a replacement. Excessive oral pilocarpine in the food can cause vomiting (as the first parasympathetic sign of toxicity); slight salivation after drug ingestion is usually a good guide that the dose of the oral pilocarpine is sufficient.

Response to lacrimogenic and lacrimomimetic therapy causes the luster to gradually return to the corneal and conjunctival surfaces. Corneal pigmentation and vascularization usually gradually improve over several months. The character of the conjunctival exudates gradually changes from a copious, thick, and tenacious exudate, to a mucus, and then serous discharge. The conjunctival inflammation gradually resolves as tear levels return to normal.

Parotid Duct Transposition

Parotid duct transposition is now quite uncommon, because of the success of lacrimomimetic drugs. Unfortunately, there are some dogs with chronic KCS that fail to respond to topical immunomodulating or oral pilocarpine therapy, and parotid duct transposition is recommended (Figure 6.3). Parotid gland and duct function should be demonstrated preoperatively by the application of 1% ophthalmic atropine (used for its bitter taste) to observe the flow of saliva from its papilla immediately





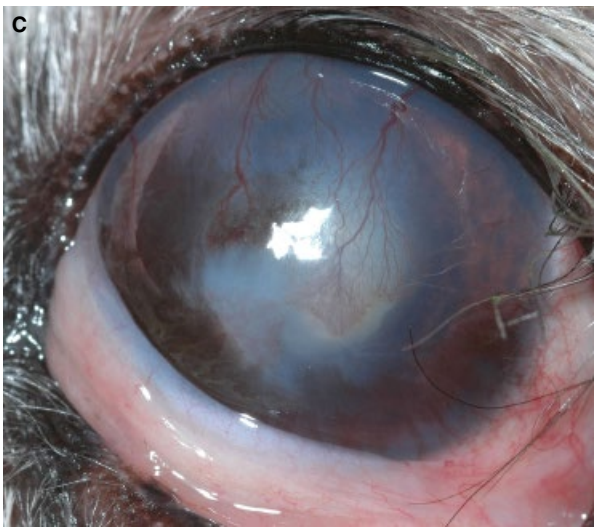




Figure 6.2 (A) Chronic KCS in a Chinese Crested Dog. Note the superficial corneal vascularization, pigmentation, and fibrosis. (B) Chronic KCS in a Shih Tzu of several months' duration. Note the vascularization and pigmentation of the cornea. (C) Chronic KCS in an American Cocker Spaniel after several weeks of topical tacrolimus. The cornea has regained its most of its luster, but conjunctivitis, keratitis, and corneal pigmentation are still present. (D) Neurogenic KCS in a Miniature Schnauzer characterized by chronic KCS and a dry nostril on the same side. Note the blepharospasm and corneal opacity. This type of KCS is secondary to parasympathetic denervation of the lacrimal gland and will often respond to oral pilocarpine therapy. Lesions causing this disease are associated with otitis media or interna and petrositis of the petrous temporal bone.



Figure 6.3 (A) Postoperative appearance several months after bilateral parotid duct transposition in a Miniature Schnauzer female with chronic KCS that failed to resolve with medical therapy. (B) The most frequent short- and long-term postoperative complication after parotid duct transposition is the onset of mineral deposits that irritate the corneal and conjunctival surfaces. These same deposits can cover the eyelids. (C) Note the mineral deposits on the eyelids in this postoperative parotid duct transposition patient. There is a conjunctival graft present on the cornea that was placed to address a previous descemetocele.

caudal of the carnassial tooth. In this procedure, performed either by oral or lateral approach, the parotid duct is relocated to the eye through a subcutaneous tunnel. The parotid duct papilla is secured in the ventrolateral conjunctival fornix. The most frequent short- and long-term postoperative complication after parotid duct transposition is the development of mineral deposits that irritate the corneal and conjunctival surfaces. If corneal ulcerations are also present, the mineral deposits can enter the corneal stroma permanently. Ligation of some, but not all, of the minor parotid gland ducts at the bottom of the gland seems the most effective at reducing flow and mineral substances. Treatment with 1–2% EDTA (ethylenediaminetetraacetic acid) ointment and eye washes is variably successful.

Qualitative Changes in Tears

Patients with qualitative tear film disorders have clinical signs that mimic KCS, but normal levels of aqueous tear production (as measured by the Schirmer tear test) (Figure 6.4). In these cases, either the mucin or lipid fractions of the tears are reduced. If the meibomian glands

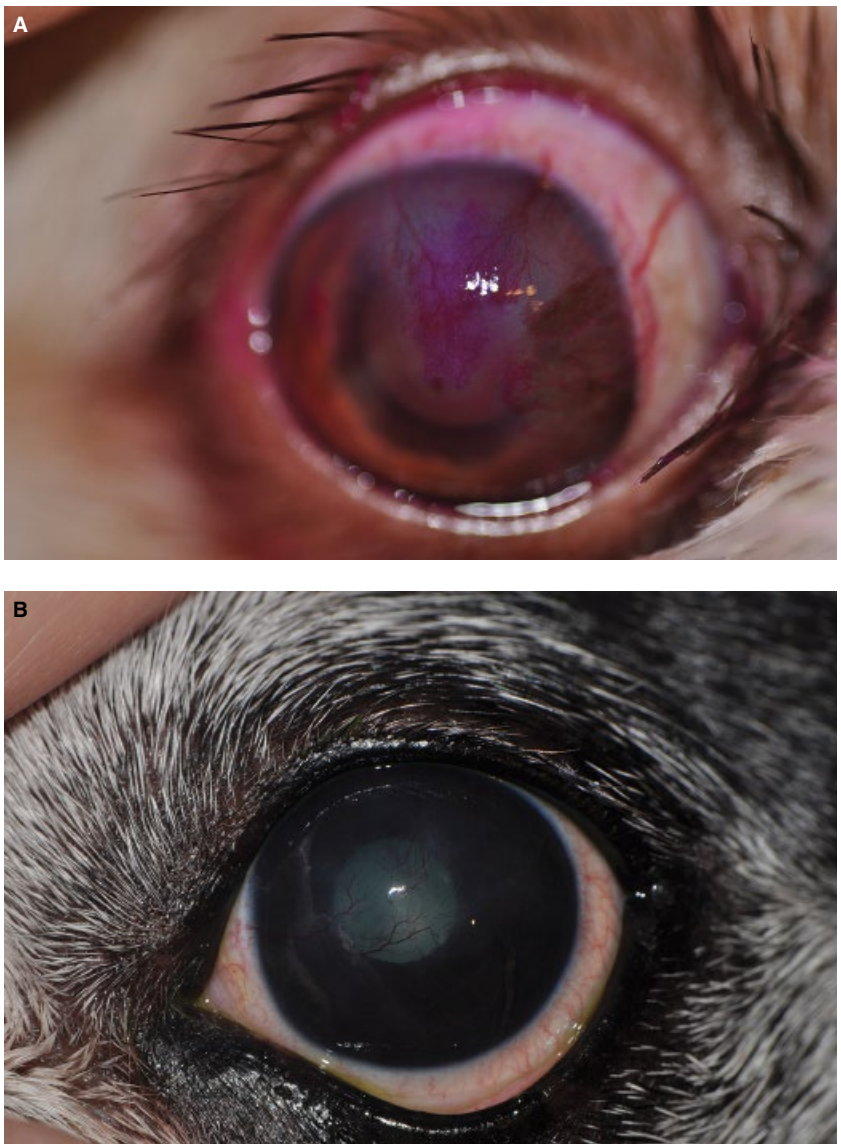


Figure 6.4 (A) Qualitative KCS in a Shih Tzu. This dog's aqueous tear production was sufficient; however, the tear quality was impaired. Note the rose Bengal retention and the corneal vascularization. (B) Qualitative KCS in a mixed-breed dog. Note the conjunctival hyperemia and the corneal vascularization and fibrosis.

are not contributing enough lipid, liquid tears will evaporate prematurely. This can be demonstrated with the tear break-up time, where topical fluorescein is applied to the ocular surface and the eyelids are held open while a cobalt blue light source illuminates the surface. The time until the tear surface breaks up or dry spots develop is measured in seconds (less than 10 seconds is abnormal). If the conjunctival goblet cells are producing inadequate amounts of mucin, the tear film will not adhere to the corneal surface appropriately. Topical rose Bengal or lissamine green stain will adhere to the corneal epithelium when the surface mucin is inadequate.

These qualitative tears disorders are treated with topical drugs to improve and prolong the tear film stability. With bacterial meibomianitis, bacterial culture may be indicated for determination of the most effective antimicrobial agent. Impacted meibomian glands can be carefully expressed to remove inspissated secretions (see also Figure 5.21). Topical specific tear replacements include mineral oil, liquid lanolin, and other petrolatum products. When mucin deficiency is diagnosed, medical therapy includes viscoelastic agents including sodium hyaluronate, chondroitin sulfate, and 1–2% methylcellulose, as well as antibacterial and sometimes anti-inflammatory drugs.

Diseases of the Nasolacrimal Drainage System

Acute epiphora produces a moist area at the medial canthi and/or lids, whereas chronic epiphora, as commonly observed in the toy and miniature breeds of dogs, causes



Figure 6.5 Young dog with medial lower entropion that has collapsed the lower lacrimal punctum, and redirected tears onto the medial canthus. With constant moisture in this area, chronic dermatitis and tear staining result. Surgical correction is indicated but varies depending on the individual's conformation. Options include medial canthoplasty or removal of a small triangular or oval section of skin immediately below the medial entropion, avoiding the eyelid margin, to evert the medial lower lid margin and open the lower lacrimal punctum.

secondary blepharitis and brownish staining of the hair. Presumably, the tear stains represent the interaction of tears, skin products, and bacteria, although no studies have been reported documenting this process.

Chronic Epiphora in Toy and Miniature Dogs

The treatment of chronic epiphora in toy and miniature breeds of dogs is challenging. Long-term systemic and topical antibiotics have reported variable success. Use of 2–2.5% medical hydrogen peroxide can be used to bleach the area. White chalk has been recommended to dry and whiten the area; possible antibacterial activity has also been suggested. Surgical procedures to correct the lower medial entropion, which causes closure of the lower lacrimal punctum, and improve the entry of tears into the lower lacrimal punctum offers a more permanent solution and is often recommended (Figure 6.5).

Acute Dacryocystitis

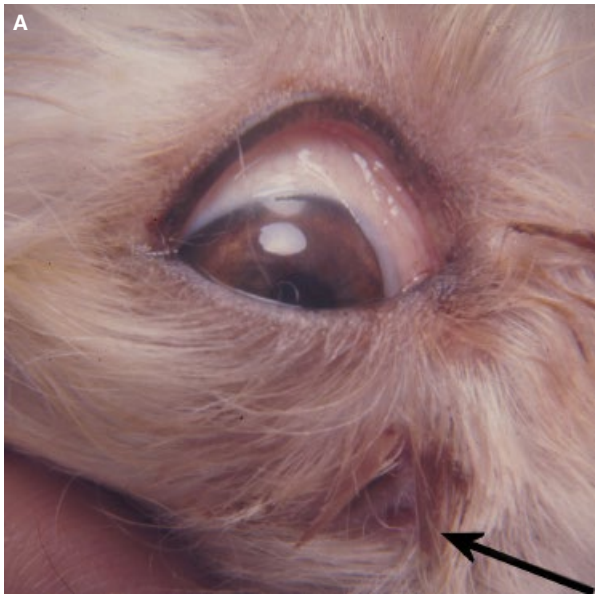
Acute dacryocystitis occurs infrequently in the dog. When it does occur, it is usually associated with a nasolacrimal duct obstruction (inflammation, foreign body migrating through lower lacrimal punctum to lodge in the lacrimal sac) and a secondary bacterial infection. Presenting signs include intermittent conjunctivitis and medial canthal swelling with considerable local pain (Figure 6.6). If flushing through the upper or lower lacrimal punctum is impossible, the nasolacrimal sac can be accessed directly (dacryocystotomy). Medical treatment consists of topical and systemic antibiotics, topical corticosteroids, and repeated nasolacrimal flushes until the inflammation has resolved and patency is maintained.



Figure 6.6 Acute dacryocystitis in this dog is characterized by painful swelling of the medial canthus and obstruction of the lower lacrimal punctum by inflammatory exudate and, not infrequently, small foreign bodies.

Chronic Dacryocystitis

Chronic dacryocystitis represents the progression of acute disease and often formation of a fistula that connects the abscessed nasolacrimal sac with the skin surface





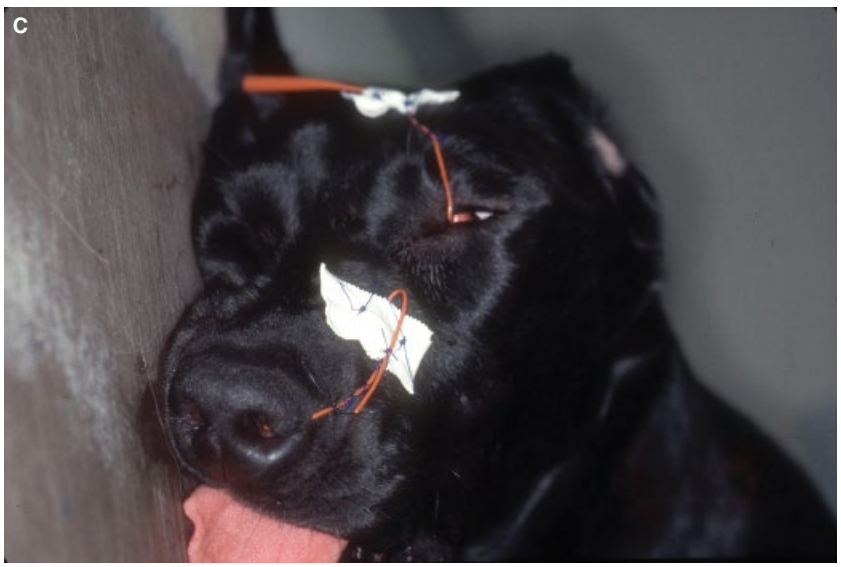


Figure 6.7 (A) Longer term dacryocystitis often develops a ventromedial fistula (arrow) which provides a bypass for tears. (B) Nasolacrimal flush performed through upper lacrimal punctum produces the extrusion of inflammatory debris (and occasionally a foreign body, i.e., plant awn) through this bypass. Additional nasolacrimal flushes may be necessary to lavage the nasolacrimal sac of all exudate and debris and then to establish patency of the entire system to the nose. (C) Treatment of chronic nasolacrimal obstruction with catheterization with monofilament nylon suture or silicone tubing which is secured by suture at the medial lower eyelid and external to the nostril.

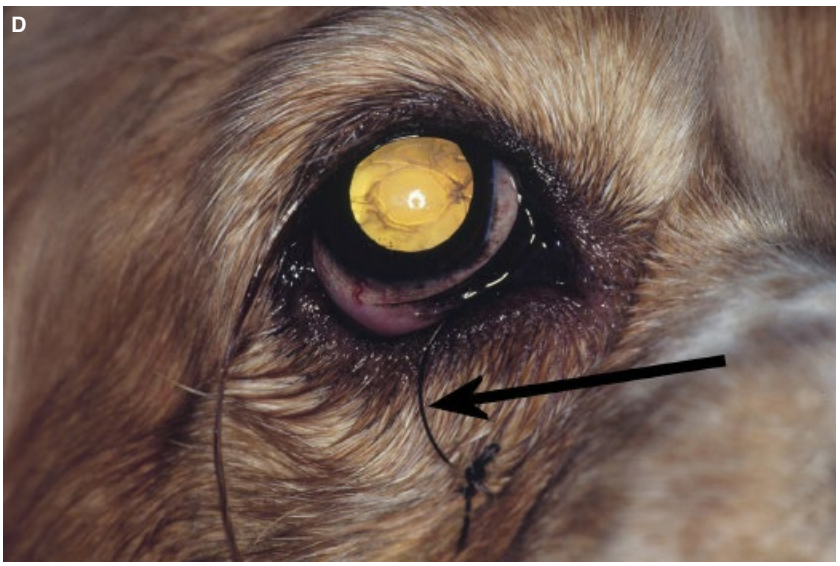


Figure 6.7 (*Continued*) (D) Close-up of suture (arrow) used to catheterize in an American Cocker Spaniel in the upper part of the nasolacrimal system. The dog had previously had cataract surgery and placement of an intraocular lens.



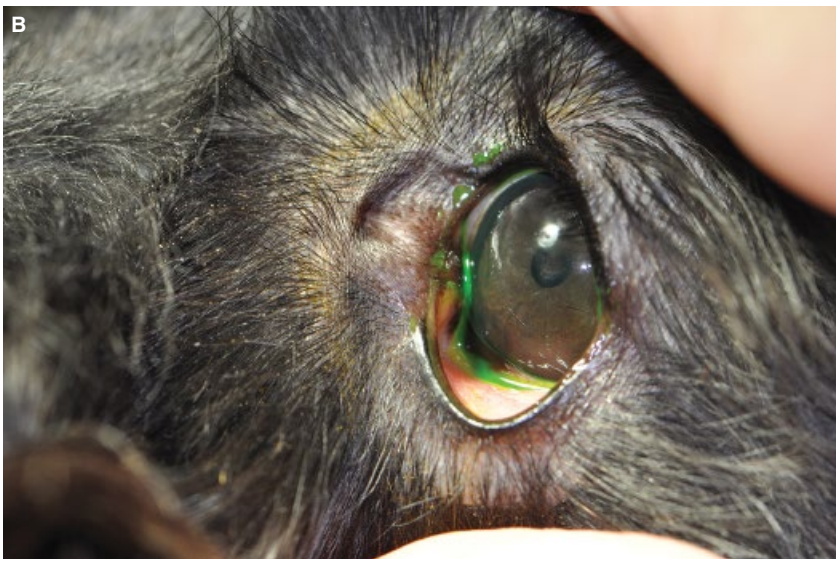


Figure 6.8 (A) Young Pekingese with a dacryocele. Communication between the nasolacrimal sac and dacryocele was confirmed by nasolacrimal flush and dacryocystorhinography. (B) Dacryops in a Scottish Terrier. Note the small swelling at the medial canthus.



Figure 6.8 (*Continued*) (C) Same patient as shown in Figure 6.8B. Note the medial canthal swelling and the lack of fluorescein exiting the nares on the same side.

in the medial canthus (Figure 6.7). Nasolacrimal flush reveals variable inflammatory debris from the opposite lacrimal punctum after the initial flush, and irrigation fluid exiting the fistula. Once patency of the entire nasolacrimal system is achieved, the fistula will usually heal by second intention. Medical treatment consists of topical and systemic antibiotics, and topical corticosteroids.

If the system patency is not maintained between nasolacrimal flushes, catheterization with monofilament nylon sutures or small silicone tubing is necessary for several weeks. Nasolacrimal catheterization, using 2-0 monofilament nylon suture or small diameter silicone tubing, is used to treat persistent nasolacrimal duct obstructions. Under sedation or short-term general anesthesia, a 2-0 monofilament tubing is carefully passed through the entire nasolacrimal system, starting at either the upper or lower lacrimal punctum. Both ends of the catheter are secured by simple interrupted sutures to the adjacent medial canthus and lateral nares, respectively. As long as the catheter is in position, topical antibiotics and corticosteroids are instilled to facilitate maintenance of patency, treat the infection, and prevent fibrosis and stenosis. An E-collar is maintained as long as the nasolacrimal catheter is in place.

Dacryoceles

Dacryoceles, also called dacryops, are cyst-like structures associated with nasolacrimal apparatus (Figure 6.8). They occur uncommonly in the dog. They can be demonstrated by contrast media and dacryocystorhinography. A more specific type, dacryocystocele, indicates cyst formation involving the nasolacrimal sac. Dacryocystocele present as a focal fluid-filled enlargement at the medial canthus. The condition is not painful, and the size of the cyst can change periodically. A nasolacrimal flush through the upper nasolacrimal punctum can cause the cyst to temporarily increase or decrease in size. Dacryocystorhinography can demonstrate a direct connection between the nasolacrimal sac and cyst. Resolution of dacryocystocele requires excision of the cyst and closure of the defect in the nasolacrimal sac wall or injection of a sclerosing agent.

7

Canine Conjunctiva and Nictitating Membrane (Nictitans)

Diseases of the canine conjunctiva and nictitating membrane (nictitans/third eyelid/membrana nictitans) are common in the dog, and for the most part easily visualized and diagnosed. Examination of both the conjunctiva and nictitating membrane is by direct inspection; occasionally some magnification is useful. Both congenital and traumatic lesions are infrequent, but inflammatory diseases are common and often secondary to lid anomalies, tear deficiency, and abnormal nasolacrimal drainage, among other causes. "Cherry eye" or inflammation and prolapse of the nictitating tear gland is common in young dogs, and surgical re-positioning of the gland beneath the nictitans is recommended. Some of these dogs are still prone to keratoconjunctivitis sicca (KCS) in the future, so continued monitoring is recommended.

Neoplasms of the conjunctiva and nictitans are less frequent than those affecting the eyelids, but are less predictable and decidedly more malignant. Most conjunctival neoplasms require a fairly wide resection, and recurrences are more likely. Adjunctive therapy is often recommended to reduce the possibility of recurrence. Neoplasms of the nictitans usually involve the tear gland, are quite malignant, and usually require excision of the entire nictitans.

Congenital or Developmental Disease of the Nictitans

Encircling Nictitans

The nictitans, as an appendix of the medial conjunctiva, functions with passive dorsolateral movements across the cornea. Occasionally, extensions from both the upper and lower margins of the free border of the nictitans occur. These mucosal extensions are often pigmented and extend for several millimeters along the middle of the bulbar conjunctiva. These are generally incidental findings that do not cause clinical signs, but when extensive they can impair nictitans movements. They occur most frequently in the American Cocker Spaniel (Figure 7.1).

Conjunctival Dermoid

As described in Chapters 5 and 8, dermoids or choristomas are normal skin that develops in abnormal locations (eyelid, conjunctiva, cornea). These raised and sometimes pigmented masses often produce long and irritating coarse hair, which prompts presentation (Figure 7.2). Treatment is careful surgical removal of the entire mass.

Eversion of Nictitans

Eversion or, less frequently, inversion of the leading margin of the nictitans occurs primarily in the large and giant breeds of dogs (i.e., German Shorthaired Pointer, St. Bernard, Great Dane, and Newfoundland). Presenting clinical signs are epiphora, limited blepharospasm, and a pink raised mass in the medial canthus or in front of the nictitans (Figure 7.3). Closer examination reveals eversion of the leading edge of the nictitans (inversion or curling inward is rare) thereby exposing the nonpigmented bulbar (deeper) surface of the nictitans.

Treatment is surgical removal of the bent or U-shaped cartilage (see Figure 7.3 insert) located immediately beneath the leading margin and the extensions of the cartilage (not the entire nictitans). Surgery is performed through a small incision on the palpebral conjunctival surface immediately over the defect and often no sutures are required for closure. Incision of the leading margin of the nictitans should be avoided. The immediate postoperative appearance is a normal nictitans. Sometimes the leading margin of the nictitans remains somewhat deformed, but after several days to a few weeks returns to normal position. Recently, a procedure utilizing thermal cautery to straighten the bent cartilage without excision of the deformed segment has been described.

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Figure 7.1 Encircling nictitans in an American Cocker Spaniel. Note the pigmented conjunctival membrane extending from the leading margin of the nictitans laterally.



Figure 7.2 Dermoid of the lateral bulbar conjunctiva in an English Bulldog puppy. Note the mass is lightly pigmented and covered by long coarse hair. Dermoids, if small, are often missed, but when the long coarse hairs begin to grow ocular irritation begins and the condition becomes apparent.

Cherry Eye (Inflammation and Prolapse of Nictitans Gland)

"Cherry eye" or the inflammation and prolapse of the tear gland of the nictitating membrane is a common disease of dogs less than 1 year of age, and affects certain breeds more commonly, especially the American Cocker Spaniel, Boston Terrier, Miniature Schnauzer, English Bulldog, and Beagle (Figure 7.4). Some of the same breeds with this disease are also predisposed to KCS.

The history is of recurrent nictitans inflammation and protusion with eventual prolapse of the gland which becomes medically nonresponsive to topical corticosteroids and antibiotics. Close inspection reveals the anterior surface of the nictitans to be normal, but with protraction of the nictitans by thumb forceps, the full extent of the inflamed and prolapsed gland protruding above the leading margins can be appreciated.

Surgical correction of "cherry eye" is usually necessary once the gland prolapse occurs. The chance of KCS developing is higher if the gland is left exposed than if it is surgically replaced. There are several surgical procedures (pocket, imbrication, anterior, posterior and intranictitans anchoring), and each has advantages and limitations. The goals of surgery are to replace the gland, reduce its inflammation, and permit continued movement of the nictitans. Recurrences can develop after surgical repositioning, particularly with very large or chronic swellings of the nictitans gland. Long-term monitoring of these dogs with Schirmer tear tests is recommended as some dogs will develop KCS even after gland replacement surgery.

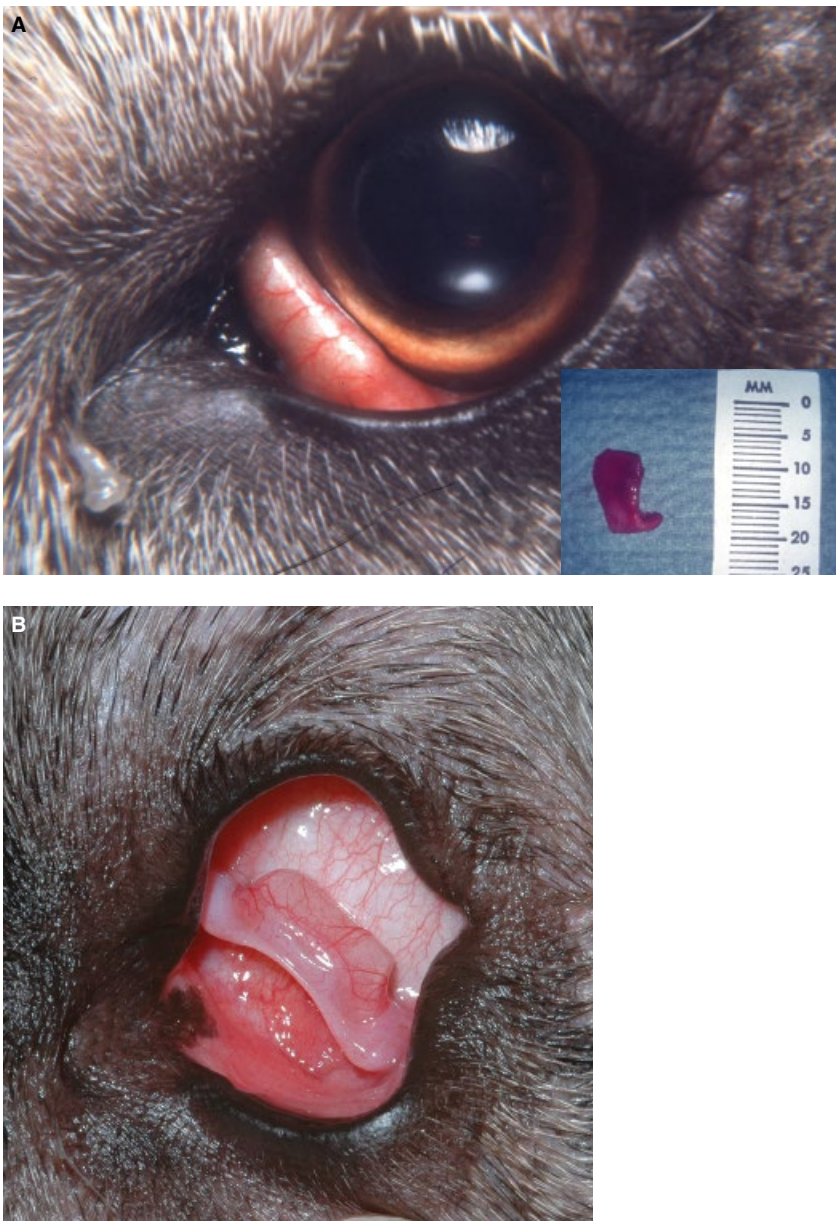


Figure 7.3 (A) Everted cartilage of the nictitans in a German Shepherd puppy. Note the leading pigmented margin is absent and folded over (everted) exposing the defective cartilage (see insert: bent cartilage upon removal). (B) Scrolled and everted nictitans cartilage in a young Great Dane.

Protrusion of Nictitans

Protrusion of the entire nictitans can be associated with persistent conjunctivitis and epiphora (Figure 7.5). Protrusion can be primary (no antecedent disease detected), or secondary to microphthalmia, enophthalmia, space-occupying orbital diseases, Horner's syndrome, tetanus, and other diseases.

Removal of the entire nictitans is not recommended for this condition, but if ocular disease and vision impairment are present, the middle section of the nictitans can be excised, thereby reducing the nictitans by one-third to half in overall size.

Plasmoma or Plasma Cell Infiltration of Nictitans

Plasma cell infiltration or plasmoma of the nictitans appears as an immune-mediated disease in which the nictitans becomes infiltrated with plasma cells and lymphocytes, with follicle formation and depigmentation (Figure 7.6). It occurs most frequently in certain breeds, such as the German Shepherd, Belgian Sheepdog, Borzio, Doberman Pinscher, and English Springer Spaniel. Plasmoma is often concurrent with pannus (chronic superficial keratitis) and/or plasma cell infiltration of the medial canthus (blepharitis). Therapy consists of topical





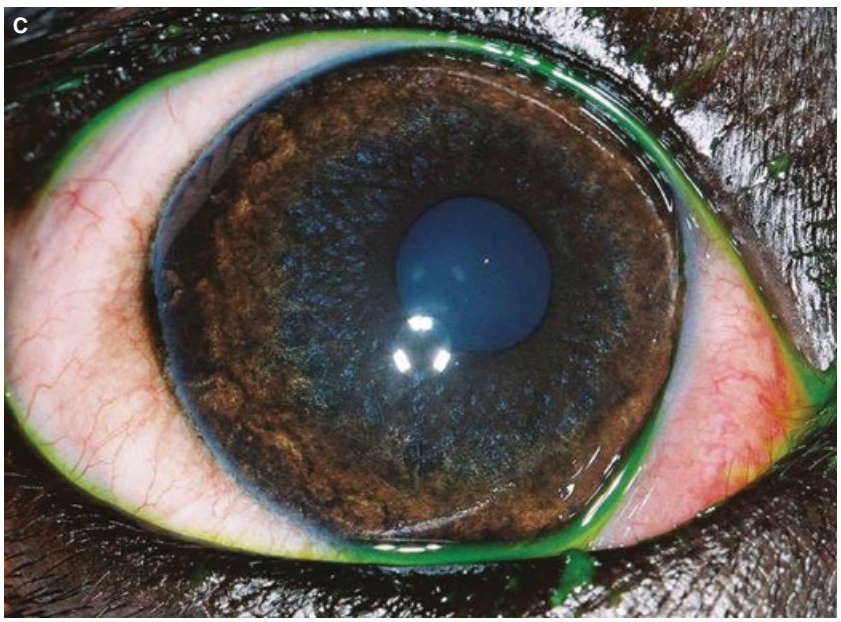
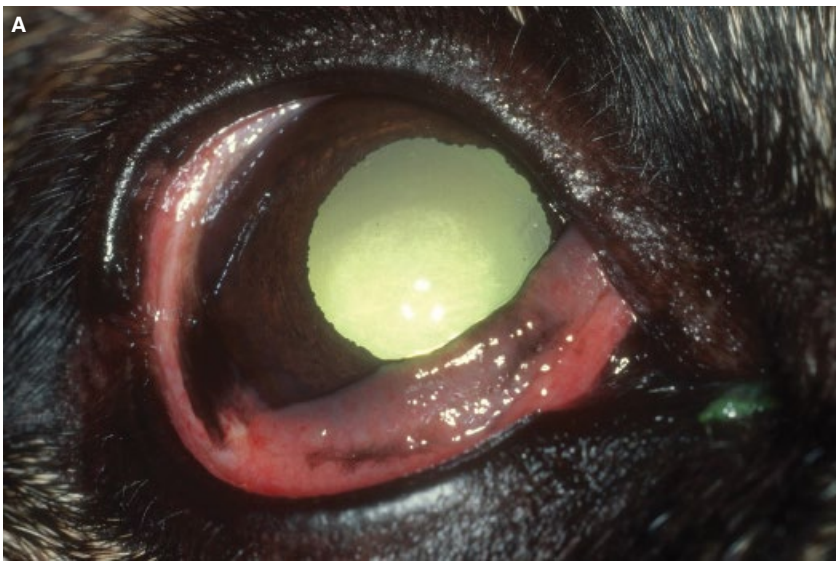


Figure 7.4 (A) Prolapse of both nictitans tear glands in a Boston Terrier puppy. The enlarged and prolapsed glands are anterior to the nictitans leading margin. (B) Close-up of a 1-year-old English Bulldog puppy with a unilateral "cherry eye." (C) Postoperative appearance of American Cocker Spaniel 1 week later using the intranictitans surgical procedure (an anchoring procedure that permits unrestricted nictitans movements).



Figure 7.5 Bilateral protrusion of the nictitans in a young Beagle with nonpigmented leading margins of both nictitating membranes. The cause was not determined.



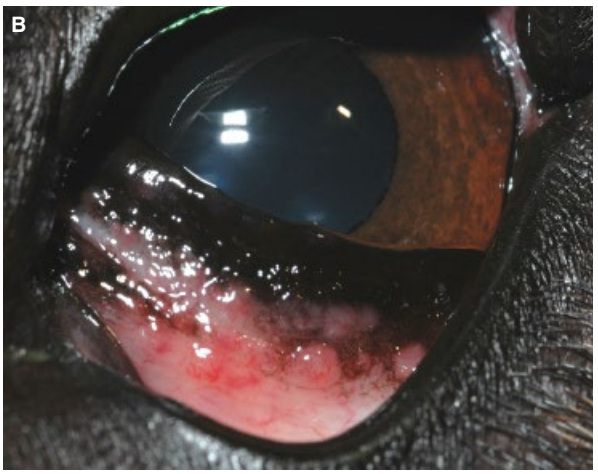


Figure 7.6 Examples of two stages of plasmoma or plasma cell infiltration of the nictitating membrane in a German Shepherd Dog. (A) The inflamed nictitans is thickened and slightly protruding, and its leading margin is losing its pigment. (B) Advanced plasma cell infiltration of the nictitans. The nictitans can become so thickened that the resulting protrusion covers some of the cornea and retraction of the third eyelid is impaired.



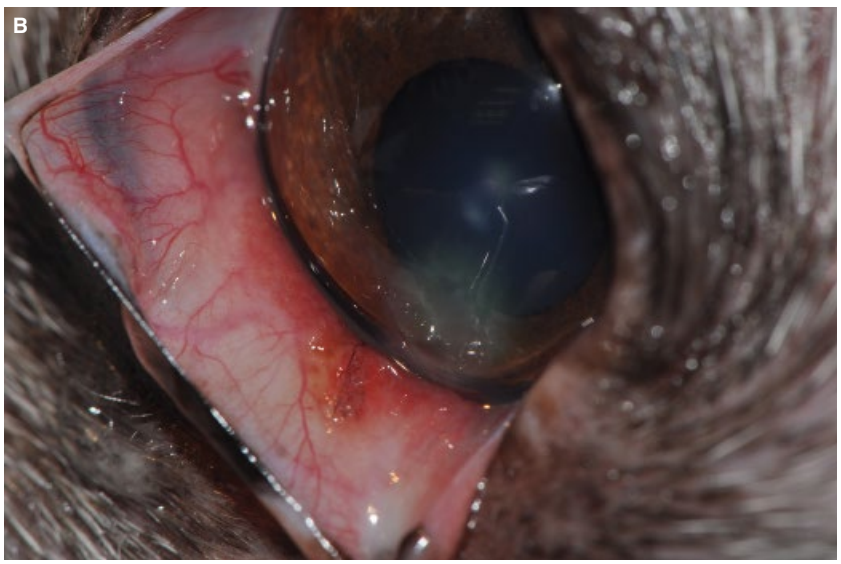


Figure 7.7 Foreign bodies (usually plant material) occasionally lodge in the deep (bulbar) fornix of the nictitating membrane. Presenting signs are lacrimation, swelling of the medial canthus, variable blepharospasm, protrusion and hyperemia of the nictitans, and often a ventromedial nonhealing corneal ulcer. Any patient with ventromedial corneal ulcer should always have a very careful examination of the nictitans fully protracted by forceps. (A) An example of foreign body in the posterior fornix of the nictitans in a Laborator Retriever mix. Often, topical anesthesia will permit grasping and protracting the nictitans with forceps to examine the entire posterior surface of the nictitans and its posterior fornix. (B) Ventromedial corneal ulcer secondary to a foreign body behind the nictitans in a mixed-breed dog.

corticosteroid and an immunomodulatory such as cyclosporine to reduce inflammation. This condition tends to be chronic and may be recurrent if medications are tapered or discontinued prematurely.

Nictitans Foreign Bodies

Most often affected dogs are the hunting and large breeds maintained outside. History includes a non-healing corneal ulcer, especially in the ventronasal quadrant (Figure 7.7).

Upon examination under topical anesthesia, protraction of the nictitans by forceps or a cotton-tipped applicator, and careful examination of the deep (bulbar) fornix behind the nictitans, reveals one or more entrapped foreign bodies. These foreign bodies are removed with fine forceps and the area flushed vigorously with sterile saline. The corneal ulcer is treated medically.

Neoplasia of Nictitans

Neoplasms of the nictitans appear less commonly in the dog than conjunctival masses, and usually present as slow-growing masses on either the anterior or posterior surface of the nictitans (Figure 7.8). Reported neoplasms include melanomas, adenocarcinomas, mastocytomas, squamous cell carcinomas, papillomas, hemangiomas, hemangiosarcomas, angiokeratomas, and lymphosarcomas.

Most neoplastic masses of the nictitans occur in older dogs (10+ years). They are usually confined to its anterior surface, and are usually malignant by histopathology. Removal of the entire nictitans is recommended.



Figure 7.8 Examples of primary neoplasms of the nictitans in the dog. (A) Adenocarcinoma of the nictitans in an old American Cocker Spaniel. The pink and ulcerated mass is arising from the nictitans tear gland and has extended over its leading margin. Axial corneal degeneration is present concurrently. (B) Hemangioma arising from the anterior surface of the lower nictitans. Recommended therapy was a combination of surgical excision with wide margins followed immediately with adjunctive therapy (cryotherapy, beta-irradiation). (C) Squamous cell carcinoma of the nictitans involving its anterior (palpebral) surface. Excision was performed involving wide margins followed by cryotherapy.

If recurrence occurs, the mass has infiltrated into the medial anterior orbit. In one study of 47 nictitans neoplasms, 8 recurred after surgical excision.

Conjunctivitis

Infectious conjunctivitides in the dog are caused by bacterial, viral, fungal, rickettsial, or parasitic agents. Fortunately, most conjunctivitides in the dog are secondary to lid defects, tear abnormalities, nasolacrimal apparatus blockages, and other eye diseases, rather than infectious causes (Figure 7.9). Primary infectious conjunctivitis appears rare in dogs. Bacterial conjunctivitis is characterized by limited blepharospasm, some eyelid edema, variable mucopurulent discharges, conjunctival hyperemia, and chemosis. Most cases of bacterial conjunctivitis are secondary to KCS. Frequent bacteria isolates include *Staphylococcus* sp. and *Streptococcus* sp.; antibiotic sensitivities are important to determine as a guide for topical therapy, especially in chronic or unusually severe cases.

Cytology in persistent and recurrent conjunctivitides can be informative as to the etiology. Broad spectrum antibiotics are generally useful. As most bacterial



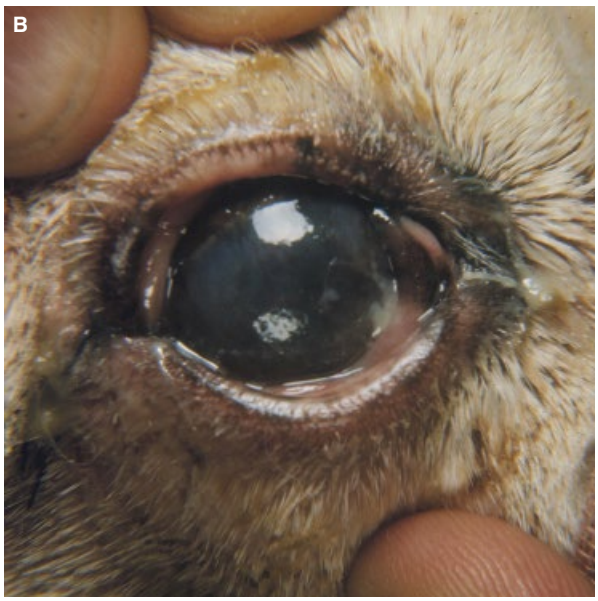




Figure 7.9 (A) Secondary bacterial conjunctivitis associated with a combined entropion–ectropion and a nictitans abnormality. (B) Bacterial conjunctivitis secondary to keratoconjunctivitis sicca (KCS) and prolapse of the nictitans gland in this English Bulldog. Thick mucopurulent discharge is present. Schirmer tests should always be part of all conjunctivitis patients' evaluations. This dog was treated for persistent conjunctivitis that only temporarily responded to topical antibiotics, and then pigmentation of the cornea started. Early KCS is often first presented for conjunctivitis when tear levels are not noticeably low. (C) Ligneous conjunctivitis in a Doberman Pinscher dog. This immune-mediated disease results in extensive membranous proliferations around the conjunctiva which can be so severe that they obscure visualization of the globe.

conjunctivitides are secondary, complete resolution of the conjunctivitis depends on elimination of the predisposing condition. Conjunctivitis can also occur as a manifestation of allergic or immune-mediated disease.

Follicular Conjunctivitis

Follicular conjunctivitis usually signals chronic inflammation and often allergic conjunctivitides. Medical history usually reveals epiphora, variable eyelid swelling and pruritus, conjunctival hyperemia and chemosis, and a persistent serous discharge (Figure 7.10). Allergic conjunctivitides are often seasonal (summer and fall).

Upon examination variable numbers of 1 mm transparent follicles are distributed in large numbers over the bulbar (deep) conjunctiva and anterior or posterior surfaces of the nictitating membrane. Conjunctival cytology reveals



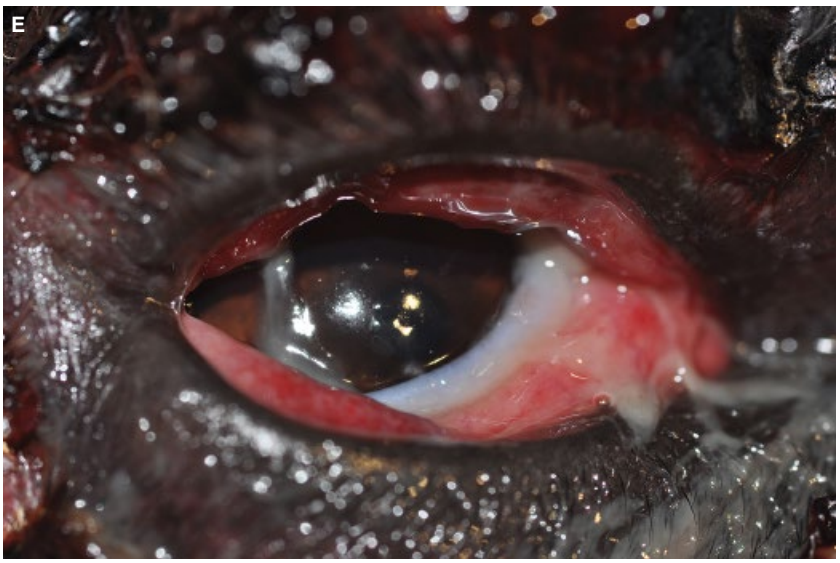


Figure 7.9 (*Continued*) (D) Conjunctivitis in an allergic dog. (E) Conjunctival hyperemia, chemosis, and hemorrhage secondary to periocular pit-viper envenomation in a Pit Bull mix.

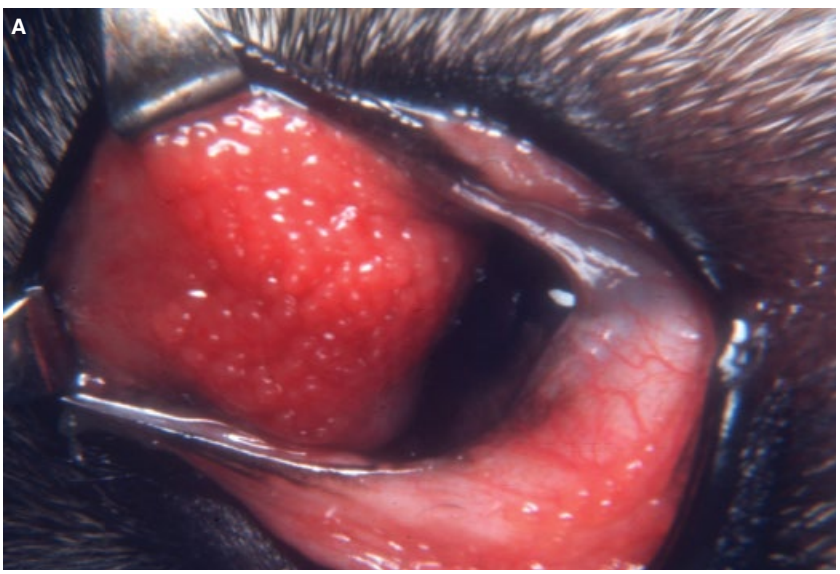


Figure 7.10 (A) Follicular conjunctivitis is characterized with the formation of follicles on the nictitans and conjunctival surfaces, and signals a nonspecific chronic inflammation. Note the numerous follicles on the nictitans posterior surface.



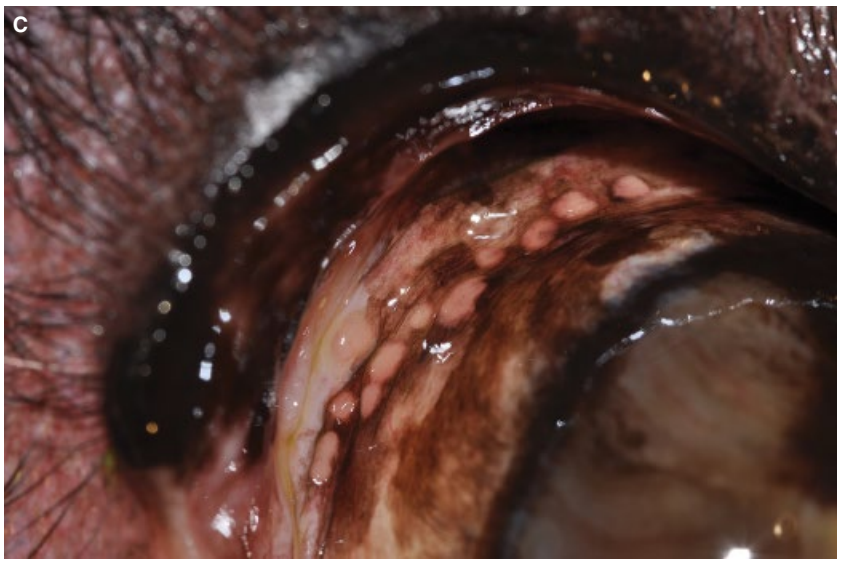


Figure 7.10 (*Continued*) (B) Lymphoid follicles on the palpebral conjunctival and anterior face of the nictitans suggest some chronicity of the conjunctival inflammation. (C) Lymphoid follicles can also take on a clear bubble-like appearance as these have in the dorsal fornix. Note the corneal fibrosis and pigmentation.



Figure 7.11 Chemosis or edema of the conjunctiva signals an acute response of the conjunctiva to an insult. Note the marked conjunctival swelling.



Figure 7.12 (A) Subconjunctival hemorrhage secondary to trauma. It appears as a swollen uniform red conjunctiva, most noticeable in the medial conjunctiva. (B) Subconjunctival hemorrhage in a dog with lymphoma. (C) Subconjunctival hemorrhage and hyphema in a dog following a trauma.

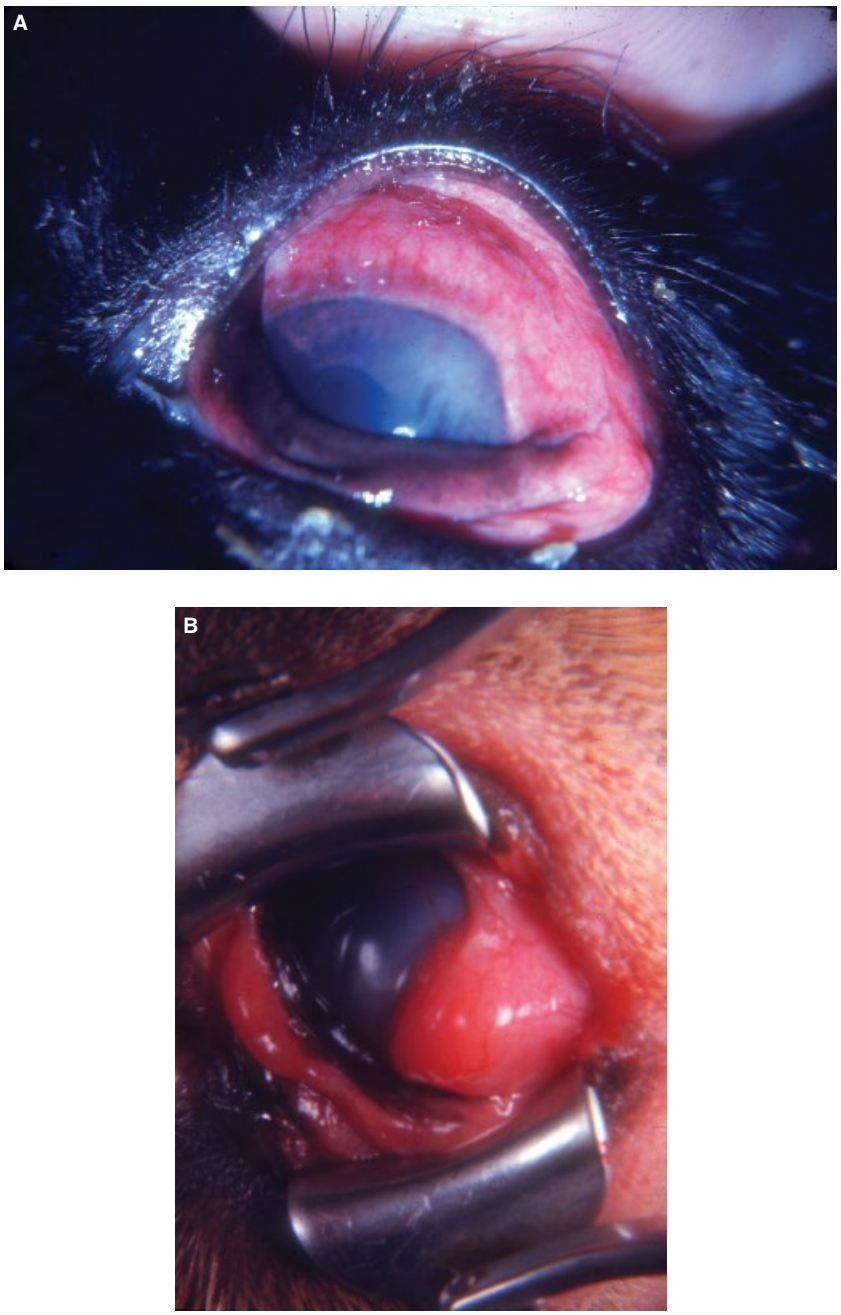


Figure 7.13 Non-neoplastic inflammatory masses of the conjunctivas. (A) Episclerokeratitis in an American Cocker Spaniel. The raised pink mass extends from the dorsal bulbar conjunctiva into the cornea. (B) Proliferative keratoconjunctivitis in a Collie. Both eyes were affected.

plasma cell, lymphocytes, and eosinophils. Treatment usually consists of topical antibiotics and corticosteroids. Manual removal of the persistent follicles can performed by debridement with a dry sterile surgical gauze square. Abrasion with copper sulfate crystals and scalpel blades are not recommended as they are too harmful.

Chemosis

An acute insult to the conjunctiva, such as an allergen, topical drugs, insect stings, or toxins, can cause the accumulation of fluids within the loosely arranged conjunctival stroma (Figure 7.11).

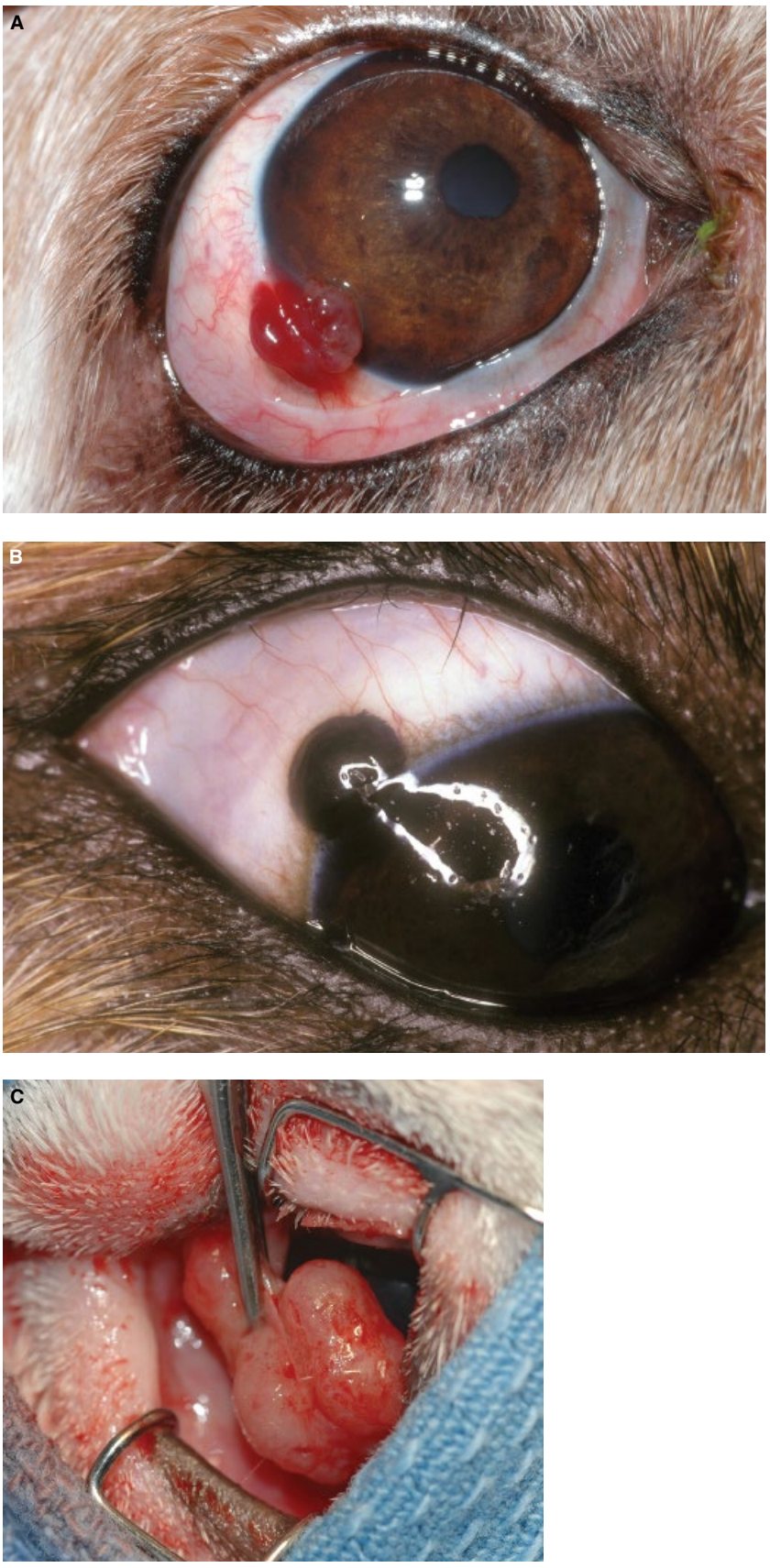


Figure 7.14 Neoplasms of the canine conjunctiva. (A) Hemangioma of the ventrolateral bulbar conjunctiva. The highly vascular red appearance is highly suggestive of vascular origin. Biopsy should be performed. (B) Limbal melanoma of the dorsal bulbar conjunctiva and sclera. The pigmented mass had been increasing in size for the past 3 months. (C) Lymphoma/lymphosarcoma can involve the conjunctiva; often it is bilateral. The conjunctiva appears thickened; cytology can be diagnostic. Most cases seem part of systemic lymphoma but one reported case appeared localized.

Subconjunctival Hemorrhage

Subconjunctival hemorrhage occurs in younger dogs, following dog fights or other trauma to the face, occasionally in association with neoplasia, Rocky Mountain spotted fever (*Rickettsia rickettsia*), ehrlichiosis, and other blood-clotting disorders (Figure 7.12).

Non-neoplastic Conjunctival Inflammatory Masses

Non-neoplastic inflammatory masses of the conjunctivas and nictitans appear to have distinct clinical features, but are grouped by histopathological findings. Masses include nodular granulomatous episclerokeratitis, fibrous histocytoma, and recurrent proliferative keratoconjunctivitis (Collie and Collie-related breeds) (Figure 7.13). Clinically these diseases may have less similar characteristics. These masses can affect the eyelids, nictitans, and, most frequently, the subconjunctiva. Nodular ocular fasciitis appears to be a separate clinical (and histologically) disease.

These non-neoplastic inflammatory masses are best biopsied to confirm the diagnosis. Most respond to intralesional and topical corticosteroids. Systemic corticosteroids or immunomodulating drugs may be necessary. Recurrence is not unusual.

Neoplasms of the Canine Conjunctiva

Conjunctival neoplasms are more malignant but less frequent than eyelid tumors in the dog, and sometimes their small size does not reflect their recurrence potential. Primary conjunctival tumors include melanomas, squamous cell carcinomas, mast cell sarcomas, papillomas, hemangiomas, hemangiosarcomas, angiokeratomas, lymphosarcomas, and histiocytomas (Figure 7.14). Masses that can be confused clinically with these neoplasia, such nodular granulomatous episclerokeratitis and nodular fasciitis, also involve the conjunctiva. As conjunctival neoplasms tend to be malignant locally (compared to most eyelid neoplasms), histopathology (especially of the surgical margins) is strongly recommended.

Treatment is generally wide excision and apposition of the conjunctival defect. Cryosurgery or beta-irradiation can also be combined with surgical excisions for adjunctive therapy.

8

Canine Cornea and Sclera

Developmental and Congenital Defects

Dermoids

Dermoids, also known as choristomas, are normal tissue in an abnormal location. They occur infrequently in puppies and are usually noticed at weaning or shortly thereafter because of the development of ocular irritation. Dermoids can be inherited in certain breeds such as the Dachshund, Dalmation, Doberman Pinscher, German Shepherd, and St. Bernard and are either unilateral or bilateral. Upon examination a mass of variable size and pigmentation with coarse hairs protruding from its surface is present at corneoscleral limbus and bulbar conjunctiva (Figure 8.1). Treatment is local excision; a superficial keratectomy is indicated if the dermoid extends onto the corneal surface. Incomplete excision usually results in regrowth.

Microcornea

Normal size globes with smaller than normal corneas are rare. Microcornea is nearly always associated with microphthalmia and other ocular anomalies. Breeds affected include Australian Shepherd (merle ocular dysgenesis), Collie, Miniature and Toy Poodles, Great Dane, Old English Sheepdog, and St. Bernard (Figure 8.2). Affected individuals usually have a shortened palpebral fissure and a prominent nictitans which partially obscures the globe.

Microcornea usually refers to a cornea that is <12 mm in horizontal diameter. Occasionally, central corneal mineralization and other ocular anomalies are present. Vision can be reduced or absent. There is no treatment available.

Persistent Pupillary Membranes

Corneal opacities and persistent pupillary membranes (PPMs) are congenital corneal and iridal anomalies (Figure 8.3). The pupil is formed during the last one-third of gestation and continues to develop for 4–8 weeks postnatally. If pupillary formation is incomplete or delayed, very fine white to pigmented remnants or strands of iris tissue originating from the iridal collarette or minor iridal arteriolar circle protrude off the surface of the iris and extend to other areas of the iris, the posterior cornea, the anterior lens capsule, or some combination thereof. If the cornea is involved, there will be deep round to oval endothelial opacities, sometimes containing pigmentation. Lens opacities involve only the anterior lens capsule and outer anterior cortex. Occasionally, corneal or lens opacities exist without the PPM attachment if the strand has previously atrophied. Generally, no treatment is necessary. Many breeds have been reported with PPMs; however, the Basenji is the only breed in which mode of inheritance has been established.

Corneal Ulcerations and Inflammation

Spontaneous Chronic Corneal Epithelial Defects

Spontaneous chronic corneal epithelial defects, corneal erosion, recurrent erosion, persistent epithelial erosion, Boxer's ulcer, indolent ulcer, refractory ulcer, or rodent ulcers are all synonyms to describe a clinical syndrome of recurrent superficial corneal ulcers that heal very slowly in the dog, and often recur. At least 24 breeds are predisposed to these ulcers. The corneal epithelium and its basement membrane appear abnormal, which accounts for the development of the erosion as well as its delayed healing (attachment) period. Most affected dogs are middle aged (5–7 years), and the Boxer breed accounts for a significant number of these ulcers (Figure 8.4).

On examination the affected cornea demonstrates a superficial corneal ulcer with an overlying lip of nonadherent loose corneal epithelium. Often, fluorescein or rose Bengal applied to these corneas will ooze or leak into stroma adjacent to the ulcer proper where the epithelium is not attached. Conjunctival hyperemia and a mild secondary iridocyclitis are often present.

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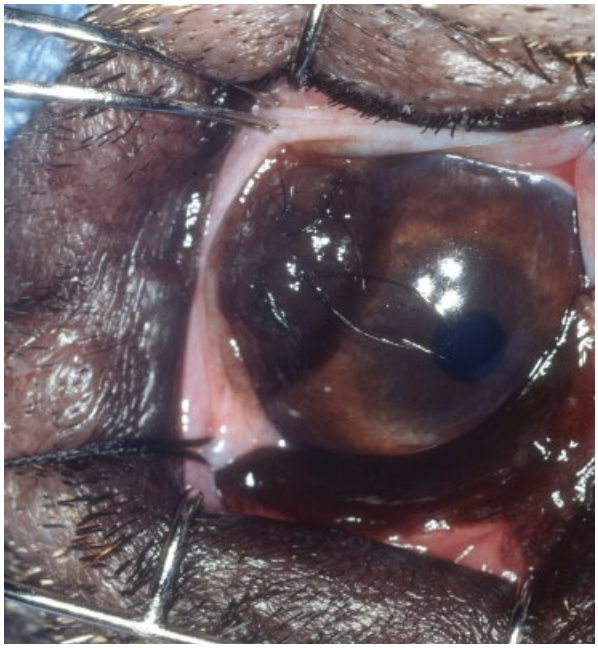


Figure 8.1 Corneoconjunctival dermoid affecting the lateral limbus in a St. Bernard puppy. A few long and coarse hairs are evident.



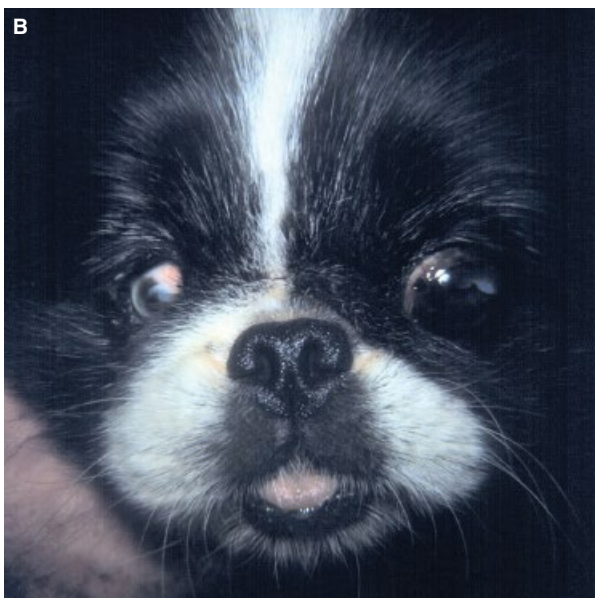


Figure 8.2 (A) Bilateral merle ocular dysgenesis in an excessively white Great Dane puppy. Note the globes are not the same size. (B) Unilateral microcornea and microphthalmia in a Boston Terrier mix puppy. The opposite globe is enlarged from congenital glaucoma. Multiple ocular anomalies are probably also present.

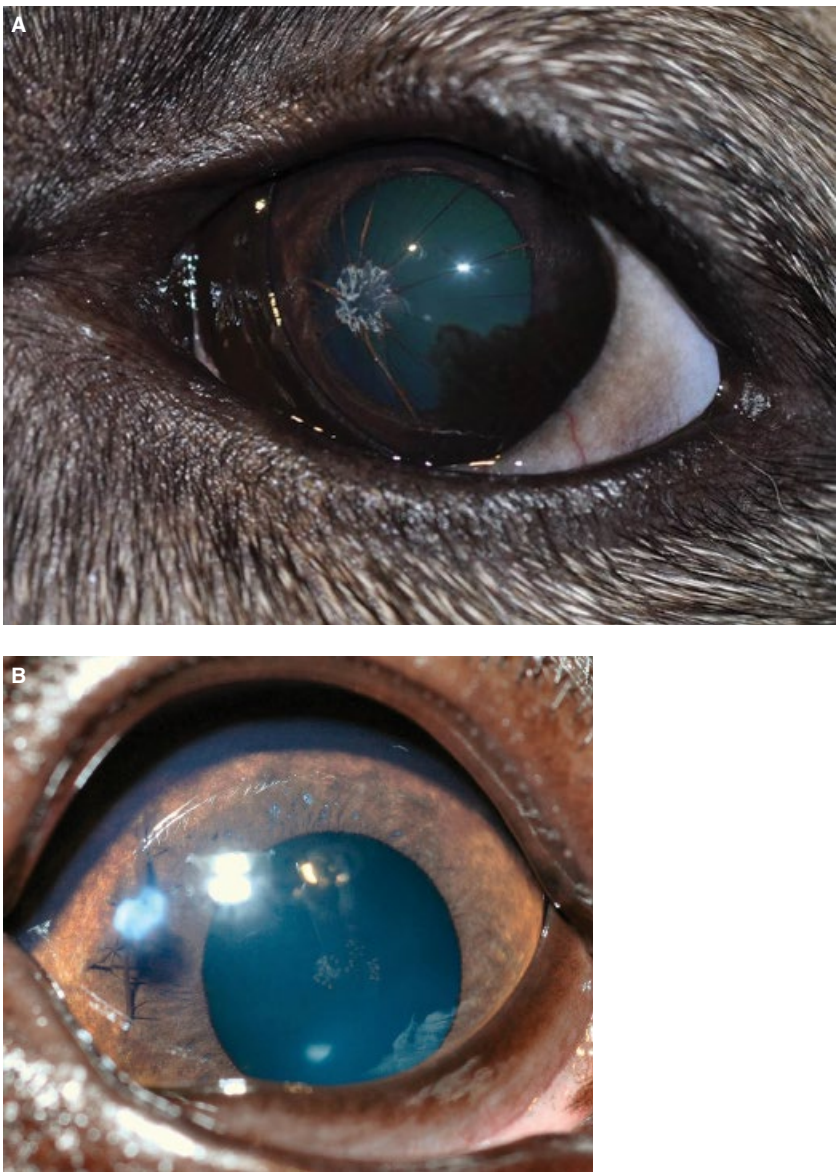


Figure 8.3 (A) Persistent pupillary membranes (PPM) in a mixed-breed dog. Note the PPMs are pigmented, originate from the middle of the iris, and form a pigmented circle on the posterior cornea. (B) PPM in a Rottweiler puppy. The pigmented PPMs are concentrated in the lateral posterior cornea with two dense corneal opacities. Note also the congenital pigment on the central anterior lens capsule and a focal anterior cortical cataract.

Treatment consists of removal of the loose epithelium under topical anesthesia with a dry cotton swab, followed by debridement of the exposed stroma to facilitate attachment. Diamond burr debridement is used often in preference to grid or multiple punctate keratotomy. Chemical debridement or superficial keratectomies are less commonly employed now. Recurrence in the ulcer in the same eye or the fellow eye is common given that these ulcers are caused by an abnormality of the epithelium and anterior stroma.

Corneal Ulcers

Corneal ulcers are characterized by the loss of corneal epithelium and varying degrees of stromal loss, which determines the depth of the wound. Application of topical fluorescein to the bulbar conjunctiva and cornea results in the dye uptake by the ulcer (Figure 8.5). As fluorescein rapidly diffuses in the stroma and even the anterior chamber, observation of the stain should occur immediately after application. Topical fluorescein, adequate illumination, and some magnification are essential aids in the diagnosis and monitoring of corneal ulcers. Corneal ulcers follow trauma, shampooing, cat scratches, exposure (as with enlarged globe, impaired blink, or corneal sensitivity), keratoconjunctivitis sicca (KCS), and other causes. In the brachycephalic breeds, exophthalmia, lagophthalmia, incomplete blinks, and other factors contribute to corneal ulcerations (see Appendix B). Corneal ulcer may progress in size and depth (with or without therapy), and changes from a shallow to a deep stromal ulcer, descemetocele, and even to corneal perforation. Iris prolapse can occur within several hours.

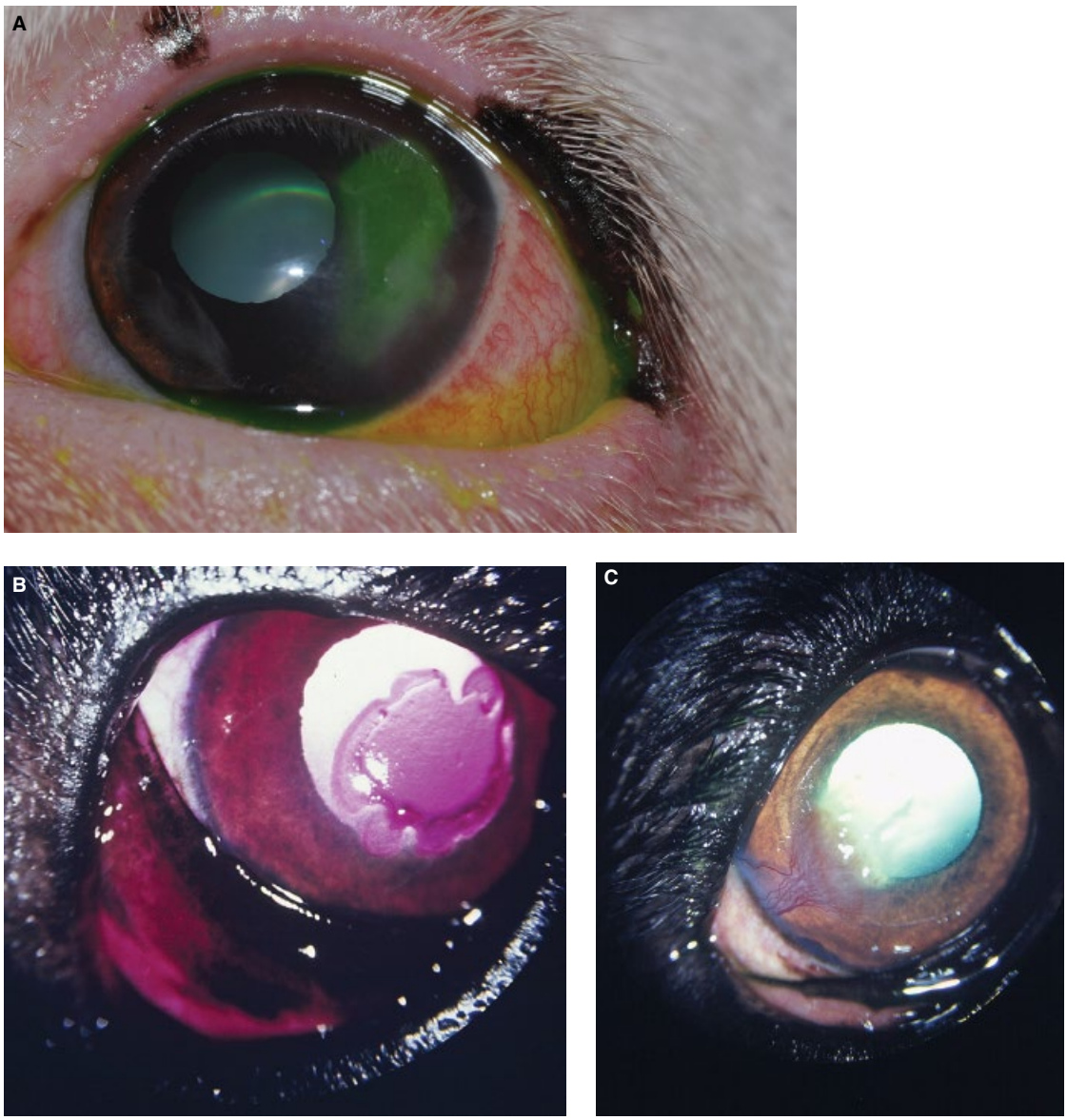


Figure 8.4 (A) Corneal erosion in a Boxer dog stained with topical fluorescein showing a loose "lip" of epithelium and some early superficial corneal vascularization from the lateral limbus. (B) Corneal erosion, stained with topical rose Bengal, demonstrates retention of stain by both the base of the erosion as well as loose epithelium surrounding the defect. (C) In this corneal erosion, healing of the ulcer has started with corneal vascularization of its medial margin.

Corneal ulcers in the dog will often become infected by opportunistic or pathogenic bacteria, with *Staphylococci* and *Streptococci* species the most commonly isolated. Viral ulcers associated with canine herpesvirus (CHV-1) have been documented in the dog, and fungal ulcers are uncommon (usually related either to a corneal foreign body or in an immunosuppressed patient).

The main goals of medical therapy are: (i) to eliminate or prevent infection of the ulcer; (ii) to control or prevent the proteolytic and enzymatic digestion of corneal stroma; and (iii) to address the reflex uveitis that invariably accompanies the ulcer. Additionally, if the ulcer has perforated or is likely to because of its deep nature or the presence of keratomalacia, surgical stabilization can be warranted.

Corneal Ulcerations in Brachycephalic Breeds

The brachycephalic breeds are predisposed to central corneal ulcers that have the tendency to progress into the deeper stroma as well as recur (Figure 8.6). These ulcers must be under close clinical monitoring as they can rapidly deteriorate. Corneal exposure, lagophthalmia, reduced central corneal sensitivity, nasal fold trichiasis,

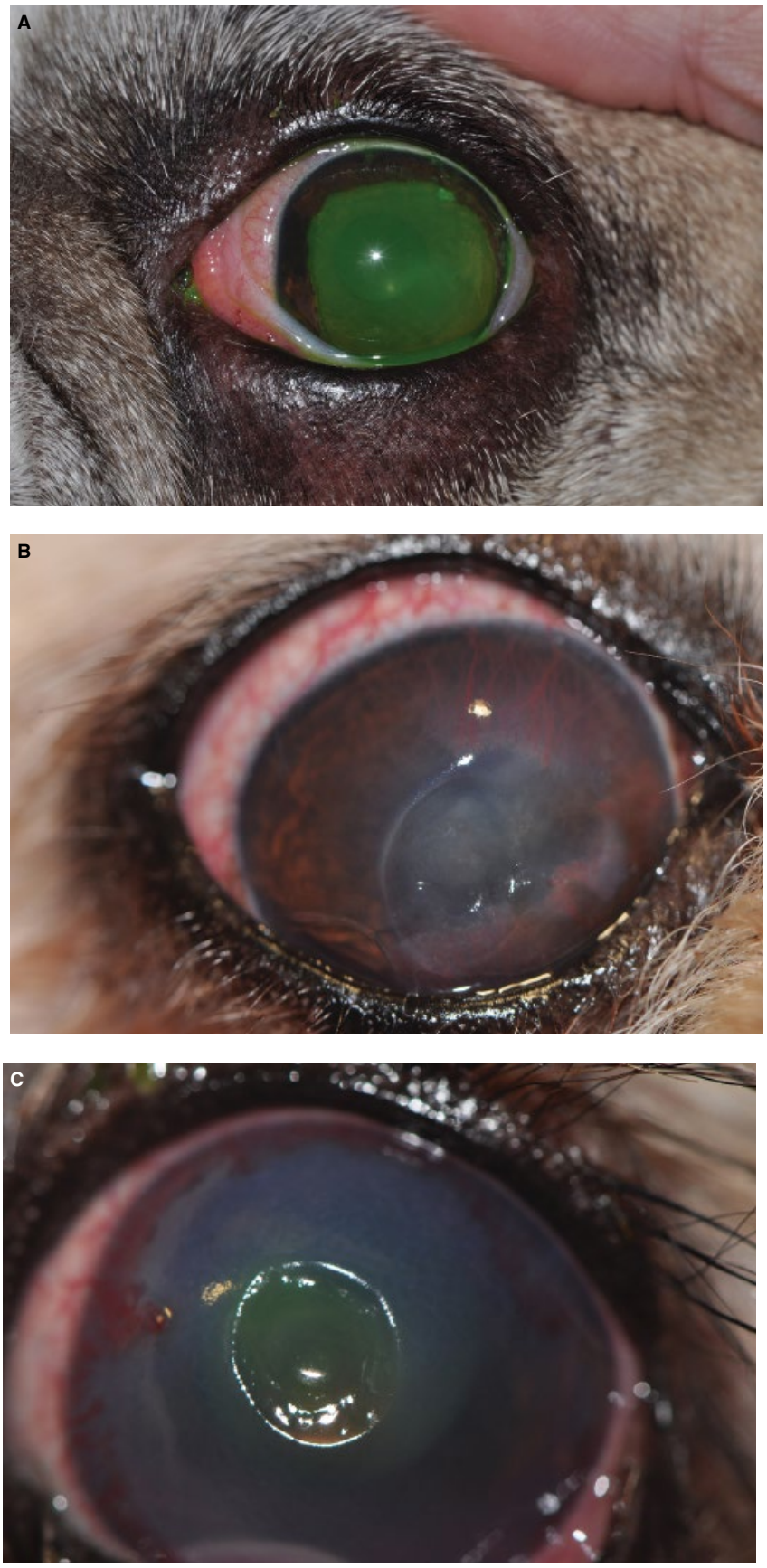
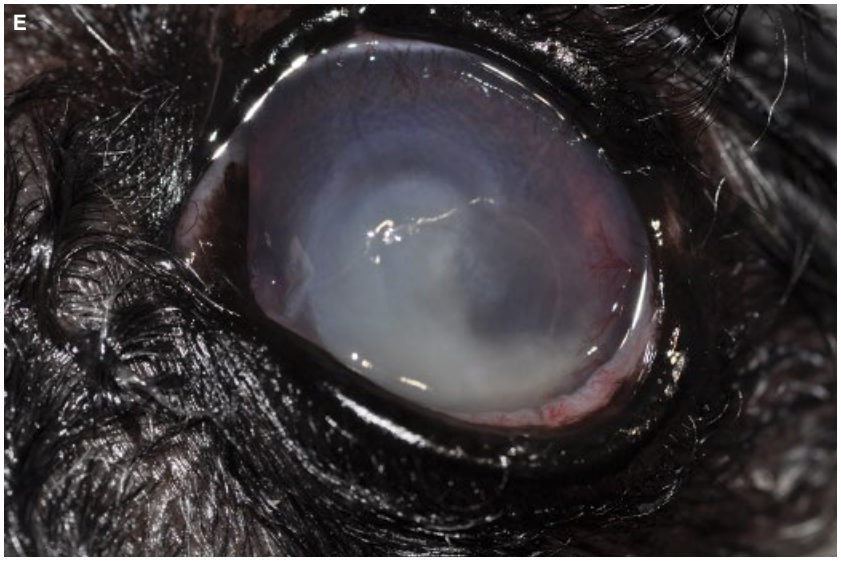


Figure 8.5 (A) A very large but superficial corneal ulcer stained with topical fluorescein. The dog's eye had developed corneal ulceration after being treated with topical corticosteroids for keratoconjunctivitis sicca. (B) Large central corneal ulcer extending to about the middle of the stroma. Note the corneal vascularization approaching the wound bed. (C) Deep stromal ulcer in a Shih Tzu dog. This ulcer extends approximately 80% into the posterior stroma, but not all the way to Descemet's membrane.





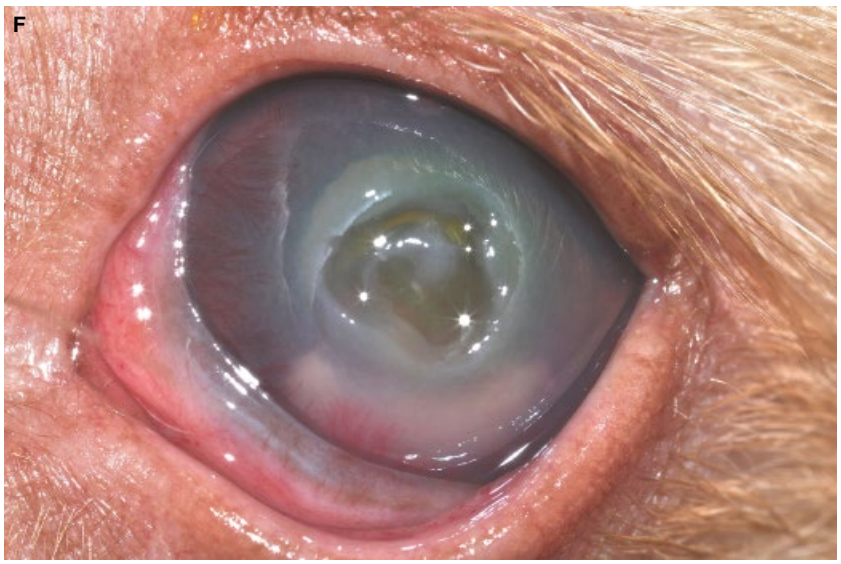


Figure 8.5 (*Continued*) (D) Deep stromal and infected corneal ulcer in a mixed-breed dog. Note the ring of perilimbal vascularization approaching the central wound and the axial cellular infiltrate and soft malacic edges. (E) Melting corneal ulcer in a dog. Note the soft, gray, corneal tissue contacting the lower eyelid margin. (F) Axial deep stromal, near descemetocele with malacic edges. There is considerable secondary uveitis as evidenced by miosis and hypopyon in the ventral anterior chamber.

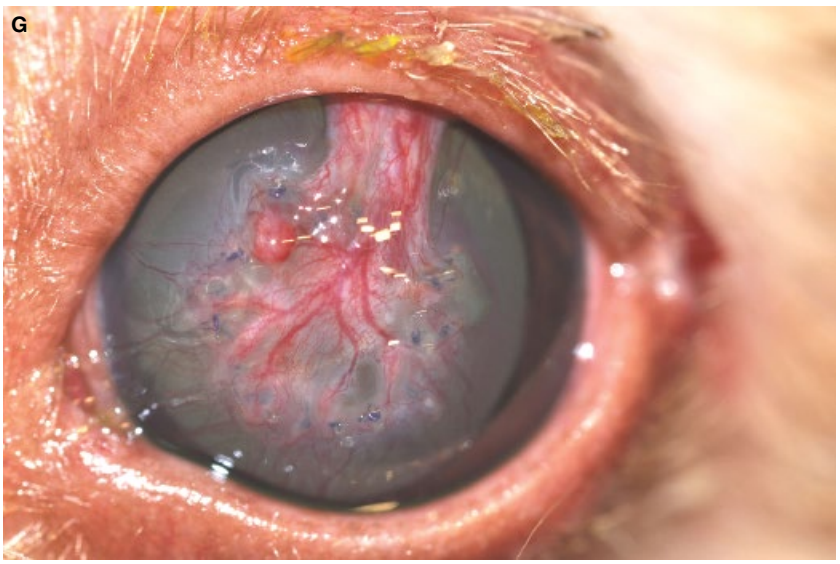






Figure 8.5 (*Continued*) (G) Same eye as in part F 3 weeks after placement of a conjunctival graft for vascular and structural support. Note the dilated pupil and resolution of the hypopyon and intracameral fibrin. (H) Conjunctival pedicle graft in another dog that has healed and is well-incorporated into the native cornea. (I) Same eye as in part H immediately following trimming of the graft pedicle.

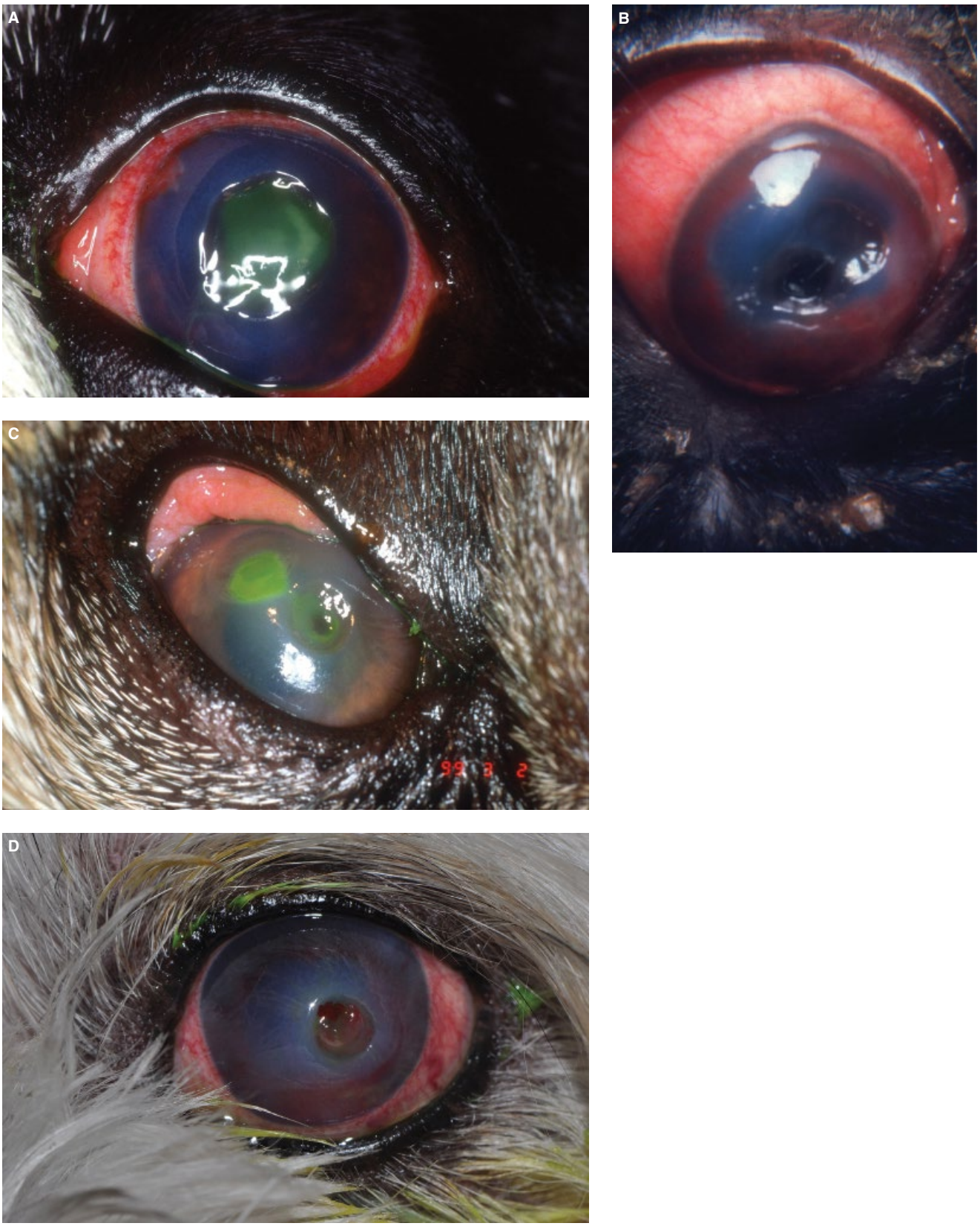


Figure 8.6 (A) Deep central corneal ulcer has been stained with topical fluorescein, the retention of which within the wound bed suggests some stroma is still present. (B) A deep corneal ulcer in a Pekingese. A portion of the ulcer is deeper than the rest (descemetocele). Corneal vascularization completely surrounds the ulceration. (C) Pekingese cornea with two ulcers: descemetocele and superficial ulcer with corneal vascularization. (D) Perforated central corneal ulcer with recent iris prolapse with hemorrhage.

distichiasis, inadequate blink rate, quantitative or qualitative tear deficiencies, and increased central corneal epithelial turnover contribute to the high prevalence of corneal ulcerations in brachycephalic breeds (see Appendix B).

The history usually includes lacrimation, blepharospasm, eyelid swelling, conjunctival hyperemia, and a corneal opacity. Because brachycephalic patients have decreased central corneal sensation (compared with mesocephalic and dolichocephalic breeds), they often will not exhibit pronounced evidence of pain. An ophthalmic examination reveals a central or paracentral corneal ulcer that retains fluorescein. The corneal stroma can be grayish, irregular, and malacic (soft or friable). When the corneal stroma is completely lost within the ulcer, descemetocele occurs (characterized with fluorescein as a circle of dye staining the sides of the ulcer wall but not its bed). With rupture of the very thin descemetocele, iris prolapse follows.

Therapy of the brachycephalic corneal ulcer includes topical antibiotics, autogenous serum, atropine (1%) or tropicamide (1%) for the secondary iridocyclitis, and systemic anti-inflammatories and analgesics. Both topical antibiotics and serum are often instilled very frequently (sometimes hourly) for the first few days, to halt ulcer progression and promote healing. For those ulcers that progress (larger and/or deeper), immediate surgical intervention is recommended (usually a pedicle conjunctival graft).

Fungal Keratitis

In contrast to the horse, dogs rarely develop fungal keratitis. The history is usually one of a corneal ulcer that persists in spite of intensive topical (and systemic) antibiotic therapy. There may be a history of trauma or a previous corneal foreign body. Affected dogs are often systemically or locally immunocompromised. Corneal ulceration is the most common clinical manifestation, but the occasional fungal corneal abscess occurs in dogs (Figure 8.7). Diagnosis is made based upon index of suspicion and the results of corneal cytology or biopsy (to demonstrate the presence of fungal elements) and culture. Therapy includes routine corneal ulcer therapy with the addition of topical antifungals, removal of the foreign and infected site, and, if necessary, a pedicle conjunctival graft. Prognosis is usually fair to good with appropriate therapy.

Pigmentary Keratitis

Pigmentary keratitis in the dog is a response by the cornea to a low grade (not sufficient to incite ulceration) and chronic insult (Figure 8.8). Pigmentary keratitis is common in dogs and occurs more frequently in certain breeds, such as the Pug, Boston Terrier, Lhasa Apso, Pekingese, and Shih Tzu (also see Appendix B). The pigment response is often associated with eyelid diseases, KCS, distichiasis, nasal fold trichiasis, and other diseases. The clinical history usually includes some ocular irritation, epiphora, conjunctival hyperemia, and a red to black tissue slowly infiltrating the peripheral cornea. The initial site of the corneal pigmentation often indicates the original position and type of the insult.

Pigmentary keratitis is most responsive to therapy before a large percentage of the cornea is involved. The source can frequently be addressed by surgery. Superficial keratectomy to remove the corneal pigmentation is not very successful as the pigment slowly returns. Topical corticosteroids, combined with topical cyclosporine or other tear stimulating and immunomodulating drugs, is a better method to address the corneal pigmentation. Early intervention and prevention of progression is usually the best strategy.

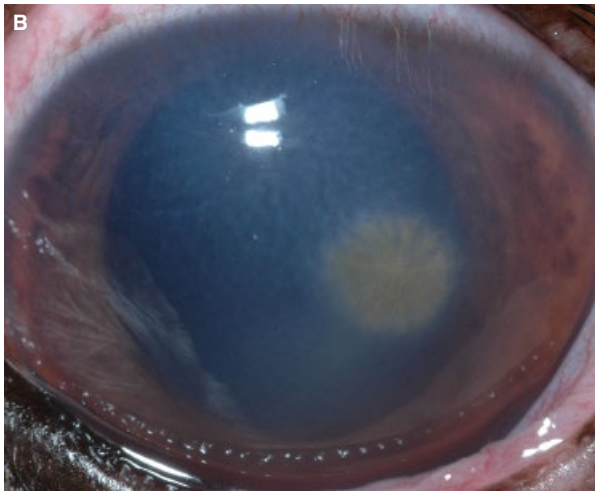
Chronic Superficial Keratitis (Pannus)

Chronic superficial keratitis or pannus is an immune-mediated corneal disease of the German Shepherd, Australian Shepherd, Greyhound, Belgian Sheepdog, and Belgian Tervuren, among other breeds. Most clinical observations on this disease are based on the German Shepherd breed. The disease is bilateral, progressive, and, if not controlled, will produce blindness. The history is usually one of a progressive corneal opacity starting at the lateral aspects of the cornea and limbus which advances across the cornea (Figure 8.9). The disease is usually more aggressive and difficult to control when it is first diagnosed in younger dogs (>4 years old) than in older dogs (>8 years old).

Chronic superficial keratitis can be divided into several clinical stages. In the first or early phase, superficial keratitis starts at the lateral and sometimes the medial limbus. Corneal edema and superficial vascularization affect the lateral cornea. In the second or progressive phase, uncontrolled superficial keratitis advances across the cornea with a central edema and vascularization. Granulation tissue proliferates and pigment develops in the superficial lateral cornea. The third or "cobblestone" phase is characterized by a rapid advancement of the keratitis and appears as focally raised red areas distributed across the cornea. Inactive or medically controlled pannus exhibits corneal scarring and residual pigmentation often concentrated at the lateral limbus.

Response to therapy depends on the age of the dog, its exposure to sunlight and other irritants, and the altitude of the patient's home range (patients at elevations of >4000 feet above sea level often have more severe disease). Medical therapy includes topical corticosteroids and





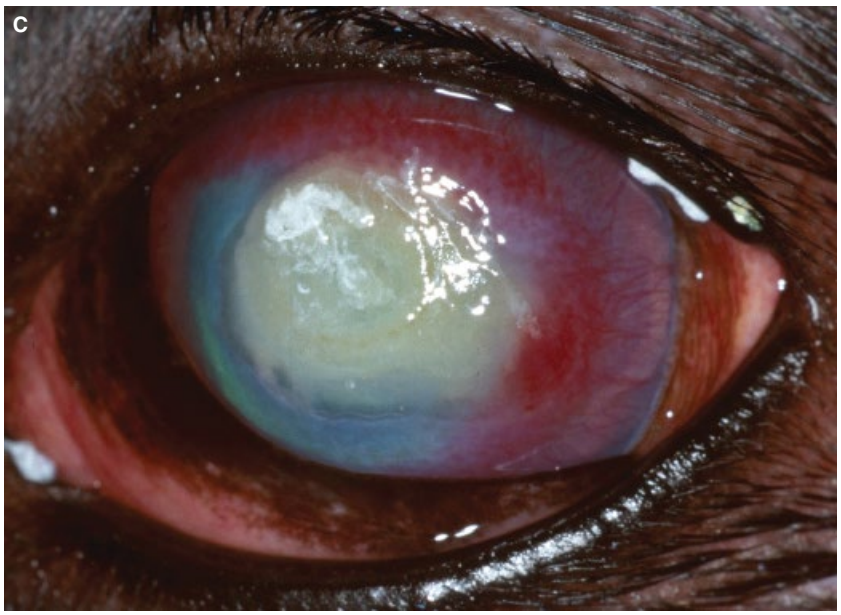


Figure 8.7 (A) Mycotic keratitis with ulceration and secondary iridocyclitis in a dog. Fungal organisms were identified on cytology. (B) Fungal keratitis in a mixed-breed dog. (C) Fungal keratitis in an aged dog that was systemically immunocompromised.



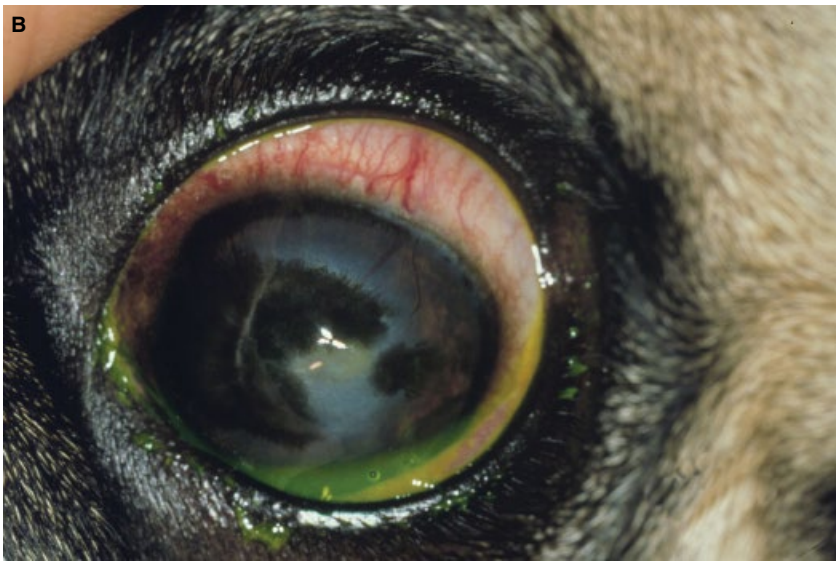
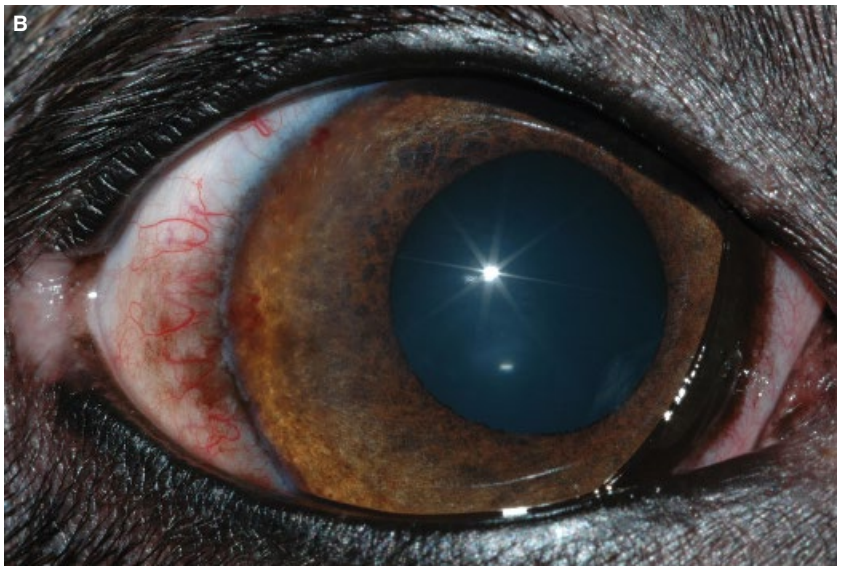




Figure 8.8 (A) Pigmentary keratitis in a Pug secondary to medial lower entropion and mild keratoconjunctivitis sicca (KCS). The pigmentation affects most of the medial superficial cornea. (B) Diffuse pigmentary keratitis secondary to a healed corneal ulcer and chronic KCS. (C) Pigmentary keratitis and conjunctival pigmentation in a Pug. Note the vascularization and axial fibrosis which was the site of a previous corneal ulceration.





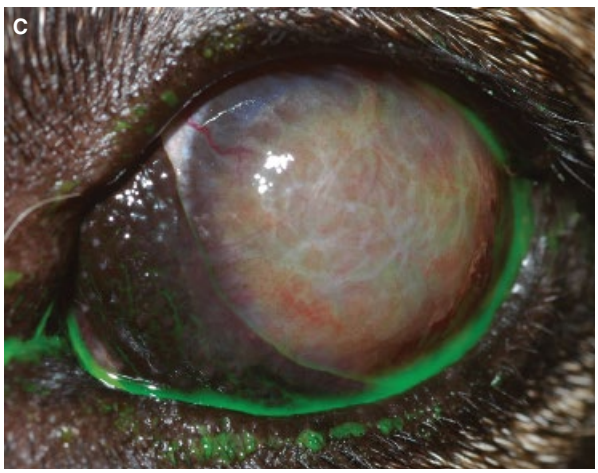


Figure 8.9 (A) Early chronic superficial keratitis or pannus in a 3-year-old German Shepherd dog is characterized by the invasion of lateral cornea with inflammatory cells (plasma cell and lymphocytes) and superficial corneal vascularization. Often a white infiltrate is present in front of the advancing superficial blood vessels and red granulation tissue. The medial canthus is also affected with the same disease process. (B) Early chronic superficial keratitis in a Greyhound dog. (C) "Cobblestone" pannus in a German Shepherd dog. Note the marked inflammation affects the entire cornea. This type of pannus is often rapidly progressive and difficult to control.

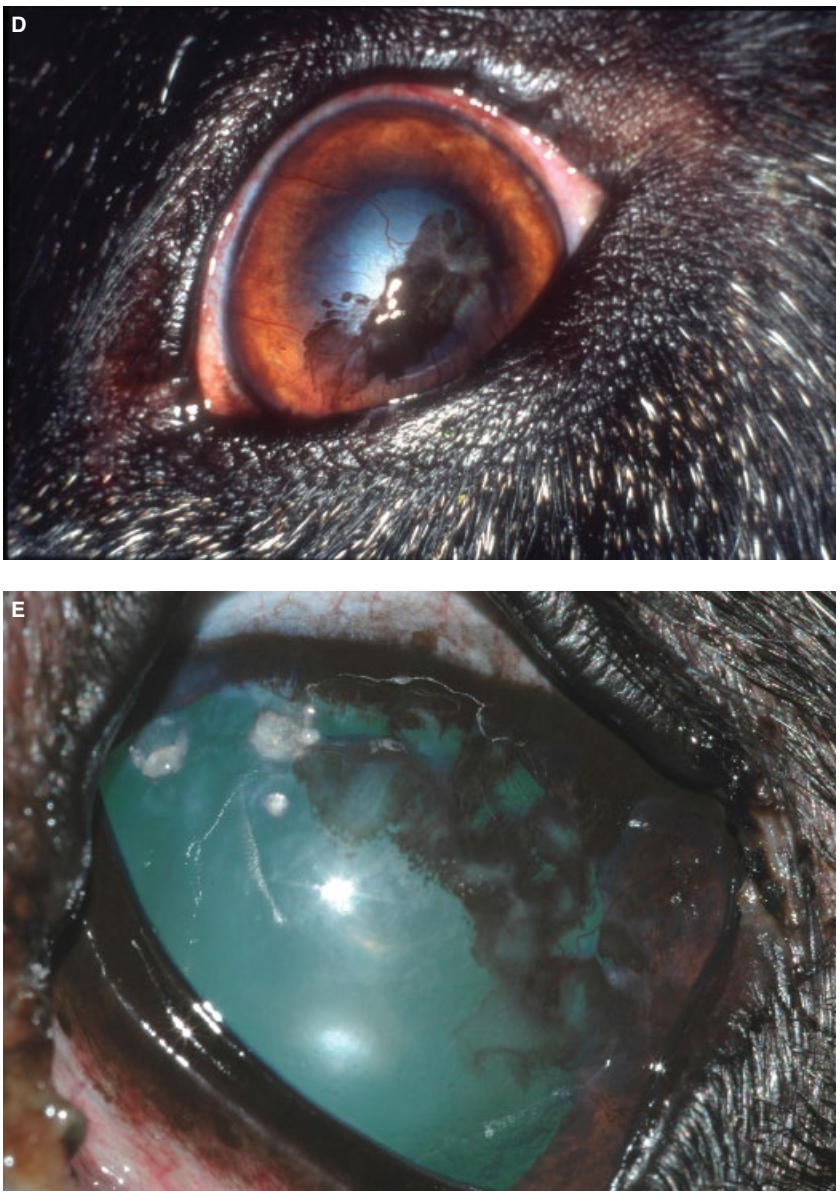


Figure 8.9 (*Continued*) (D) The eye with chronic superficial keratitis in a German Shepherd has been treated and controlled by topical corticosteroids for several years. Although some corneal pigmentation, vascularization, and scarring are present, the majority of the cornea is clear. (E) In some cases of pannus, foci of stromal deposits and corneal degeneration occur, especially at the leading margins of the inflammation.

immunomodulators to effect (once to several times daily). As the disease cannot be eliminated, therapy is adjusted to control the inflammation so that the minimal amount of medication necessary to keep the inflammation in check is maintained. Beta radiation and superficial keratectomies can be used for refractory cases, but these modalities are not without limitations.

Neuroparalytic Keratitis

In neuroparalytic keratitis the function of the facial nerve is temporarily or permanently lost resulting in an impaired or absent protective blink reflex (Figure 8.10). The facial nerve dysfunction can cause drooping and lack of movement of the upper and lower eyelids, ear, and lip, and lack of nostril movements during breathing. Tear production, as measured by the Schirmer's tear test, is normal, increased, or subnormal. Reduced tear levels signal additional damage to the parasympathetic fibers of the facial nerve (a higher or more central lesion).

Treatment is supportive and consists of topical lubricants and care for any exposure related ulcers that have developed. If tear production is impaired, oral or topical pilocarpine (1–2 drops of 2% ophthalmic pilocarpine/10 lb body weight orally in the food twice daily or 1 drop of 0.1% ophthalmic pilocarpine applied to the affected eye twice daily) to directly stimulate the lacrimal gland is sometimes helpful. Tarsorrhaphies (either partial temporary or permanent) may also be indicated.



Figure 8.10 (A) Neuroparalytic keratitis in a dog with loss of facial nerve innervation and the blink reflex. The central cornea has developed malacia. (B) Neuroparalytic keratitis in a mixed-breed dog. There is extensive corneal vascularization and an axial corneal ulceration.

Neurotropic Keratitis

In neurotropic keratitis, corneal sensitivity is reduced or lost due to lesion(s) of the ophthalmic division of the trigeminal (fifth) cranial nerve (Figure 8.11). The blink reflex is intact, but corneal sensitivity as tested by a pointed piece of cotton or by the Cochet-Bonnet corneal aesthesiometer is decreased or absent. Tear production can be decreased or normal, but distribution of the tears on the ocular surface is always abnormal. Treatment is supportive and should be directed towards protecting the cornea from exposure.

Immune-mediated Keratitis

Punctate keratitis is an unusual form of corneal dystrophy or an immune-mediated keratitis in the Shetland Sheepdog and Longhaired Dachshund (Figure 8.12). The history typically begins with blepharospasm, lacrimation, and conjunctival hyperemia in a relatively young dog (~2 years old).

Examination reveals punctate corneal lesions affecting the epithelial and anterior stromal layers, distributed throughout the cornea. The lesions may contain a depressed center and often retain topical fluorescein. Corneal vascularization may or may not be present. Treatment is usually topical corticosteroids and/or cyclosporine, but recurrences are common.

Other forms of immune-mediated keratitis manifest with corneal vascularization with or without cellular infiltrate. The designation of immune-mediated requires ruling out other causes of corneal inflammation (KCS, qualitative dry eye, foreign bodies or other sources of chronic irritation). Rarely, corneal ulcerations will occur with this form of keratitis. Treatment requires topical immunosuppressives or immunomodulators.

Florida Spots

A unique corneal disease characterized by one or more anterior stromal opacities occurs in Florida and other

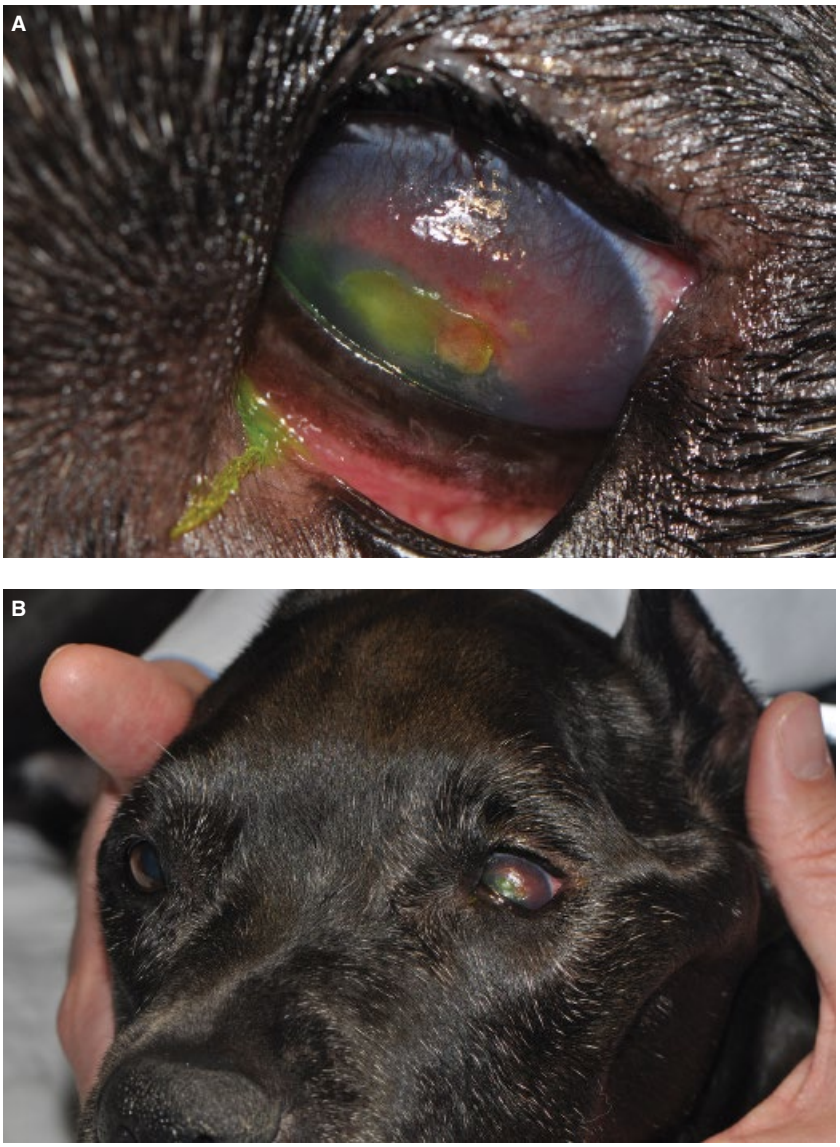
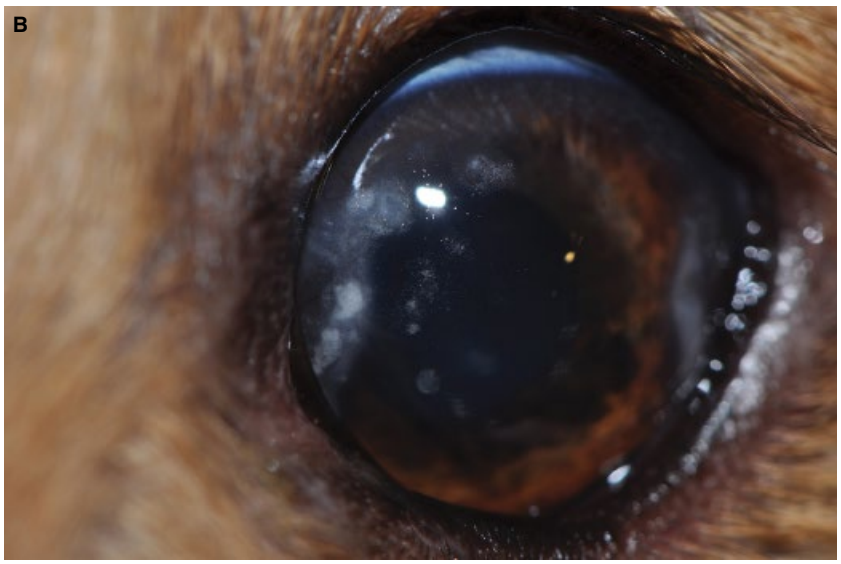


Figure 8.11 (A) Neurotropic keratitis in a dog secondary to the loss of trigeminal cranial nerve function. Corneal sensitivity is absent as demonstrated by the lack of blink after corneal touch. (B) Same dog as in part A. Note the marked atrophy of the temporalis and masseter muscles.



Figure 8.12 (A) Multiple epithelial and stromal opacities characterize the punctate keratitis in the Shetland Sheepdog.



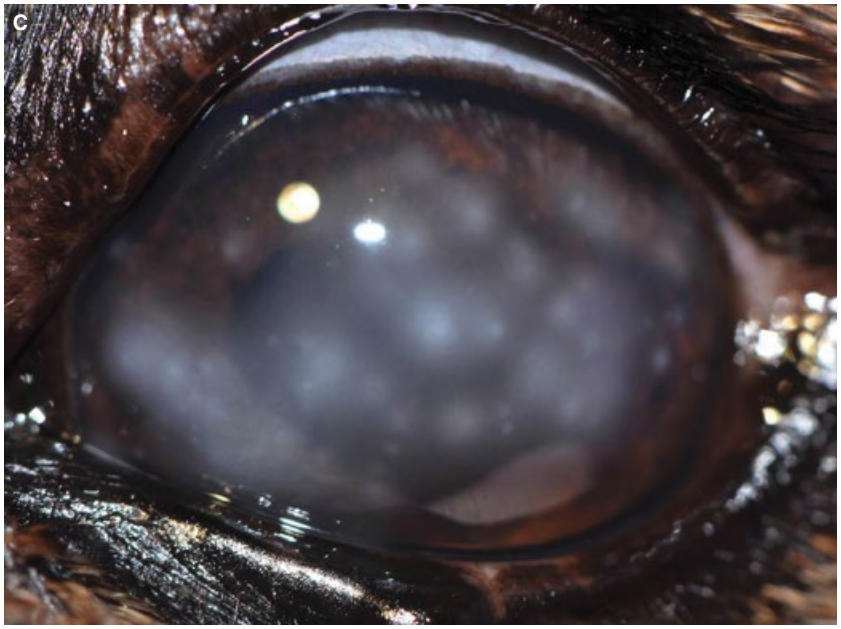




Figure 8.12 (*Continued*) (B) Punctate keratitis in a Shetland Sheepdog. (C) Punctate keratitis in a Miniature Dachshund. (D) Immune-mediated keratitis not associated with tear film abnormalities in a young Shih Tzu.

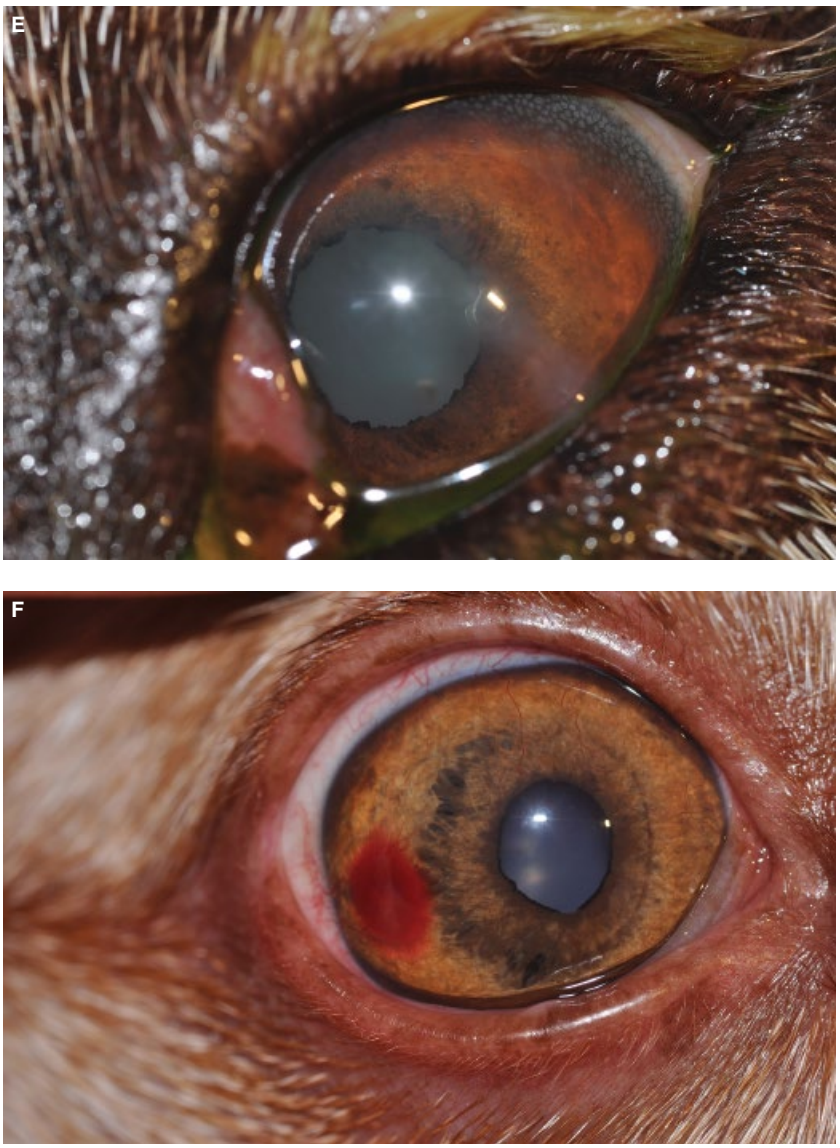


Figure 8.12 (*Continued*) (E) Immune-mediated keratitis in a Labrador Retriever mix. Note the perilimbal infiltrate. (F) Intrastromal hemorrhage in a dog with focal keratitis. Initially, blood vessels walls in newly formed corneal vessels are fragile and may hemorrhage, especially if systemic hypertension is present.

tropical Caribbean areas and has been reported mostly in the dog, but also in the cat and horse. Affected dogs are of variable ages and are often housed out of doors. The opacities, which are more dense in their centers and have a halo-like, or indistinct border, are located in the anterior or middle stroma and are devoid of superficial vacularization (Figure 8.13). Signs of inflammation and discomfort are not present. Treatment is neither necessary nor successful.

Corneal Trauma and Lacerations

Corneal lacerations occur most frequently in young dogs (Figure 8.14). Cat scratches are a common cause, especially in dogs less than 1 year of age. Corneal lacerations vary in location (central, paracentral, or limbal), depth (partial or full thickness) and prognosis (simple or complicated with iris prolapse, lens injury, or endophthalmitis). When corneal integrity is threatened or lost, apposition of the wound is recommended. Lacerations with hyphema must be carefully managed as intraocular damage may be present.

Corneal Foreign Bodies

Corneal foreign bodies are usually of plant material (rather than metal or glass), and as such tend to be irritating and can potentially introduce an infectious agent (Figure 8.15). The history is usually acute onset blepharospasm, elevation of the third eyelid, lacrimation, and eyelid edema.

Identification of the foreign body on ophthalmic examination usually requires topical anesthesia and



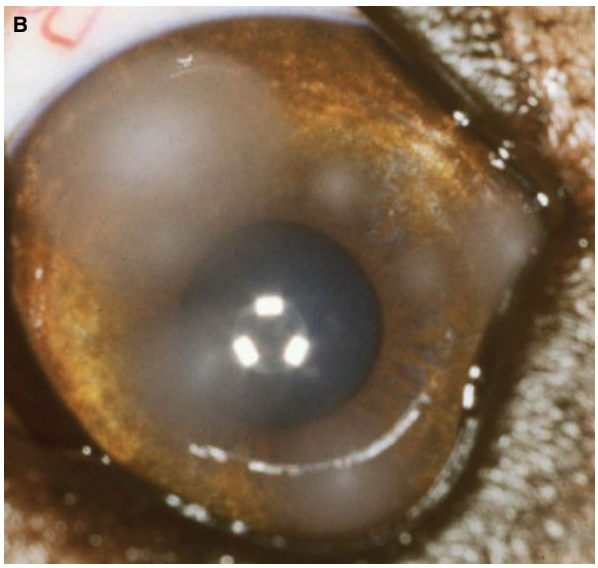


Figure 8.13 (A) Florida keratopathy in a 3-year-old Siberian Husky dog. The corneal spots are located primarily in the anterior stroma and incite limited to no inflammatory response. (B) Florida keratopathy in another dog. Note that the opacities incite no corneal response.



Figure 8.14 (A) Appearance of an acute, full-thickness, central corneal laceration in a dog with resultant iris prolapse. Note the hyphema in the anterior chamber.



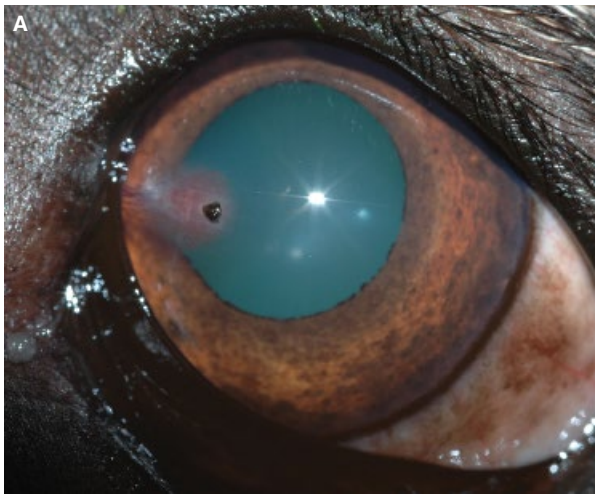




Figure 8.14 (*Continued*) (B) Postoperative appearance of the dog in Figure 8.14A after replacement of the iris and apposition of the cornea wound by simple interrupted absorbable sutures. (C) Partial-thickness corneal laceration secondary to an encounter with a cat. Note the miosis signaling reflex uveitis and the dorsal conjunctival ulceration. (D) Full-thickness corneal laceration in a young mixed terrier.



Figure 8.14 (*Continued*) (E) Healed corneal laceration in a young Miniature Dachshund following cat claw induced trauma.



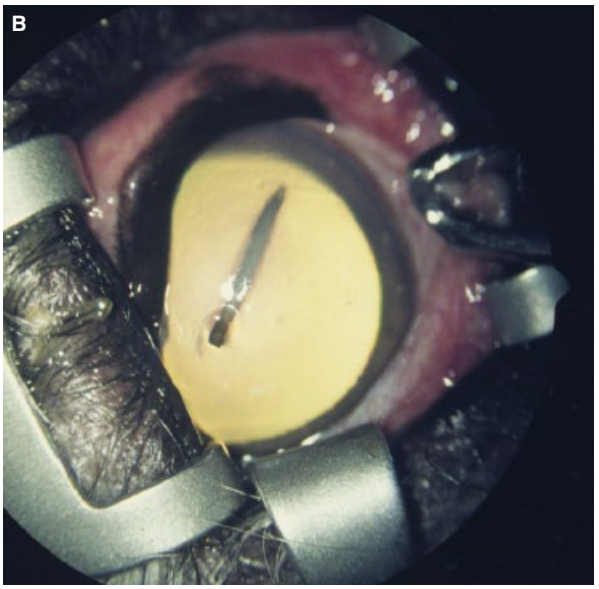




Figure 8.15 Corneal foreign bodies in the dog. Organic or plant material incites the most intense inflammation. (A) A small organic foreign body adhered to the superficial cornea in a dog. Note the approaching blood vessels. (B) A penetrating corneal foreign body (wood sliver) in a dog. Portions of the foreign body are protruding above the anterior corneal epithelium as well as beyond the posterior corneal surface and into the anterior chamber. (C) Small organic foreign body (part of a leaf) adhered to the cornea. The presence of the superficial corneal vessels suggests the foreign body has been present about 2 weeks.

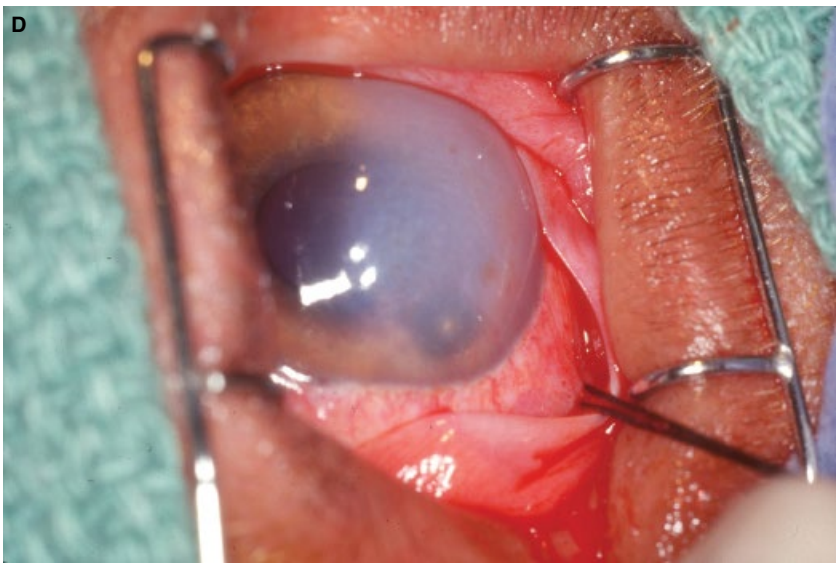


Figure 8.15 (*Continued*) (D) Intense corneal inflammation and iridocyclitis in a dog. A lead pellet is embedded in the posterior cornea (4 o'clock position).

occasionally sedation. The extent and depth of the foreign body within the cornea must be accurately determined before attempts at extraction are initiated. The corneal foreign body may be superficial and easily removed by thumb forceps or a strong stream of sterile wash. However, the foreign body may be firmly embedded within the cornea and require a superficial keratectomy for total removal. Foreign bodies that traverse the cornea and penetrate the anterior chamber will require full-thickness repair of the corneal breach. Foreign bodies that penetrate the anterior lens capsule usually require extraction of the lens (via phacoemulsification) as well as removal of the foreign body and repair of the corneal wound in order to prevent phacoclastic uveitis (a form of lens-induced uveitis). It may be difficult to localize the precise location of the foreign body within the cornea or determine if the anterior chamber has been breached, especially if a significant amount of corneal edema is present. If a fibrin clot has formed within the anterior chamber, especially if it is adhered to the corneal endothelium, it is very likely that the object has entered the anterior chamber.

Corneal Dystrophies

Corneal dystrophies are common in the dog with nearly 30 breeds predisposed. Corneal dystrophies are classified by their depth. Epithelial and basement membrane dystrophies result in recurrent or refractory corneal erosions and ulcerations (Boxer ulcers). Stromal dystrophies occur at different depths but all appear as opacities or deposits within the stroma. Endothelial dystrophies result in chronic and progressive corneal edema. Dystrophies differ from corneal degenerations, as they are bilateral, occur in certain breeds, do not typically incite inflammation, and, although progressive, rarely cause blindness.

Corneal Stromal Dystrophies

The corneal stromal dystrophies are painless corneal opacities consisting of lipids and cholesterol material (Figure 8.16). They are often breed-specific, with predictable ages of onset. The pet owner usually observes a central corneal opacity, but no evidence of pain. The opacities appear as gray to white crystalline opacities, more often in the anterior stroma but in more advanced stages can involve all levels of the stroma. Treatment by superficial keratectomy is not usually necessary unless vision is impaired, which is rare.

Corneal Endothelial Dystrophies

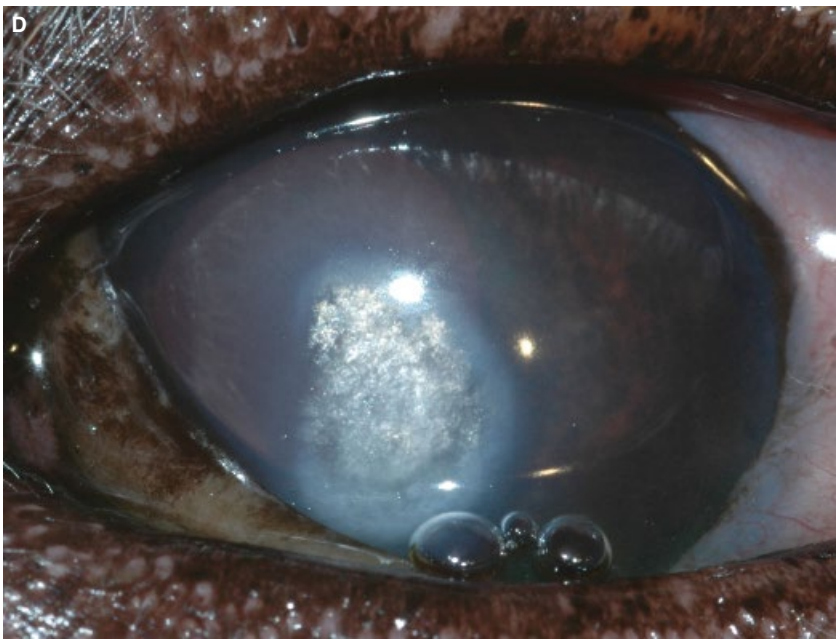
Corneal endothelial degeneration occurs most frequently in the Boston Terrier, Dachshund, and Chihuahua breeds in middle to advanced age (Figure 8.17). Females are more commonly affected than males. The history is one of slowly progressive corneal edema in the absence of signs of pain or irritation.

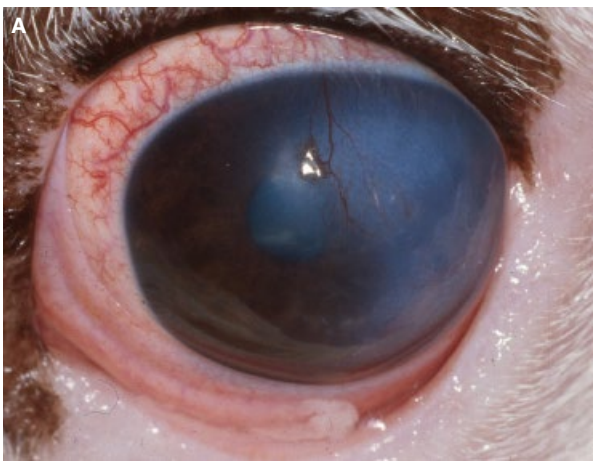
Upon examination, in early cases of corneal endothelial dystrophy the lateral or central cornea is edematous, but the remainder of the cornea is relatively clear. As the disease progresses, the cornea becomes completely edematous sufficient to cause vision impairment. Vesicles or bullae can form within the edematous surface epithelium and often rupture, resulting in corneal ulceration and acute pain.

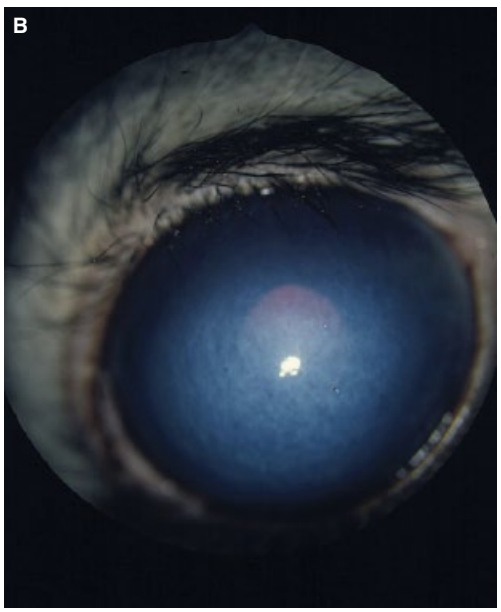


Figure 8.16 Canine corneal stromal dystrophies. (A) Lipid stromal corneal dystrophy in a dog. The lipids deposits appear as white opacities within the different layers of the stroma. (B) Inherited lipid stromal dystrophy affecting the central cornea in a Beagle. (C) Very faint stromal dystrophy of the central cornea. No corneal blood vessels were present.

Figure 8.16 (*Continued*) (D) Very dense corneal stromal dystrophy affecting the cornea. If this dense dystrophy affects the central cornea, vision can become impaired. Drug-induced mydriasis can improve vision.







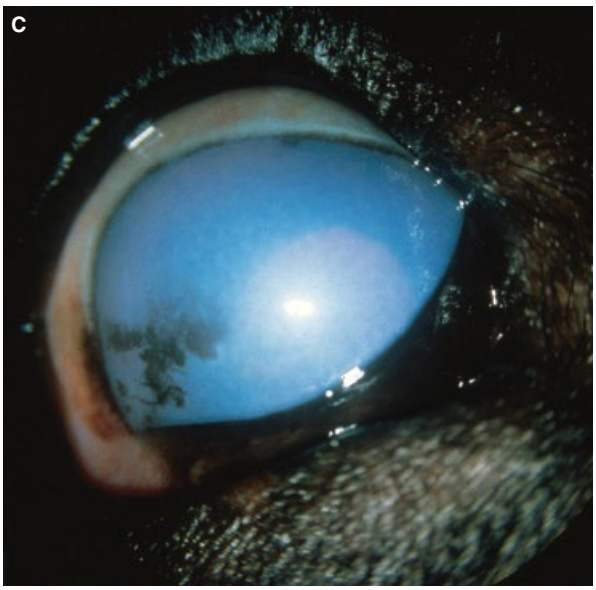
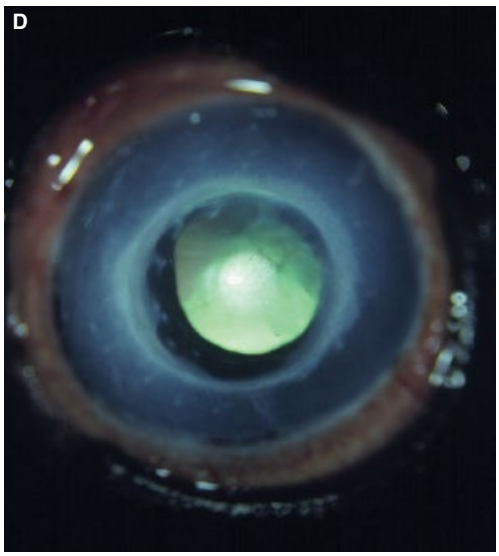


Figure 8.17 (A) Endothelial corneal dystrophy in an 11-year-old Boston Terrier. Most of the edema is in the lateral cornea. (B) Endothelial corneal dystrophy in an 10-year-old Boston Terrier. The progressive corneal edema has started centrally and is gradually expanding. (C) Advanced corneal endothelial dystrophy in a Boston Terrier. The edema has affected the entire cornea, caused blindness, and has resulted in the formation of recurrent bullae.





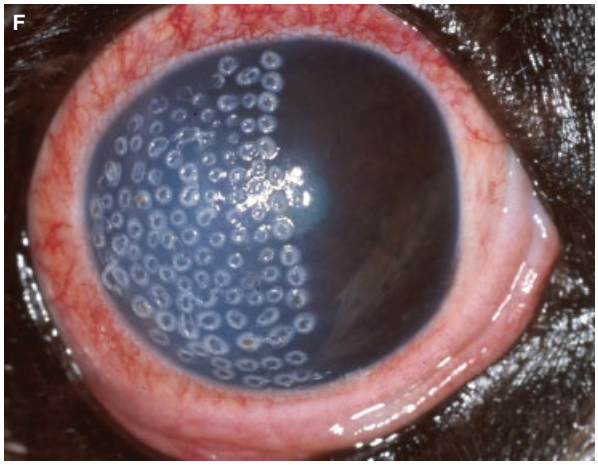


Figure 8.17 (*Continued*) (D) After treatment of the condition by full-thickness keratoplasty, the appearance of the eye 5 months postoperatively. The donor cornea has maintained the central cornea clear and the dog visual. (E) A Boston Terrier after superficial keratectomy and placement of a Gunderson conjunctival flap. (F) Boston Terrier corneal endothelial dystrophy immediately following thermokeratoplasty.

Medical therapy is limited and typically consists of the use of hyperosmotic fluids (5% NaCl solution or ointment, 40% glucose, or 100% anhydrous glycerin) to decrease the fluid accumulating in the cornea and minimize the formation of bullae. However, these drugs can be irritating and are short-acting. Surgical treatment is recommended early and includes thermokeratoplasty, very thin permanent complete bulbar conjunctival grafts (Gunderson flaps), or full-thickness keratoplasty. As an alternative to full-thickness keratoplasty in affected eyes, thermokeratoplasy (corneal diathermy or the Salaras procedure) can be used. In this procedure, superficial burns result in scar tissue that minimizes the potential space between the corneal lamellae to prevent formation of corneal bullae.

Corneal Degenerations

At first glance, corneal degenerations appear similar clinically to corneal dystrophies, but there important differences. Degenerations are often unilateral, occur secondary to other ocular or systemic disease, and often are vascularized and partially pigmented (Figure 8.18).

The clinical history includes previous ocular or corneal inflammation, trauma, or surgery. The corneal lesion, often containing triglycerides and cholesterol or mineral (calcium), contains tiny gray to white crystalline appearing opacities, superficial vascularization, and pigmentation. Treatment is not usually necessary for the corneal lesions, but can be necessary if systemic illness is

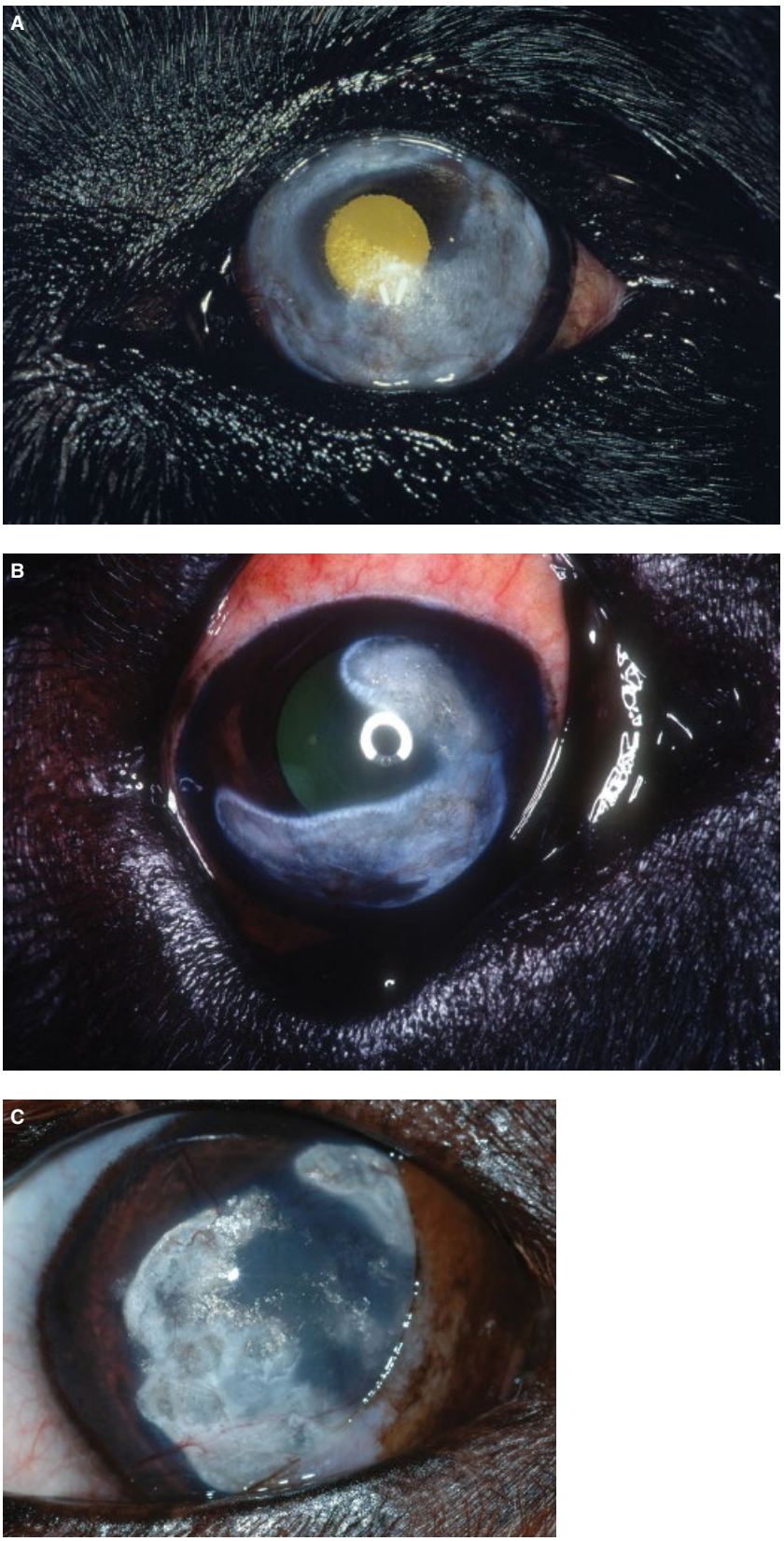


Figure 8.18 (A) Corneal degeneration associated with hypothyroidism in a female Rottweiler. Note the degeneration has not affected the central cornea and has superficial vascularization. (B) Corneal degeneration in a dog following trauma and corneal ulceration. (C) Severe lipid degeneration in the cornea of Golden Retriever. Note the vascularization and the different densities of the deposits.







Figure 8.18 (*Continued*) (D) Age-related corneal degeneration. The white opacities in the anterior stroma are likely mineral and predispose this cornea to ulceration. (E) Age-related degeneration, or mineralization, of the cornea in an elderly dog with an associated deep corneal ulceration. (F) Corneal degeneration related to the presence of an epibulbar melanoma.

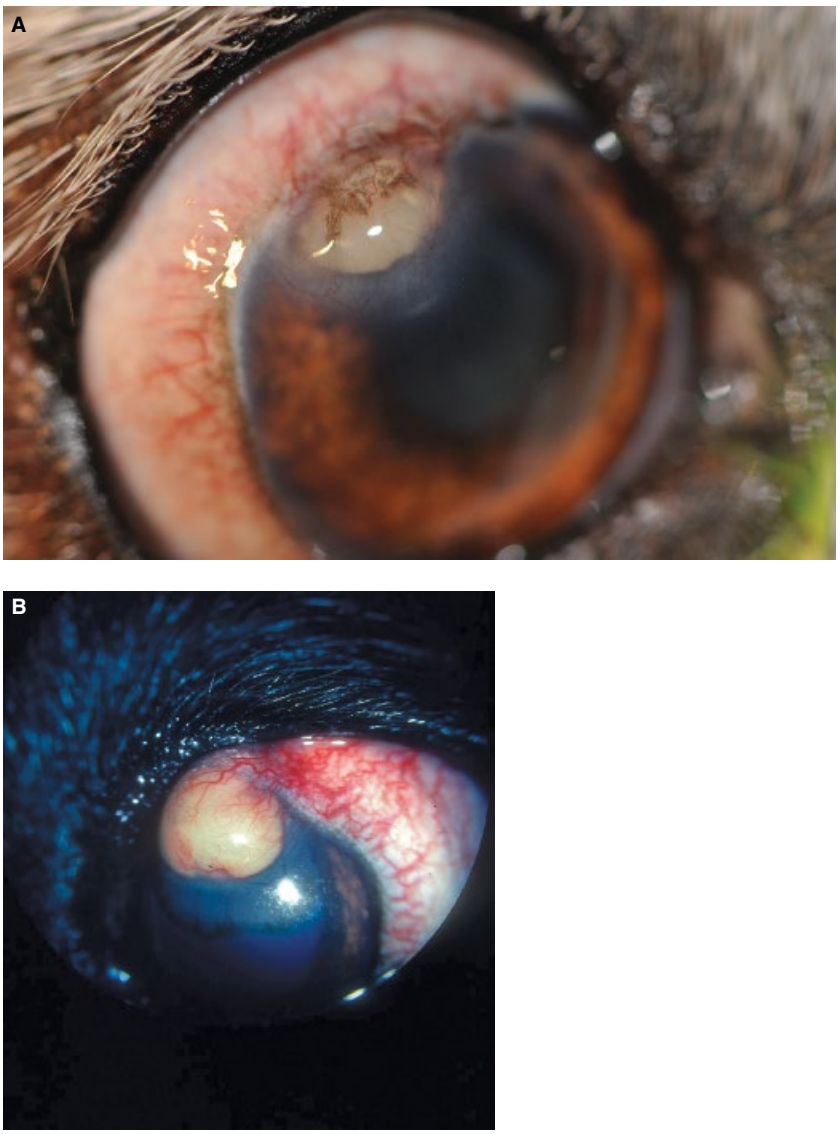


Figure 8.19 (A) Corneal cyst in a dog following corneal ulceration. The opaque cyst is raised above the corneal surface and contains superficial blood vessels. (B) Corneal cyst in a dog.

implicated in the pathogenesis. Hypothyroidism and hyperlipoproteinemia have been associated with lipid keratopathy or degeneration.

Corneal Cysts

Corneal cysts, like other cysts of the body, arise from the epithelium and usually possess clear to light yellow fluid (Figure 8.19). Corneal cysts are rare in dogs, and follow corneal trauma, ulceration, or surgery. These cysts are usually unilateral and, once formed, tend to persist and may expand. Examination reveals a solitary, raised, clear to white or light yellow lesion that if aspirated provides a clear fluid with a few nonkeratinized squamous epithelial cells. Treatment is usually by superficial keratectomy.

Limbal or Epibulbar Melanomas

Limbal or epibulbar melanomas primarily affect the middle-aged dog, and the German Shepherd breed appears predisposed (Figure 8.20). The dorsolateral limbus is most often affected. In younger affected dogs (2–4 years) these tumors grow more rapidly and are locally invasive. Examination reveals a smooth round to oval pigmented mass beneath the bulbar conjunctiva and extending onto the cornea. These tumors are usually quite superficial but occasionally extend into the deep layers of the sclera (necessitating some type of scleral graft after tumor excision). Ultrasonography and gonioscopy are essential to determine the exact depth of the tumor and whether or not scleral graft material will be needed at the time of surgical excision.

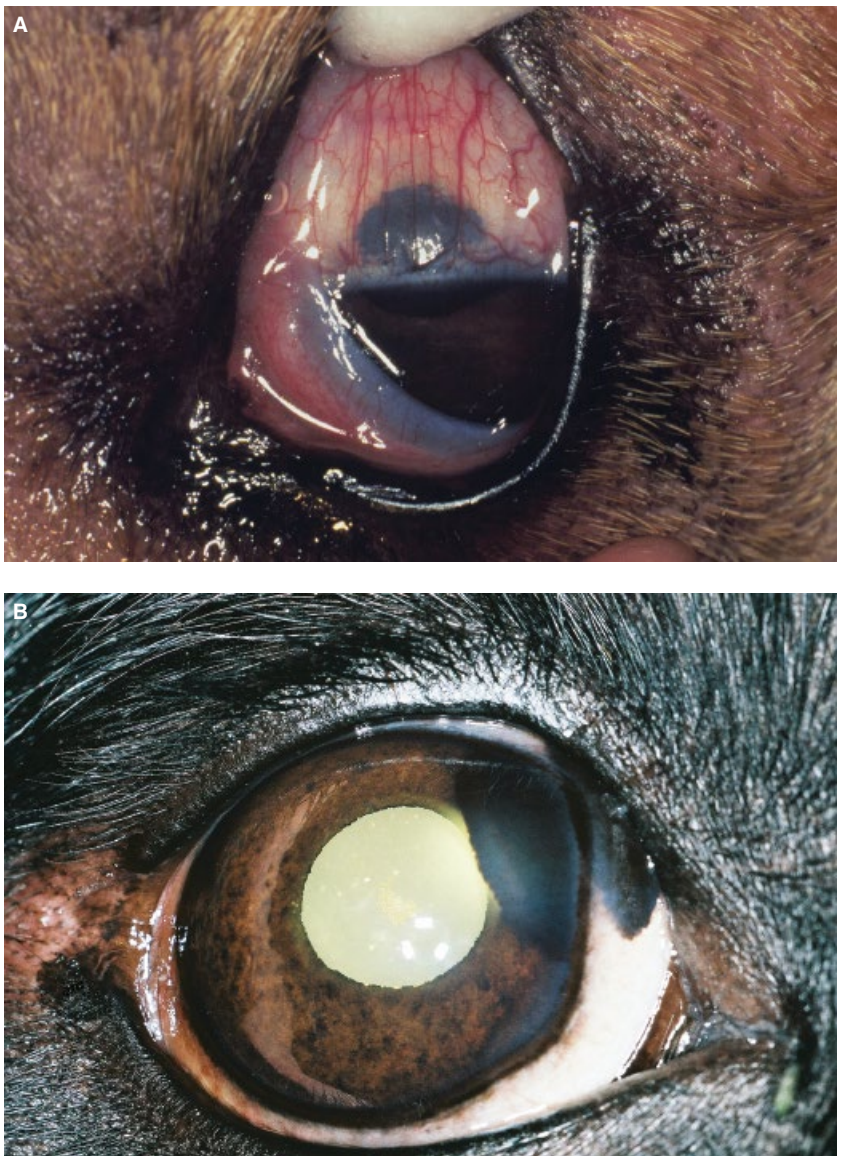


Figure 8.20 (A) Limbal melanoma in a German Shepherd Dog affecting the dorsal sclera and subconjunctival tissues. The tumor is beneath the dorsal bulbar conjunctiva and within the limbal sclera. (B) Limbal melanoma in a dog affecting the dorsolateral sclera. The tumor has extended along the corneal endothelium.

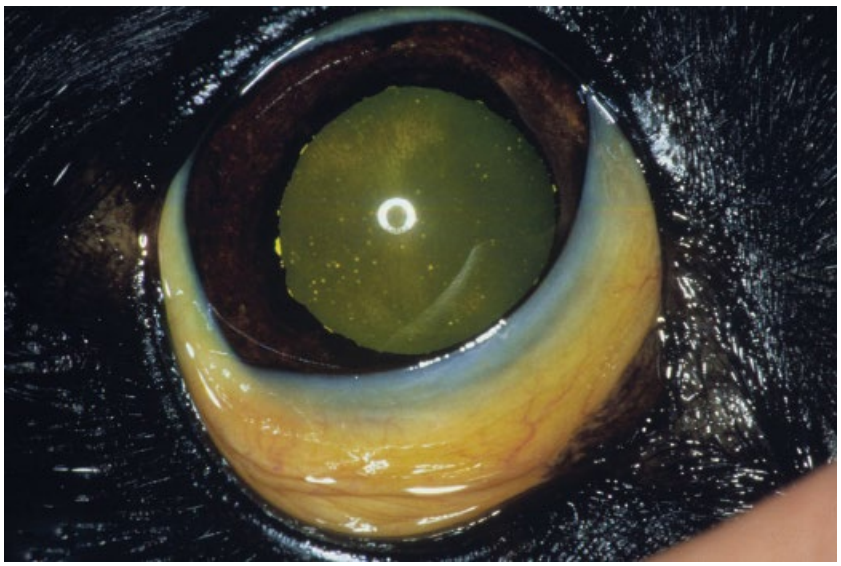


Figure 8.21 Scleral and conjunctival icterus in a dog. The yellow discoloration has affected the conjunctival and scleral tissues.

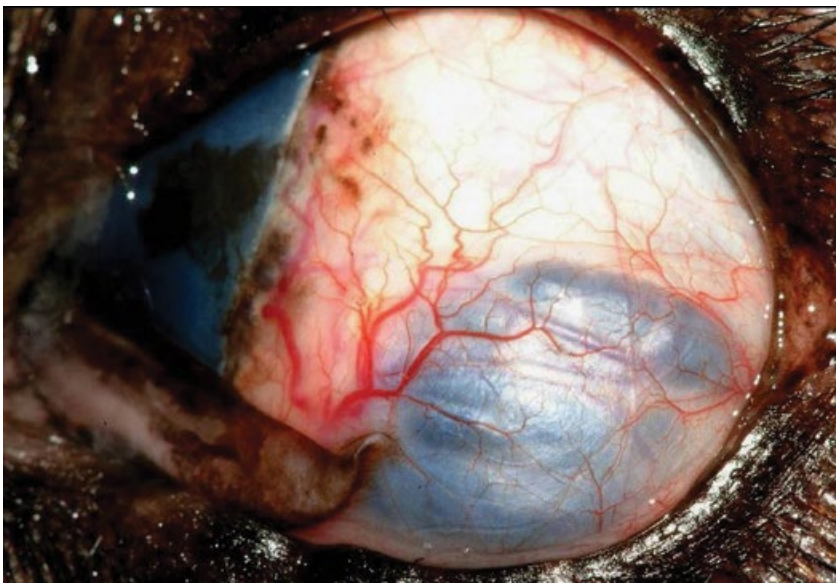


Figure 8.22 Staphyloma. Uveal tissue is visible beneath the thinned and weakened sclera.





Figure 8.23 (A) Histiocytoma of the dorsal episclera in a Boston Terrier dog. The mass has also extended onto the cornea. (B) Proliferative keratoconjunctivitis in a Collie. Both eyes were affected similarly. Sometimes the nictitans and eyelids are also involved.







Figure 8.23 (*Continued*) (C) Nodular granulomatous episcleritis in a Collie dog. (D) Episcleritis in a mixed-breed dog. Note the raised perilimbal scleral inflammation and the corneal edema adjacent to it. (E) Focal nodular episcleritis in a German Shepherd Dog.



Figure 8.23 (*Continued*) (F) Same dog as in Figure 8..23E in profile.

Treatment options vary with the rapidity of growth and the age of the patient. Therapy includes temporization, local excision, local excision with scleral graft, Nd:YAG and diode laser ablation, cryotherapy, and occasionally enucleation if there has been intraocular extension.

Scleral Diseases

Icterus sufficient to cause a yellow discoloration of the sclera is infrequent in the dog (Figures 8.21 and 8.22). Nevertheless, dogs with liver disease and elevated bilirubin may show this clinical sign.

Non-neoplastic or Inflammatory Lesions

Clinical diseases grouped in this classification include nodular fasciitis, fibrous histiocytoma, proliferative keratoconjunctivitis, limbal granuloma, pseudotumor, and Collie granuloma, as well as the nodular and diffuse forms of episcleritis. Nodular fasciitis can also affect the nictitans and subcutaneous tissues of the eyelid, and is characterized histologically by an abundance of fibroblasts with reticulin fiber formation. Corneoconjunctival histocytomas contain histiocytes, fibrocytes, plasma cells, and lymphocytes. The proliferative keratoconjunctivitis of the Collie and Shetland Sheepdog can involve the nictitans, eyelids, conjunctivae, and cornea, and may be bilateral (Figure 8.23). Episcleritis usually presents with engorged episcleral vessels and ciliary flush. Perilimbal corneal edema may be present concurrently. The etiology of these inflammatory lesions of the sclera is unknown, but an immune-mediated role is suspected.

Typically, the history is one of variable irritation, blepharospasm, and an enlarging pink mass involving the episcleral tissues and adjacent cornea. Gonioscopy and ultrasonography help in establishing the borders and depth of the mass. Treatments include topical and systemic corticosteroids, local excision, cryotherapy, systemic immunomodulating medications, or a combination of tetracycline and niacinamide. Recurrence is common.

9

Canine Glaucomas

We have used the term glaucomas because this group of ocular diseases, which affects the entire eye, includes at least 25 different types of glaucoma documented in humans and nearly as many types reported in the dog. Traditionally, the glaucomas are divided into primary, secondary, and congenital forms. The human and canine glaucomas generally have elevated intraocular pressure (IOP), optic nerve damage, and loss of retinal ganglion cells. In canine patients presented to veterinary college hospitals in North America over nearly a 40-year span, glaucoma patients represented about 2% of the total canine population and occurred in 45 breeds of dogs. Primary and secondary types occurred with equal frequency. The primary glaucomas are divided into narrow/closed angle glaucomas and open angle glaucomas. The majority of the breed-related primary glaucomas have not been investigated in any detail to document the distinction. In the USA, the most frequent type of primary glaucoma in the dog is narrow/closed angle glaucoma; in humans in the USA, the most frequent primary glaucoma is open angle. In Asian countries, the most frequent type of glaucoma in humans is narrow/closed angle. In secondary glaucomas, the ocular hypertension is attributed to another disease. In the dog, the most frequent cause of secondary glaucoma is lens displacement (lens luxation). The rarest form of glaucomas are congenital, affecting young puppies with anomalous outflow pathways and sometimes other ocular anomalies.

Diagnostic aids essential in the clinical management of the canine glaucomas include tonometry, gonioscopy (to examine the anterior chamber, or iridocorneal, angle) and ophthalmoscopy. In general, medical and surgical therapy must anticipate that the underlying outflow disease is progressive, therefore the maintenance of "safe" IOP changes over time.

Surgical therapy of the primary glaucomas is aimed at either decreasing aqueous humor production (usually by laser cyclophotocoagulation) or increasing aqueous humor outflow (anterior chamber shunts). Treatment for the secondary glaucomas requires concurrent treatment of the underlying etiology and the ocular hypertension.

Optic Nerve Head Changes in the Glaucomas

The optic nerve head/optic disc/optic papilla is the focus of injury with elevated IOP as it mostly contains the axons of the retinal ganglion cells as they exit the scleral sieve known as the lamina cribrosa. The peripheral retinal ganglion cells in the ocular fundus are the most sensitive to elevated IOP as they travel the greatest distance to converge at the optic nerve head and lamina cribrosa, and bend the greatest angle at the disc.

The normal canine optic nerve head is quite variable in appearance. It is located in the tapetal fundus, nontapetal fundus, or at their junction. When the optic disc is located in the tapetal fundus, it is usually surrounded by a pigmented ring. The canine optic disc is myelinated, but variably so, which accounts for its shape and size. From its surface and periphery emerge 15–20 primary arterioles (light red), and 3–4 primary retinal veins (darker red). Often, a complete or incomplete venous circle is near the center of the disc, which is the physiologic pit (optic cup) and remnants of the primary hyaloid system (Bergmeister papilla).

The optic nerve head or papilla changes in glaucoma appear to be influenced by the degree of IOP elevation and duration of the elevation. In primary open angle glaucoma, the papilla develops an enlarging central optic cup that signals progressive degeneration (Figure 9.1). In narrow/closed angle glaucoma, the rise in IOP often appears acutely (even within hours) and with considerably higher IOPs (50–80 mmHg) (Figure 9.2). The result is optic nerve atrophy. Infarcts in the peripapillary region and ischemia of individual short ciliary arteries, which supply the posterior segment (retina and choroid), have been associated with these high spikes in IOP. These infarcts appear as wedge-shaped hyperreflective areas in the tapetal ocular fundus. When viewing the ocular fundus by ophthalmoscopy in a potential glaucoma patient, it is important to measure IOP by tonometry before any interpretations are made.

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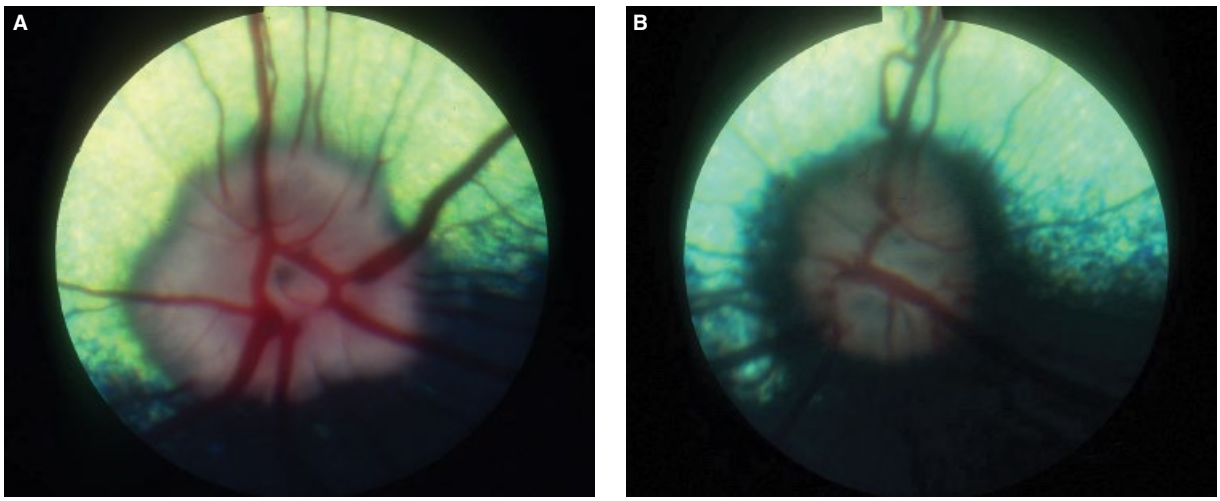


Figure 9.1 Optic nerve head and primary open angle glaucoma in the Beagle. (A) Early optic nerve head changes in primary open angle glaucoma. Note the depression or enlargement of the central optic nerve head nerve cup. (B) More advanced optic nerve head changes in primary open angle glaucoma. The optic nerve head is smaller than normal, its myelin lost, and has become pigmented.

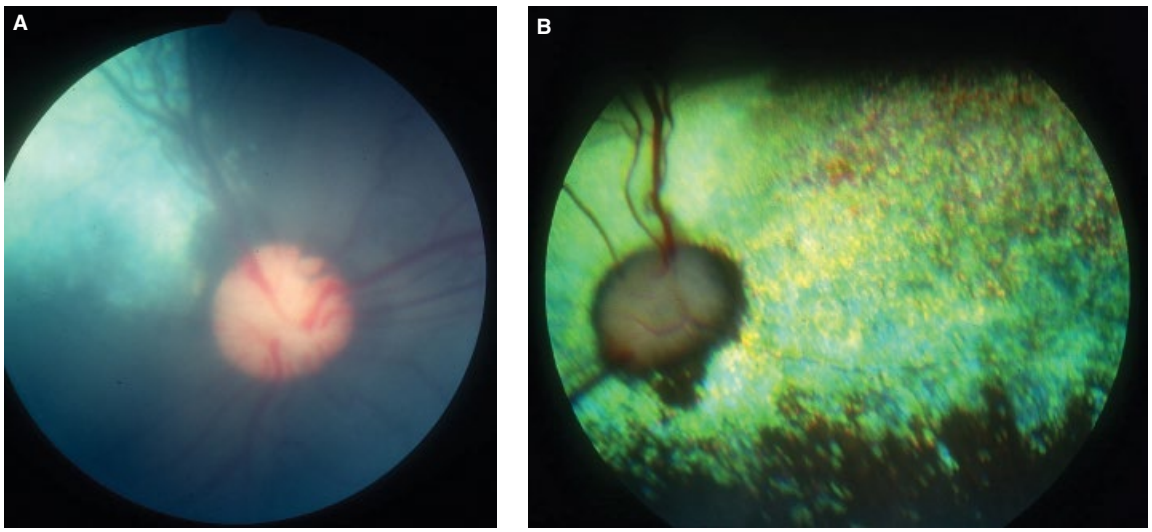


Figure 9.2 Optic nerve head changes in primary narrow/closed angle glaucoma (PCAG) in the dog. (A) Corneal edema develops when intraocular pressure (IOP) exceeds about 40 mmHg, which makes direct ophthalmoscopy more difficult because of the hazy view. In acute PCAG, the optic nerve head is swollen (papilledema) because of impaired axoplastic flow. Primary retinal blood vessels are constricted. (B) In advanced PCAG, the optic nerve head appears atrophied and most of the retinal blood vessels have disappeared. Because of the retinal degeneration, the tapetal fundus is hyperreflective.

Congenital Glaucoma

The congenital glaucomas affect young puppies which usually present with a unilateral rapidly enlarging globe (Figures 9.3 and 9.4). Anomalous aqueous outflow pathways are usually present, therefore both eyes should be evaluated closely; gonioscopy can be very helpful.

Upon presentation, megaloglobus (or buphthalmia) is present, the pupil is usually dilated, the episcleral vessels are enlarged, and lens luxation or subluxation is frequent. The puppy sclera is highly elastic (accounting for the greater degree of buphthalmia than that seen in an adult animal), but advanced retinal and optic nerve degeneration are usually present.

Short-term medical therapy is recommended to determine if the size of the globe reduces and if return of vision is possible. With an enlarged globe, lagophthalmia and persistent exposure keratitis occur, often necessitating



Figure 9.3 Congenital glaucoma in a Dalmatian puppy. The globe is enlarged and the cornea ulcerated due to exposure.



Figure 9.4 Congenital glaucoma in a Jack Russell Terrier puppy. The globe is enlarged and the cornea is edematous.

enucleation. Prophylactic therapy by the medical reduction of IOP for the fellow eye is also recommended.

Primary Narrow/Closed Angle Glaucoma

Primary closed angle glaucoma (PCAG) is the most common form in the canine and has been reported in at least 11 breeds including the American Cocker Spaniel, Samoyed, and Chow Chow. This condition is bilateral, but usually presents asymmetrically and months may separate recognition of clinical signs in both eyes (Figure 9.5).

Owners may observe the onset of PCAG as a series of self-limiting attacks of mydriasis, corneal edema, episcleral congestion, and perhaps increased sensitivity to touch about the eye and head. Presumably during these episodes IOP is also elevated, but within hours returns to normal levels. However, eventually the ocular hypertension and the clinical signs persist, initiating presentation to the veterinarian. Gonioscopy reveals an iridocorneal angle that is either narrow or closed. The opposite eye, which is often still asymptomatic, may also have narrow iridocorneal angle.









Figure 9.5 (A) Bilateral PCAG in the Siberian Husky. Advanced glaucoma in the right eye; early glaucoma in the left eye. (B) PCAG in the Chinese Shar Pei. The left eye has absolute glaucoma. (C) Acute congestive PCAG in the American Cocker Spaniel. Note the corneal edema, dilated pupil, and episcleral congestion. IOP is 66 mmHg. (D) Chronic PCAG in an American Cocker Spaniel. Note the buphthalmos, corneal edema, and episcleral injection.

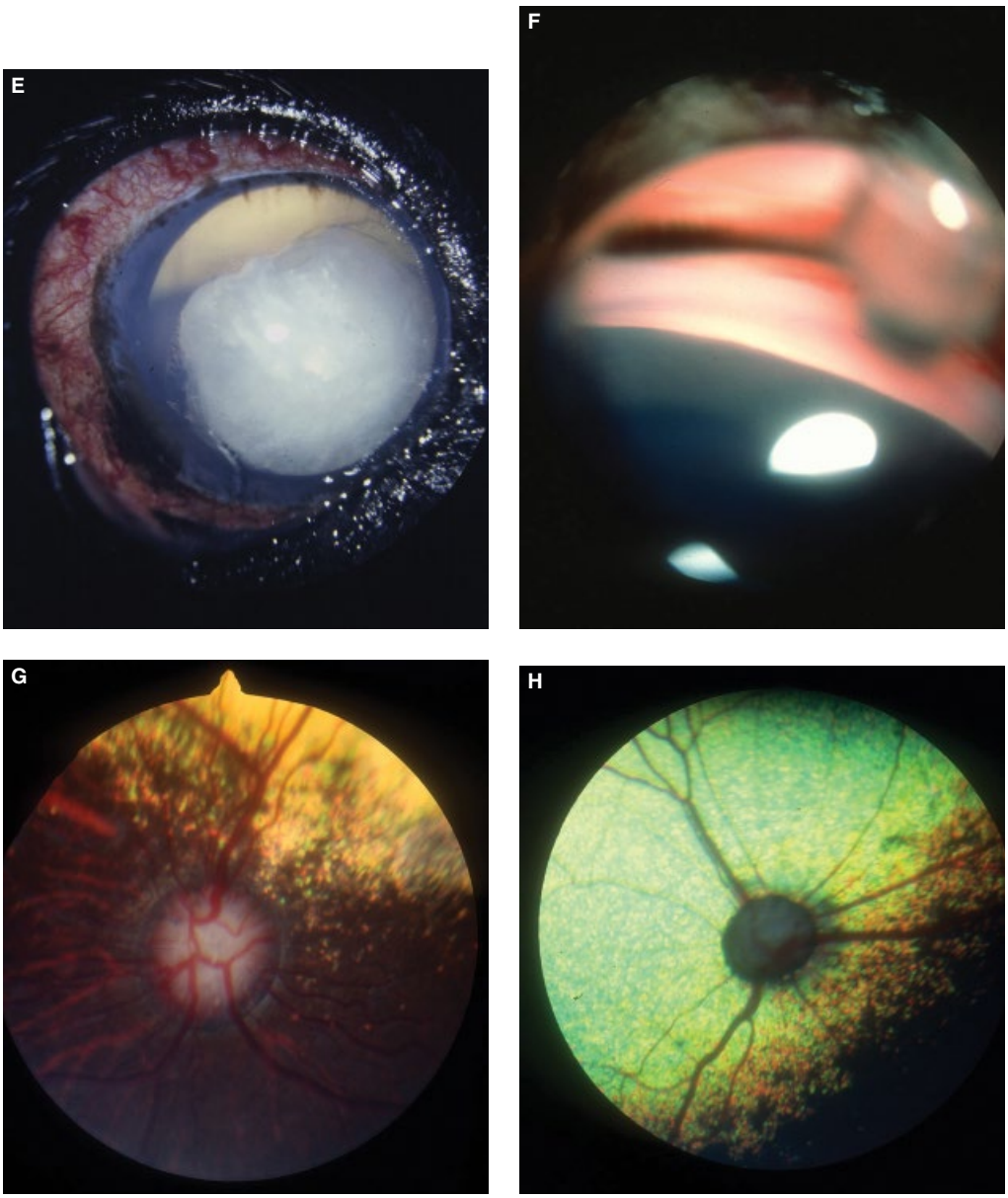


Figure 9.5 (*Continued*) (E) Advanced PCAG in the American Cocker Spaniel; note the enlarged globe, prominent and congested episcleral blood vessels, dilated pupil, and a cataractous and luxated lens within the anterior chamber. (F) Gonioscopic appearance of PCAG. The entire aqueous outflow apparatus is obstructed from direct observation by the base of the iris. (G) Fundus appearance of PCAG patient with optic nerve head atrophy. The papilla is smaller than normal, depressed, and some of the lamina cribrosa is exposed. (H) Fundus appearance of PCAG patient with advanced optic disc degeneration. The disc is reduced in size, myelin has been nearly lost, and the numbers of retinal blood vessels reduced to only the large primary arteries and veins.

Chronic PCAG results in blindness. Unfortunately, it is not unusual for a patient to present with chronic, end-stage disease in one eye and acute disease in the other. Signs of the advanced state include vision loss, fixed and dilated pupil, episcleral venous congestion, moderate to advanced megaloglobus, corneal edema and striae, lens luxation, cataract formation, vitreal degeneration and liquefaction, and optic nerve head and retinal degeneration. The optic nerve appears dark and depressed. The atrophied retina in the tapetal areas appears as hyperreflective areas with variably pigmentation and generalized loss of retinal vasculature.

Treatment depends on the stage of the disease and in the IOP and the response to medical therapy. Medical treatment usually involves polypharmacy with various combination of prostaglandins, miotics, beta antagonists,

alpha agonists, carbonic anhydrase inhibitors (both systemic and topical), and corticosteroids (iridocyclitis is often present concurrently). Acute disease can be treated with intravenous mannitol. Laser cyclophotocoagulation and anterior chamber shunts are the currently recommended surgical approaches. Prophylactic therapy of the asymptomatic eye with miotics or beta antagonists can delay the onset of the glaucoma for several months to as long as 2½ years.

Primary Closed Angle Glaucoma with Pectinate Ligament Dysplasia

PCAG with pectinate ligament dysplasia (persistent mesodermal remnants or goniodysgenesis) is typified in the glaucoma in the Basset Hound (Figure 9.6). Predisposed breeds include the Bouvier des Flandres, Flat Coated Retriever, Great Dane (in England), among others. The effect of this anomaly on the genesis of



Figure 9.6 PCAG with pectinate ligament dysplasia: (A) PCAG with pectinate ligament dysplasia in the Basset Hound eventually affects both eyes. In this dog, advanced glaucoma affects one eye; the opposite eye has early glaucoma (note the bilateral but unequal mydriasis). (B) The Basset Hound glaucomatous eye often has concurrent iridocyclitis. The cause of the inflammation has not been ascertained, but greatly complicates the medical control of IOP in these eyes. Note the conjunctival hyperemia (ciliary flush). A prostaglandin analogue has been administered which is responsible for the miosis present.

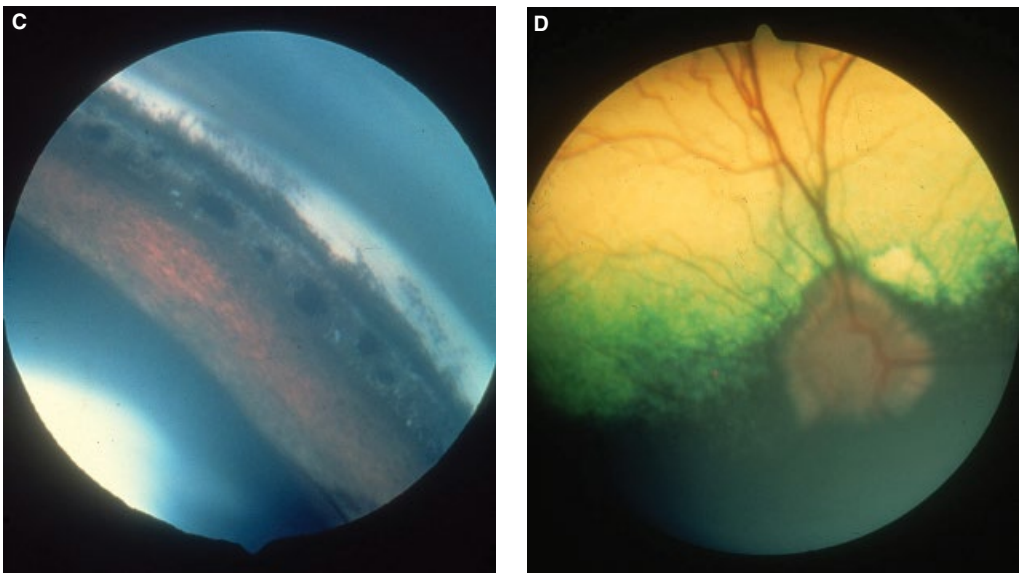


Figure 9.6 (*Continued*) (C) Gonioscopic appearance of a Basset Hound with PCAG and pectinate ligament dysplasia. Note the persistent bands of pectinate ligaments and irregular oval to round "flow holes." (D) Ocular fundus appearance of a middle-aged Bassett Hound with early PCAG with pectinate ligament dysplasia. Central excavation of the optic disc is present as well as retinal degeneration immediately dorsal of the disc and generalized reduction of the retinal blood vessels.

ocular hypertension is probably related to the extent or percentage that this anomaly affects the outflow pathways; in any event, only the most severely affected eyes appear to develop glaucoma. There can be additional iridocorneal angle abnormalities behind these anomalous pectinate ligaments. The clinical and histopathologic significance of a few dysplastic pectinate ligaments in an asymptomatic eye is not known.

PCAG with pectinate ligament dysplasia presents in the Basset Hound as an acute high pressure glaucoma often with iridocyclitis. Sometimes the history suggests multiple self-limiting bouts of conjunctival redness, and corneal edema. Clinical signs include blepharospasm, eyelid swelling, ciliary flush, episcleral congestion, diffuse corneal edema, pupil is slightly dilated or normal size, lens subluxation, megaloglobus, and variable optic nerve head and retinal degeneration. Aqueous flare may be present. Gonioscopy reveals a narrow angle with short stout pectinate ligaments, a restricted opening to the iridocorneal cleft, and large areas of solid pigmented bands with "flow holes" bridging the cleft opening, or a closed angle.

When presented with the advanced form of this glaucoma, the globe can be quite enlarged. The other clinical findings are similar to the initial presentation, but more severe. Gonioscopy, if possible, through an edematous cornea, reveals a closed angle.

Treatment of the Basset Hound PCAG with pectinate ligament dysplasia is difficult because of the combination of the glaucoma and iridocyclitis. The same medications and surgeries are employed as presented for PCAG. With the combination of both glaucoma and iridocyclitis, medical management is more difficult.

Primary Open Angle Glaucoma

Primary open angle glaucomas (POAG) occur in several breeds of dogs, but have been studied in depth in the Beagle (Figure 9.7). POAG is inherited in the Beagle as an autosomal recessive trait. The course of this form of glaucoma often spans several months to 2–3 years. Most early POAG are asymptomatic for months to a few years, except for slight mydriasis and episcleral venous congestion. Once IOP exceeds about 30 mmHg, progressive mydriasis, globe enlargement, lens subluxation, and narrowing to closure of the iridocorneal angle occur.

The ophthalmic examination results vary with the stage of the glaucoma. In the early stage when the dog is 1–3 years old, the eye appears normal; however, there is mild IOP elevation (20–25 mmHg) and a diurnal variation in IOP that exceed 8–10 mmHg. The iridocorneal angle and ciliary cleft are open and gonioscopically normal in appearance. Once the clinical signs of glaucoma develop (IOP 30–40 mmHg), mydriasis, episcleral venous congestion, slight corneal edema, slight megaloglobus, lens subluxation, and, by gonioscopy, an iridocorneal angle that is either open or narrowed. By ophthalmoscopy, the optic nerve and retina can demonstrate some early degeneration.

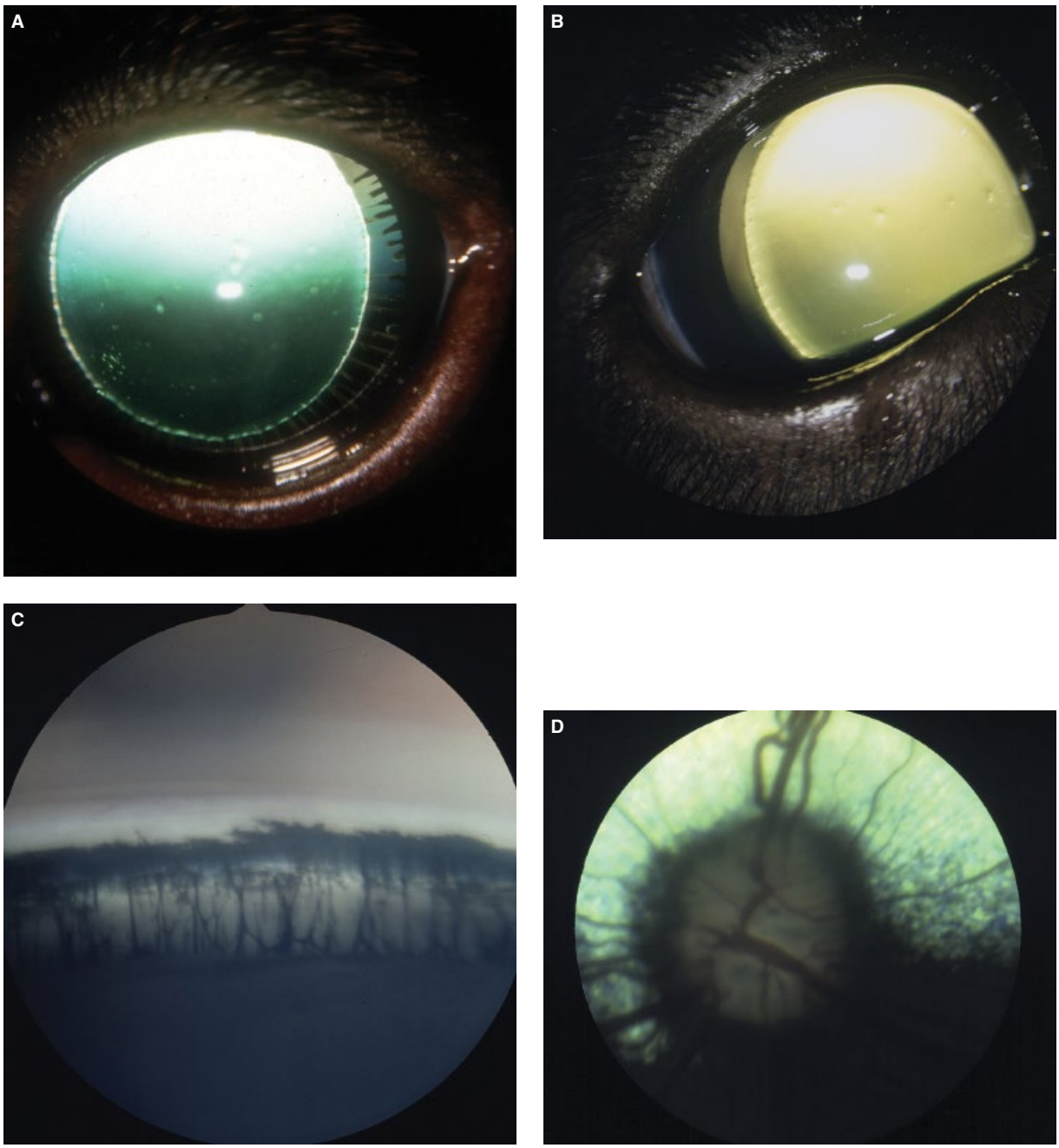


Figure 9.7 (A) In POAG in the Beagle the enlargement of the globe is slow. With slight globe enlargement, the periphery of the lens becomes visible, along with elongated ciliary processes from the increased zonulary tension. (B) As POAG advances, the globe enlargement continues and eventually lens subluxation occurs. Note the lateral aphakic crescent in the pupil where there is no lens. (C) By gonioscopy, the iridocorneal angle and opening of the ciliary cleft are open and devoid of abnormalities. However, with megaloglobus, iridocorneal angle and ciliary cleft closure results. (D) Optic nerve head degeneration in a Beagle with moderately advanced POAG. Note the flattened optic disc surface, loss of myelin, and "baring" of the lamina cribrosa. The number of small retinal blood vessels is also reduced.

In advanced glaucoma when the dog is 3–6 years old, these clinical signs progress. The megaloglobus is profound with persistent mydriasis, and the corneal edema is more dense and striae begin to form. The pupil fails to respond to light. The lens is subluxated; total displacement and cataract formation are not uncommon. The optic nerve and retinal degeneration are advanced, and vision is lost.

Treatment of POAG depends on the stage of diagnosis. This form of the disease is more medically responsive

than the narrow/closed angle types if it is diagnosed in its early stages. Once the iridocorneal angle has narrowed or closed, medical management is difficult, and surgery should be considered.

Comparisons of Human and Canine Glaucomas

Clients are often informed about glaucoma because they have been diagnosed with glaucoma or they know someone with the disease. As a result, pointed questions can be expected. The more we educate our pet owners about their pet's disease, the more realistic their expectations become. In North America, the frequency of both human and canine glaucomas is 2%, but the most frequent type of human primary glaucoma is POAG, whereas in the dog it is PCAG. Open angle glaucoma in humans is usually diagnosed in their annual eye examination, usually early in its process, when it can be treated with long-term topical drugs with preservation of vision and high success rates. Unfortunately, the same is not true for the dog; the most frequent type is the narrow/closed angle type, it is presented with one eye in advanced stage of the disease, and long-term medical control is impossible.

If the same type of glaucoma (i.e., PCAG) is compared between humans and dogs, the differences are comparable. In humans with narrow/closed glaucoma, 50% of these patients have marked vision loss in one eye on the first visit to the ophthalmologist and blindness is frequent. Like dogs, their response to medical therapy is poor. Most MD ophthalmologists in America and Europe rarely encounter patients with narrow/closed angle disease, so based on their experiences with the open angle form, they fail to appreciate a realistic outcome in the dog.

Secondary Glaucomas

Secondary glaucomas include a group of diseases with elevated IOP that can be attributed to a distinct cause. In the management of these glaucomas, successful treatment of the cause in the early stages can eliminate the glaucoma. In advanced secondary glaucoma, the changes to the eye may be irreversible and hence more difficult to treat.

Lens Displacement or Lens Luxation

Lens displacement or lens luxation is the most frequent secondary form of glaucoma in the dog (Figure 9.8). Due to a structural defect in the lens zonules (zonular dysplasia), it affects Terrier breeds most frequently (e.g., Jack Russell or Parson Russell Terriers, Wire Hair Fox Terrier, Smooth-haired Fox Terrier, Sealyham Terrier), but is also inherited in the Border Collie and Tibetan Terrier. The inherited forms of lens dislocation usually occur in younger to middle-aged individuals (2–5 years old) and are often bilateral. Lens luxation also occurs in dogs that have advanced cataracts (hypermature stage) and are aged (8–10 years old).

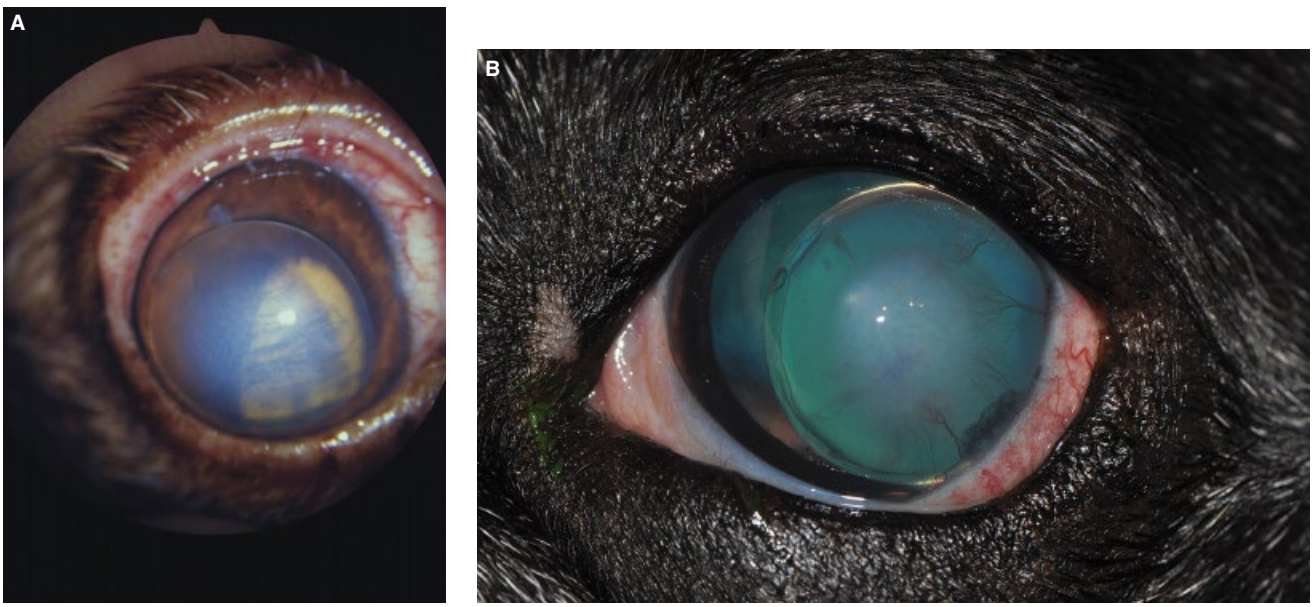


Figure 9.8 Lens luxations or displacements in the dog. (A) Anterior lens luxation or displacement into the anterior chamber in a Brittany Spaniel. Note the iris and oval pupil are obscured and behind the lens. (B) Anterior lens luxation in a Boston Terrier. Note the axial corneal edema where the lens has made contact with the corneal endothelium.



Figure 9.8 (*Continued*) (C) Lens subluxation in a dog. Although the lens is partially in the patellar fossa, it is often tilted. Note the large medial aphakic crescent. (D) Posterior luxation of a cataractous lens with secondary glaucoma in a mixed-breed Terrier-cross dog.

The lens displacement can be subluxation (within the patella fossa), complete luxation into the anterior chamber (usually with the vitreous still attached), or complete luxation into the posterior vitreous (often lying on the ventral retina). The affected eye can be asymptomatic or demonstrate repeated "attacks" of ocular hypertension (conjunctival redness, corneal edema, dilated pupil, and temporary loss of vision).

The effect of lens displacement on IOP varies with lens position and the condition of the vitreous (normal consistency or liquefied). Anterior lens luxation usually increases IOP by blockage of the pupil with the lens and adherent vitreous obstructing anterior flow of aqueous humor. There can also be subtle iris bombé with iridocorneal angle closure. The effect of lens subluxation on IOP is more variable; phacodenesis and iridodenesis contribute to pupillary blockage; a low grade iridocyclitis may also be present. Vitreous can enter the pupil and anterior chamber through the aphakic area created by the torn zonules. In the vitreal or posterior lens luxations the anterior face of the vitreous is torn and the lens often lies on the ventral ocular fundus. Increased IOP results from movements of the lens throughout the anterior and vitreal chambers, displacement of vitreous into the anterior chamber, and the accompanying iridocyclitis.

Reports and experience to date suggest that early lens removal (by cryoextraction or phacoemulsification) yields the best results and preserves vision the longest.

Often, IOP elevations postoperatively require persistent antiglaucoma therapy for maintenance of vision in the long term. Success rates for surgical removal of displaced lenses are lower than for phacoemulsification of cataracts, with glaucoma and retinal detachments being potential complications. If possible, it is preferable to phacoemulsify the lens contents (in the bag), and to leave the lens capsules "in situ" so as to minimize any vitreous changes.

Anterior Uveitis and Secondary Glaucoma

The second most frequent form of secondary glaucoma is anterior uveitis (iridocyclitis), often in chronic or recurrent forms (Figure 9.9). Anterior uveitis affects IOP and aqueous humor dynamics as a result of inflammatory cells, proteins, and debris in the aqueous outflow channels, from synechiae formation by the adherence of the inflamed "sticky" iris to the lens or cornea, or from pupillary occlusion. Uveitic glaucoma is common in cases of chronic anterior uveitis, such as following corneal perforation, uncontrolled lens-induced uveitis, systemic diseases, uveodermal syndrome (Vogt–Koyanagi–Harada syndrome) and other forms of autoimmune uveitis, and pigmentary uveitis in the Golden Retriever breed (Figure 9.10).

Clinical signs in affected animals are a combination of those of anterior uveitis and glaucoma: some irritation, blepharospasm, lacrimation, deep corneal vascularization, conjunctival hyperemia and episcleral venous congestion,

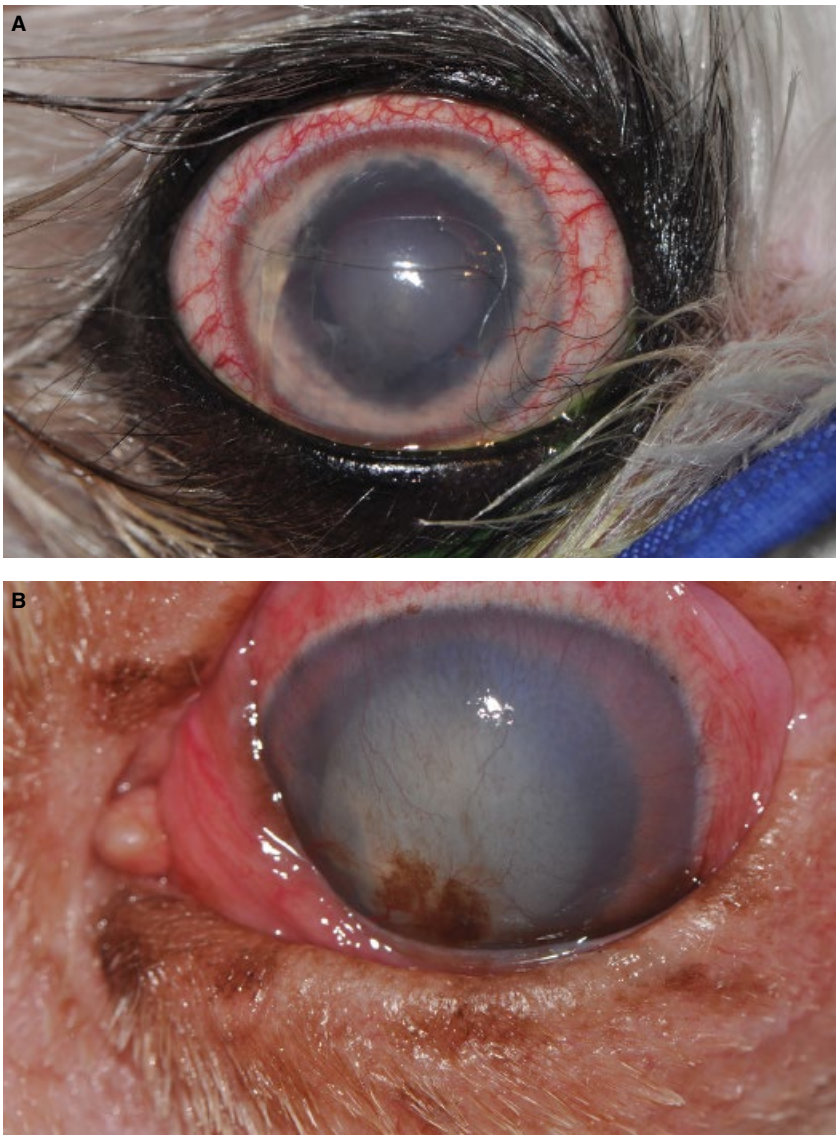


Figure 9.9 Cataract formation, resorption, lens-induced uveitis, and glaucoma. (A) Severe anterior uveitis and secondary glaucoma. Moderate buphthalmia is also present. (B) Glaucoma secondary to lens-induced uveitis. The cornea is edematous and heavily vascularized.







Figure 9.9 (*Continued*) (C) Secondary glaucoma in an eye with hypermature cataract and intraocular hemorrhage. Note the iris is darker than normal reflecting increased pigmentation from chronic uveitis. The lower eyelid margin mass is incidental. (D) Hypermature cataract, lens-induced uveitis, and recent secondary glaucoma. The cataract is reduced in size overall and its anterior surface is flat. (E) Severe anterior uveitis with secondary glaucoma.

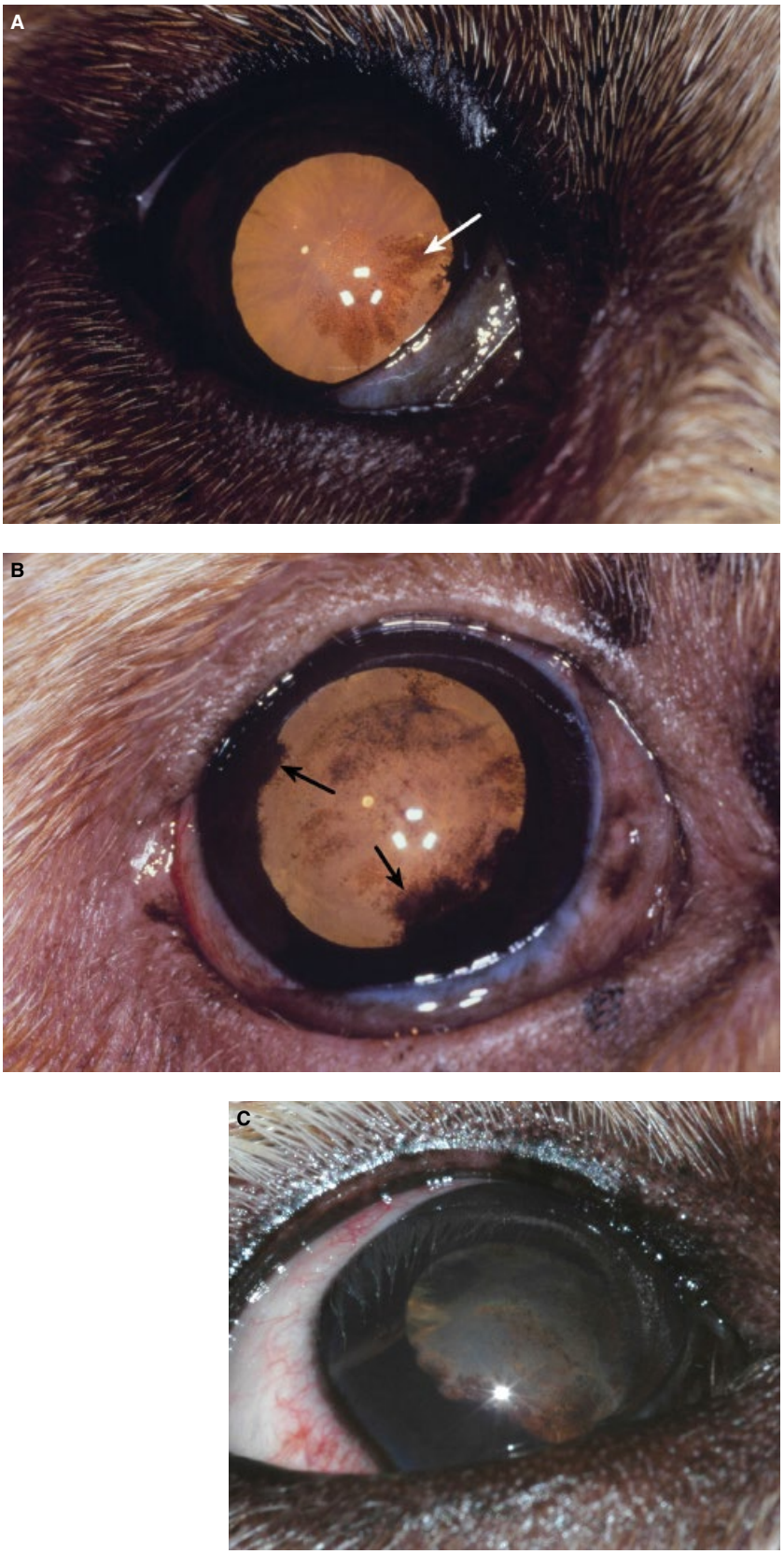


Figure 9.10 Golden Retriever chronic uveitis/uveal cysts syndrome. About 50% of Golden Retrievers with this eye disease will develop glaucoma; close monitoring is advised. (A) Note the numerous pigment deposits (arrow) on the medial anterior lens capsule as well as limited posterior synechiae. (B) More advanced case. Large number of pigment deposits and several posterior synechiae (arrows) are present. (C) Advanced case of pigmentary uveitis and secondary glaucoma.

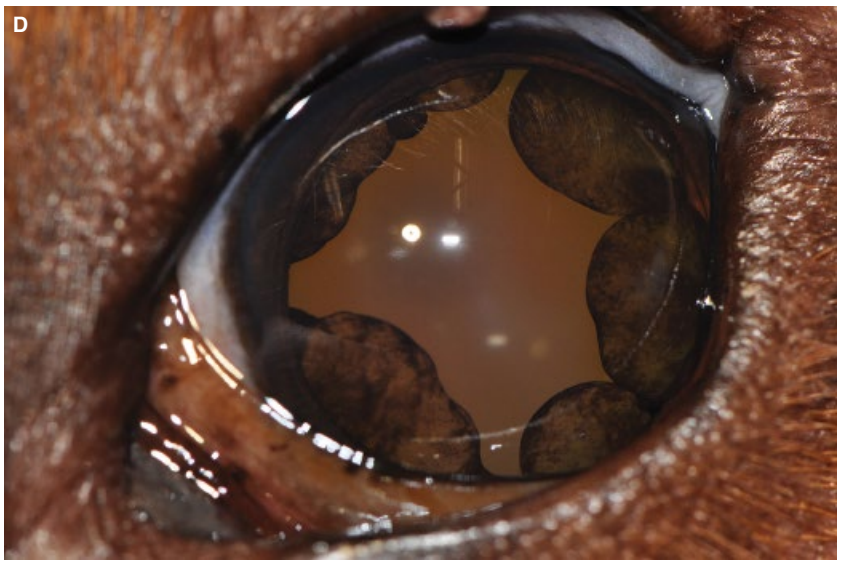


Figure 9.10 (*Continued*) (D) Multiple anterior uveal cysts behind the iris in a Golden Retriever.

corneal edema, midrange pupil, pigment spots on the anterior lens capsule, aqueous flare, and elevated IOP.

Treatment consists of topical and systemic corticosteroids, and nonsteroidal anti-inflammatory agents (for the anterior uveitis), and drugs to lower IOP. Prognosis is usually guarded.

Aphakic and Pseudophakic Glaucomas

The incidence of glaucoma after cataract surgery is variable, but ranges from 5% to 15% (Figure 9.11). The glaucoma results from: (i) pupillary blockage and iris bombé with posterior synechiae; or (ii) the interference of aqueous outflow through the iridocorneal angle secondary to the formation of peripheral anterior synechiae from the postoperative anterior uveitis and formation of preiridal vascular membranes. A unique form of the pupillary blockage glaucoma is malignant glaucoma or aqueous misdirection in which vitreous is forced through a small pupil into the anterior chamber restricting aqueous humor flow into the anterior chamber, which is redirected into the vitreal space. Surgical establishment of a patent pupil is essential and must be performed early. Unlike in humans, laser photocoagulation of pupillary membranes is difficult in the dog as the membranes are thicker and tougher.

Cataract surgery usually proceeds the development of glaucoma by several weeks to months. Vision impairment or blindness is usually present. Treatment should be directed towards controlling the anterior uveitis and lowering the IOP.

Traumatic Glaucoma

Traumatic secondary glaucoma is uncommon in the dog, in contrast to humans. The trauma can be either blunt or penetrating. The canine eye response to direct trauma is often phthisis bulbus (atrophy of the eye) because of the resultant anterior uveitis rather ocular hypertension. One of the more common examples of trauma causing secondary glaucoma is a penetrating corneal wound caused by a cat scratch and resultant severe anterior uveitis (Figure 9.12). Treatment is directed at both the anterior uveitis and ocular hypertension.

Hyphema and Secondary Glaucomas

Hemorrhagic secondary glaucoma is rare in the dog, and usually follows repeated bouts of intraocular hemorrhage, which usually does not clot (Figure 9.13). As in trauma, the canine response to intraocular hemorrhage is more often phthisis bulbus (atrophy of the eye) rather than ocular hypertension. Intraocular hemorrhage can also occur within a glaucomatous eye secondary to rhegmatogenous retinal detachment and giant retinal tears. Ultrasonography is an essential diagnostic aid for this condition as visual inspection of the deeper ophthalmic structures is not possible. Treatment consists of drugs to reduce IOP. The anterior chamber hemorrhage can be cleared, at least temporarily, by the intracameral injection of TPA (25 μg tissue plasminogen activator). Generally, the prognosis for return of vision is poor.





Figure 9.11 (A) Secondary aphakic glaucoma after cataract surgery in a dog. Note the globe enlargement, corneal edema, iridal swelling, irregular and fixed pupil, pupillary membrane formation, and intraocular hemorrhage. (B) Pseudophakic glaucoma in a Boston Terrier. Note the dorsal corneal incisions in both eyes, the result of phacoemulsification lens removal surgery. The glaucoma resulted from gradual closure of the iridocorneal angle and ciliary cleft several months after phacoemulsification for cataract.



Figure 9.12 Traumatic glaucoma in a dog secondary to corneal laceration (cat scratch) and secondary anterior uveitis. Note the corneal scar and anterior synechiae (adhesion between iris and cornea). The glaucoma resolved once the uveitis was medically controlled.

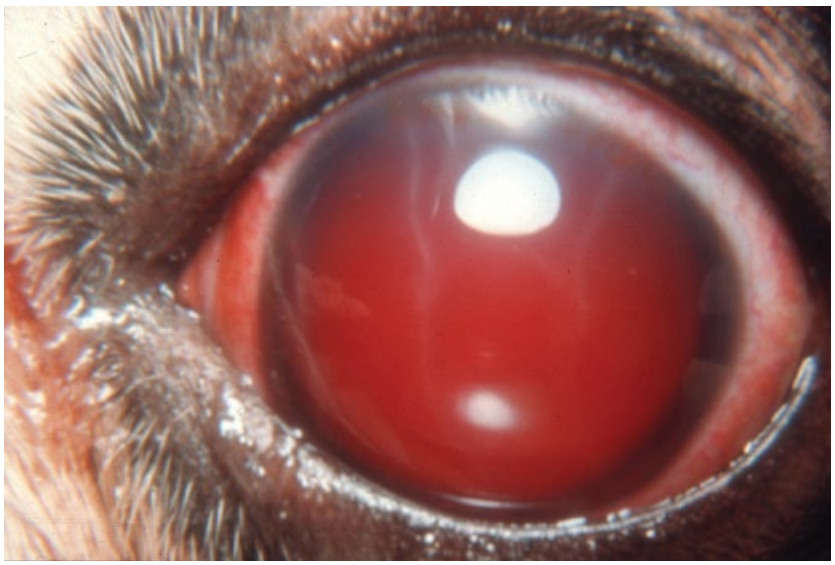


Figure 9.13 Secondary glaucoma from intraocular hemorrhage in a Boston Terrier dog. The source of the hemorrhage was a complete retinal detachment with a giant retinal tear.

Pigmentary or Melanocytic Glaucoma

Pigmentary or melanocytic glaucoma occurs in middle-aged Cairn Terriers and other isolated breeds (Figure 9.14). Prominent areas of pigment deposition develop within the filtration angle, episcleral and subconjunctival tissues, tapetal fundus, and even the optic nerve. It is thought these aggregates of melanocytes interfere with aqueous humor outflow and result in slow onset chronic glaucoma.

The ophthalmic examination reveals episcleral congestion, slight to moderate buphthalmia, a dilated pupil, and areas of pigmentation scattered throughout the eye. Gonioscopy reveals diffuse pigmentation within the filtration angle and outflow channels into the anterior sclera. Treatment consists of drugs to lower IOP and often filtration surgery. Because the pigmented cells continue to increase, long-term prognosis is very poor.

Intraocular Neoplasia and Secondary Glaucoma

Both primary and secondary intraocular neoplasms can present as an overt mass, persistent uveitis, recurrent to persistent intraocular hemorrhage, and/or secondary glaucoma. Anterior uveal melanomas and adenomas/adenocarcinomas of the ciliary body represent the majority of the primary intraocular neoplasms in the dog (Figures 9.15, 9.16, 9.17, 9.18, and 9.19).

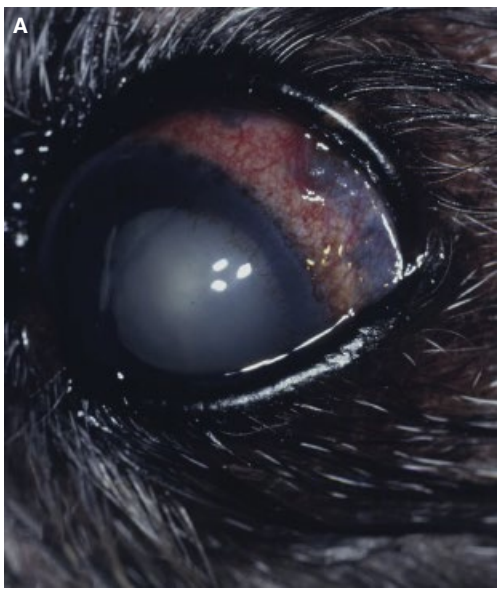


Figure 9.14 (A) Melanocytic secondary glaucoma in a Cairn Terrier associated with pigment cell proliferation and alterations in the aqueous humor outflow pathway. Note the congested conjunctival blood vessels, dilated pupil, mature cataract, and pigmentation beneath the bulbar conjunctiva.



Figure 9.14 (*Continued*) (B) Profile view of a Cairn Terrier with pigmentary glaucoma. Note the pigment deposition in the sclera and the episcleral injection.

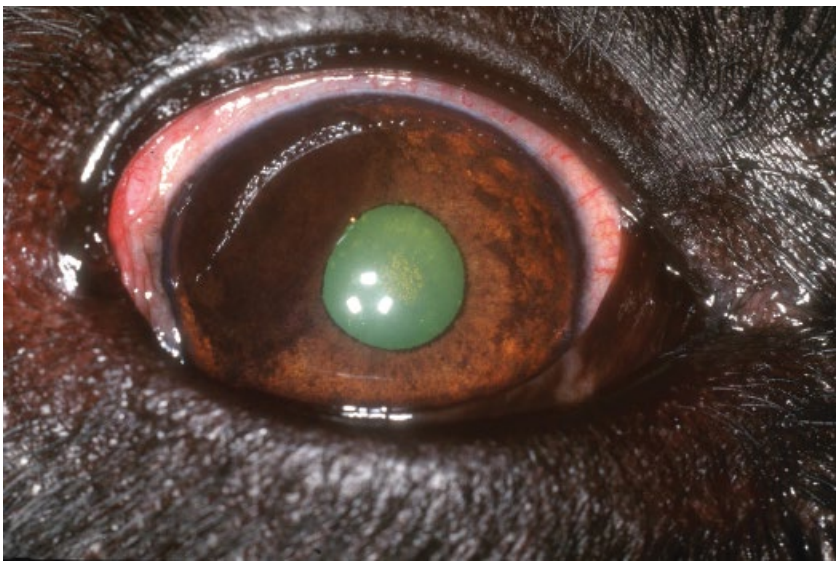


Figure 9.15 Secondary glaucoma and malignant melanoma of the ciliary body. The tumor has developed in the ciliary body and extended onto the surface of the iris.

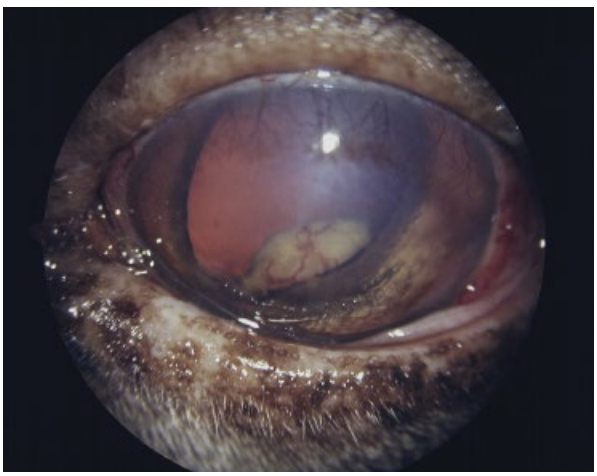


Figure 9.16 Secondary glaucoma and ciliary body primary adenocarcinoma. The white mass has emerged from behind the iris into the pupil and has blood vessels on its surface.

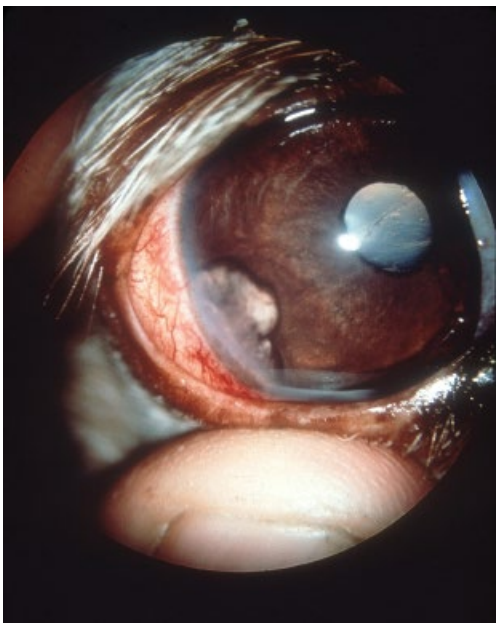


Figure 9.17 Secondary glaucoma and metastatic nasal adenocarcinoma.

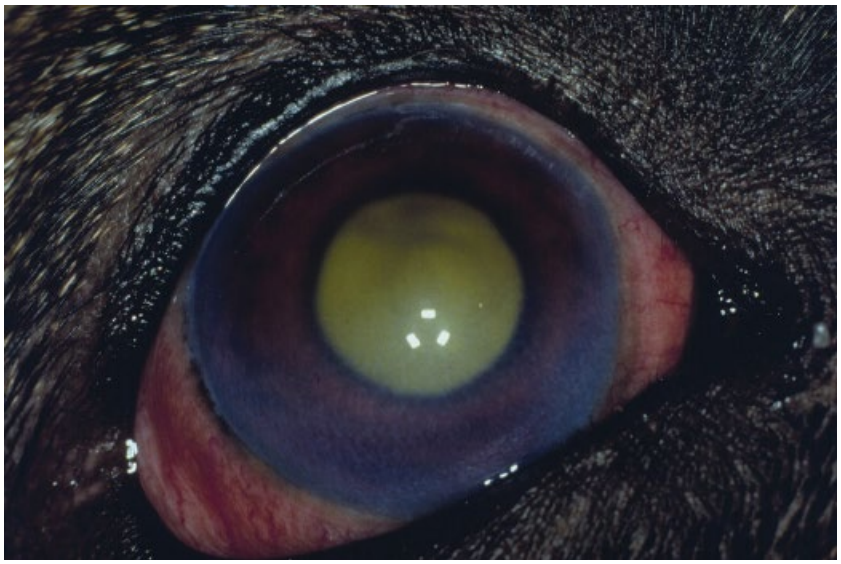


Figure 9.18 Glaucoma secondary to anterior uveitis and lymphoma in a dog. Note the enlarged globe, corneal edema, and swollen iris. The glaucoma resulted from obstruction of the iridocorneal angle and ciliary cleft by inflammatory and neoplastic cells.



Figure 9.19 Glaucoma secondary to anterior uveitis and lymphoma in a dog. Note the extensive intraocular hemorrhage.

The history indicates a slow onset of buphthalmia, episcleral congestion, persistent inflammation, and/or intraocular hemorrhage. Upon examination, a pigmented or nonpigmented mass is observed arising from the iris or from the ciliary body (appearing in the pupil or at the base of the iris). Evidence of inflammation (usually associated with tumor necrosis), and hemorrhage may be present. Inspection of the outflow pathways by gonioscopy and the entire eye by ultrasonography is important. Treatment, once glaucoma has occurred, is usually by enucleation.

Surgical and Laser Treatment for Canine Glaucoma

There are several surgical procedures used to treat the glaucomas. They are generally divided into those that increase the outflow of aqueous humor and those that reduce the rate of aqueous humor formation. The most frequent procedures currently used are anterior chamber shunts (to increase outflow) and laser cyclophotocoagulation (to decrease the rate of formation) (Figure 9.20).

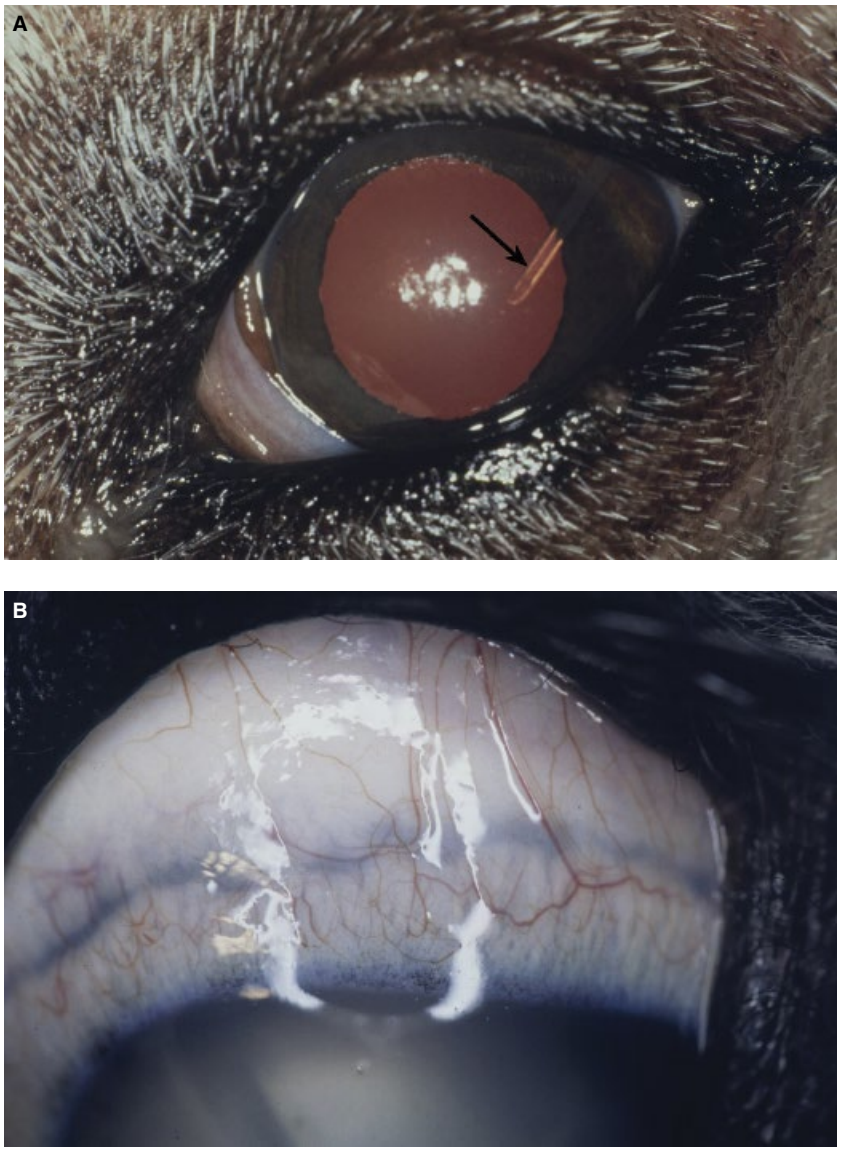


Figure 9.20 (A) Treatment of PCAG with an anterior chamber shunt. The silicone drainage tube (arrow) is visible within the anterior chamber exiting through the dorsolateral limbus into the retrobulbar space. (B) The effects of laser cyclophotocoagulation on the sclera when used for treatment of canine PCAG. Note an irregular row of laser "burns" 4–6 mm posterior to the limbus.

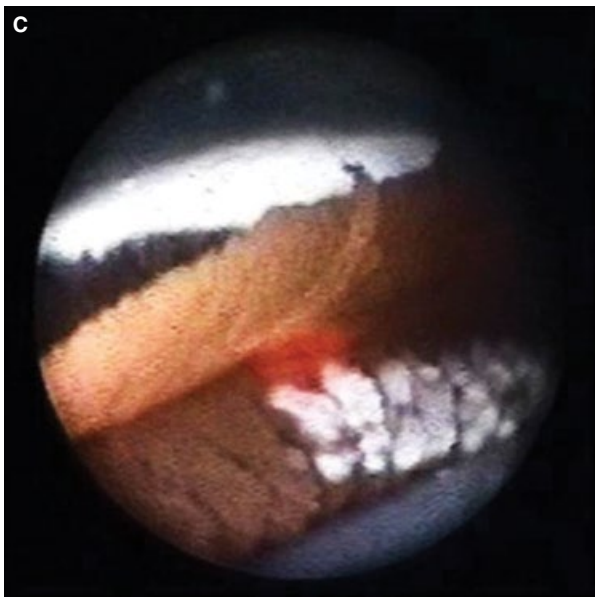


Figure 9.20 (*Continued*) (C) Intraoperative view of endolaser photocoagulation near a ciliary body process. Note the charred ciliary process after photocoagulation.

Anterior chamber shunts are usually positioned about 10–12 mm posterior to the limbus, but fibrosis about the implant can eventually cause failure. The tubing within the anterior chamber bypasses the impaired aqueous humor pathways and carries the aqueous humor to the device's episcleral base and into the subconjunctival space. The aqueous humor is absorbed by conjunctival capillaries. Fibrosis commonly occurs around the implant and is a cause of failure. It appears to be stimulated by components within the aqueous humor. Often, shunt placement is performed in combination with laser cyclophotocoagulation and achieves the short-term aim of fluid diversion before the effects of the laser destruction are clinically apparent.

In transcleral laser cyclophotocoagulation the probe is directed a few millimeters posterior of the limbus through the bulbar conjunctiva and sclera toward the ciliary body. As a result of laser cyclophotocoagulation, release of uveal pigment, mild hemorrhage, and cyclitis result. The success rate of laser cyclophotocoagulation for IOP control is better than for maintenance of vision. Recently, endolaser photocoagulation has been used with encouraging results. The laser is inserted through the sclera or through the pupil with aphakes or pseudophakes, and the ciliary processes are photocoagulated individually under direct observation.

10

Canine Anterior Uvea

The canine uveal tract is divided into anterior (iris and ciliary body) and posterior (choroid) portions. The anatomic divisions are also useful clinically, as often the anterior and posterior uvea are affected by separate clinical disorders.

Congential Variations and Disorders

Heterochromia Iridis

Heterochromia iridis indicates multiple colors within one iris or between the irides. In many breeds, a unilateral completely blue iris is acceptable for that breed standard; in other breeds a unilateral blue iris is rejected for registration. In some breeds, such as the Siberian Husky, both irides can be blue (Figure 10.1). Blue irides in certain breeds, such as the Dalmatian, have been associated with deafness and cardiac defects. The ocular fundus in eyes with blue irides can be subalbinoid with limited to no pigmentation of the nontapetal fundus visible with ophthalmoscopy. The tapetum lucidum is usually present, appearing as a yellow–green triangular area (the tapetal fundus), but is occasionally absent (atapetal).

Heterochromia iridis is also associated with the merling gene and coat color in many breeds. In heterozyzous dogs (Mn), heterochromia irides can appear as completely blue, or as partially brown with focal areas of blue and white. The ocular fundi in these dogs can be totally pigmented, subalbinoid with or without a tapetal fundus, and either pigmented or hypopigmented nontapetal fundus and a normal to markedly reduced tapetal fundus.

In those merled breeds when the merling genes are homogyzous (MM), multiple ocular anomalies (merle ocular dysgenesis) occur with heterochromia iridis in puppies with excessively white hair coats (Figure 10.2; see also Figure 4.1). These abnormalities include microphthalmia, pupillary size and shape abnormalities, focal iridal hypoplasia and colobomas, cortical cataracts, equatorial staphylomas, retinal dysplasia and retinal detachments. Iris color is usually blue but occasionally yellow or very light brown. The ocular fundus is usually subalbinoid with a lightly pigmented nontapetal fundus, and either atapetal or a yellow–green tapetal fundus.

Persistent Pupillary Membranes

Persistent pupillary membranes (PPMs) are the most frequent congenital defect of the canine anterior uvea (Figure 10.3; see also Figure 8.3). Pupil formation occurs in the last one-third of gestation, but, not infrequently, pups at 12–14 days postnatal have some lingering pupillary "tags." By 4 months or with maturation of the eye, any PPMs remaining are usually considered abnormal.

PPMs usually originate from the collarette region on anterior surface of the iris; this is also the area of the minor arteriolar circle of the iris. PPMs appear as fibrous to pigmented bands of variable width which attach to other areas of the iris, the posterior surface of the cornea, and/or the anterior lens capsule. Those attaching to the cornea and the lens produce posterior corneal opacity and anterior capsular cataract, respectively. PPMs that connect to different areas of the iris cause pupillary shape abnormalities and irregular dilation. Progression of the corneal and lens opacities with PPMs is unlikely. Treatment is not usually necessary. Doppler ultrasound can be used to determine if PPMs have blood flow.

Iridal Nests or Iridal Arrests

Iridal nests or iridal arrests are punctate foci of pigmented tissues that reside on the central anterior lens capsule, the result of congenital deposition of cells during embryologic development and/or the formation of the pupil (variation of PPM) (Figure 10.4). They do not cause cataract formation. They are variable in size and number of pigmented cells, but do not adversely affect vision in the dog. Treatment is generally not required. If the dog's day vision is impaired, drug-induced mydriasis may improve vision.

Iridal Hypoplasia and Colobomas

Colobomas are defects of congenital origin (Figure 10.5). Typical colobomas occur at the 6 o'clock position and

Color Atlas of Veterinary Ophthalmology, Second Edition. Kirk N. Gelatt and Caryn E. Plummer.
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Figure 10.1 (A) Heterochromia iridis in a Siberian Husky. Many breed standards allow one or both eyes to be blue. (B) Heterochromia iridis is also associated with merling in the dog. Combinations of brown, blue, and white are common.

atypical colobomas occur at any other position. Iridal colobomas affect heterochromic eyes more frequently, especially if the globe is microphthalmic and has other anomalies. Iridal hypoplasia is a congenital thinning of the iris tissue. Often, the tissue can be transilluminated revealing a reflection from the fundus.

Inflammations of the Anterior Uvea

Inflammations are the most frequent group of acquired diseases of the canine anterior uvea. These inflammations (anterior uveitis or iridocyclitis) are infectious (virus, bacterial, fungal, parasitic, other), traumatic, neoplastic, toxic, metabolic, or autoimmune in origin. The key clinical question is to separate those iridocyclitides associated with other eye diseases from those secondary to systemic diseases (which often affect both eyes). In addition, simultaneous involvement of the posterior uvea, the choroid, signals panuveitis and a more serious clinical disease involving the retina and optic nerve.

Acute Uveitis

The clinical signs of acute uveitis include photophobia, tearing or lacrimation, blepharospasm, eyelid edema, and a "red" eye (Figure 10.6). The ophthalmic examination findings include aqueous flare, ciliary flush and episcleral injection, conjunctival hyperemia, variable corneal edema, miosis, iris swelling, deep corneal vascularization, hypopyon, hyphema, keratic precipitates, and decreased intraocular pressure. When both eyes are involved and systemic disease is suspected, a complete

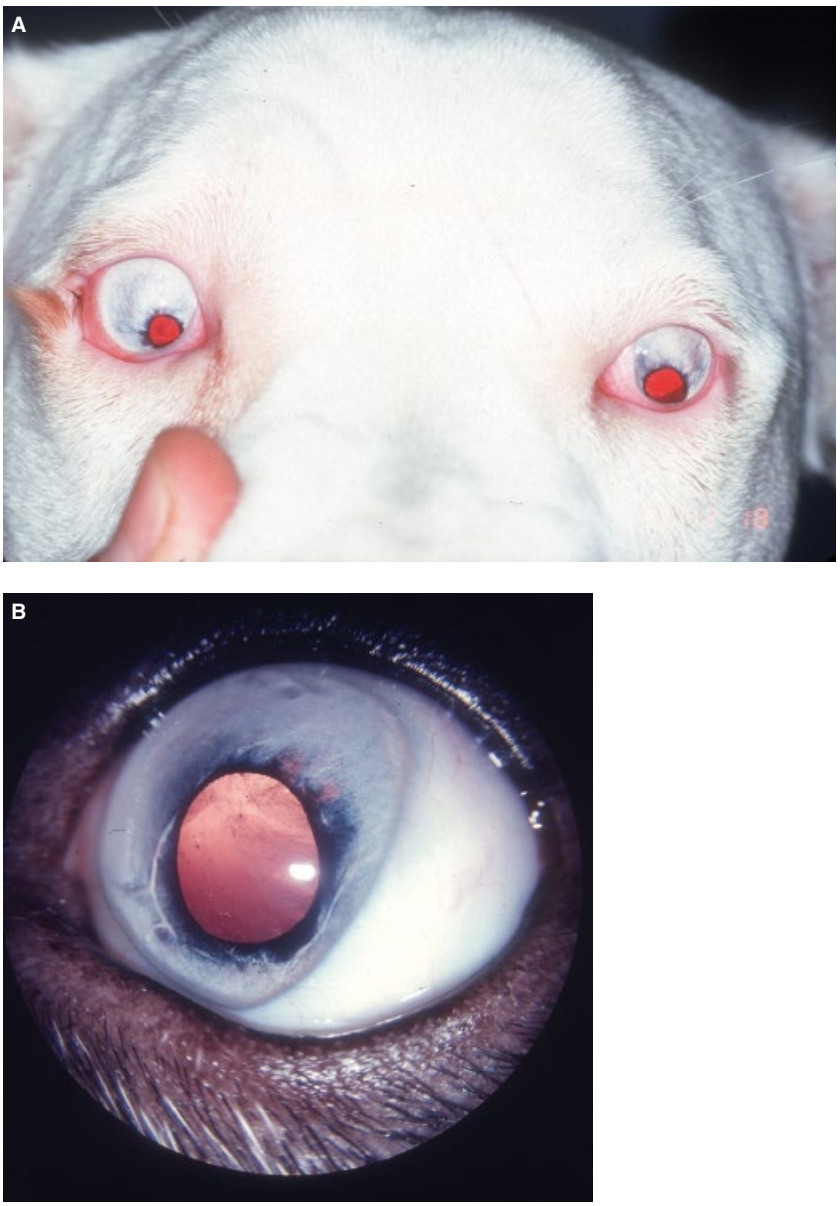


Figure 10.2 (A) Heterochromia iridis is also associated with microphthalmia and other ocular anomalies in the homozygous merle (excessively white) dogs. This syndrome is termed merle ocular dysgenesis. Note the malformed pupil and unequal size globes in this Great Dane puppy. (B) Heterochromia iridis in an excessively white Australian Shepherd puppy with bilateral microphthalmia. The central iris is hypoplastic and contains some persistent pupillary membranes. Early cataract is present as well, noted as dark wisps within the lens viewed via retroillumination.

blood count, serum biochemical profile, urine analysis, and chest and abdominal imaging are usually included in the initial workup. If the patient resides or has traveled to geographic locations with a high incidence of certain infectious diseases, specific testing for evidence of infection or exposure to these infectious agents (serology, polymerase chain reaction, etc.) is indicated as well.

The treatment and prognosis of acute iridocyclitis depends on its cause and response to therapy. Treatment is directed at the suspected cause as well as nonspecific therapy for the anterior uveal inflammation. Pupillary dilation is important to reduce the chance of posterior synechiae and cataract formation, stabilize the blood–aqueous barrier, and decrease the pain associated with iridal and ciliary body musculature spasms. Atropine (1%) is recommended to effect (maximal mydriasis), but should not be instilled excessively as it significantly decreases tear production. Anti-inflammatory agents including topical, systemic, and subconjunctival corticosteroids, as well as topical and systemic nonsteroidal agents, decrease the uveal inflammation, miosis, and pain. Topical and/or systemic antibiotics, and systemic antifungals, can be utilized when infectious disease is suspected.

Chronic Uveitis

Chronic inflammations of the anterior uvea are less frequent than the acute forms, but are noted with increasing frequency in clinical practice, and generally

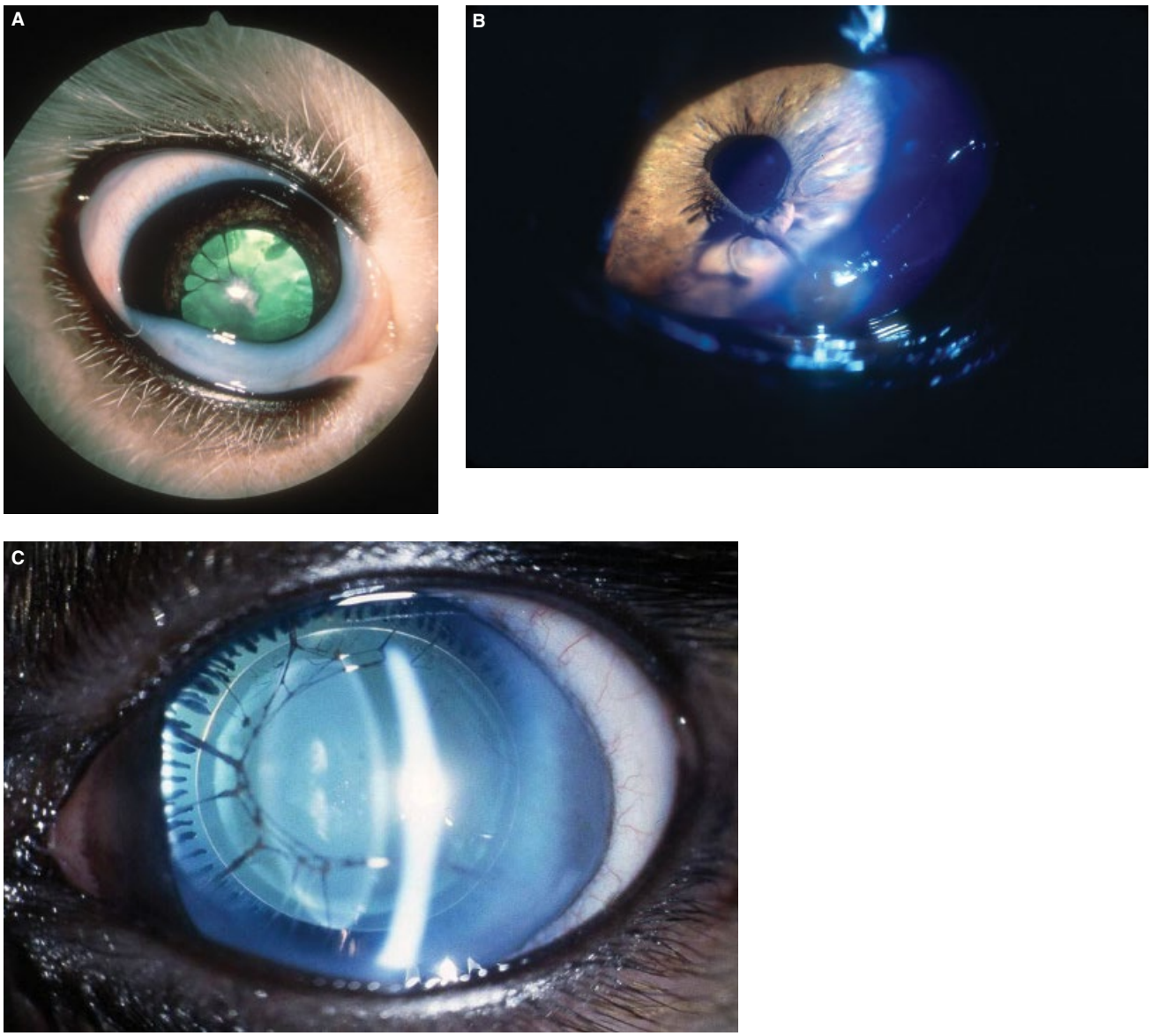


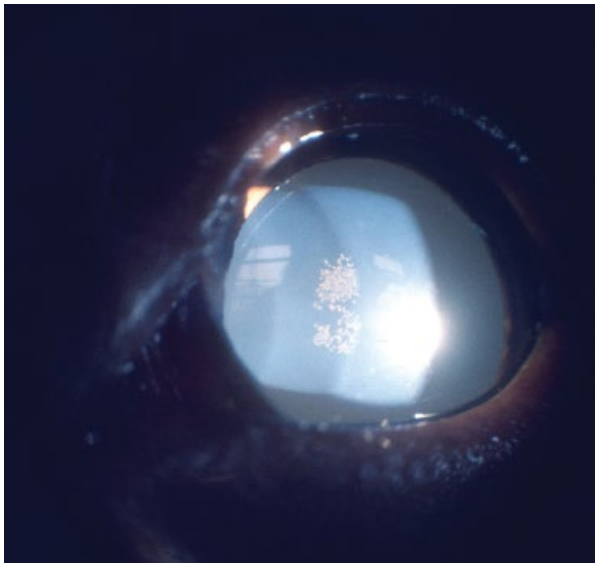
Figure 10.3 (A) Persistent pupillary membrane (PPM) and secondary axial anterior capsular cataract in an Old English Sheepdog puppy with microphthalmia. Note the base of the PPMs is on the anterior surface of the iris (collarette region). (B) Rather large PPMs that are adherent to the posterior cornea. (C) Aniridia, the developmental absence of the iris, is illustrated here. PPMs are all that exist of the iris. The equator of the lens and the ciliary processes are visible. A nuclear cataract is present. Some remnants of the iris are usually present microscopically.

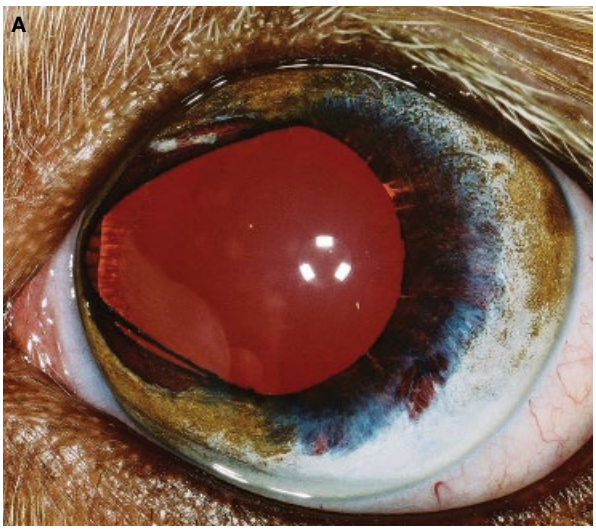
lead to the development of serious complications, including synechiae formation, cataract formation, retinal degeneration and detachments, and glaucoma (iridocorneal closure and peripheral anterior synechiae formation, or pupillary occlusion/annular posterior synechiae/iris bombé), at high frequencies.

The history is usually one of protracted anterior uveitis that responds only partially to intense or long-term therapy. Some of the more common causes of chronic uveitis in the dog include cataract formation (usually cataract hypermaturity), systemic mycotic infections (blastomycosis, cryptococcosis, and coccidioidomycosis), uveodermatologic syndrome (mainly in the Arctic breeds), and other forms of autoimmune uveitis, Golden Retriever uveitis (pigmentary uveitis; see Figure 9.10), and neoplasia (either systemic or local) (Figure 10.7).

The observations from the ophthalmic examination of eyes with chronic uveitis include those seen in acute iridocyclitis, as well as posterior synechia formation, irregular and fixed pupil, pigment deposits from the iris on the anterior lens capsule, cataract formation, either loss or hyperpigmentation of the iris, ectropion uvea

Figure 10.4 Large number of iridal nests or arrests on the central anterior lens capsule. They do not cause cataract formation.





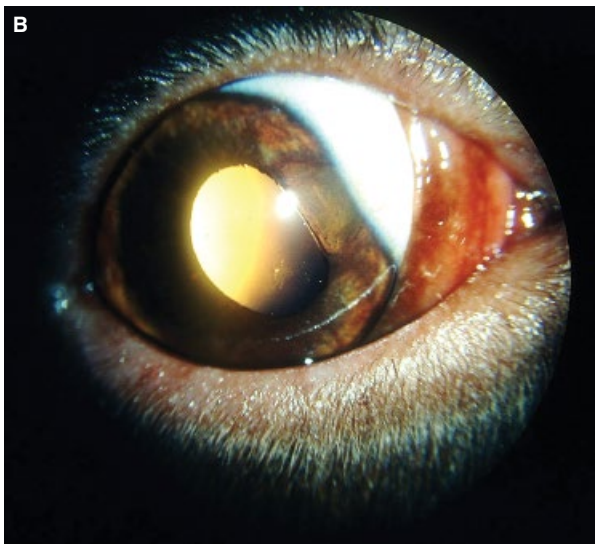
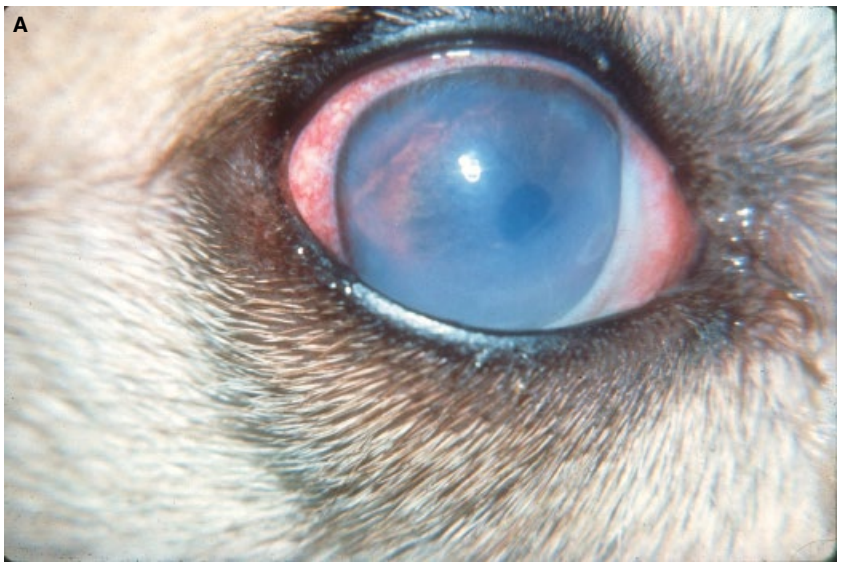
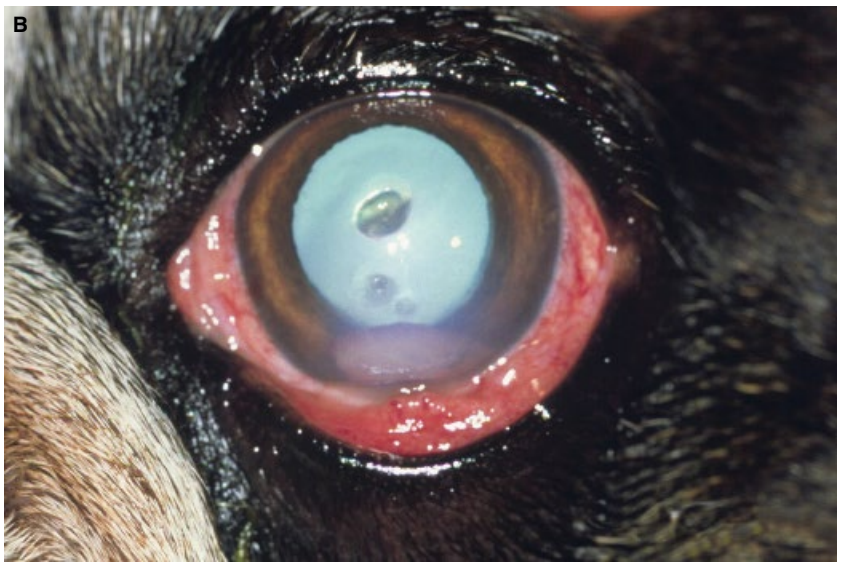




Figure 10.5 (A) Atypical iridal coloboma in a young Australian Shepherd puppy at the 9 o'clock position. The defect is nearly full-thickness and partially exposes the posterior iridal pigmented epithelium. (B) Sector iridal hypoplasia in a puppy. The affected iris is a lighter brown and has distorted the pupil. The adjacent sclera is also malformed. (C) Iris hypoplasia in a young mixed-breed dog. The defects in the iris at 9 o'clock position are not full-thickness. The pigmented posterior epithelium can be visualized through the thin or absent iris stroma.





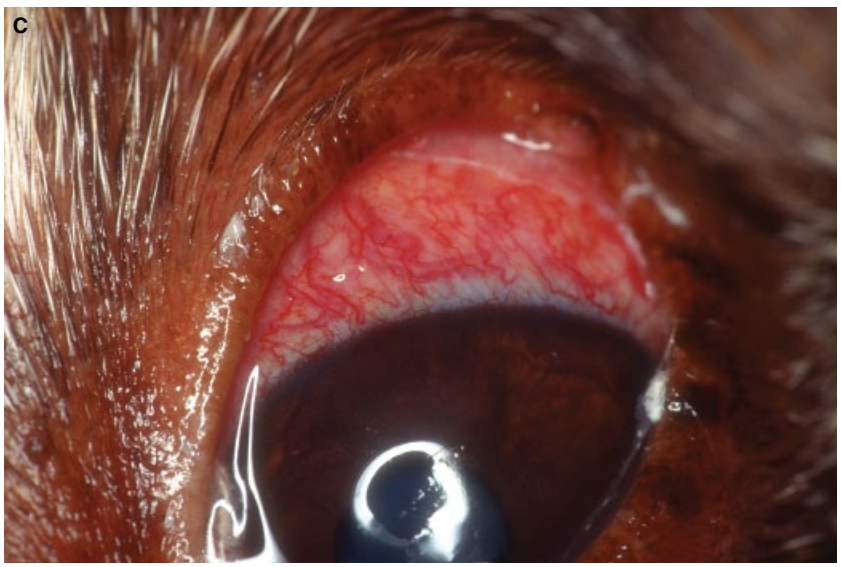
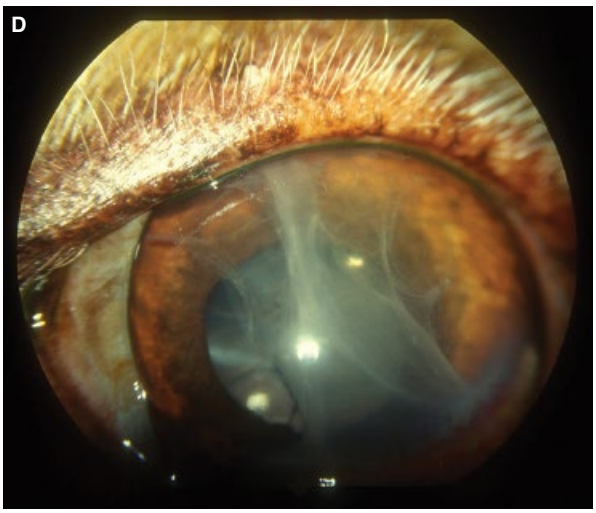


Figure 10.6 (A) Acute iridocyclitis of less than 24 hours' duration. Note the conjunctival hyperemia, miosis, and profound corneal edema. (B) Acute iridocyclitis secondary to a central deep corneal ulcer. Note the swollen iris, hypopyon, and aqueous flare. The pupil has been dilated pharmacologically. (C) Ciliary flush in acute anterior uveitis represents hyperemia of the deep perilimbal or circumcorneal anterior ciliary vessels which are important indicators of intraocular inflammation.



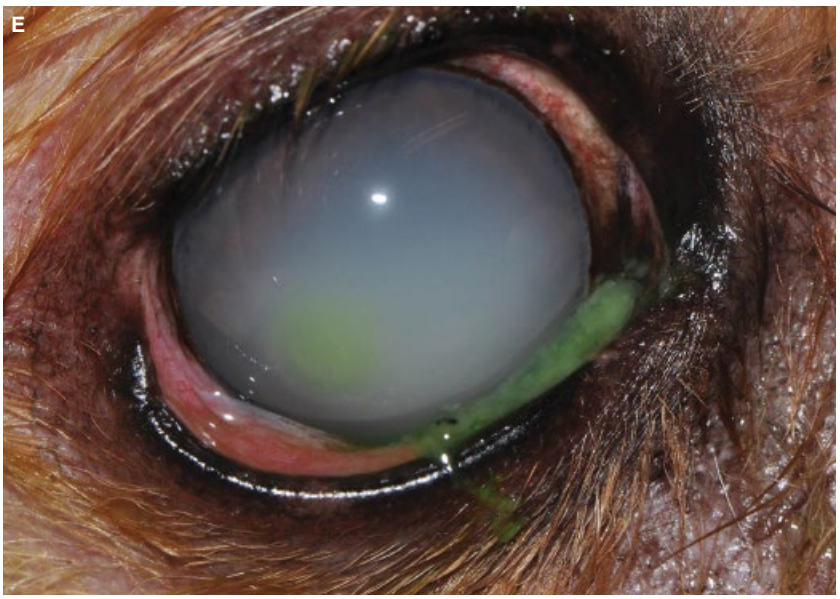


Figure 10.6 (*Continued*) (D) Clotted fibrin in the anterior chamber of a dog following phacoemulsification of a hypermature cataract 5 days previously. (E) Lipemic aqueous in a dog with hyperlipoproteinemia. If blood lipid levels are elevated and the patient develops uveitis, lipid can leak into the eye and result in a milky color to the aqueous humor. This opacity is more dense than aqueous flare. In this case, the dog has a corneal ulcer (note fluorescein retention) and reflex anterior uveitis. The finding of lipemic aqueous should initiate a systemic workup.

(distortion or eversion of the pupillary iris revealing pigment from its posterior surface), decreased intraocular pressure (until secondary glaucoma develops), secondary glaucoma, and phthisis bulbus (atrophy of the globe). Inflammatory membranes can cross the pupil and adhere to the anterior lens capsule and extend onto the iridal surface (as preiridal fibrovascular membranes). Pupillary seclusion, usually from annular (360°) posterior synechiae formation, and formation of iris bombé lead to secondary glaucoma.

The goals of therapy are to reduce the anterior uveal inflammation, dilate and move the pupil to avoid posterior synechiae formation, and eliminate the inciting cause. Medical therapy is usually similar to that for acute uveitis, but can include the addition of immunodulators or immunosuppressives, such as cyclosporine or azathioprine, for certain cases. Other therapy includes target therapy for the underlying etiology if it has been determined.

Specific Types of Anterior Uveitis

Paired with often characteristic systemic clinical signs, anterior uveitis is part of many systemic diseases, and these ocular signs are very helpful in establishing the clinical diagnosis. Laboratory tests can help to confirm the specific disease.

Anterior Uveitis and Rocky Mountain Spotted Fever

Rocky Mountain spotted fever (RMSF) is caused by *Rickettsia rickettsii* and carried by ticks, *Dermacentor andersoni*, *D. variabilis*, and *Amblyomma americanum*.

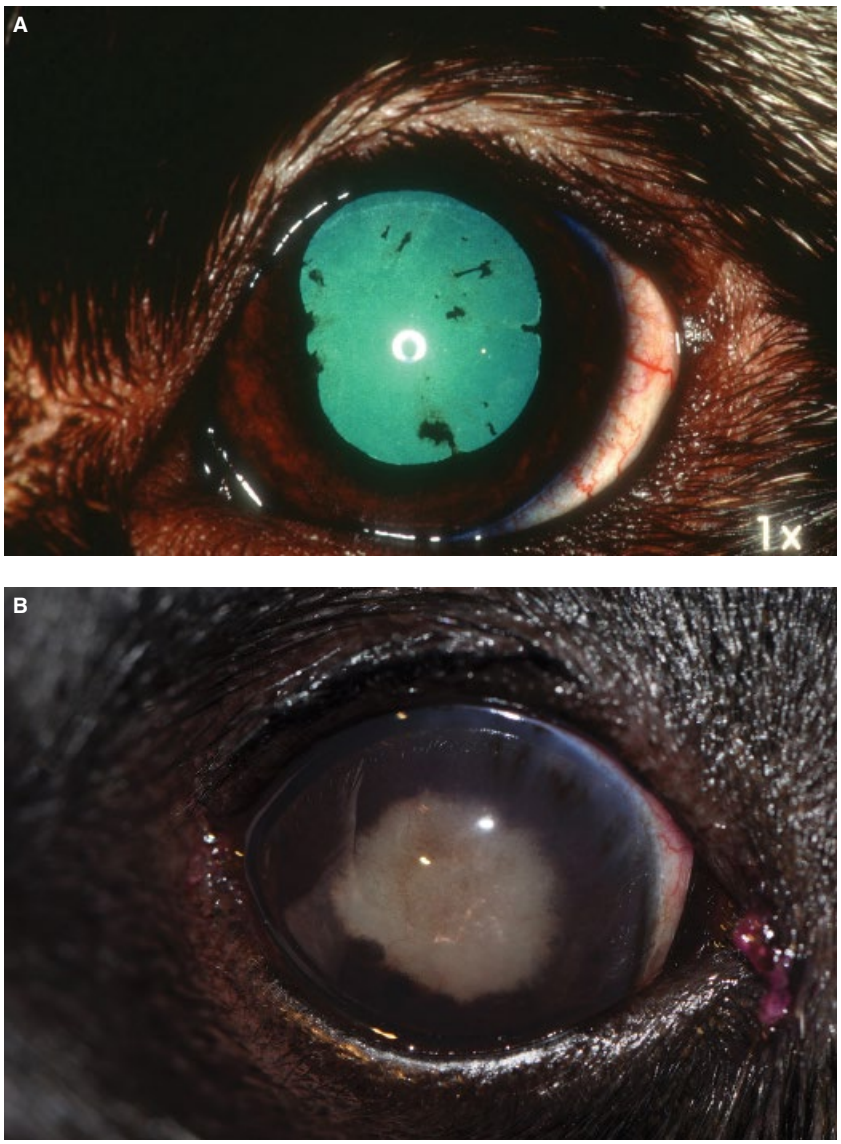


Figure 10.7 (A) Uveodermatologic syndrome in an Atika during a remission phase. Aggressive systemic and topical corticosteroids and azathioprine have markedly reduced the uveitis. Note the pigment foci on the anterior lens capsule and mulitfocal posterior synechiae. (B) Chronic anterior uveitis has distorted and fixed the pupil with nearly complete posterior synechiae. A hypermature cataract has resulted from the chronic intraocular inflammation.

It produces a vasculitis and platelet deficiency that is shown clinically as diffuse hemorrhages that affect many organs. Hemorrhages and inflammation can affect the conjunctiva, anterior and posterior uvea, retina, and optic nerve head (Figure 10.8; see also Figure 3.15E). The uveitis usually develops 2–3 weeks postinfection. Serology tests, especially when paired, are useful in making the diagnosis. There are several other tick-borne infections that can cause uveitis, including ehrlichiosis, anaplasmosis, and borreliosis.

Anterior Uveitis and Heartworm (*Dirofilaria immitis*)

The immature (fourth stage) larvae of *Dirofilaria immitis* is the most frequent intraocular parasite in the dog in North America (Figure 10.9; see also Figure 18.18). However, since long-term prophylactic therapy for heartworm is common, this parasite is now an infrequent cause for anterior uveitis. The history is usually an outside dog, not on heartworm preventative, and an unilateral "red" eye.

Ophthalmic examination reveals signs of anterior uveitis, pronounced and often focal corneal edema, aqueous flare to frank fibrin, miosis and iridal swelling, and a translucent mobile parasite within the anterior chamber. During the examination and upon exposure to light, the parasite may disappear through the pupil into the posterior chamber or even the vitreous.

Treatment is to surgically extract the intact viable parasite through a small limbal incision into the anterior chamber, and treat the anterior uveitis medically with mydriatics and anti-inflammatories (corticosteroids and nonsteroidal anti-inflammatories). Some residual corneal edema and/or scarring may persist.

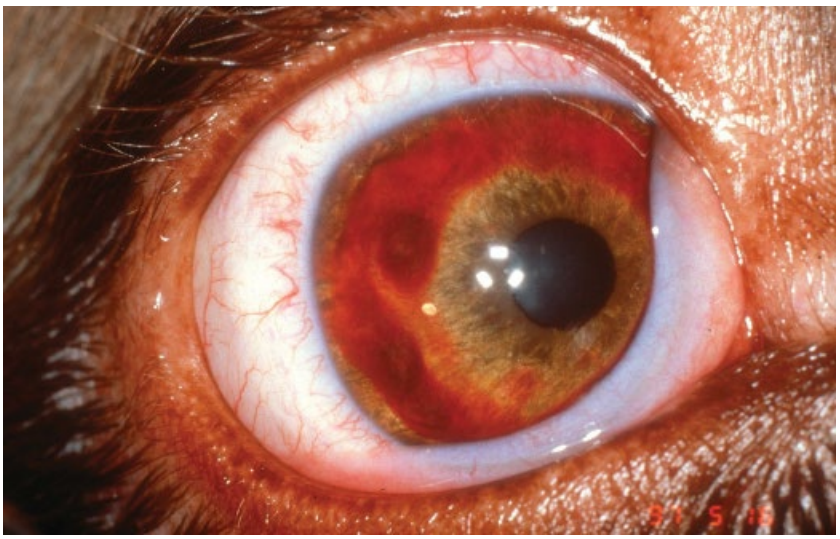


Figure 10.8 Anterior uveitis with hemorrhage of the iridal surface in a dog infected with *Rickettsia rickettsii.*



Figure 10.9 Heartworm (*Dirofilaria immitis*) in the anterior chamber of a dog. The parasite appears as a mobile translucent worm. Note the marked iridocyclitis including hyperemia of the conjunctiva, ciliary flush, nictitans protrusion, and corneal edema and vascularization.

Anterior Uveitis Secondary to Infectious Canine Hepatitis

Infectious canine hepatitis (ICH) was a common cause of anterior uveitis in the 1960s and 1970s and corneal edema was a prominent feature of the disease (Figure 10.10). The disease occurred primarily in puppies. Although the naturally occurring disease produces the ophthalmic disease in about 20% of affected animals, the most frequent cause of the ICH anterior uveitis during these decades was the vaccine (canine adenovirus-1; CAV-1). With the development of new ICH vaccines in the 1980s, which utilize canine adenovirus-2 (CAV-2), the incidence of eye disease has decreased significantly.

The anterior uveitis develops about 2–4 weeks after the natural disease (during the recovery phase), and about 1–3 weeks following CAV-1 vaccination. The anterior uveal inflammation and corneal edema result from an immune-complex Arthus reaction. The virus persists within the anterior uvea and corneal endothelium after the initial viremia. A localized immune process occurs 1–3 weeks later in the anterior segment, and is particularly damaging for the corneal endothelium.

The most striking aspect of this form of anterior uveitis is the severe corneal edema, which may not resolve. The condition can affect both (10–30%) eyes. In a few affected animals, persistent corneal edema, secondary glaucoma, and phthisis bulbus (atrophy of the globe) result. Treatment is aimed at the anterior uveitis.



Figure 10.10 Anterior uveitis secondary to infectious canine hepatitis has profound corneal edema because of the persistence of virus particles within the cornea endothelium.

Anterior Uveitis and the Mycoses

The mycoses are a frequent cause of anterior and posterior uveitis in the dog, and are somewhat geographic in their distribution (Figure 10.11). In the southwest regions of America, coccidioidomycosis (*Coccidioides immitis*) predominates; in the river valleys (Mississippi/Tennessee/Ohio), blastomycosis (*Blastomyces dermatitis*) occurs. Cryptococcosis (*Cryptococcus neoformans*) is ubiquitous.

Affected dogs may not demonstrate systemic clinical signs. With coccidioidomycosis and blastomycosis, the organisms enter the body through the respiratory tract and reach the uveal tract via systemic circulation. Cryptococcosis generally affects the central nervous system (CNS), and the organisms enter the optic nerve head and retina via extension from the CNS. Aspergillosis affects the German Shepherd Dog breed primarily.

Blastomycosis presents with either loss of vision or with a "red" eye. Often both eyes are involved and approximately 50% of the dogs with systemic blastomycosis will have ocular disease. Both anterior (30–50% of cases) and posterior uveitis (22–43%) are likely; panuveitis occurs in about 26–72% of the patients and has the poorest prognosis. An intense anterior uveitis with marked aqueous flare and often hypopyon is typical. Posterior synechiae and cataract formation are likely, particularly if medical therapy is delayed. Vitreal inflammatory debris is common and can be aspirated to aid diagnosis. Posterior uveitis and chorioretinitis can result in focal granulomas, retinal hemorrhages, and exudative retinal detachments.

Therapy for blastomycosis is both expensive and extended. Posterior segment inflammation and retinal detachment have a poor prognosis for response to therapy and restoration of vision. In addition to symptomatic therapy for the ocular inflammation, specific antifungal therapy with anti-infective agents is necessary.

Lens-induced Uveitis

Lens-induced uveitis (LIU) is common in dogs and is present in all cataractous eyes (as demonstrated by applanation tonometry), especially those in which the cataracts progress to hypermaturity (Figure 10.12). Probably during cataract formation, the lens capsule increases its permeability allowing low molecular weight lens proteins access to the anterior segment. Some clinical studies, using either applanation tonometry or fluorophotometry, suggest that nearly all of the stages of cataract formation (incipient, immature, mature, and hypermature) demonstrate lower intraocular pressure and a leaky blood–aqueous barrier (suggesting diffusion of lens material through an intact anterior lens capsule and the resultant lens-induced uveitis).

LIU occurs in two clinical forms. Phacolytic uveitis is associated with cataract hypermaturity; histologically, lymphocytes and plasma cells are the main inflammatory cells present. The second type of LIU is less frequent; phacoclastic uveitis is associated with anterior capsular tears (usually in young dogs with the capsular tears caused by penetrating cat claws).

Presenting signs are those of acute to chronic anterior uveitis in the presence of cataract. The breeds of dogs most often affected are those with the inherited or primary cataracts. Ophthalmic findings include conjunctival hyperemia, ciliary flush, miosis, variable aqueous flare to frank fibrin, slow and/or incomplete mydriasis after topical 1% tropicamide, and low intraocular pressure (<10 mmHg).

LIU requires constant monitoring and intermittent to continuous topical (and sometimes systemic) medications. Topical medications include the anti-inflammatories (corticosteroids and/or the nonsteroidals), and mydriatics (usually 1% tropicamide or 1% atropine). LIU must be under medical control before cataract surgery in dogs, and can contribute to lower success rates if it is not. In those patients unable to have cataract surgery, LIU necessitates lifelong medical therapy and regular re-examination and tonometry. Chronic LIU is associated with a high incidence of secondary glaucoma in the dog (see Figure 9.9).

Pigmentary Uveitis, Anterior Uveal Cysts, and Secondary Glaucoma

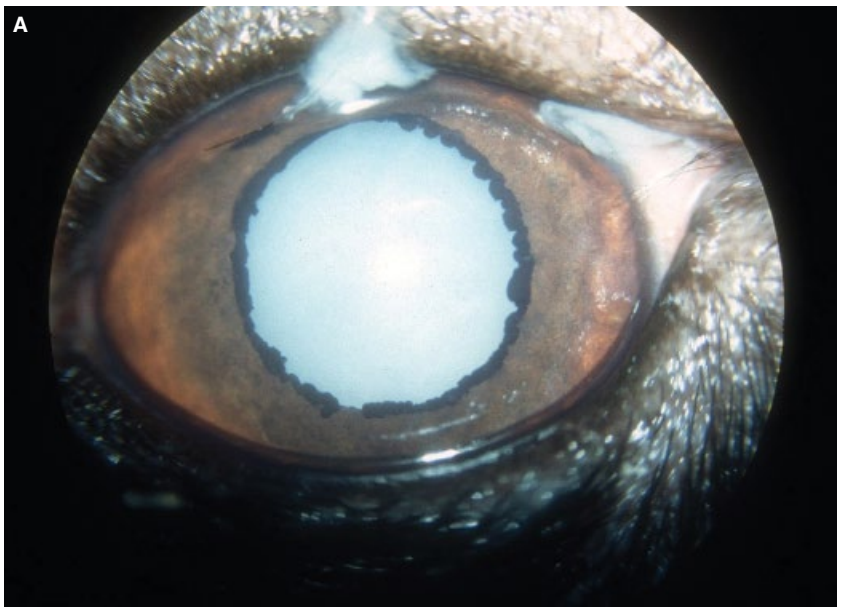
A recently reported syndrome of pigmentary uveitis, anterior uveal cysts, and secondary glaucoma occurs in



Figure 10.11 (A) Mycotic iridocyclitis and posterior chorioretinitis in a dog caused by *Blastomyces dermatitis*. Note the swollen iris, irregular pupil, and exudative retinal detachment (arrow). (B) Mycotic iridocyclitis and posterior chorioretinitis in a young German Shepherd Dog. The yellow–white fundus reflex (leukocoria) is caused by the infiltration of inflammatory cells (vitritis) and *Aspergillus* sp. organisms. (C) Coccidioidomycosis is not uncommon in California and southwest America. Note the nictitans protrusion and anterior uveitis in this dog from Arizona. Source: (C) Photo courtesy of Dr. Ron Sigler.

Golden Retrievers, and is not associated with systemic disease (Figure 10.13; see also Figure 9.10). Most affected dogs are middle aged or older (average 8 years). The disease is characterized by chronic anterior uveitis with the development of pigmented deposits on the anterior lens capsule, often in a radial fashion. These melanin deposits are from either the posterior iris surface or, less likely, the ciliary body. Anterior uveal cysts develop and seem to contribute to the development of glaucoma. Cataract formation with posterior synechiae formation and secondary glaucomas occur in more than half of affected dogs.

The clinical history is one of persistent and long-term eye irritation and anterior uveal inflammation. Pigmented deposits are scattered on the anterior lens capsule resulting in a classic appearance. Aqueous flare and formation of inflammatory membranes develop, proceeding the onset of glaucoma. The formation of posterior synechiae is common as the disease progresses, and causes cataract formation. Glaucoma develops secondary to deposition of pigment within the trabecular meshwork, peripheral anterior synechiae formation, and forward displacement of the basal iris (exacerbated by uveal cysts).



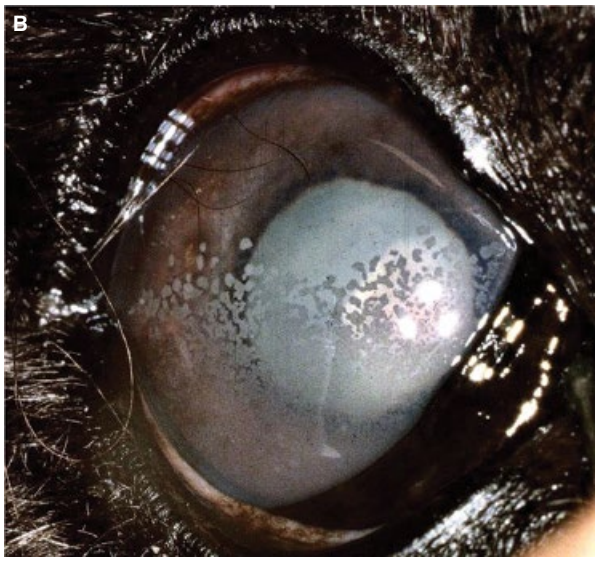


Figure 10.12 (A) Lens-induced iridocyclitis and cataract in an American Cocker Spaniel. The chronic uveal inflammation has resulted in ectropion uvea or eversion of the iridal pigment epithelium into the pupil. (B) Diabetic cataracts in this Miniature Schnauzer have resulted in severe lens-induced uveitis. Note the keratic precipitates present on the corneal endothelium.

This condition is chronic and eventually becomes refractory to topical and systemic anti-inflammatory and mydriatic medications. As a result, the prognosis for long-term vision is poor.

Anterior Uveitis and Uveodermatologic Syndrome

Uveodermatologic syndrome, or Vogt–Koyanagi–Harada syndrome, occurs primarily in the Arctic breeds, but has been reported sporadically in non-Arctic individuals. Breeds affected most frequently include the Akita, Siberian Husky, Samoyed, and the Shetland Sheepdog (Figure 10.14; see also Figures 5.19, 10.7, 18.26, and 18.27). Affected dogs are usually young (average 3 years of age), and the condition eventually affects both eyes. The skin lesions can precede or follow the ophthalmic signs. Histopathology suggests an immune-mediated destruction of the melanocytes within the eye and the dog's skin.

The skin lesions are erosive and inflammatory, and loss of pigmentation of the nasal planum, lips, scrotum, footpads, and eyelids (especially the margins) is typical. The ophthalmic findings include marked blepharitis with loss of pigmentation, persistent anterior uveitis with loss of iridal pigmentation, posterior synechiae and secondary cataract formation, pigment foci on the anterior lens capsule, posterior choroiditis, loss of pigmentation of the nontapetal fundus, retinal detachment, and secondary glaucoma (either from peripheral anterior synechiae and iridocorneal closure, or annular (360°) posterior synechiae and iris bombé).

The prognosis of this condition is guarded overall and is poor for vision in the long term. Treatment of

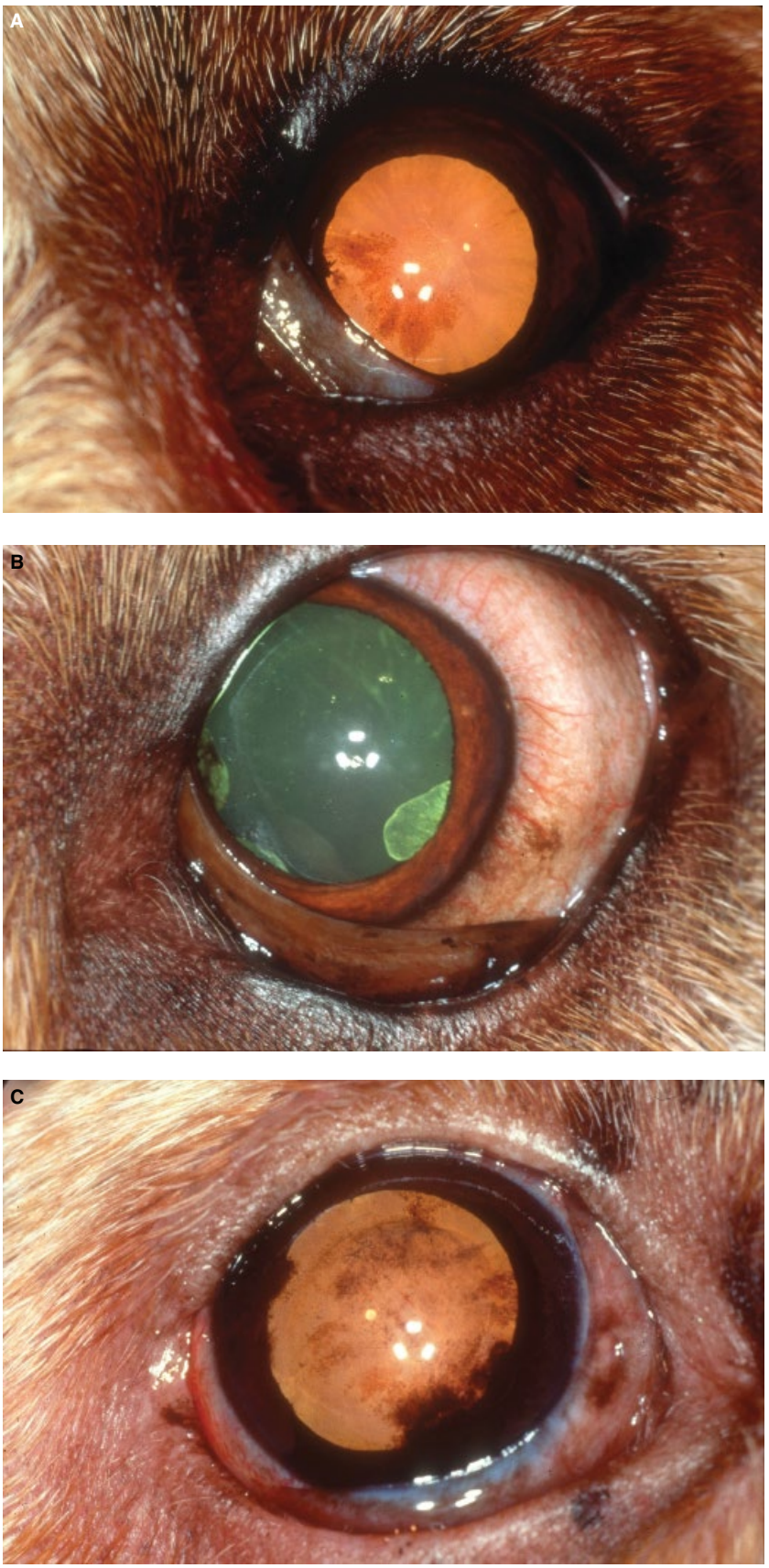
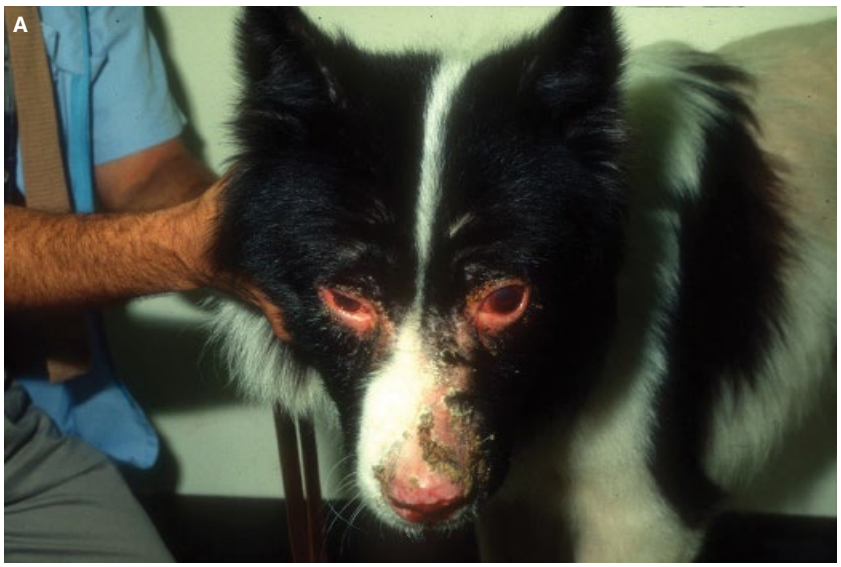


Figure 10.13 (A) Early pigmentary anterior uveitis in a Golden Retriever is characterized by the appearance of pigmented spots on the anterior lens capsule. (B) Pigmentary uveitis in a Golden Retriever with multiple anterior uvea cysts (probably of ciliary body origin) and a single posterior synechia (8 o'clock position). (C) As pigmentary uveitis in the Golden Retriever progresses, posterior synechiae begin to form resulting in cataract formation. Courtesy of John S. Sapienza.





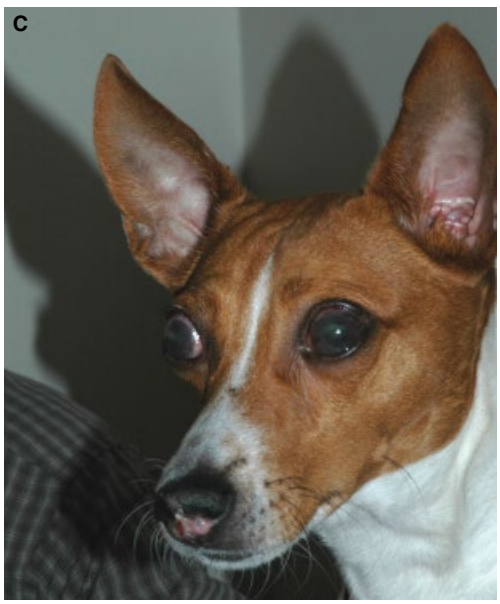


Figure 10.14 (A) Uveodermatologic syndrome (UDS) affects the pigmented tissues in the body and eye. In this Akita dog, the inflamed areas that have partially lost their pigmentation include the lips, nose, eyelids, and irides. Note the buphthalmia of the left eye, the result of secondary glaucoma. (B) A closer view of the eye of another affected Akita shows the loss of pigmentation and diffuse blepharitis affecting both eyelids. (C) Dog with UDS with bilateral secondary glaucoma.

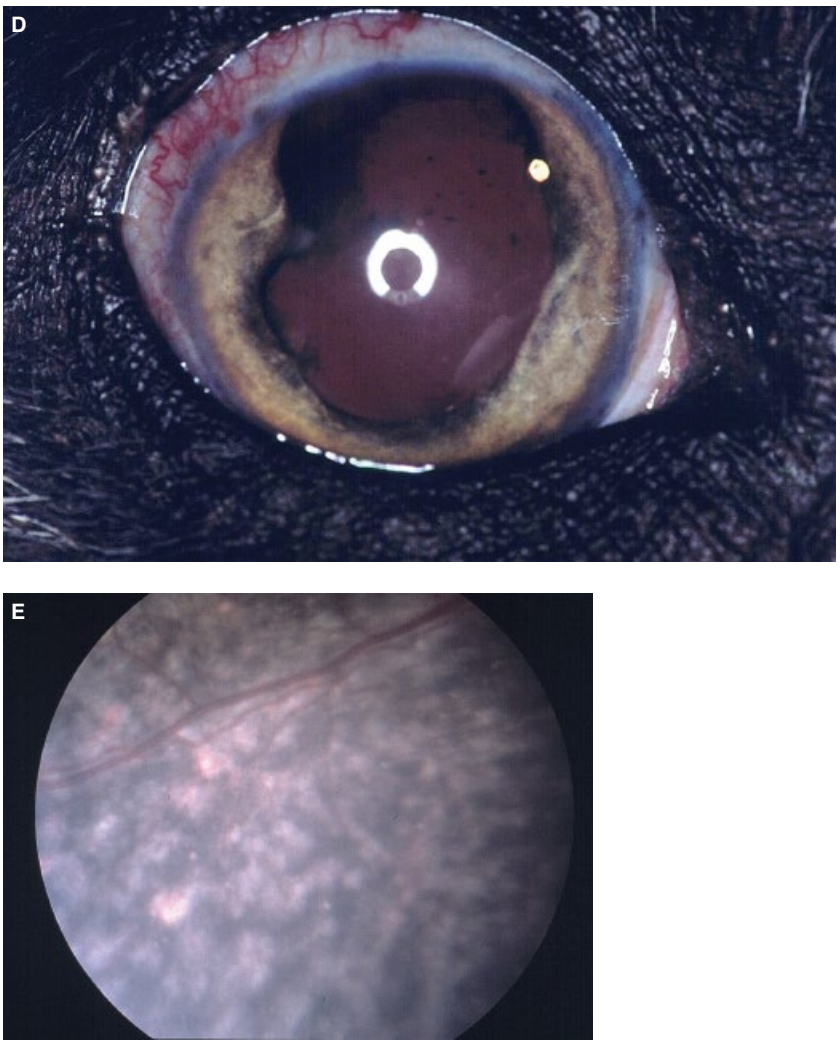


Figure 10.14 (*Continued*) (D) In another dog with UDS the chronic iridocyclitis has resulted in a lighter brown iris (due to destruction of the pigment cells), posterior synechiae, and secondary glaucoma. Note the episcleral congestion and slightly enlarged globe. (E) Loss of pigmentation of the nontapetal fundus also occurs with UDS. Note the exposure of the deeper choroidal vasculature, loss of pigmentation, and pigment clumping.

uveodermatologic syndrome is difficult but attempts to halt the immune-mediated destruction of melanin in the eye and skin. Topical and systemic corticosteroids along with mydriatics are used for the panuveitis with variable success. Systemic immunomodulators or immunosuppressive medications may be helpful as adjunctive therapy.

Iridal Degenerationsand Iridal Atrophy

Senile iris atrophy is not uncommon in older dogs, and appears more frequently in the Toy and Miniature Poodles, Miniature Schnauzer, and Chihuahua breeds (Figure 10.15). Pupillary abnormalities, color changes, and full-thickness holes affect the iris. The loss of the iridal tissues cause the brown iris color to become lighter; if the atrophy extends to the posterior iridal pigment layer, the iris will appear dark brown to black. Senile iris atrophy does not usually cause secondary problems (i.e., secondary glaucoma).

In Toy and Miniature Poodles, senile iris atrophy often affects the pupillary border and the iridal sphincter musculature. The result is a slightly dilated pupil with a scalloped border. If the ability to constrict the pupil is impaired or eventually lost, the dog becomes sensitive to and avoids bright illumination and sunlight.

Senile iris atrophy can also involve the iridal substance and results in full-thickness holes that permit direct observation of the periphery of the lens. Impaired pupillary light reflex, increased sensitivity to light, and variable mydriasis are frequent findings. There is no treatment for senile iris atrophy.

Iridal or Uveal Cysts

Uveal cysts arise from the posterior surface of the iris (usually black cysts) or from the ciliary body (usually light brown or translucent cysts). These cysts occur

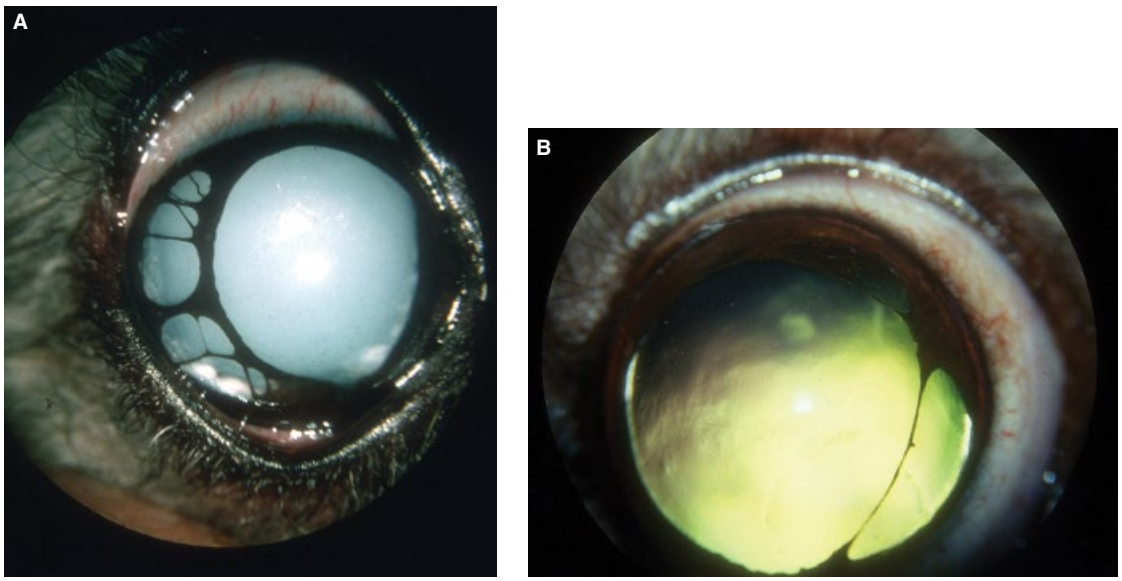


Figure 10.15 (A) Advanced senile iris atrophy in a Miniature Poodle. Note the majority of the lateral iris has atrophied, leaving intact primarily the iridal sphincter area. A dense mature cataract is also present. (B) Senile iris atrophy affecting primarily the iris sphincter. This dog was highly sensitive to bright light.

more often in older dogs, and inflammation and trauma are suggested causes. Uveal cysts, or the predisposition to their development, are believed to be inherited in some instances. Iris cysts appear as fixed (attached to the posterior surface or stuck in the anterior chamber angle), or floating and mobile smooth variably pigmented masses. They are usually translucent with intense and focused illumination, and can be distinguished from anterior uveal melanomas. Iris cysts do not require treatment, but if numerous they can be aspirated from the anterior chamber, or deflated by noninvasive laser photocoagulation.

The Golden Retriever is prone to uveal cysts, which have more significance in this breed and can signal serious eye disease (see Figure 9.10 and 10.13). Ciliary body cysts in the Golden Retriever and Great Dane have been associated with persistent anterior uveitis, and secondary cataract formation and glaucoma. Histologically, ciliary cysts are more often attached to the ciliary processes, and often filled with periodic acid–Schiff positive material. The significance of this material within the ciliary body cysts is unknown.

Anterior Uveal Trauma

Foreign bodies that penetrate the cornea to enter the anterior chamber, the iris, and even the lens constitute serious insults to the eye, and are considered true ophthalmic emergencies. The composition of the foreign body is important; generally, copper, iron, and organic foreign bodies require removal. Lead shot may not require extraction from the eye, as it seems not to incite chronic inflammation (Figure 10.16; see also Figure 8.15). If the anterior lens capsule is penetrated by the foreign body, removal of the lens capsule contents by phacoemulsification is recommended. Delay increases the likelihood of the development of medically refractory lens-induced uveitis, secondary glaucoma, phthisis bulbus, and blindness.

The history is usually one of trauma, and the development of acute severe eye pain and irritation, eyelid swelling, nictitans protrusion and inflammation, conjunctival hyperemia and swelling. One or more foreign bodies can reside within the cornea, anterior chamber, or deeper within the eye. The ocular media is fairly clear initially and can permit direct inspection of the foreign body and damaged ocular tissues. However, within hours to a few days the secondary inflammation and corneal edema can become severe and interfere with examination of the eye. Both ultrasonography (nonmetallic foreign bodies) and radiography (metallic foreign bodies) provide additional information. Once a foreign body has entered the anterior segment, surgical extraction is usually necessary.

Hyphema

Hyphema or hemorrhage within the anterior chamber is not infrequent in dogs, and results from congenital anomalies, uveitis, trauma, neoplasia, systemic hypertension, chronic glaucoma and buphthalmia, retinal detachment,

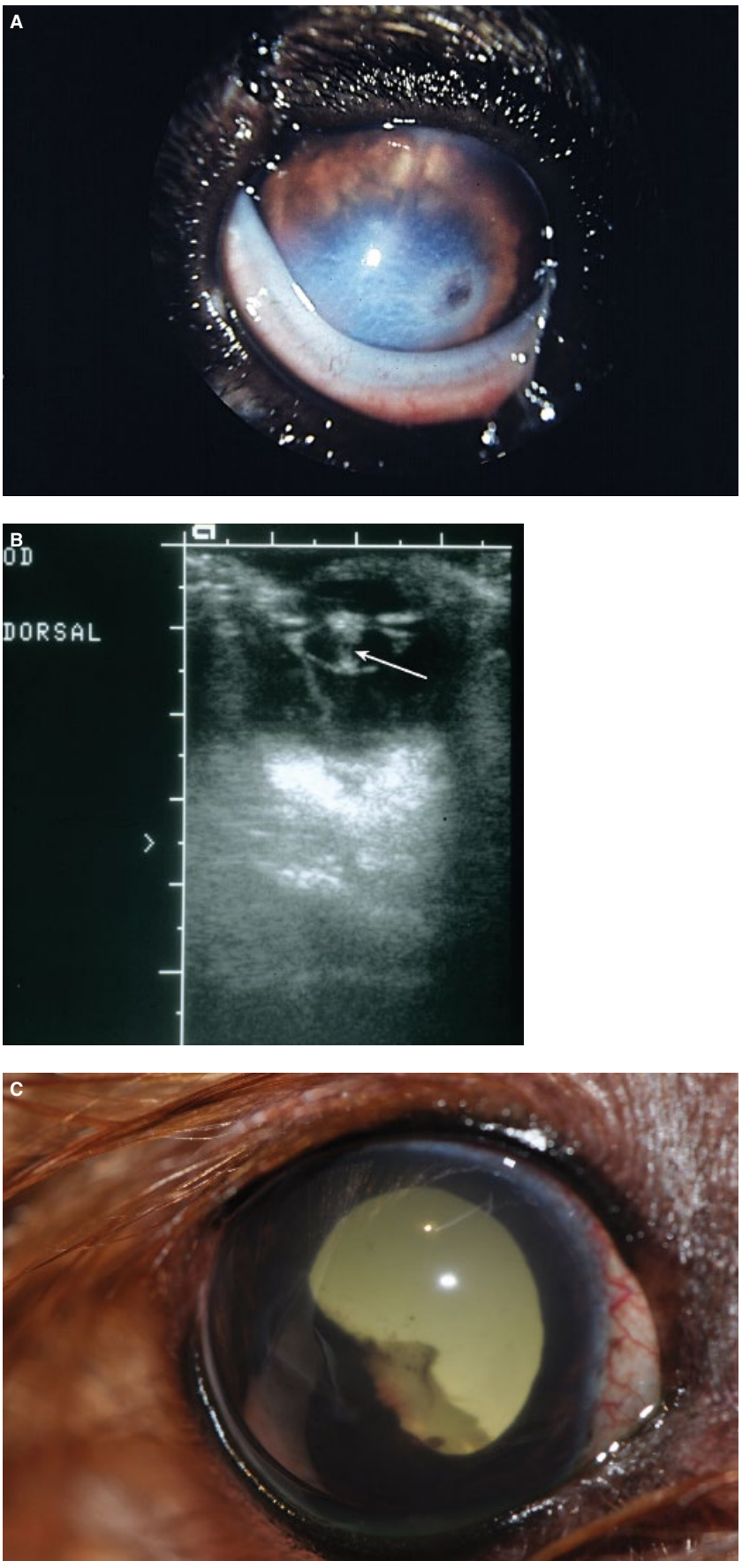


Figure 10.16 (A) Penetrating intraocular lead shot that has traversed the cornea, lens, and posterior segment. The entry of the shot is near the center of the cornea. (B) Ultrasonographic appearance of pellet's pathway (arrow) through the lens and intravitreal hemorrhage. (C) Anterior and posterior uveitis in a dog following an encounter with a cat which resulted in a corneal laceration (healed) and an anterior lens capsule rupture. Note the posterior synechia and the exudate adjacent to the lens. The fundic reflex is obscured by inflammatory exudate as well.

and vascular disorders (coagulation and platelet defects, and hyperviscosity syndromes) (Figure 10.17; see also Figure 3.15). Hemorrhage in the anterior chamber usually arises from iris and ciliary body blood vessels. However, hyphema can result from retinal and choroidal vessels which produce intravitreal hemorrhage that enters the anterior chamber.

The clinical history is variable, dependent on the cause of the hyphema. Congenital ocular anomalies, such as Collie eye anomaly or vitreoretinal dysplasia, can result in complete retinal detachments and present as hyphema that does not clot in young puppies. Trauma from a blunt or penetrating object can also present with hyphema that clots. Persistent and/or recurrent hyphema (which does not clot) in older dogs can signal intraocular neoplasia, systemic hypertension, retinal detachment, and giant retinal tears.

The appearance of the hyphema provides clues as to the source and cause of the intraocular bleed. Hyphema, consisting of nonclotted blood that fills the anterior chamber, signals persistent or recurring intraocular hemorrhage secondary to coagulation and platelet disorders, and retinal detachments. Hyphema that consists of several layers of different colors of clotted blood (top is red and bottom layer is purple) suggests recurrent bouts of hemorrhage and failure of clotting. Focal areas of clotted hemorrhage within the anterior chamber often result from trauma and iridocyclitis, and signal resolution of





Figure 10.17 (A) Hyphema in a dog following trauma. The intraocular hemorrhage has stopped and clotted. (B) Hyphema and subconjunctival hemorrhage in a dog secondary to thrombocytopenia.

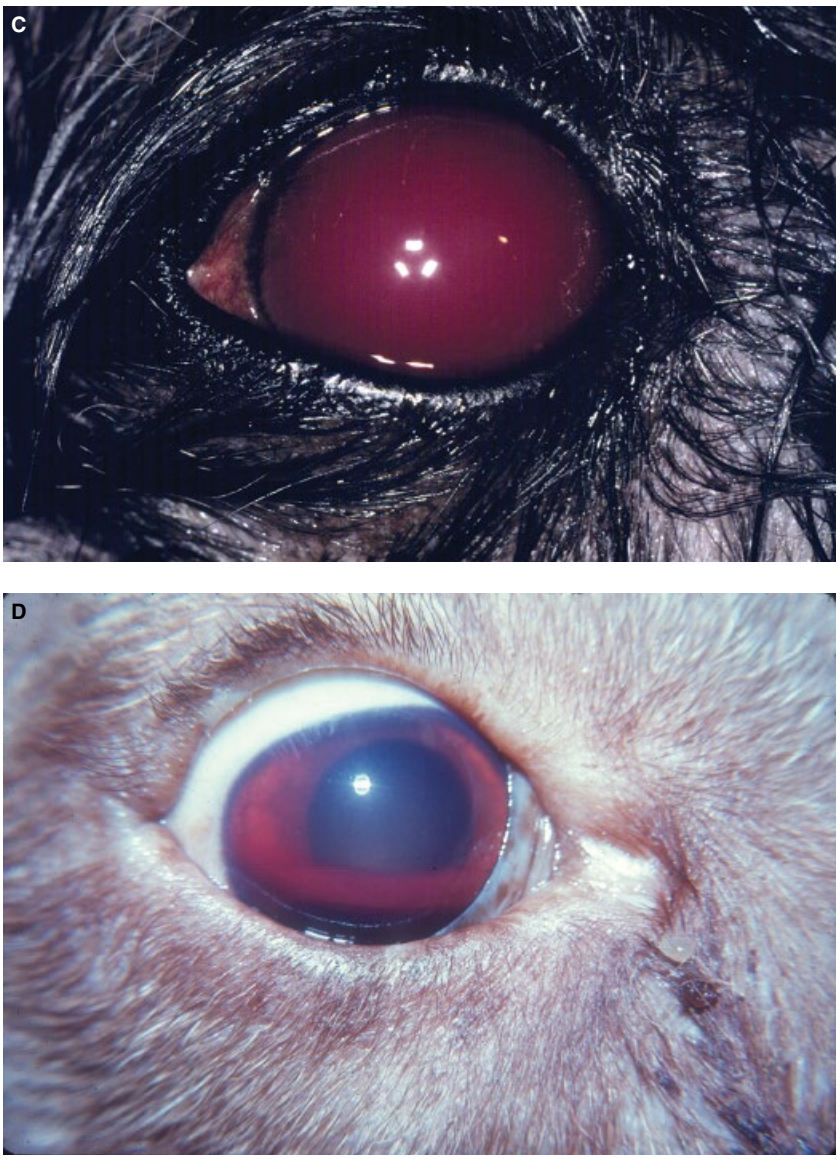


Figure 10.17 (*Continued*) (C) Hyphema secondary to retinal detachment in a dog. Presumably retinal vessels bled into the vitreous and eventually the hemorrhage entered the anterior chamber. Retinal detachment has detected by ultrasonography. (D) Recurrent hyphema in a Chesapeake Bay Retriever with metastatic choroidal hemangiosarcoma. Note the different layers (and colors) of blood settled in the ventral anterior chamber. Hence, the need to examine hyphema with ophthalmic ultrasonography.

the original source of hemorrhage. When the intraocular tissues cannot be adequately examined by direct observation because of the amount of blood in the anterior chamber, ultrasonography is recommended for examination of the deeper ocular tissues.

Treatment of hyphema is directed toward the concurrent uveitis present. Direct removal of hyphema is infrequently attempted in dogs, unless the hemorrhage is causing increased intraocular pressure. Anterior chamber blood is believed to exit through the aqueous outflow pathways (intact erythrocytes rather than by hemolysis). Intracameral injection of 25–50 µg tissue plasminogen activator (Activase-Genentech, San Francisco, CA) can dissolve blood clots of less than 10–14 days' duration.

Anterior Uveal Tumors

Anterior segment (iris and ciliary body) neoplasia is not uncommon in dogs, but choroidal or posterior segment neoplasms are rare (in contrast to humans). Most of these neoplasms are pigmented, and classified as either melanocytomas or malignant melanomas. The latter are decidedly more invasive, and have the tendency to metastasize. Nonpigmented neoplasms are less frequent and of variable malignancy. While some affected dogs are presented because the owner noticed the mass, most patients are presented for intraocular hemorrhage, secondary glaucoma, and blindness (see Figures 9.14, 9.15, 9.16, 9.17, and 9.18).

Melanocytic Neoplasms

Melanocytomas, sometimes referred to as simply melanomas, are the most frequent pigmented intraocular neoplasm in the dog and tend to occur most frequently in older dogs (10 years and older). The highest incidence of anterior uveal melanomas occurs in the German Shepherd, Boxer, and Labrador Retriever breeds (Figure 10.18). These anterior uveal neoplasms can be very destructive to the eye causing hyphema, iridocyclitis, lens subluxation, glaucoma, and even retinal detachments, but they rarely metastasize (less than 5%). Malignant forms represent a smaller group (5–10%) of these tumors. The histologic appearance of uveal melanomas is not predictive of biologic behavior.

Usually, the patient presents because the owner has noted a color change in the eye, or that it appeared inflamed or enlarged. Examination reveals a pigmented mass (amelanotic melanomas are rare, but do occur) extending from the iris surface, from the anterior chamber angle, or through the pupil arising posterior to the iris. Concurrent intraocular hemorrhage, iridocyclitis with low intraocular pressure, or globe enlargement (buphthalmia with elevated intraocular pressure) are common. Other diagnostics, such as ultrasonography and gonioscopy, provide additional information about the mass's consistency and borders. A complete blood count, general chemistry, urine analysis, as well as thoracic and abdominal radiographs are considered a baseline workup. Enucleation is the usually treatment; however, with the low metastatic rate, some masses have been successfully treated by single or repeated diode laser photocoagulation or iridocyclectomy.



Figure 10.18 (A) Anterior uveal melanoma in a dog. The pigmented mass involves the dorsolateral quadrant of the iris. (B) Malignant melanoma of the ciliary body in a dog. The pigmented mass has caused secondary glaucoma, and has extended beyond the limbal sclera into the lateral subconjunctival tissues.



Figure 10.18 (*Continued*) (C) Anterior uveal melanocytoma melanoma in a dog. The pigmented mass involves the dorsolateral dorsomedial quadrant of the iris and is beginning to expand beyond the confines of the globe. (D) Amelanotic melanoma of the anterior uvea in a dog. This was a particularly aggressive neoplasm and resulted in severe glaucoma. The exudate in the anterior chamber was a mixture of inflammatory neoplastic cells.

Ciliary Body Neoplasms

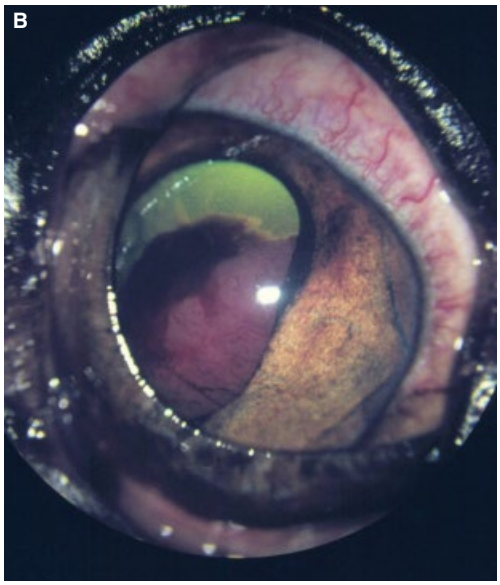
Ciliary body adenomas and adenocarcinomas are the second largest group of primary intraocular neoplasms in the dog (Figure 10.19; see also Figure 9.16). The German Shepherd and American Cocker Spaniel breeds seem predisposed. Older dogs are usually affected (8 years of age on average). As the majority appear to arise from the nonpigmented epithelium of the ciliary body, they appear clinically as white or pink tumors. The metastatic rate appears low.

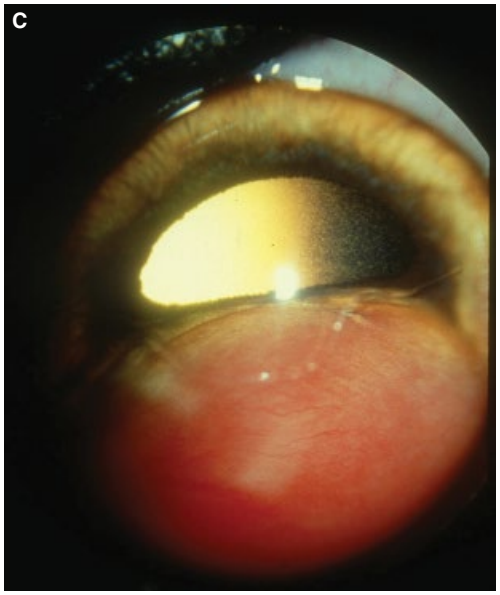
The owner usually notices a color change in the eye, or the mass can cause hyphema, anterior uveitis, and/or secondary glaucoma. Examination reveals a white to pink mass extending or encroaching into the anterior chamber through either the pupil or the base of the iris. Occasionally, blood vessels are visible on the surface of the mass. Both gonioscopy and ultrasonography provide additional information about the tumor's size and border. Enucleation is the recommended treatment.

Metastatic Neoplasms

The canine anterior uvea is an infrequent site for metastatic neoplasms. Most of these tumors spread hematogenously to the anterior uvea, although direct extension from adjacent structures, such as the sinus and nasal cavities, can occur (Figure 10.20; see also Figure 9.17). The most frequent metastatic tumors to the anterior uvea are hemangiosarcoma and transmissible venereal tumor. Other metastatic adenocarcinomas have occurred from distant organs such as the mammary gland, urinary bladder, thyroid gland, adrenal gland, nasal cavity, kidney, and pancreas.







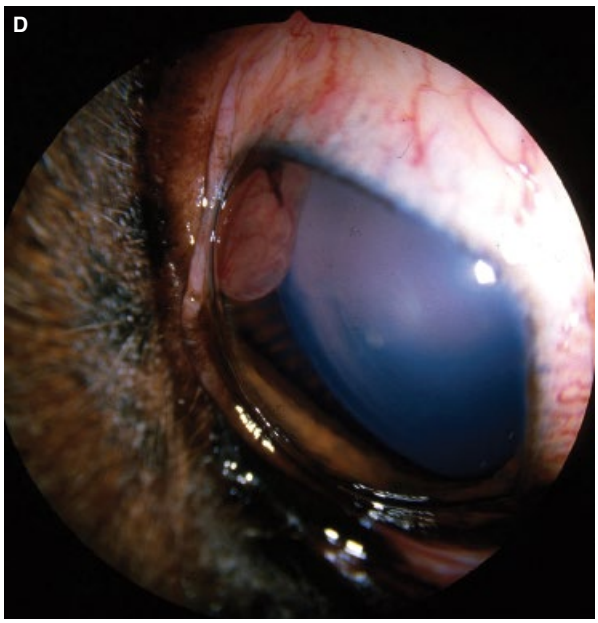


Figure 10.19 (A) Ciliary body adenoma. This mass was expansive, but benign. (B) Primary ciliary body adenocarcinoma in a dog. Note the nonpigmented mass within the distorted pupil. An area of hemorrhage covers part of the dorsal surface of the mass. (C) Very large ciliary body adenocarcinoma in a dog. The extent of the mass was best visualized with ultrasonography. (D) Ciliary body adenoma. Note that the mass is posterior to the iris and is displacing the iris forward allowing visualization of the adjacent ciliary body processes.

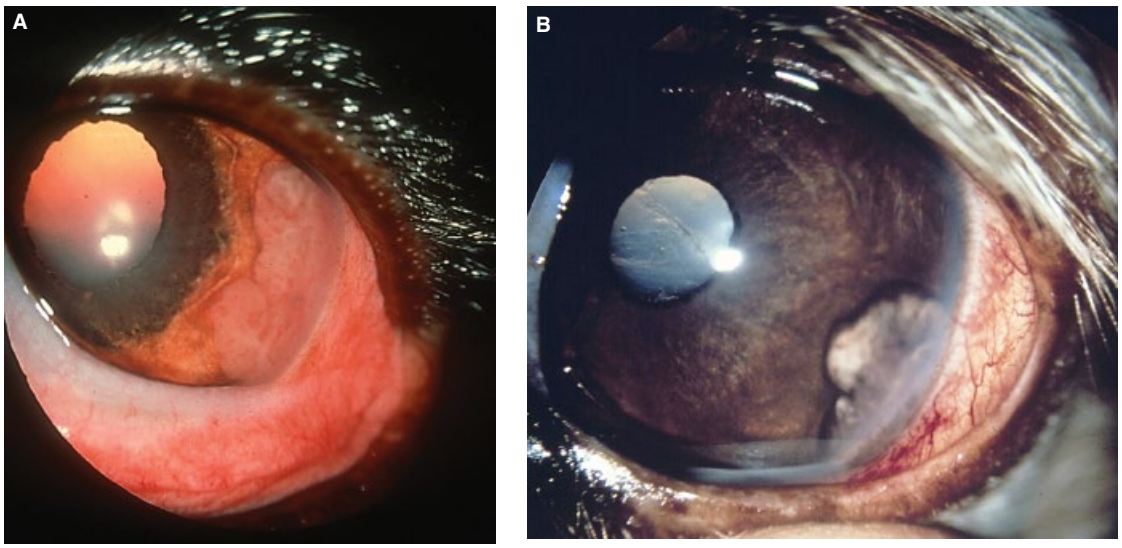


Figure 10.20 (A) Metastatic adenocarcinoma of the ciliary body that has extended forward of the iris and into the anterior chamber. The primary mass was an adenocarcinoma of the mammary gland. (B) Metastatic adenocarcinoma of the ciliary body at the base of the iris. The primary mass was a nasal adenocarcinoma that also involved the retrobulbar tissues.

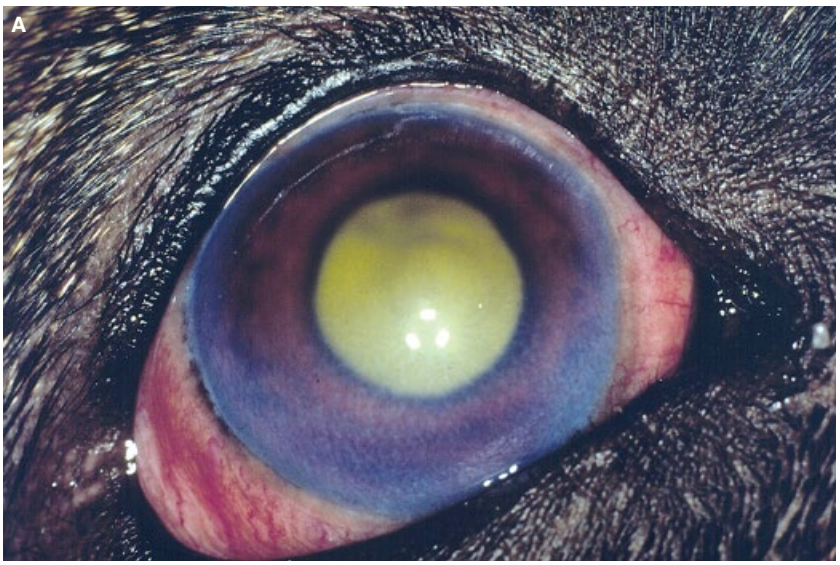
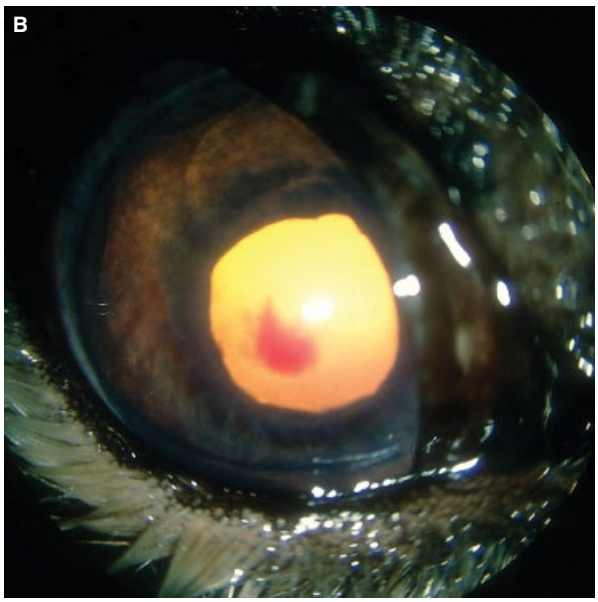


Figure 10.21 Lymphoma affects the orbit and many tissues of the eye. The most frequent form is anterior uveitis and infiltration of the uveal tract. (A) Iridocyclitis and uveal lymphoma in a dog. Note the circular peripheral corneal infiltrates, iridal swelling with ectropion uvea, and vitreal opacification with inflammatory and neoplastic cells.

The owner usually notes a color change in the eye, signs of anterior uveitis, hyphema, glaucoma, or blindness (often secondary to retinal detachment). The mass is usually nonpigmented and can be observed on the iridal surface, extending through the pupil, or the base of the iris into the anterior chamber. Gonioscopy and ultrasonography provide additional information relative to the tumor's size and borders. Once the diagnosis of a metastatic tumor is determined, periodic monitoring, chemotherapy, or euthanasia are possible options.

Lymphoma

Anterior uveal lymphoma is the most frequent secondary intraocular neoplasm in the dog. About 33–37% of



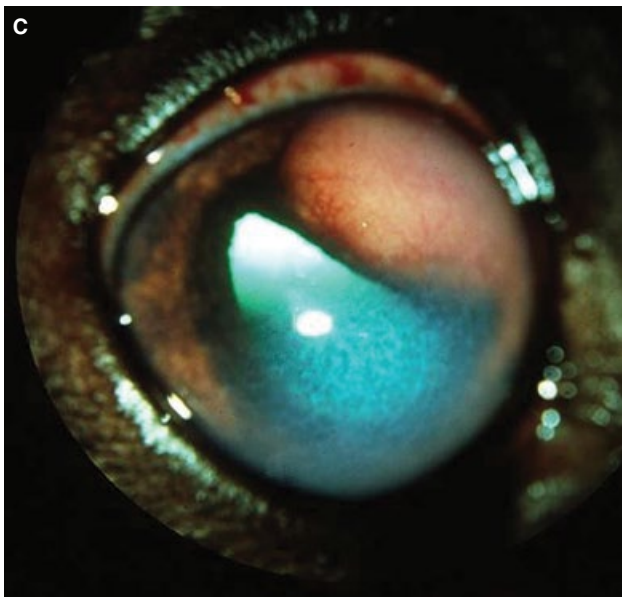


Figure 10.21 (*Continued*) (B) Hyphema on the central anterior lens capsule, anterior uveitis, and an irregular pupil from posterior synechiae in a dog with lymphoma. (C) Lymphoma in this dog has resulted in a focal mass lesion within the iris stroma as well as anterior uveitis. Note the distortion of the pupil.

dogs with systemic lymphoma have ophthalmic involvement and it is often bilateral. Anterior uveitis with either hypopyon or hyphema is the most frequent clinical abnormality (Figure 10.21; see also Figures 9.18 and 9.19). Other clinical signs include interstitial keratitis, conjunctival infiltrates and masses, intraretinal and subretinal hemorrhages, and secondary glaucoma. The interstitial keratitis can appear as a circular dense white band of neoplastic lymphocytes 1–2 mm from the limbus. The iris can be diffusely or focally enlarged with either lymphocytic or, more frequently, lymphoblastic lymphosarcoma. If one or both eyes are affected, the success or lack of therapy can be monitored by periodic ophthalmic examinations.

11

Canine Lens and Cataract Formation

The lens is a unique structure that is transparent in its normal state and functions to focus light onto the retina. It is made up of very regularly arranged lens fibers that are derived from the lens epithelium, which continues to produce new fibers throughout the animal's life. These fibers become densely packed within the lens nucleus (center) in later life which results in nuclear sclerosis, a normal aging change. It is enclosed by a thin lens capsule that isolates it from the body but maintains limited permeability to allow passage of essential nutrients. The lens capsule varies in thickness (anterior > posterior) and is suspended by dozens of tiny zonules (tertiary vitreous) from the ciliary body posterior to the iris. The lens is isolated from the rest of the body and immune system before birth and therefore is not recognized as self. When lens fibers become abnormal (cataractous) or the lens capsule is damaged or breached, a significant immune-mediated uveitis results, which is referred to as lens-induced uveitis.

Congenital Abnormalities

Developmental defects in the lens can involve the lens itself, persistent vascular systems designed to provide nutrition to the developing lens that fail to regress, or the zonules. The most common defects are smaller than normal lens (microphakia usually combined with microphthalmia) and cataract formation. Congenital cataracts are discussed later in the section on cataract.

Microphakia

Microphakia is a smaller than normal lens, and probably occurs more frequently than reported. Microphakia occurs in most, if not all, microphthalmic globes (Figure 11.1). Congenital cataracts are often also microphakic. In a study on Miniature Schnauzer congenital cataracts, ultrasonographic globe measurements from growing and adult normal and cataractous dogs revealed that the majority of cataractous eyes were slightly smaller than age-matched controls and the affected lenses were correspondingly reduced in size. In dogs with multiple ophthalmic anomalies, such as those in homozygous merle dogs or breeds with inherited retinal dysplasia and retinal detachments, microphakia is often present (see Figures 4.1 and 10.2).

Examination of an affected lens reveals reduced lens circumference and/or reduced thickness (anteroposterior). Ultrasonography (a-scan mode) measurements are the most accurate clinical (*in vivo*) method to measure the lens anteroposterior length (axial length). Other abnormalities include persistent pupillary membranes, persistent hyaloid remnants (persistent hyperplastic tunica vasculosa lentis, PHTVL and persistent hyperplastic primary vitreous, PHPV), and lenticonus or lentiglobus. If the microphakic lens is cataractous, the opacity remains stationary in size or progresses depending on the areas of the lens affected. Treatment is not usually attempted provided clinical vision is present.

Lens Colobomas

Colobomas of the canine lens are rare. They appear clinically as a notch or indentation of the lens equator (Figure 11.2). They can be typical (located at the 6 o'clock position) or atypical (all other sites). Lens colobomas appear directly related with the focal absence of zonules and an associated coloboma in the ciliary body and often the iris. Cataract may not be present.

Lenticonus and Lentiglobus

In lenticonus and lentiglobus the anterior and/or posterior surface of the lens protrudes in either a conical (conus) or more generalized (globus) projection. Posterior lenticonus is the most common form and occurs occasionally with congenital cataracts (inherited congenital cataracts in the Miniature Schnauzer or persistent hyaloid remnants (PHTVL and/or PHPV) (Figure 11.3).

The clinical appearance of posterior lenticonus, as viewed anteroposteriorly, is a circular and often cataractous outcropping of the posterior lens and capsule.

Color Atlas of Veterinary Ophthalmology, Second Edition. Kirk N. Gelatt and Caryn E. Plummer.
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Figure 11.1 Microphakia in a dog. Note the lens periphery is smaller than normal, and elongated ciliary processes surround the lens equator (arrows). A complete hypermature cataract is also present.

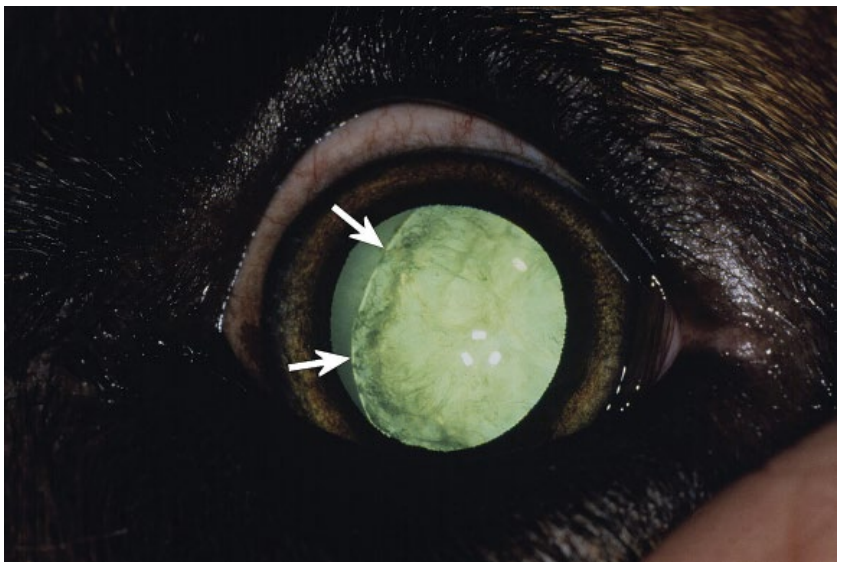


Figure 11.2 Lens coloboma in a puppy. The coloboma is a flattened area devoid of zonulary attachments (arrows). An incomplete and immature cataract is also present.

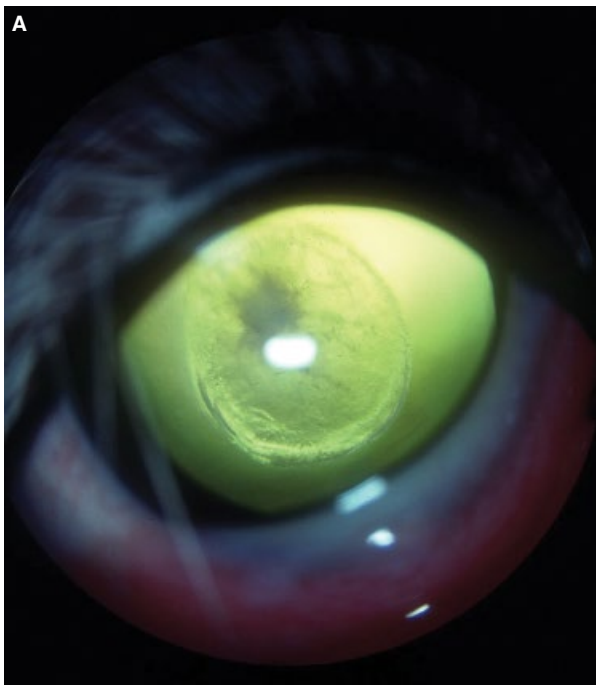


Figure 11.3 (A) Lenticonus in an Old English Sheepdog puppy. The very discrete focal cataract affects the posterior cortex and posterior lens capsule.

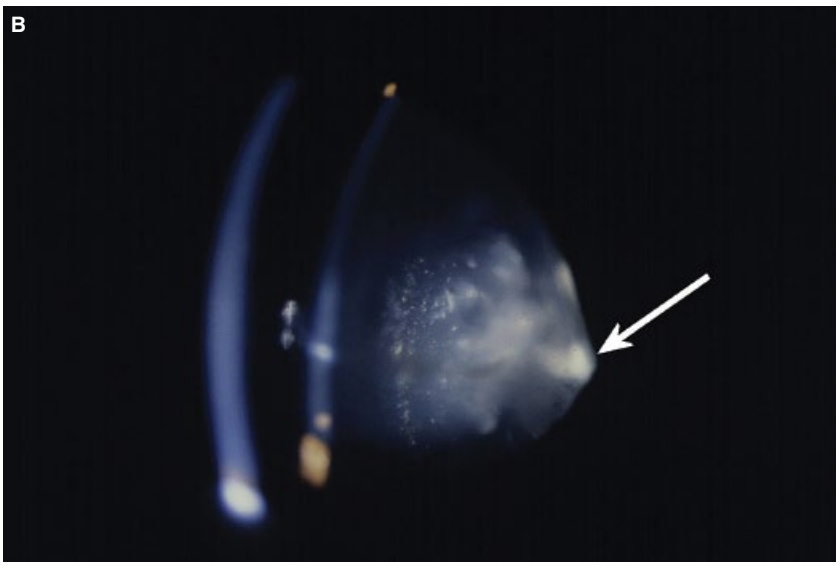


Figure 11.3 (*Continued*) (B) Slit lamp biomicroscopic view of posterior lenticonus in a Miniature Schnauzer cataract (nuclear and posterior cortex). The posterior protrusion from the caudal lens extends into the anterior vitreous (arrow).

Posterior protrusion of the polar area of the lens into the anterior vitreous can be confirmed with slit lamp biomicroscopy. If the lens is cataractous, ultrasonography can be used to detect posterior lenticonus.

In the evaluation of congenital cataracts for surgery, special attention is directed at the posterior lens. Because posterior lenticonus often signals a thin and weak polar posterior lens capsule and possible anterior vitreal abnormalities, cataract surgery in these eyes presents additional risks.

Persistent Pupillary Membranes and Anterior Cataracts

As noted in Chapter 10 on the canine anterior uvea, persistent pupillary membranes (PPMs) are the most frequent anterior uveal anomaly in the dog. They represent an abnormal development of the pupil, and arise from the anterior surface (collarette) of the iris (Figure 11.4; see also Figures 8.3 and 10.3). This distinguishes the PPM from posterior synechia which implies the adherence of the pupillary or posterior surface of the iris to the lens, and suggests past or current iridocyclitis.

PPMs extend from the collarette area to the anterior lens capsule, posterior cornea, or to an adjacent collarette area. PPMs extending to the anterior lens capsule appear as fine gray to white strands or larger bands of pigmented tissue. They can contain blood vessels, which hemorrhage when torn by traction of miosis or mydriasis, or laser or surgical incision. Anterior capsular or subcapsular cataracts, secondary to PPMs, usually do not progress. If small and focal, these cataracts may have limited to no appreciable effect on clinical vision. If larger, dense, and within the pupil, an adverse effect on vision (particularly in bright light, when miosis further limits the axial field of view) can occur; in these patients long-term drug-induced mydriasis and/or cataract surgery may be beneficial.



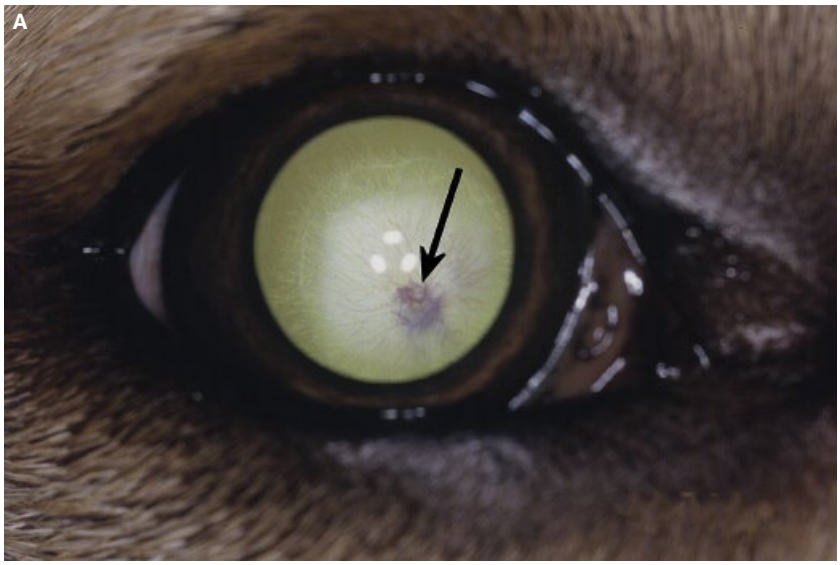
Figure 11.4 Old English Sheepdog puppy with four persistent pupillary membranes (PPMs) which extend from the collarette area of the anterior iris and insert on the anterior lens capsule causing cataract formation. Additional cataract formation is occurring in the lens cortices.

Persistent Hyaloid and Posterior Cataracts

Persistent hyaloid remnants (PHPV and/or PHTVL) are an infrequent condition in puppies, and affect one or both eyes (Figure 11.5; see also Figure 12.1). This disease has been investigated in the Doberman Pinscher and Staffordshire Bull Terrier. Other breeds affected include the Bouvier des Flandres and the Standard Schnauzer.

Persistent hyaloid remnants, when the sole abnormality, are characterized by a posterior polar cataract with blood vessels extending from the optic nerve head to the polar posterior lens capsule. The cataract is usually limited in size and density, permitting ophthalmoscopy and examination of the ocular fundus. Treatment is usually not indicated unless some daytime vision impairment occurs. Topical mydriatics can permit the dog to see "around" the opacity.

The disease in the Doberman Pinscher is more severe and usually includes other developmental anomalies



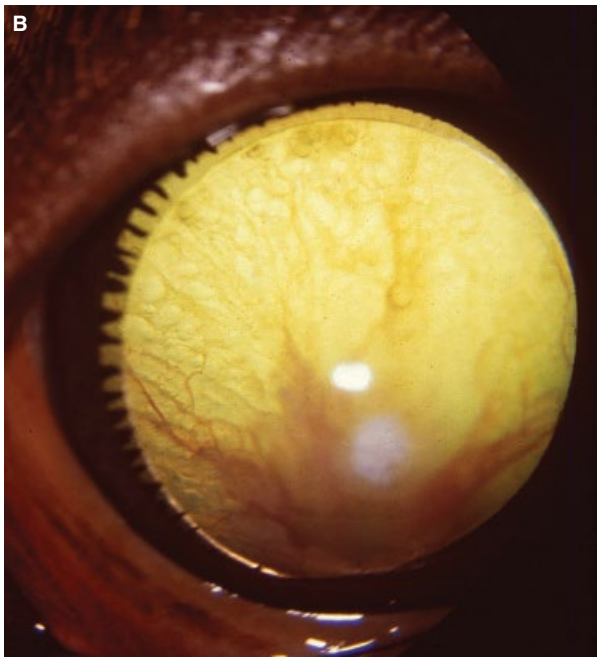


Figure 11.5 (A) Persistent hyaloid vascular remnants in a Labrador Retriever puppy. Note the red hyaloid blood vessels (arrow), and posterior cortical and capsular cataract. (B) Persistent hyperplastic tunica vasculosa lentis in a hound dog. The surrounding mat of blood vessels are perfused and posterior capsular cataract is present.

including persistent pupillary membranes, secondary cataracts with anomalous posterior lens capsules, posterior lenticonus, and retinal dysplasia. As a result, cataract surgery in this breed is hazardous and yields low success rates.

Cataracts

Cataracts are defined as an opacity of the lens or its capsule. Cataracts are frequently encountered in dogs, and are a leading cause of blindness in purebred dogs. Cataracts are classified clinically by age of onset (congenital, adult, and senile), position within the lens (anterior capsule, anterior cortex, nuclear, posterior cortex, and posterior lens capsule; also polar, axial, sutural, zonular, and equatorial), stage of maturity (incipient, immature, mature, hypermature, Morganian), etiology (inherited, traumatic, toxic, metabolic, inflammatory), and appearance (spike, wedge, spoke, cuneiform, sunflower, stellate, punctate, and purverulent). Cataracts are caused by abnormalities of the lens fibers and/or capsule and must be distinguished from nuclear sclerosis, a normal aging change, and minor lens imperfections which can be detected with slit lamp biomicroscopy (Figure 11.6). These imperfections include zones of discontinuity between the anterior and posterior cortical and nuclear (adult, fetal, and embryonal) regions, anterior and posterior lens sutures, Mittendorf's dot (remnants of posterior hyaloid on the posterior lens capsule), and arcuate line of Vogt (circular line of the anterior aspects of Cloquet's canal).

Lens Changes and Aging

Nuclear sclerosis is a normal aging change of the lens produced by the compaction of the central lens fibers (Figure 11.7). It is often confused with cataract formation, but it produces no significant impairment of vision. When it is particularly dense, affected individuals can exhibit subtle decreases in depth perception. The condition produces a gray to bluish color (caused by light scattering) to the center or nucleus of the lens, but does not impede direct or indirect ophthalmology and visualization of the ocular fundus, except when very dense in the very elderly dog.

Inherited Cataracts in the Dog

Inherited cataracts occur in a great number of dog breeds. More than 90 breeds are predisposed to cataract development and the mode of inheritance has been documented in about 20 breeds. Although both lenses become cataractous in the inherited types, the time of onset and degree of cataract formation can vary between eyes (see Appendix C).

Classifying cataracts by their stage of maturity is very useful as it correlates to clinical findings and effects on vision (Figure 11.8). There are four main stages of cataract: incipient, immature, mature, and hypermature. In the

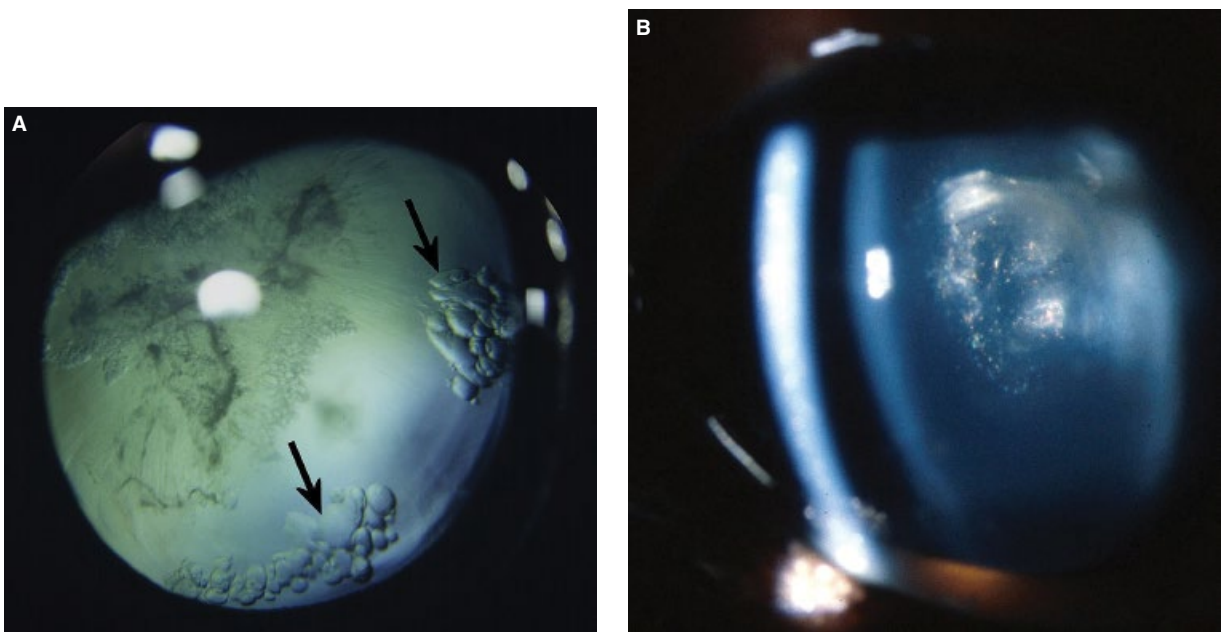


Figure 11.6 (A) Cataract formation is first evidenced as the formation of vacuoles (arrows) within the lens. These vacuoles gradually enlarge and form "water clefts." (B) More extensive cataract formation in a Chesapeake Bay Retriever. The cataract formation is concentrated in the lens nucleus, but has extended into the posterior cortex.

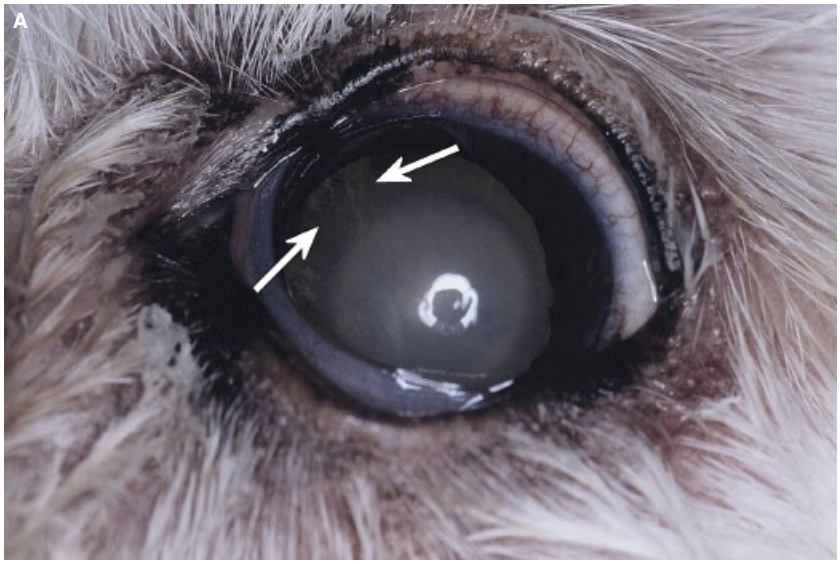






Figure 11.7 (A) Nuclear sclerosis of the lens in a 10-year-old dog. The gray translucent zone is confined to the lens nucleus. Cortical cataract formation is also starting (arrows). (B) Nuclear sclerosis in an aged mixed-breed dog. Note the strand of iris indicating atrophy along the ventral pupil. (C) In this example of nuclear sclerosis, the fundic reflex is easily seen.

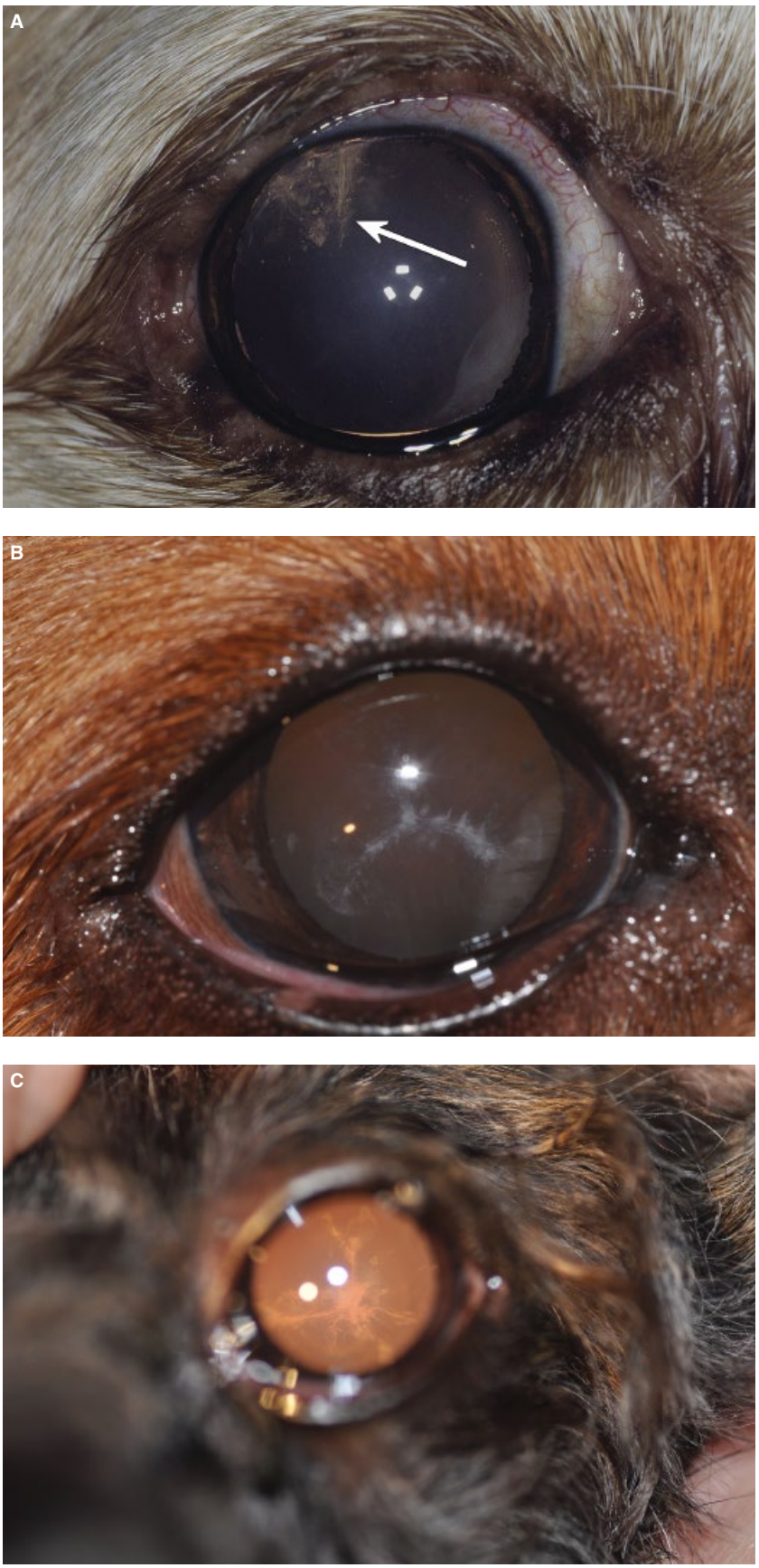
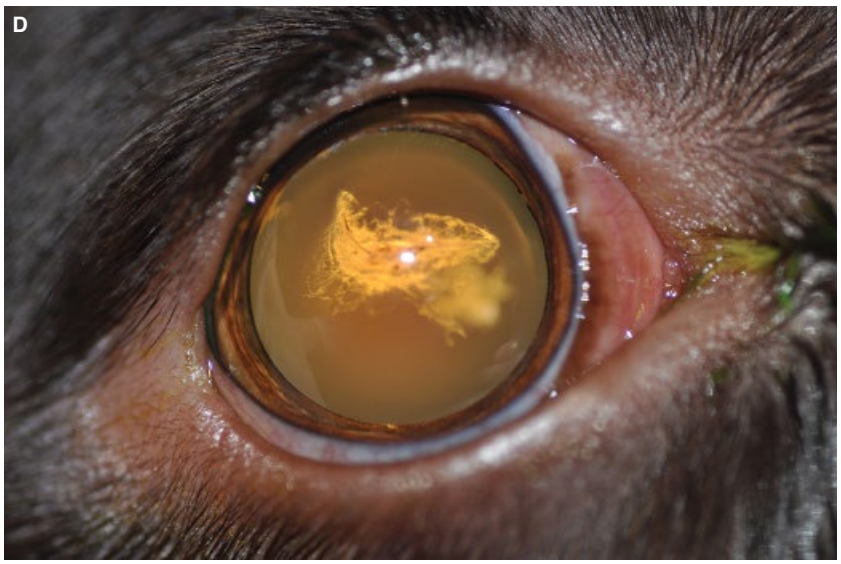
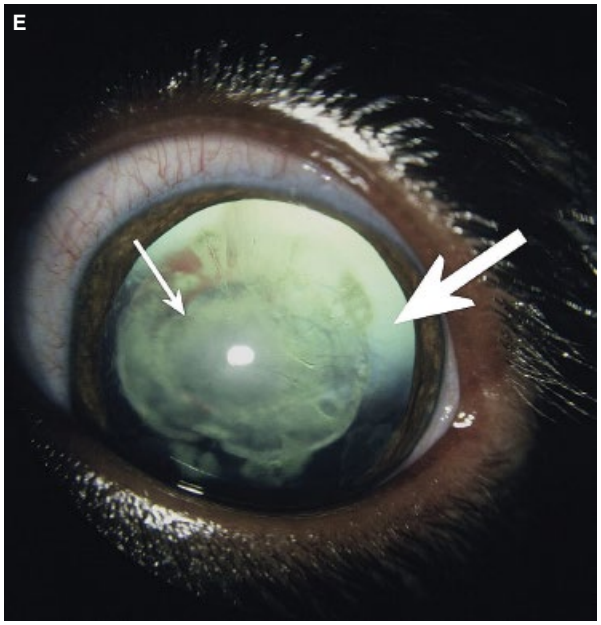


Figure 11.8 Cataract formation is classified by stages of maturity. (A) Incipient: Cataract formation is just starting (arrow), and affects limited areas of the lens. Tapetal reflection and vision are normal. (B) Incipient: Cataract formation has occurred along the anterior sutures in this lens. (C) Incipent: Cataract formation has occurred along the posterior sutures in this lens.





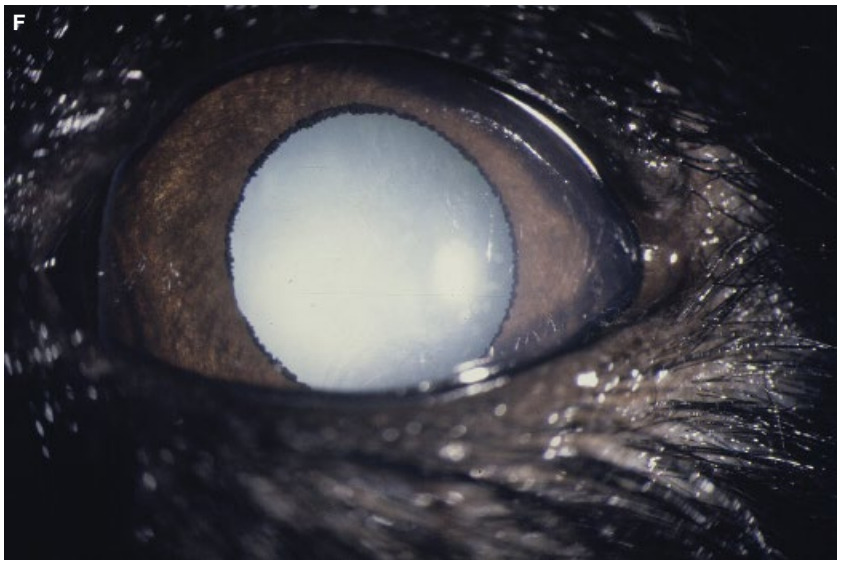


Figure 11.8 (*Continued*) (D) Incipient: There is early cataract formation in both the anterior and posterior aspects of this lens. Note the difference in the focus for the two regions of the lens. (E) Immature: Both normal (large arrow) and cataractous areas (small arrow) are present. The tapetal reflection and vision are normal. (F) Mature: The entire lens is cataractous and may be swollen (intumescent). Both tapetal reflection and vision are absent.





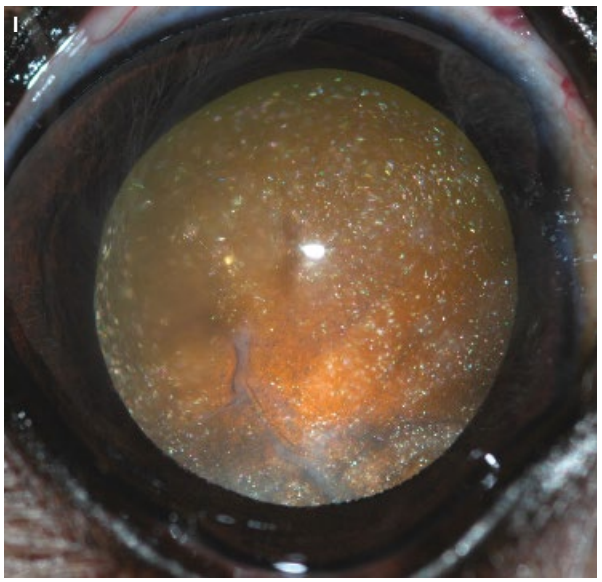


Figure 11.8 (*Continued*) (G) Hypermature: The entire lens is cataractous but lens volume is reduced. The anterior lens surface may be irregular and wrinkled. Tapetal reflection and vision are usually absent. Lens-induced iridocyclitis is usually present. Note the ciliary flush indicating iridocyclitis. (H) Hypermature: Resorbing lens material will often take on a "sparkly" appearance in a hypermature cataract. (I) Hypermature: In this example of a hypermature cataract, most of the lens material has resorbed leaving only a smattering of sparkly particles and capsular plaques.

incipient form, only a small part of the lens is involved (10–15%), the tapetal reflect is normal, vision is unaffected, and the ocular fundus is easily visualized. In the immature cataract, there are both normal and cataractous areas of the lens, the tapetal reflect is present, clinical vision is either normal or impaired (can be improved by mydriasis), and the ocular fundus can be visualized through the clear areas of the lens. In the mature cataract, the entire lens is cataractous, the lens can be swollen (intumescent), the tapetal reflection is absent, and the ophthalmoscopic view of the ocular fundus is not possible. In hypermature cataracts, the lens is reduced in size, the anterior lens capsule is irregular and wrinkled, the tapetal reflection may not be visible, and ophthalmoscopy is limited.

Experience and research suggest the cataract patient of choice is the dog with immature cataracts, rather than mature or hypermature cataracts. Lens-induced uveitis (LIU) develops in dogs during cataract formation via the leakage of lens material through an intact anterior lens capsule. Cataract surgery in dogs with established LIU and mature and hypermature cataracts yields lower success rates and higher rates of complications.

Primary Cataracts in Selected Breeds

Cataracts are presumed to be inherited if there is no pre-existing condition or history that could explain the presence of cataract in an individual patient, such as diabetes mellitus or chronic uveitis. Purebred dogs with inherited cataract will often develop lenticular opacities at specific ages or in specific locations in the lens. Most have bilateral involvement. The lag time between the initial detection of early cataract formation and blindness varies by breed, but can be years. Cataracts that involve the equatorial region (periphery of the lens) are expected to progress (all lens growth occurs in this area). Nuclear cataracts (within the center of the lens) are congenital and do not usually progress and may even gradually become relatively smaller as the lens enlarges.

In the establishment of inherited cataracts, periodic eye examinations have proven extremely useful in detecting the development of the cataract at its earliest stages and monitoring the progression of these changes. It is important to examine certain breeds with established ages of onset during this "window of time" to detect early cataract formation. Additionally, determining the initial site of cataractogenesis is very useful in many breeds in the determination of the heritable cataracts (Figure 11.9).

For instance, in the Afghan Hound and Standard Poodle, the first detectable site of cataract formation is the lens equator, and cataract progression will follow. In the American Cocker Spaniel, anterior and posterior cortical involvement of the lens signal inherited cataract in this breed. These cataracts either quickly (a few months) or slowly (a few years) reach maturity and cause blindness.

In the Miniature Schnauzer inherited congenital cataract, initial cataract formation occurs within the lens nucleus. Cortical involvement is variable and progression of the cataract is likely over the next few years. The Boston Terrier has two forms of inherited cataracts: nuclear cataracts in puppies, and equatorial cataracts in adult dogs. Both are progressive and eventually involve the entire lens.

The adult Golden and Labrador Retrievers have similar cataracts that are inherited in an autosomal dominant fashion, either a posterior polar non-progressive cataract (heterozygous affected) or a generalized cortical and progressive type (homozygous affected). Golden Retrievers also have congenital cataracts. In the Siberian Husky, cataract usually develops in the first 1–3 postnatal years. Cataract formation starts in the posterior cortex and progression is variable.

Secondary Cataracts

Diabetes mellitus is the most frequent cause of metabolic cataracts in the dog (Figure 11.10; see also Figure 18.21). Diabetic dogs are the second largest group of patients presented for cataract surgery. Once the diagnosis of diabetes mellitus and insulin treatment have started, approximately 50–70% of the dogs will develop cataracts within 6–12 months. Cataract formation is related to the inability of the lens to metabolize glucose at sufficient levels, resulting in the accumulation of intracellular sorbitol. Sorbitol eventually changes the osmotic balance

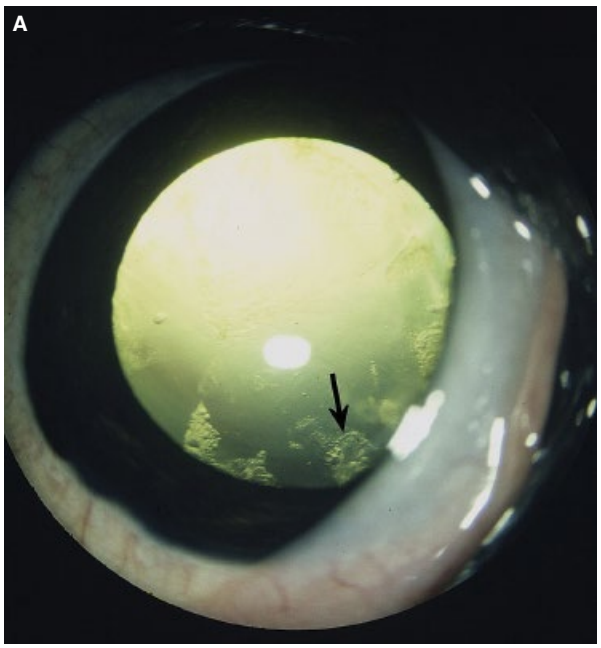
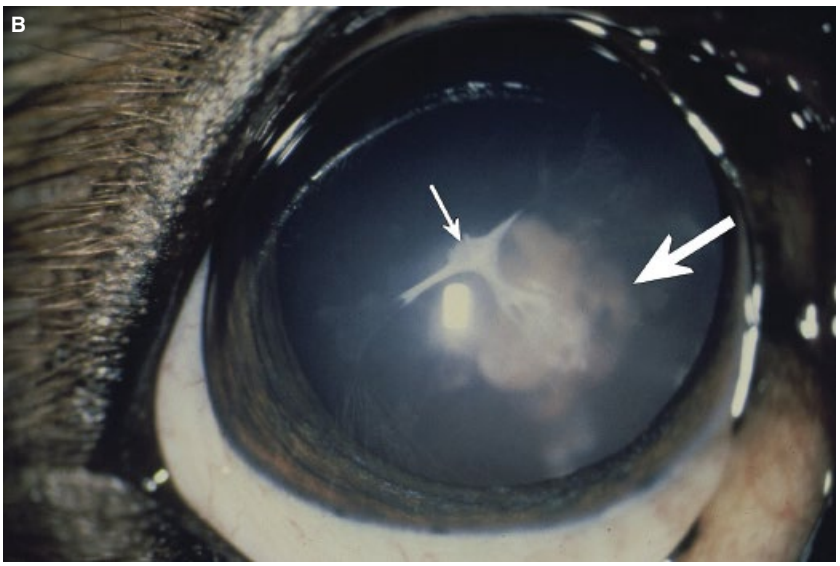
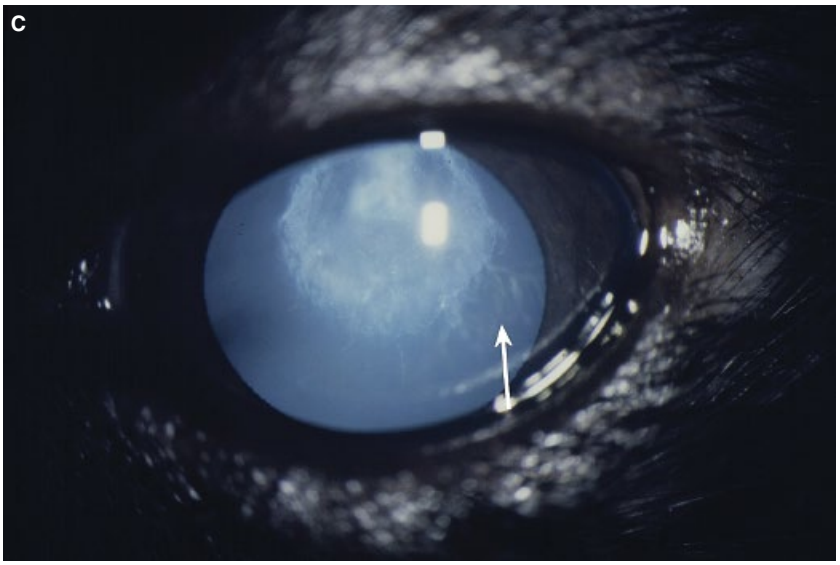
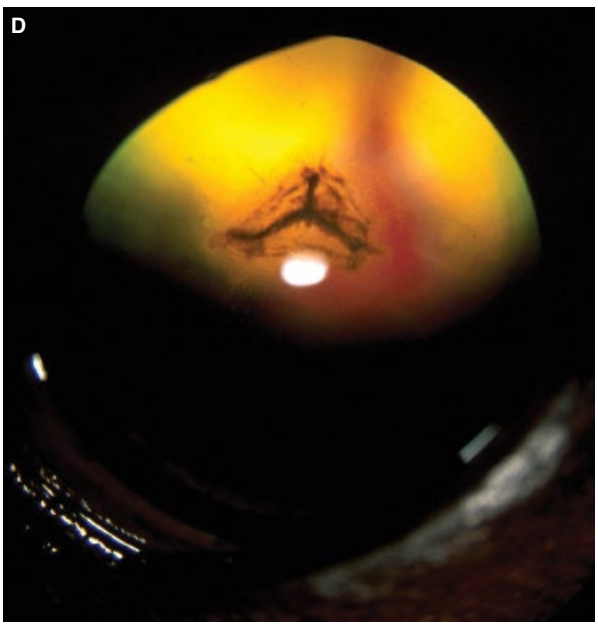


Figure 11.9 The age of onset and area(s) or region of the lens first involved in cataract formation are often seen in certain breeds. (A) Early cataract formation (i.e., presence of vacuoles, arrow) in a 2-year-old Afghan Hound. The first sign of cataractogenesis is in the lens equator.







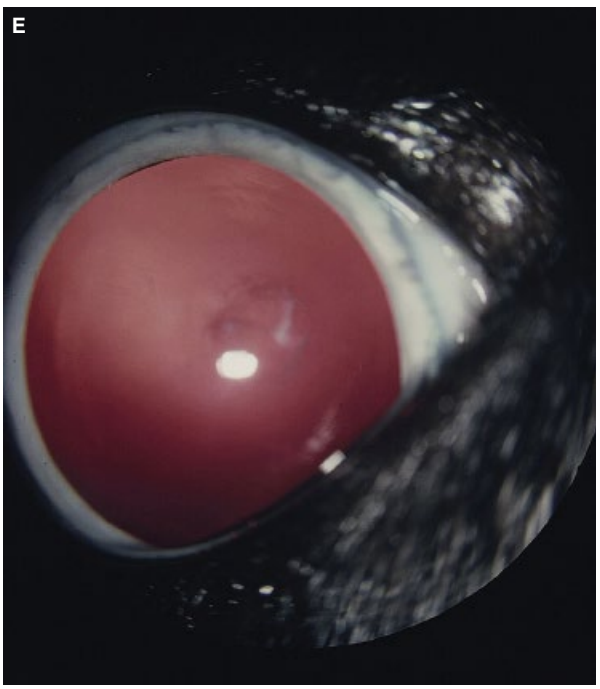


Figure 11.9 (*Continued*) (B) Cataract formation affecting affecting the anterior (small arrow) and posterior (large arrow) cortices in an American Cocker Spaniel. (C) Cataract formation primarily involving the nucleus in a Miniature Schnauzer. Some early cortical cataract formation (arrow) has also started and cataract progression will occur. (D) Posterior polar cataract formation in the Golden Retriever (and Labrador Retriever). This cataract does not usually progress to any appreciable extent and does not cause blindness. It can cause vision impairment in bright sunlight. (E) Posterior cortical cataract formation in the Siberian Husky. Progress is variable and often slow.



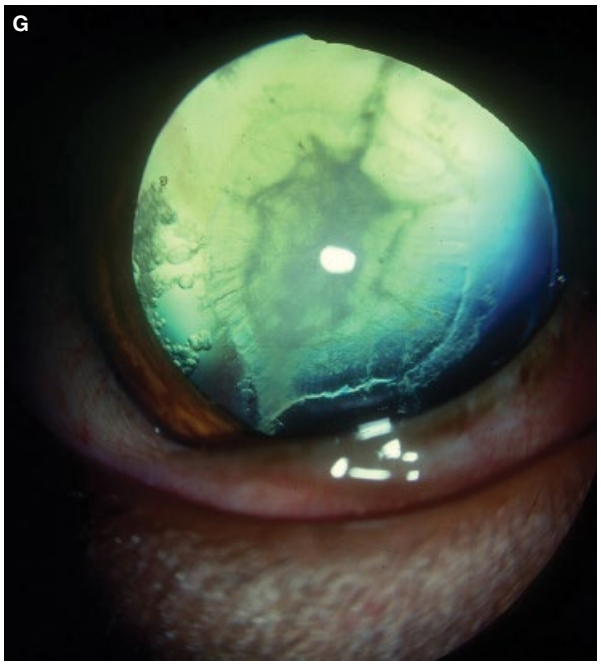


Figure 11.9 (*Continued*) (F) Inherited cataracts in the Bichon Frise. Cataract formation starts in both the anterior and posterior cortices. (G) Cataract formation affecting nucleus and cortices in the Chesapeake Bay Retriever.

within the lens fibers and causes fiber swelling, rupture, and death. Both age of the dog and the levels of the blood glucose influence onset and progression of the cataractogenesis.

The earliest sign of diabetic cataract formation is the development of vacuoles within the lens equator. The cataract often rapidly advances to cortical involvement, and cataract maturation. Often, lens swelling is marked and results in water clefts within the suture lines (intumescent cataract). Because of rapid swelling of the lens during diabetic cataract formation, ruptures of the anterior or equatorial lens capsule can occur, and require immediate cataract removal before other complications develop. Cataract surgery in diabetic dogs is as successful as with primary or inherited cataracts.

Cataracts Secondary to Inflammation

Cataracts, secondary to iridocyclitis of any etiology, are a common form of secondary cataract in dogs (Figure 11.11; see also Figure 10.12). Often, these cataracts are associated with posterior synechiae, inflammatory membranes, and contain deposits of pigmented iridal tissues on the anterior lens capsule (usually torn iris tissue from posterior synechia). The incidence of cataract development increases with the chronicity of the uveitis.

Cataracts and Trauma

Injury to the lens can follow penetrating as well as blunt trauma to the eye (Figure 11.12). Penetrating injuries of the lens are most common, especially following cat scratches, and pellets or buckshot from shotguns.

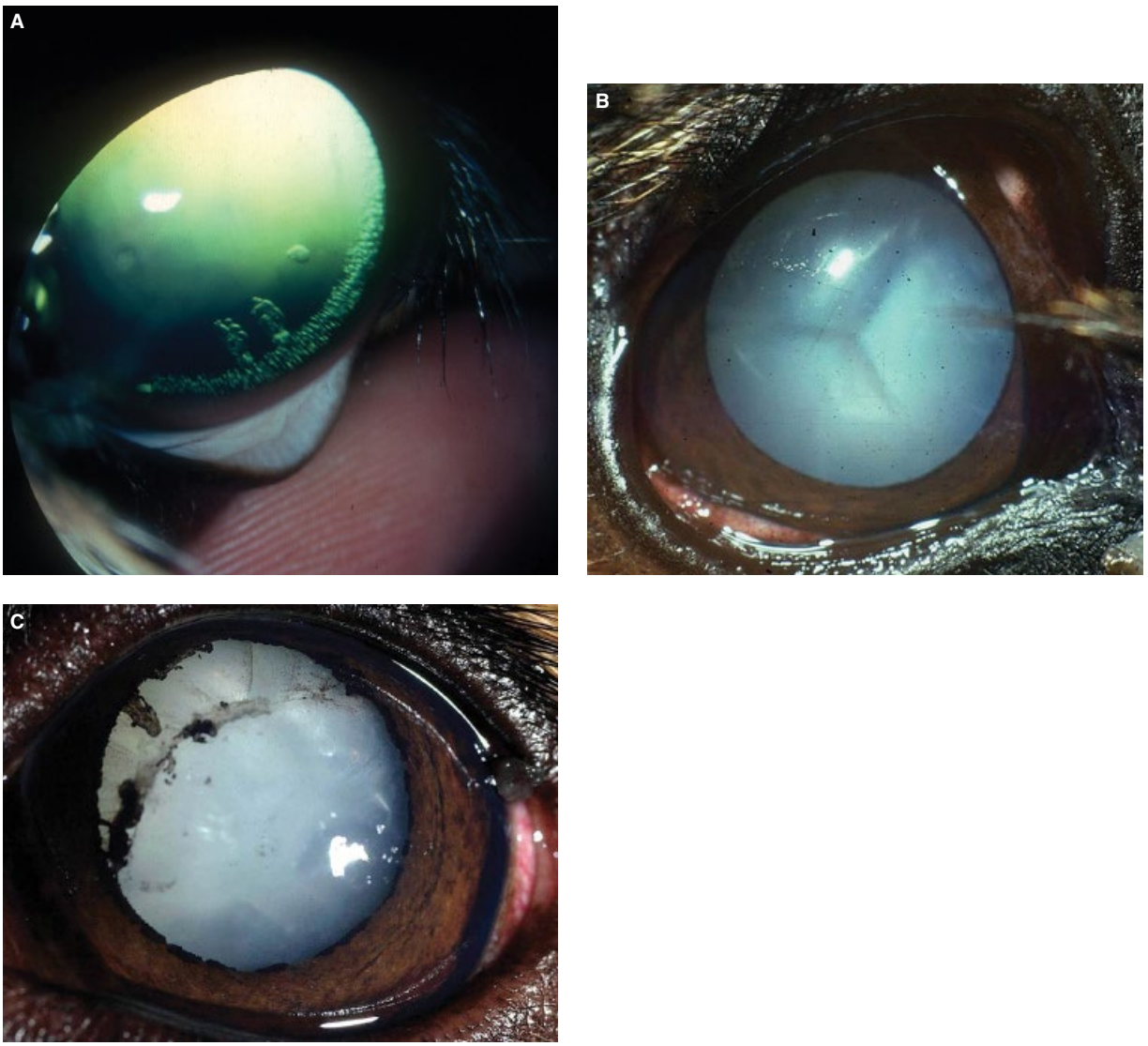


Figure 11.10 (A) Diabetes mellitus first affects the lens equator and is seen as vacuole formation within the lens equator. (B) Intumescent (swollen) diabetic cataract. (C) Intumescent diabetic cataract that has ruptured its capsule due from excessive swelling. The pigment on the dorsomedial aspect of the lens is the location of the rupture at the equator.

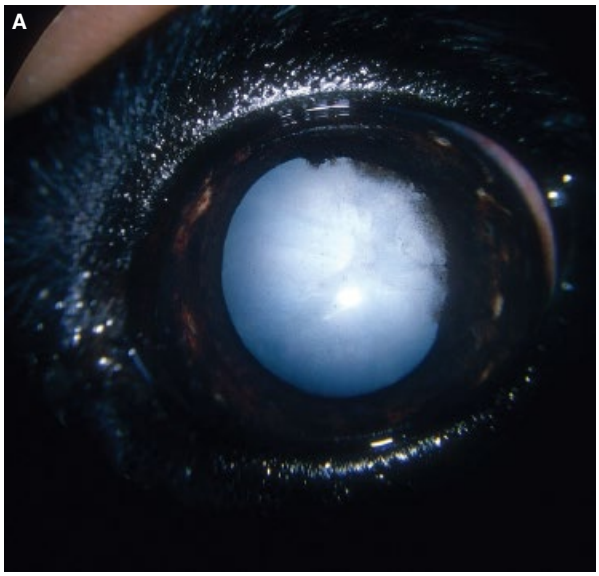
Laceration of the anterior lens capsule by cat scratches occurs most commonly in puppies and dogs younger than 1 year of age. The laceration is usually near the limbus, and the corneal wound may be self-sealing. Lacerations of the lens capsule longer than 1.5 mm with evidence of active leakage of lens material are candidates for immediate lensectomy (phacoemulsification). With good illumination and some magnification, the tear may be detected; if lens material is protruding through a capsular rent, a tear should be suspected.

Medical treatment (rather than lens removal) is not recommended as the LIU will usually become refractory to therapy, and phthisis bulbus or end-stage glaucoma is the final result.

With shotgun pellets (birdshot or buckshot) penetrating the cornea, the corneal wounds are usually self-sealing, appearing as slightly tan or brown focal depressions. The lens should be carefully inspected and, if penetrated, lens removal (by phacoemulsification) is recommended. Intraocular hemorrhages are common both in the anterior chamber and in the vitreous.

Cataract Resorption

The potential for spontaneous cataract resorption is greater in those breeds with inherited cataracts that occur in the first few years of life. It is not unusual for cataracts in older dogs (over 5 years) to progress from incipient, immature, mature, to hypermature, but resorption of the cataracts sufficient to restore vision is rare (Figure 11.13).



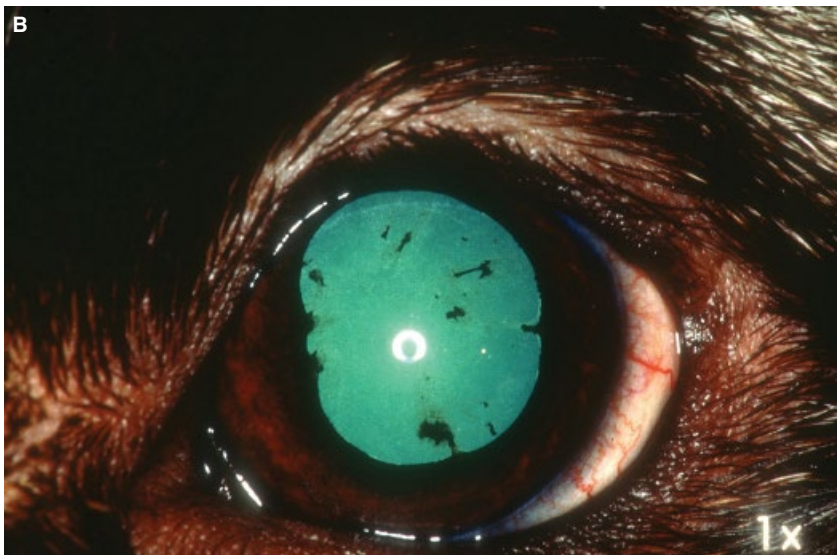
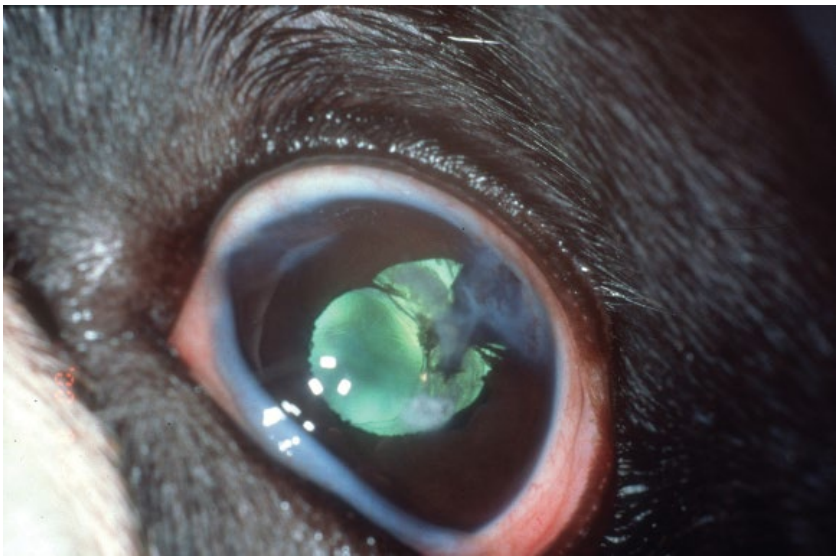
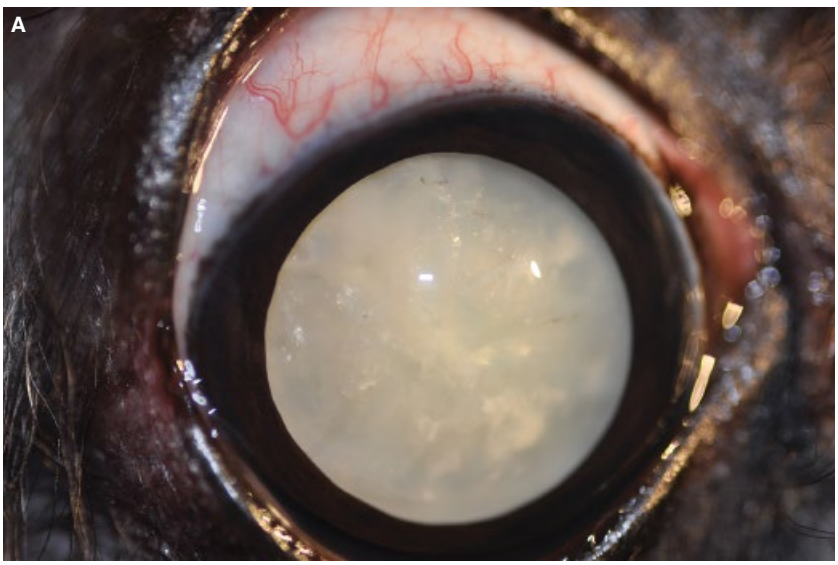


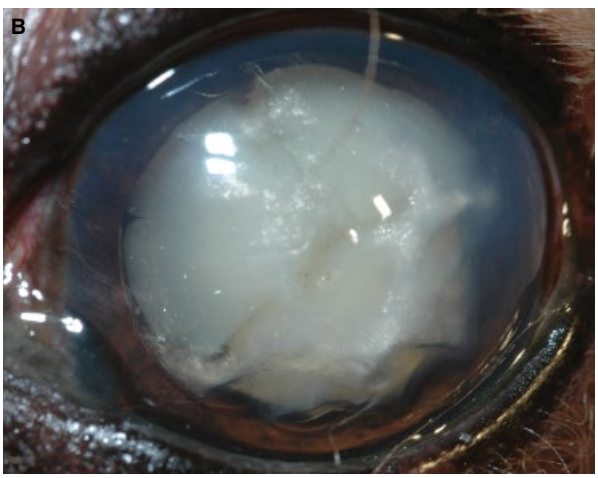


Figure 11.11 (A) Cataract can develop secondary to iridocyclitis from the formation of posterior synechiae, possible changes in the aqueous humor composition, and the formation of pre-iridal and pupillary membranes. Note the irregular pupil, several posterior synechiae, and a complete mature cataract. (B) Early cataract formation with multiple posterior synechiae. (C) Early anterior capsular cataract in a Golden Retriever with pigmentary uveitis. There is a uveal cyst in the ventral anterior chamber.

Figure 11.12 Lens injury, either penetrating or blunt, can cause cataract formation. In this dog the immature and incomplete cortical cataract developed following blunt trauma, as well as iridocyclitis, and the formation of posterior synechiae causing an irregular pupil shape.







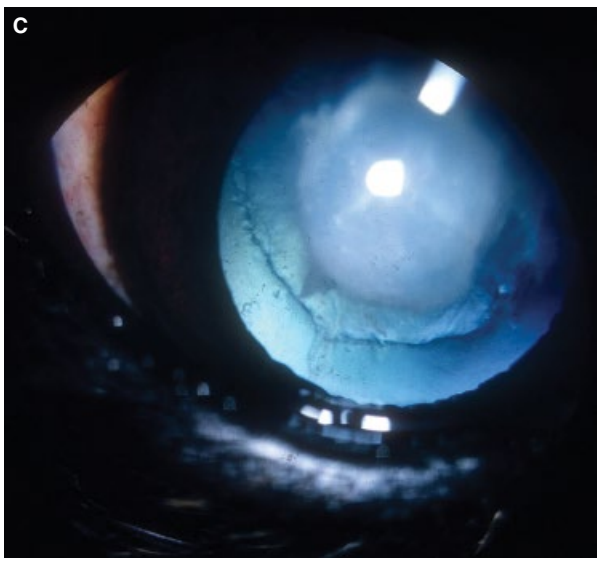
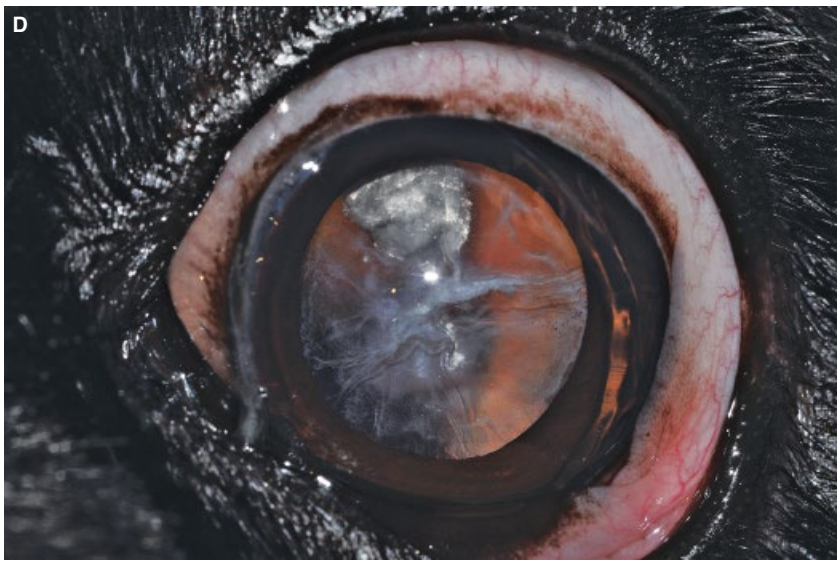
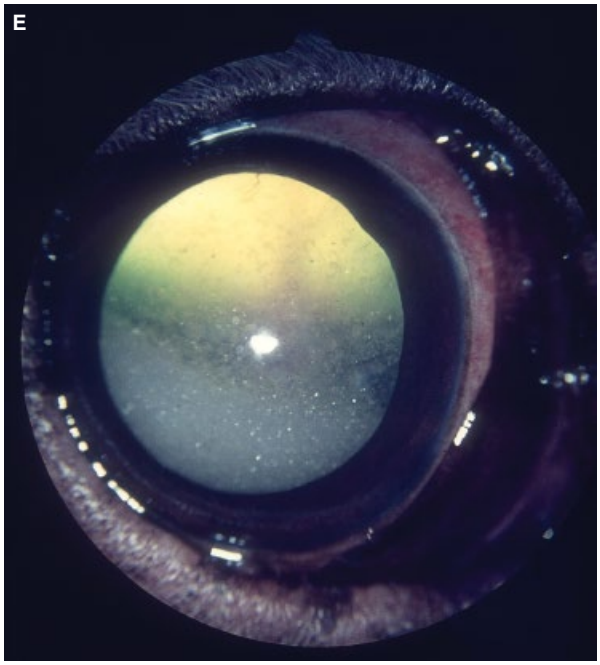


Figure 11.13 (A) Early resorbing hypermature cataract. (B) Resorbing hypermature cataract. Note the wrinkling of the lens capsule. The ventral aspect of the lens has subluxated and is sitting in front of the iris. (C) Early cataract resorption involving mostly the lens cortex. Overall the lens is reducing in size. The nucleus is still fairly opaque. Advanced cataract resorption. Most of the lens has resorbed, except for some sparkly abnormal lens fibers adhered to the remaining capsule.





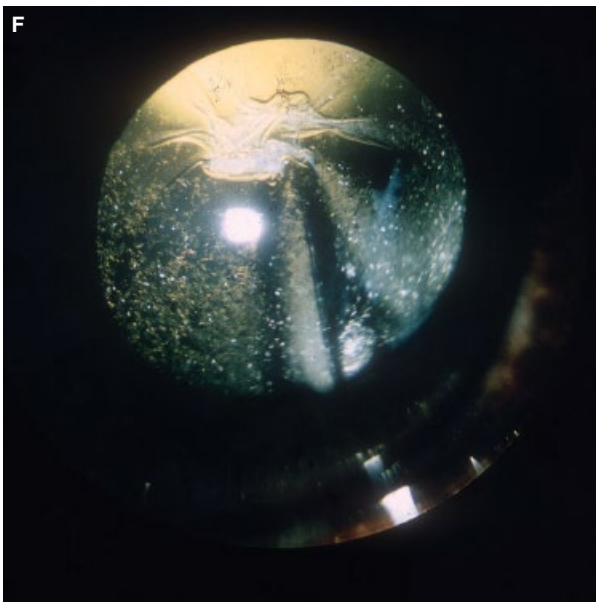


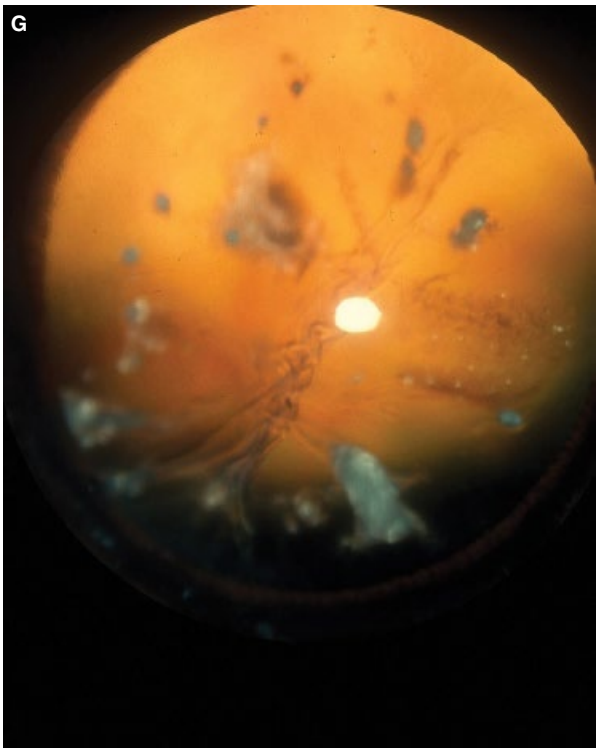
Figure 11.13 (*Continued*) (D) More advanced cataract resorption affecting the cortex (nearly resorbed) and the nucleus (partialy present). With drug-induced mydriasis, the dog is visual. Wrinkles in the lens capsule are present. (E) Cataract resorption. Residual lens material has gathered in the ventral aspect of the lens capsule. The dorsal aspect is clearing. (F) End-stage cataract resorption, with the nearly empty lens capsular bag and several small lens remnants. The dog is visual.

Cataract resorption is more common when cataract formation is rapid. Intumescent cataracts often develop rapidly and then resorb over time. The intumescence disappears and the anterior lens surface changes from convex to plano to even concave, as the lens shrinks. Resorption occurs initially in the equatorial and cortical areas, as the nucleus is most resistant to degradation. Proteases have been identified within the lens that degrade lens proteins into smaller molecular weights lens proteins which are small enough to transverse the anterior lens capsule. LIU will be present during lens resorption and requires monitoring and usually requires treatment with topical corticosteroids.

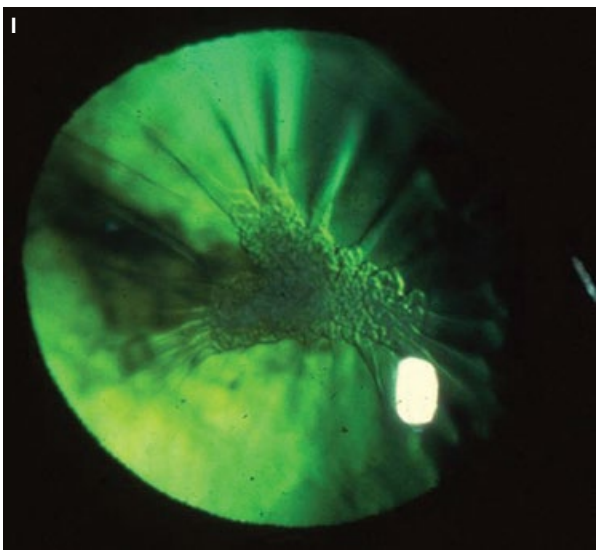
Lens Luxation or Displacement

Lens luxations or displacements occur when their zonular attachments weaken and rupture. Zonules, which maintain the lens in position, can be weakened and even transected by abnormal development, degeneration, trauma, rupture, or some combination. Lens luxations

Figure 11.13 (*Continued*) (G) Further clearing of the lens. Sometimes the entire capsular bag is devoid of any lens material and totally clear. (H) A large capsular wrinkle and a few residual cataractous plaques remain in this resorbing lens. (I) Wrinkling of the lens capsule in a completely resorbed cataract. This animal is essentially aphakic and hyperopic (far-sighted).







occur most frequently in the terrier breeds as a result of zonular dysplasia, and inheritance has been documented in the Smooth-haired Fox Terrier, Wire Hair Fox Terrier, Sealyham Terrier, Jack Russell Terrier, and in non-terrier breeds, such as the Border Collie, German Shepherd, and Tibetan Terrier (reportedly not a terrier breed). The age of onset of those breeds with inherited lens luxation is usually 3–5 years old.

When the lens is unstable, it may luxate completely anteriorly (into anterior chamber forward of the iris), posteriorly (behind the iris into the vitreal chamber), or partially in either direction (subluxation). The major complication of lens luxation is secondary glaucoma, either as a result of anterior uveitis or mechanical obstruction of the drainage apparatus. Experience suggests early lens removal provides the best treatment for this condition, but glaucoma and retinal detachments after lens removal are still possible.

Lens Subluxation

In lens subluxation, a zone or quadrant of zonular attachment to the lens is lost, and an aphakic crescent often results (Figure 11.14). In regions where the zonules are

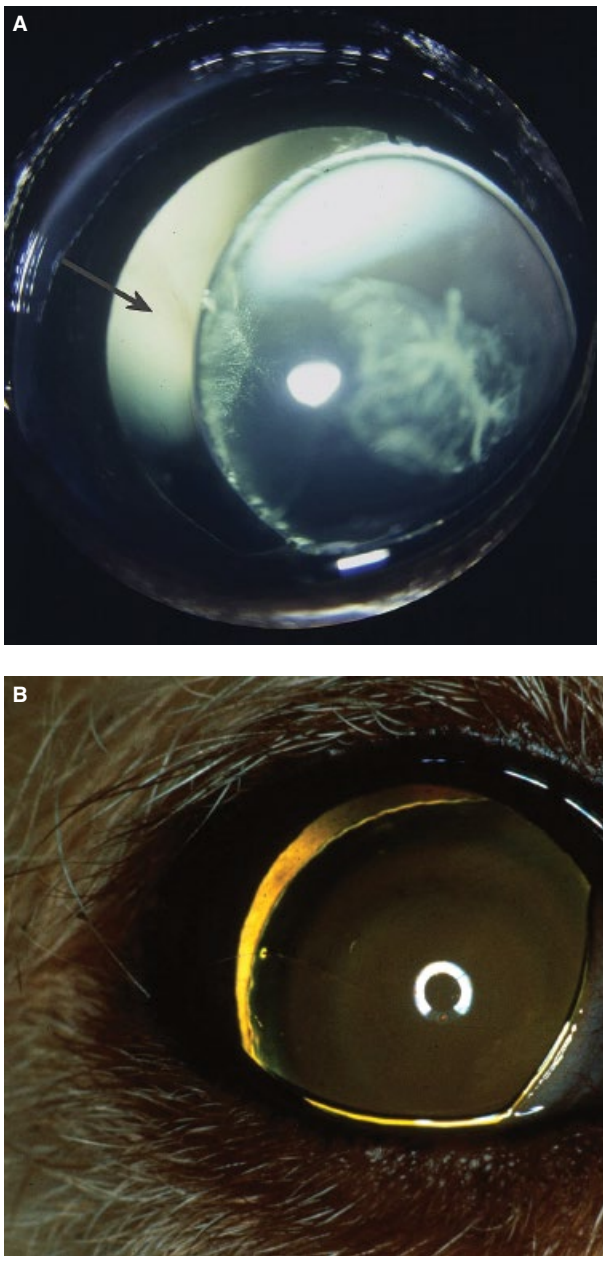


Figure 11.14 (A) Lens subluxation or the focal loss of zonulary attachments to the lens results in an unstable lens, sometimes tilted, occupying part of the pupil. The remaining pupil, devoid of lens (arrow), is referred to as aphakic crescent. Incipient cataract is present in the dislocated lens. (B) Medial subluxation. An aphakic crescent is present dorsolaterally.



Figure 11.15 (A) In anterior lens luxation all of the zonulary attachments are lost, and the lens is displaced into the anterior chamber as in this Smooth-haired Fox Terrier dog. Since the anterior vitreous is usually still attached to the posterior lens capsule, pupillary block secondary glaucoma can result. (B) Anterior lens luxation in a Chinese Crested Dog. Note the dyscoria behind the lens. (C) Same dog as in Figure 11.15B. Both lenses were luxated in the anterior chambers. The right lens was in contact with the corneal endothelium resulting in edema.

lost, the vitreous, often degenerated and appearing as white irregular strands, protrudes into the pupil and sometimes into the anterior chamber. Iridodenesis (tremor of the iris), phacodenesis (movements of the lens), a tiled lens, and an irregular depth of the anterior chamber are other common clinical signs. Secondary glaucoma can develop from intermittent pupillary blockage by the unstable lens, vitreous in the anterior chamber, iridocyclitis, or a combination thereof.

Anterior Luxation

In anterior lens luxation, all of the zonulary attachments to the lens equator are lost and the lens becomes displaced into the anterior chamber (Figure 11.15). The anterior aspects of the vitreous usually remain attached to the posterior lens capsule and can block the transpupillary flow of aqueous humor. As a result, an immediate secondary iris bombé develops that is partially masked

by the lens within the anterior chamber. Another secondary type of glaucoma can develop (malignant glaucoma or aqueous misdirection glaucoma), in which aqueous humor enters the vitreous and worsens the vitreous displacement within the pupil and anterior chamber.

The initial clinical history is eye irritation, blepharospasm, episcleral injection, and variable corneal edema. Detection of the clear lens within the anterior chamber can be easily missed; however, the pupil is usually partially distorted. Corneal edema can make this diagnosis more difficult. If the lens is cataractous, detection of anterior lens luxation is usually easier.

Posterior or Vitreal Luxation

Posterior lens luxation or intravitreal luxation results when the lens devoid of its zonular attachments moves posteriorly through the anterior vitreal membrane or face, which is liquefied or torn (Figure 11.16). The vitreous is usually degenerated (syneresis) allowing the loose lens to lie on the ventral retina. Because of the vitreal loss and liquefaction, the possibility of acute secondary glaucoma is lower than with anterior luxation because if the lens remains in the vitreous, it does not physically obstruct aqueous humor outflow. Retinal detachment as well as displacement of the lens into the anterior chamber can occur. When the vitreous has undergone syneresis, medical management with miotics to maintain the lens in the vitreal chamber can be considered. The main risk with miotic therapy is if the loose lens occludes the pupil. Mild to moderate uveitis often accompanies lens luxation, both anterior and posterior, and should be addressed with medical therapy.

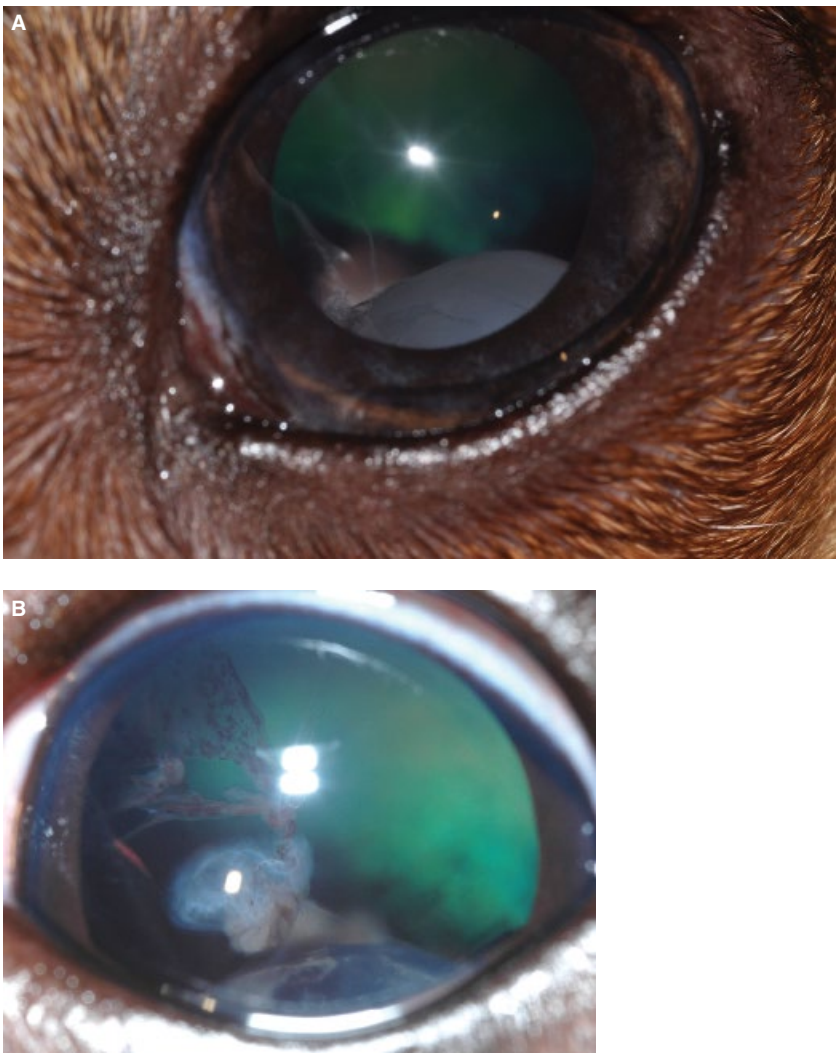


Figure 11.16 (A) In posterior luxations, all of the zonular attachments have been lost and the lens traverses the anterior face of the vitreous. Often, the lens lies on the vitreal floor and may be mobile. (B) Posterior luxation. In this example the lens is barely visible behind the pupil on the ventral floor of the vitreal chamber. A retinal detachment has occurred and hemorrhage and vitreal degeneration are present.

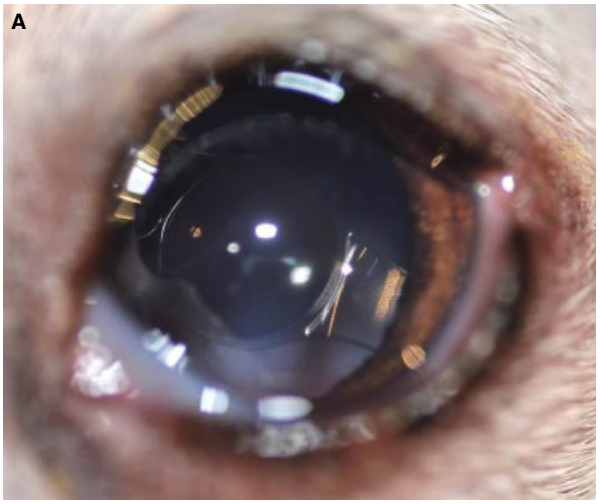






Figure 11.17 (A) Intraocular lens (IOL) in an eye following cataract removal via phacoemulsification. The central round area of the IOL is called the "optic" and the peripheral offshoots are termed the "haptics," which maintain the IOL in position within the capsular bag. (B) Pseudophakic dog 1 year after phacoemulsification cataract removal. The IOL is centered and there is mild postoperative capsular fibrosis surrounding the haptics and the periphery of the optic. There are areas of posterior synechia ventrally where the iris is adhered to the lens capsule as a result of uveitis. This eye is inflamed and hypertensive. (C) Extensive posterior synechiae and posterior capsular fibrosis in a post-phacoemulsification eye.



Figure 11.17 (*Continued*) (D) Capsular fibrosis surrounding an IOL in a Boston Terrier 2.5 years following phacoemulsification and IOL placement.

Pseudophakes

Most dogs undergoing lens extraction surgery, especially phacoemulsification, will have an intraocular lens (IOL) placed (Figure 11.17). Most IOLs are placed within the lens capsule "bag," if it is intact and stable enough in its position and orientation to accept the implant. Occasionally, when the lens capsule is absent or unstable (following intracapsular lens extraction for luxated lenses), veterinary ophthalmologists will place "sulcus" lenses that are secured to the globe at or near the equator. The presence of an IOL improves the patient's visual acuity significantly compared to the aphakic state.

12

Canine Vitreous

The vitreous is a transparent gel that fills the posterior globe, bordering the posterior lens, and the retina and optic nerve head. It is the largest structure of the eye in both size and volume (about two-thirds of the eye). Its volume varies depending of the size of the eye and the species; for instance, dog (1.7 ± 0.86 mL) and horse (26.15 ± 4.87 mL). The majority of the vitreous is formed during the development of the eye, and in the adult is normally devoid of any vasculature. It primarily consists of water (99%), collagen fibers combined with hyaluronic acid (which form into a gel), infrequent hyalocytes, and few migrating macrophages. It is attached to the ocular structures it borders near the ora ciliaris retinae (the vitreous base) and the posterior lens capsule (ligamentum hyaloideocapsulare).

The vitreous changes considerably during embryogenesis and development of the eye. It initially consists of what is referred to as the primary vitreous, which is formed prenatally and consists mainly of the hyaloid artery system. This system regresses postnatally leaving minimal remnants, Mittendorf's dot (former attachment immediately caudal of the central posterior lens capsule), and Bergmeister's papilla (former attachment to the optic nerve head). The adult gel is called the secondary vitreous and the tertiary vitreous is the lens zonules.

The vitreous gel over time and aging undergoes variable syneresis (or liquefaction). Diseases of the vitreous are mostly extensions from other adjacent tissues (inflammatory cells, foreign bodies, parasites or infectious agent, and hemorrhage).

Congenital Abnormalities

Congenital abnormalities of the vitreous are the results of persistence, hyperplasia, or proliferation of the primary vitreous (hyaloid vasculature). The extent of the opacity created in the posterior lens capsule, lens, and anterior vitreous impacts the effect on vision.

Persistent Hyaloid Remnants

Hyaloid remnants are the remaining hyaloid vasculature which initially provided the prenatal blood supply to the posterior lens (Figure 12.1; see also Figure 11.5). In the latter part of the prenatal and early postnatal eye development, after the production of aqueous humor has begun, atrophy of hyaloid vasculature occurs. Hyaloid remnants are not usually associated with visual impairment unless very extensive.

Hyaloid remnants consist of either small fibrous remnants located at the axial posterior lens capsule or larger axial cataracts involving the posterior cortex and posterior lens capsule with small but functional persistent hyaloid blood vessels that extend from the optic nerve head to the posterior lens capsule. In the former, the clinical appearance is a small, dense, round to oval, posterior capsular opacity that does not progress, and can reduce in size and density during the first 2 years of life. In the latter, the persistent hyaloid vasculature appears as prominent red vessels within the posterior lens capsule and the posterior lens cortex, with varying amounts of opacification of the lens in the same area. If the axial cataract is not large, direct inspection as well as ophthalmoscopy reveal a fibrous band or perfused red blood vessels extending from the optic nerve head to the posterior lens surface. Intralenticular hemorrhage can also occur with persistent hyaloid blood vessels.

Progression of these cataracts is unlikely and so cataract surgery is rarely performed. If cataract surgery is attempted, defects in the posterior lens capsule as well as hemorrhage from the patent hyaloid vessels are frequent intraoperative complications. Ultrasonography and Doppler ultrasonography are useful to evaluate the morphology of the posterior lens and blood vessel perfusion. If visual impairment is demonstrable during daytime or with bright illumination, topical mydriatics can be helpful.

Color Atlas of Veterinary Ophthalmology, Second Edition. Kirk N. Gelatt and Caryn E. Plummer.
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Persistent Hyperplastic Tunica Vasculosa Lentis

This condition occurs sporadically in dogs, and most often is unilateral (Figure 12.2). In some breeds, such as the Doberman Pinscher and Staffordshire Bull Terrier, the condition is inherited and affects both eyes. The inherited syndromes vary from a few axial posterior capsular fibrovascular spots to severe intralental and retrolental pigmentation and hemorrhage, microphakia or spherophakia, cataract formation, and microphthalmia. Cataract surgery is usually avoided in these breeds because of the possibility of intraocular hemorrhage, posterior capsular defects, vitreous loss, and retinal detachment. Affected dogs should not be used as breeding animals.

Vitreal Opacities

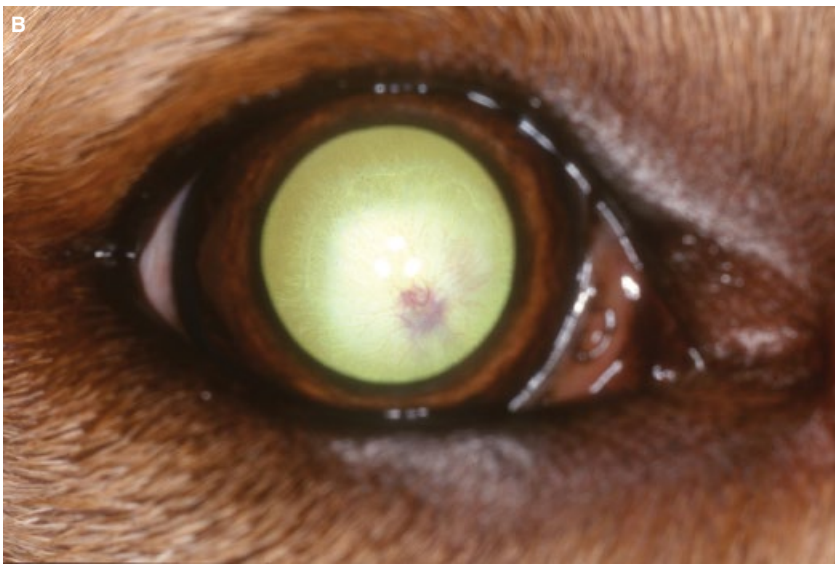
Asteroid hyalosis is a form of vitreal degeneration (Figure 12.3). In asteroid hyalosis, round to oval, gray to white opacities (asteroid bodies) are suspended within the gel vitreous. These bodies consist of calcium and phospholipids. Asteroid hyalosis does not usually produce visual impairment, but in some dogs the number



Figure 12.1 Hyaloid remnants in a young puppy. Located at the posterior pole of the lens, these hyaloid remnants appear as a white strand attached to the axial posterior lens capsule.



Figure 12.2 (A) Persistent hyperplastic tunica vasculosa lentis (PHTVL) in a young Visla dog. With miosis this opacity effectively prevents vision.



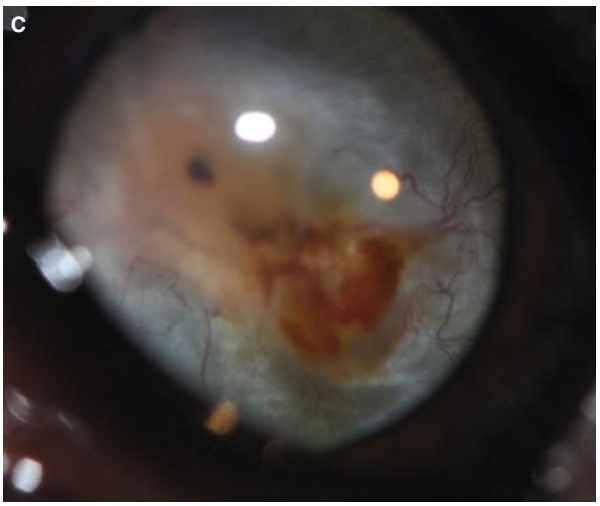


Figure 12.2 (*Continued*) (B) PHTVL in another dog. (C) Extensive PHTVL which resulted in considerable visual impairment.

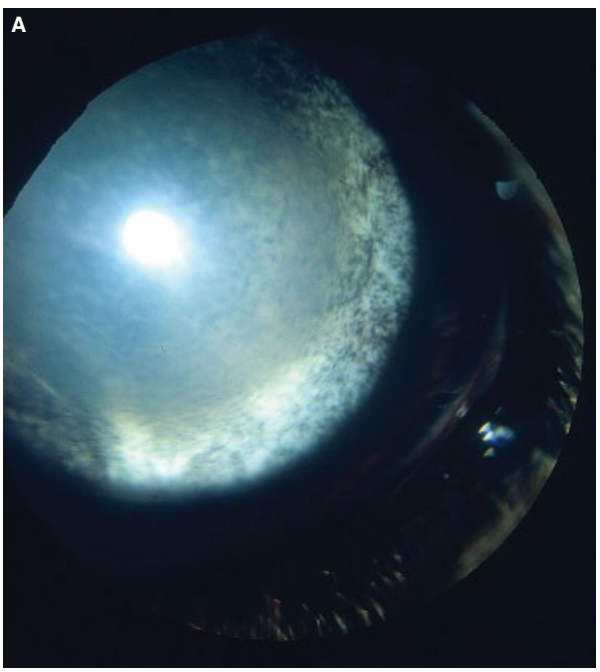
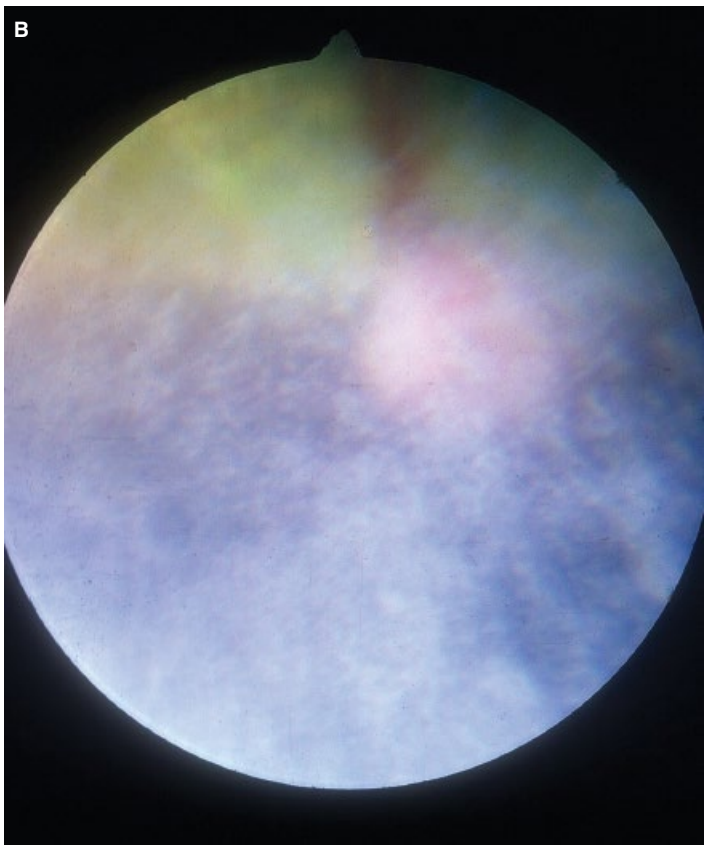


Figure 12.3 (A) Asteroid hyalosis of an aged dog. Note the numerous white to yellow minute opacities suspended within the anterior vitreous. As the vitreous is more than 10 mm in length, only some of these opacities are in focus.



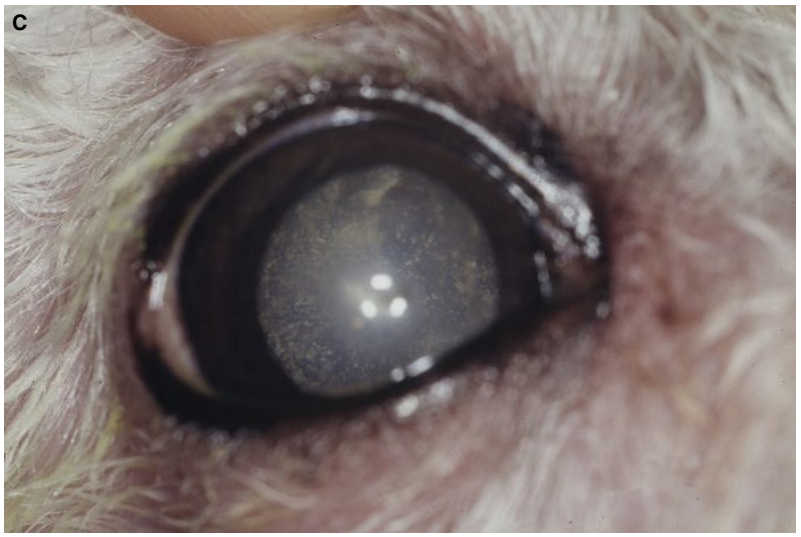




Figure 12.3 (*Continued*) (B) Asteroid hyalosis in an aged dog as seen by direct ophthalmoscopy, near to level with the ocular fundus. (C) Asteroid hyalosis in a dog. Note the focus of this image is posterior to the iris and lens. (D) Asteroid hyalosis in vitreous that has become displaced into the anterior chamber following lens extraction. The anterior chamber is deep because no lens is present to support the iris leaflets.

of asteroid bodies can be in the hundreds. There is no treatment for this condition.

Vitreal Inflammation

Vitritis is the infiltration of inflammatory cells from adjacent inflammations of the pars plana of the ciliary body (pars planitis), the retina and choroid (chorioretinitis), and posteriorly the optic nerve head (optic neuritis or papillitis), and infrequently with neoplasia (Figure 12.4). As the vitreous is normally clear, infiltration with inflammatory cells produces distinct floaters that are often adjacent to the inflamed areas or larger hazy regions. With time, focal areas of the vitreous undergo syneresis, and these floaters exhibit more motion, and gravitate to the most ventral parts of the vitreal space. Vitritis is common with systemic fungal infections (blastomycosis, coccidiomycosis, cryptococcosis) and prototheca, and these organisms can often be demonstrated on cytology following needle aspiration of the vitreous (hyalocentesis: insertion of the hypodermic needle is through the pars plana of the ciliary body to avoid a hole in the peripheral retina) performed through the sclera and pars plana ciliaris. Parasites can also infect the vitreous and sometimes break through the vitreal face, enter the posterior chamber, pupil, and anterior chamber.

Examination of vitritis includes focal illumination and magnification (slit lamp biomicroscopy) for its anterior

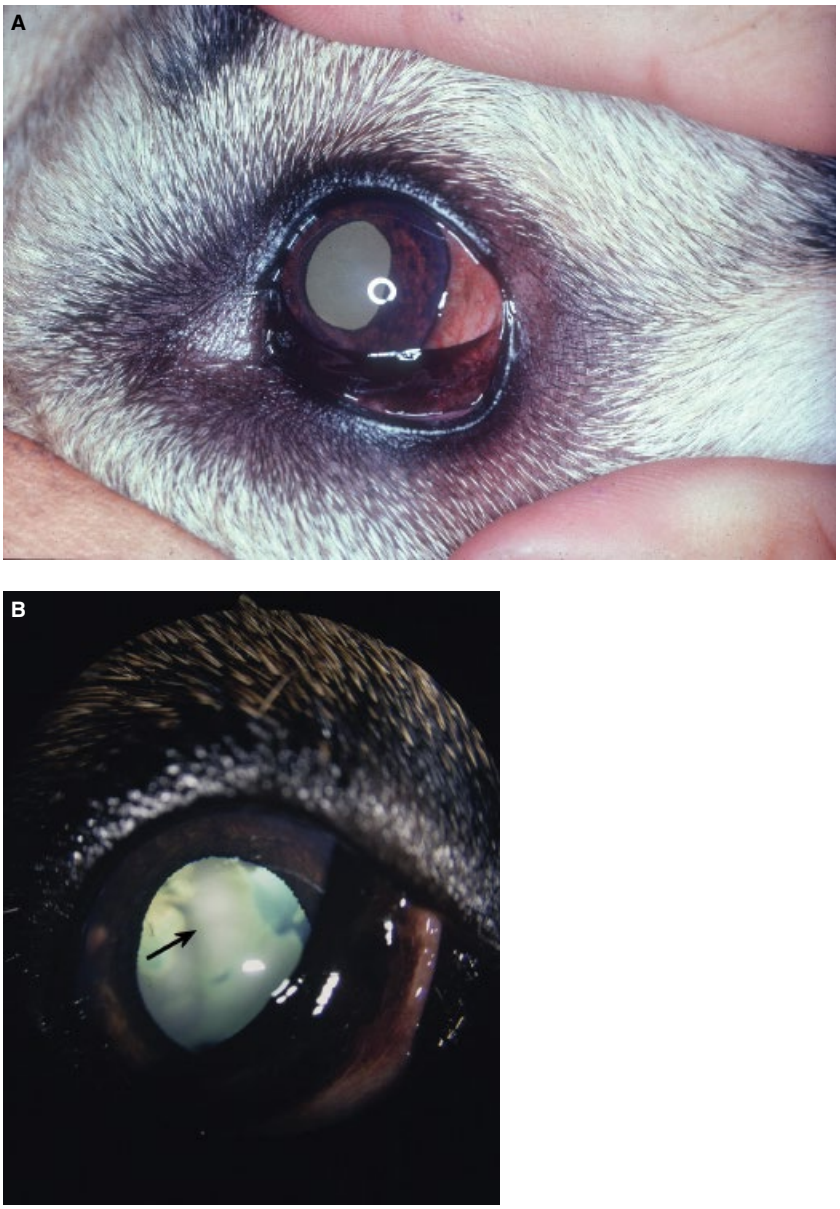


Figure 12.4 (A) Vitritis or hyalitis in a dog associated with systemic aspergillus infection. The inflammatory exudates in the vitreous produce a white ocular reflection (leukocoria) and prevent inspection of the ocular fundus. (B) A dog with fungal endophthalmitis. Note the clotted inflammatory material within the anterior vitreous (arrow).

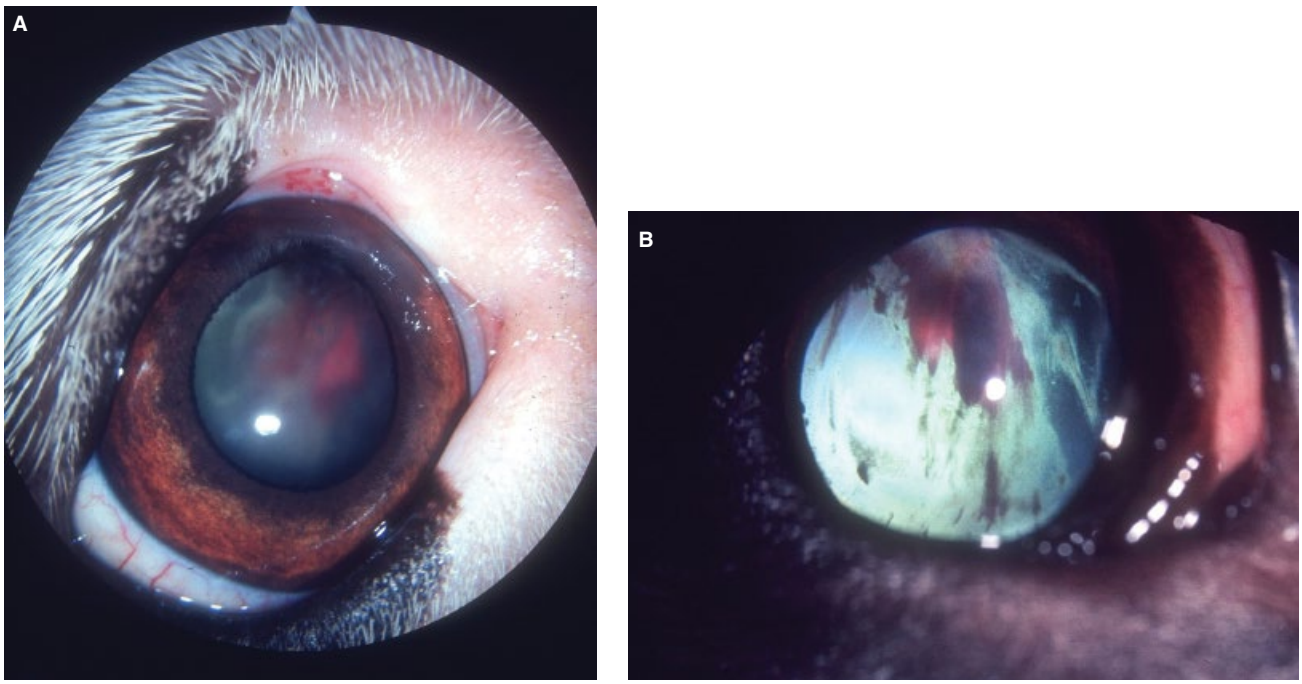


Figure 12.5 (A) Vitreal hemorrhage in a dog following leadshot penetration of the posterior segment. Note the vitreal hemorrhage suspended in the dorsomedial vitreous. (B) Resolving vitreal hemorrhage. Note the strands indicating vitreal degeneration.

portion, and ophthalmoscopy or slit lamp biomicroscopy with the Hruby lens for the posterior aspects. Vitreal paracentesis or hyalocentesis can be used to aspirate liquified vitreous and inflammatory debris. The aspirate can be examined microscopically for inflammatory and neoplastic cells as well as cultured and antibiotic sensitivities determined. Ultrasonography (b-scan) can also be used to examine the vitreous, especially when the cornea or lens is opaque and direct observation is impossible. Treatment of vitritis is directed toward the primary condition. Inflammatory cells within the vitreous are slow to resolve (generally several weeks to months).

Vitreal Hemorrhage

Vitreal hemorrhage can follow congenital eye diseases (Collie eye anomaly, retinal dysplasia), blunt or penetrating trauma, primary or secondary intraocular neoplasia, systemic hypertension, blood clotting disorders, and can be spontaneous in the Beagle (Figure 12.5). Hemorrhage can be suspended within the vitreous gel, develop clots within the vitreous body, or occur between the "solid" posterior vitreous and anterior limiting membrane of the retina ("keelboat" hemorrhage). Vitreal hemorrhage is either clotted (associated with trauma and neoplasia, or a resolving insult) or unclotted (often persistent or active disease, as with Collie eye anomaly, retinal detachment, systemic hypertension, and blood clotting disorders).

If possible, the cause for the hemorrhage should be ascertained and treatment directed to its cause. Exit of blood from the vitreous may require many weeks to months because erythrocytes must degenerate and be removed by macrophages. Residual ferrin can persist indefinitely within the gel vitreous. Usually, no treatment for intravitreal hemorrhage is administered. Sequelae of vitreal hemorrhage include syneresis, and the formation of fibrinous to fibrous membranes and traction bands.

13

Canine Ocular Fundus and Optic Nerve

Diseases of the ocular fundus include those that affect the retina, choroid, and optic nerve head (also referred to as the posterior segment). They are congenital or developmental in origin, or inflammatory, traumatic, neoplasitic, or degenerative. For examination purposes, the canine ocular fundus is divided into tapetal fundus, nontapetal fundus, optic nerve head, and the retinal vasculature. A complete fundic examination evaluates the size and shape of the optic nerve head, the perfusion of the retinal arterioles and venules, and the state and position of the retina. There are many variations in the normal ocular fundus that will become apparent on ophthalmoscopy. It is important to perform routine ophthalmoscopy on all patients so that these variations can be documented, which will facilitate the early detection of developing pathology.

Recent advances in the development of specific DNA mutation tests for retinal diseases in purebred dogs have markedly increased our knowledge of these diseases and our ability to detect carrier and affected dogs prior to the onset of vision impairment.

Normal Ocular Fundus and Variations

The canine ocular fundus has considerable variations, and this often confuses the novice attempting to learn ophthalmoscopy at the same time as diagnosing fundus disease. Those breeds with blue irides or heterochromia iridis, and breeds with merling (or dappling) can have markedly different appearances of the ocular fundi between fellow eyes but may nevertheless be normal in both eyes (Figure 13.1).

The tapetum develops during the first 16 weeks of life through a series of color changes (black or gray to lavender or purple to blue followed by the adult coloration of yellow–orange or yellow–blue–green). The tapetum is a roughly triangular area in the dorsal ocular fundus that is thought to assist vision in reduced illumination. The retinal pigment epithelium in the tapetal fundus has limited melanin in its cytoplasm, permitting the deeper tapetal layer (within the anterior choroid) to be visible behind the retina. In the merled and toy breeds, the tapetal fundus can be markedly reduced in size, occur as multiple tapetal "islands," or be totally absent.

The nontapetal fundus is normally dark brown to black and derives its color from the melanin granules within the retinal pigment epithelium. When heterochromia iridis is present, the nontapetal fundus is only lightly, or partially, pigmented, permitting visualization of the deeper choroidal blood vessels ("tigroid" nontapetal fundus), or nonpigmented (allowing visualization of the deeper choroidal blood vessels against the white scleral background). The junction between the nontapetal and tapetal fundus is often not a distinct line, but a gradual transition in the nontapetal pigmentation and increased thickness and color of the tapetum. Within the tapetal fundus, slightly temporal and dorsal to the optic disc is the area centralis (a cone-rich area).

The optic nerve head (also called the papilla or optic disc) can be located within the tapetal fundus, within the nontapetal fundus, or at the junction of the two. When the optic nerve head is in the tapetal fundus, it is often surrounded by a pigment ring. Its surface is myelinated, and from its periphery arise the primary retinal arteries and veins. The shape of the pink to white disc ranges from round to oval to quite irregular. There is often a central depression, the physiologic cup, which was formed by the prenatal atrophy of the hyaloid system.

The canine ocular fundic vasculature is holangiotic, and consists of about 15–20 arterioles, and 3–4 primary larger arteries and veins. The arterioles are lighter red while the veins carrying deoxygenated blood are darker red. Sometimes, a partial to complete venous circle is apparent on the disc's surface.

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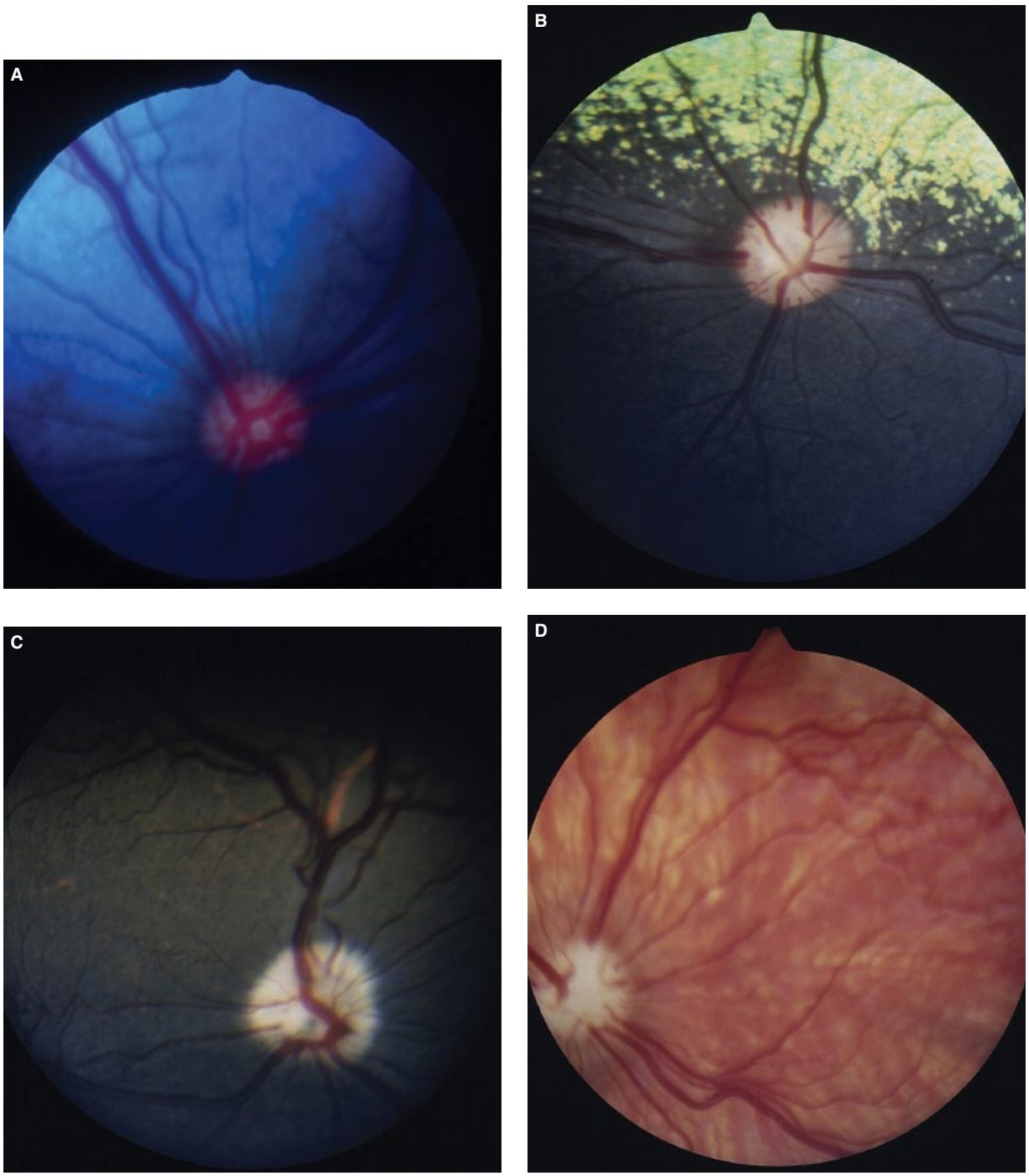


Figure 13.1 There are considerable normal variations of the ocular fundus and optic nerve head or disc variations among more than 250 recognized dog breeds. Coat color and degree of pigmentation have a significant impact on the appearance of the fundus. (A) Ocular fundus of a 10-week-old puppy. The tapetal fundus is blue and will continue to change color until eye development is complete (about 16 weeks of age). (B) The most common ocular fundus appearance in the dog. The dorsal triangular tapetal fundus is yellow–green; the nontapetal fundus is very dark brown or nearly black; the optic disc is at or slightly below the junction of the tapetal and nontapetal fundi; and the retinal blood vessels emerge from the disc's surface and periphery. Position of the optic disc varies by breed and may be present totally within the tapetal fundus, the nontapetal fundus, or at their junction. (C) Completely pigmented ocular fundus, without any observable tapetal fundus in a mature Boston Terrier. A light orange blood vessel adjacent to the dorsal retinal blood vessels is within the chorioid. (D) A sub-albinoid ocular fundus in a dog with heterochromia iridis. No tapetum is observable. Sometimes some pigmentation will be present in the nontapetal fundus. Because of the paucity of pigmentation, the deep choroidal larger diameter blood vessels can be clearly visualized.

Congenital Diseases

Congenital diseases of the canine fundus are not uncommon and occur mostly in selected breeds. Because the canine ocular fundus does not reach maturity until about 15 weeks of age, ophthalmoscopic examinations in puppies at weaning time (about 6–8 weeks of age) does not allow detection of all congenital anomalies. Subsequent re-examination at 4–6 months of age, when congenital defects are more apparent, is advisable. The continuing development of DNA or genetic tests has been beneficial to confirm clinical diagnoses as well as detect carriers of selected diseases, to permit rationally programmed elimination of a disease. Total reliance on DNA testing is not recommended as periodic ophthalmic examinations are indispensable to detect new variations of the disease or a new mutation.

Collie Eye Anomaly

Collie eye anomaly (CEA) is one of the most investigated posterior segment anomalies in the dog. In the 1960s and early 1970s, this anomaly was also called posterior scleral ectasia or posterior staphyloma. This defect appears inherited as an autosomal recessive trait; the optic nerve defects (optic nerve colobomas or "pits") can be transmitted as an autosomal dominant trait. Recently, a study in Sweden challenged the mode of inheritance.

This disease affects Rough and Smooth Collies (80–90% of individuals of these breeds are affected), Shetland Sheepdog (5% in the USA to 60% in the UK), Border Collie (<5%), Australian Shepherd (<5%), and, more recently, has been reported in the Nova Scotia Duck Tolling Retriever and the Lancashire Terrier.

By ophthalmoscopy, the ocular fundus disease is characterized as normal, "go normal," or affected. The "go normals" are adult dogs that as puppies had very small focal areas of choroidal hypoplasia that eventually pigmented and now appear normal (although these dogs breed as affected animals). CEA manifests with blood vessel tortuosity, focal choroidal hypoplasia lateral to the optic disc (100%), posterior colobomas (papillary or peripapillary staphylomas or "pits") (10–20%), retinal detachments (5%), and intraocular hemorrhage (Figure 13.2).

Vermiform streaks or retinal folds can also affect the ocular fundus but appear unrelated to CEA but instead to unbalanced growth between the sclera and the inner retina. They resolve (or disappear) with maturity. They appear as gray to white irregular lines within the retina.

Recommendations for the past 45+ years have been to breed only mildly affected dogs (with only choroidal hypoplasia) and to avoid use of the more severely affected Collies (those with colobomas or staphylomas). Many progressive Collie breeders are now breeding normal eyed Collies (many are carriers) with the eventual goal of eliminating the disease from the breed. In other breeds with this defect affected dogs should be neutered, and both parents of affected offspring should not used for breeding. There is a commercially available DNA test for CEA (CEA-CH: intronic mutation in *NEHJ1)* which

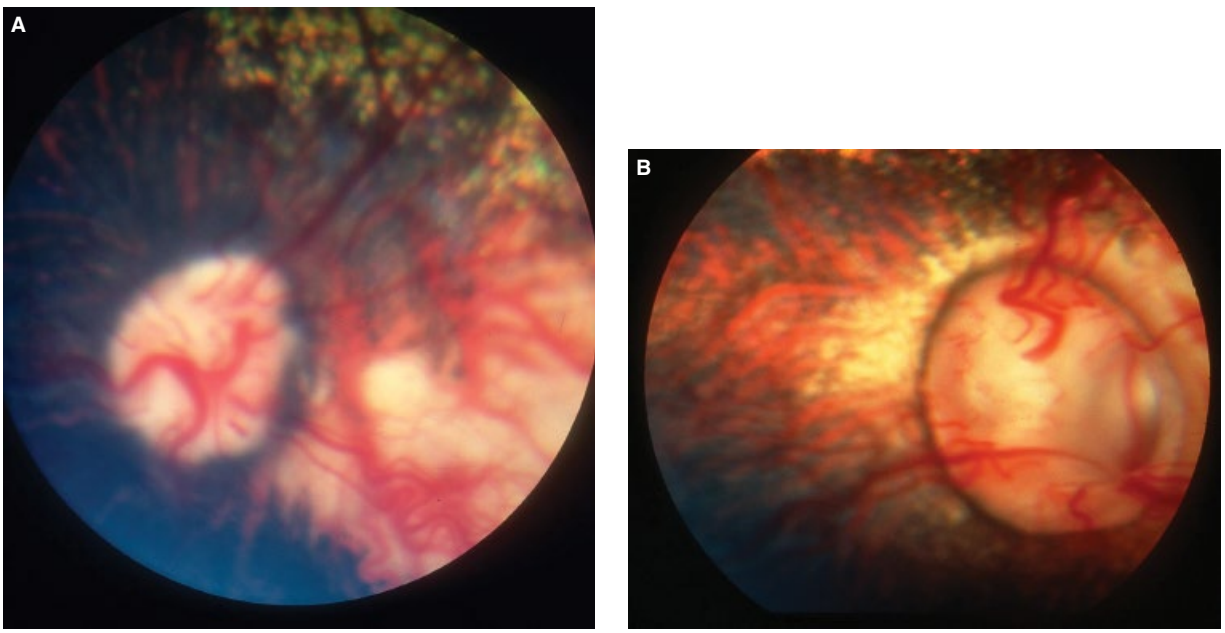


Figure 13.2 Collie eye anomaly (CEA). (A) The basic abnormality of Collie eye anomaly is focal choroidal hypoplasia lateral to the optic disc. (B) The more severe defect, optic nerve coloboma, affects about 20–30% of Collies.

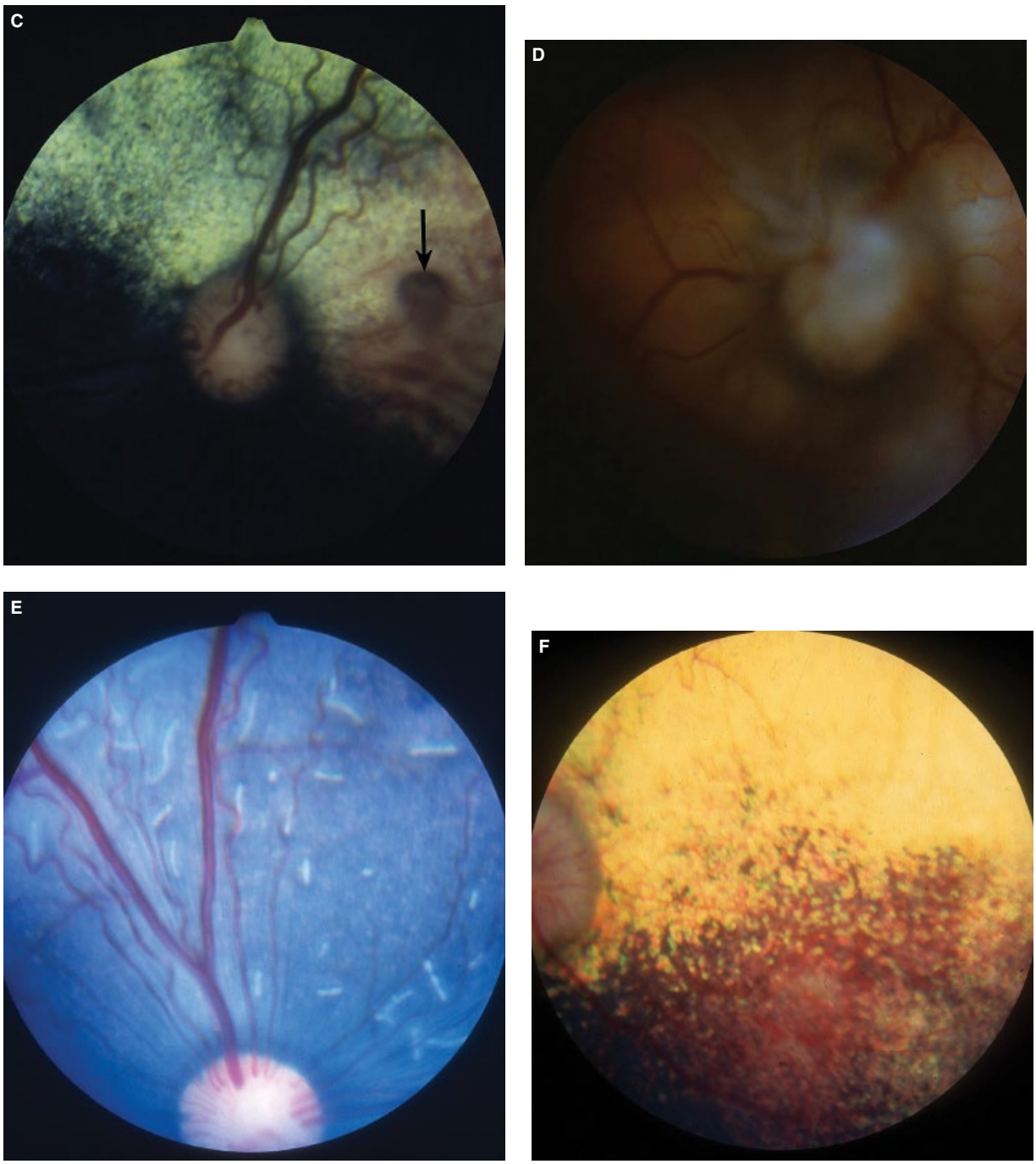


Figure 13.2 (*Continued*) (C) Colobomas (arrow) can also occur near but distinct from the optic disc. Note the focal choroidal hypoplasia area. (D) Retinal detachments develop in about 2–5% of affected Collies, usually near the optic disc and often associated with optic disc colobomas. The detached and edematous retina is protruding forward of the optic disc. (E) Vermiform streaks are occasionally observed in young Collie puppies. These streaks represent an imbalance of growth between the retina and outer choroid and sclera. They disappear over several weeks. (F) Young (8 weeks) Collie with very small choroidal hypoplasia. This lesion is quite small and a candidate for "go-normal." As adults, the "go-normal" Collies appear normal but breed as CEA affected dogs. This puppy also has early progressive retinal atrophy (rod–cone dysplasia) and is night blind.

helps detect the carriers and "go normal" dogs, to facilitate a CEA-free breeding program (for more information see Optigen, www.optigen.com and other sites).

Retinal Dysplasia

Retinal dysplasia in many breeds seems to be increasing, and is of increasing concern for the conscientious purebred dog breeder and fancier. The causes of retinal dysplasia are genetic or viral (herpesvirus), or the result of irradiation, certain drugs, hypovitaminosis A, or intrauterine trauma (Figure 13.3). There are three categories of retinal dysplasia: (i) focal or multifocal; (ii) geographic; and (iii) the most severe, total retinal dysplasia with retinal detachment.

Breeds commonly affected with focal or multifocal retinal dysplasia include American Cocker Spaniel, Beagle, Labrador Retriever, Rottweiler, and Yorkshire Terrier. This form of retinal dysplasia does not produce clinical vision impairment. Both the tapetal and nontapetal fundi are affected. In the tapetal fundus, lesions appear as hyperreflective to pigmented "Y," "X," or irregular streaks or folds, and most frequently affect the area dorsal to the optic nerve head. In the nontapetal fundus, affected areas are gray to white and similar in shape.

Those breeds commonly affected with geographic retinal dysplasia include the Cavalier King Charles Spaniel, English Springer Spaniel, and the Labrador Retriever. Affected dogs have normal clinical vision or demonstrate vision impairment to blindness. Blind dogs can also have nystagmus, microphthalmia, intraocular hemorrhage, cataract formation, and retinal detachment. Less affected dogs show focal areas of retinal dysplasia, most frequently in the central tapetal fundus, which by ophthalmoscopy appear as large hyper- and hyporeflective areas with variable and often dense pigmentation. The retinal blood vessels can be attenuated in the involved areas. There is no treatment, and affected dogs should not be used as breeding animals.

Total retinal dysplasia with retinal detachment (or nonattachment) affects the Bedlington Terrier, Akita, Chow Chow, Doberman Pinscher, Labrador Retriever, Sealyham Terrier, and Samoyed. Affected puppies usually show esotropia, smaller than normal palpebral fissures, dilated pupils, smaller than normal eyes, protruding nictitating membranes, rotary nystagmus, blindness, and intraocular hemorrhage. They will often have microphthalmia and cataract formation in addition to detachments and posterior segment hemorrhage. The Labrador Retriever and Samoyed breeds, which have the more severe forms of this disease, can also have skeletal abnormalities. Affected dogs as well as the parents of affected puppies should not be used as breeding animals. DNA tests are available for many of these mutations.

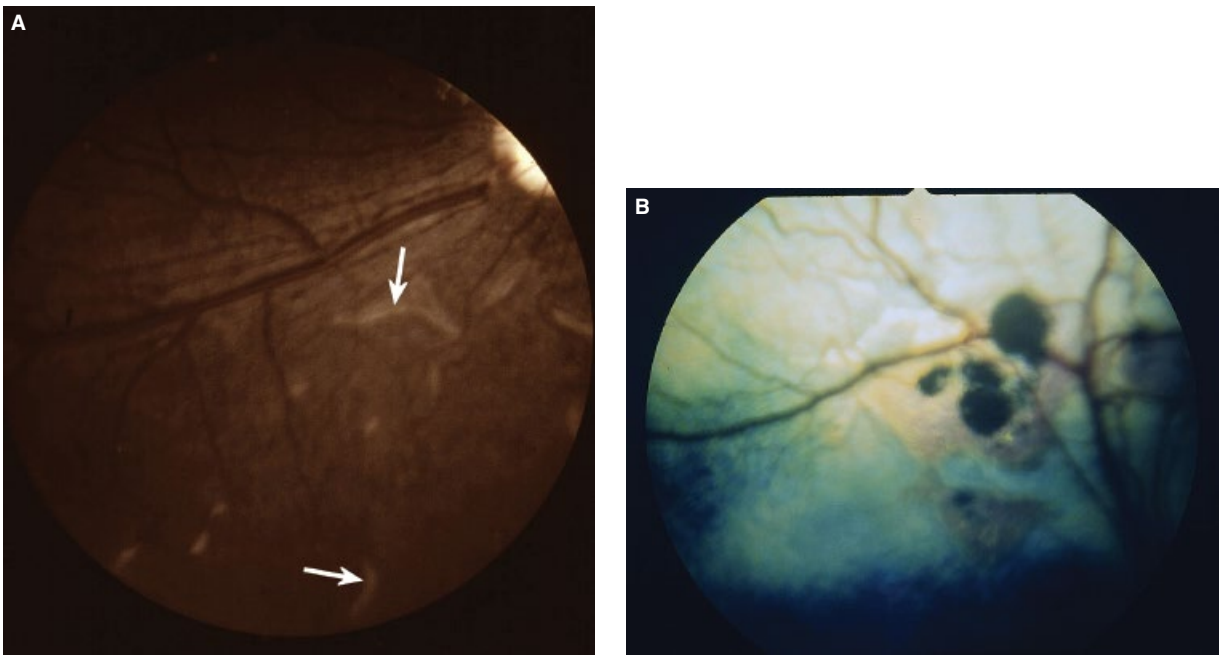


Figure 13.3 Retinal dysplasia occurs in several different forms in specific breeds (focal or multifocal, geographic, or total with retinal detachment), and may not impair vision. (A) Focal retinal dysplasia in the American Cocker Spaniel. The non-pigmented areas (arrows) are located within the nontapetal fundus. Vision appears normal. (B) Geographic retinal dysplasia in a Labrador Retriever. The dysplastic area is dorsal of the disc and contains areas of hyperreflectivity and pigmentation, and can be confused with previous chorioretinitis. Vision can be normal or subtle visual impairment can be demonstrated for visually critical tasks.

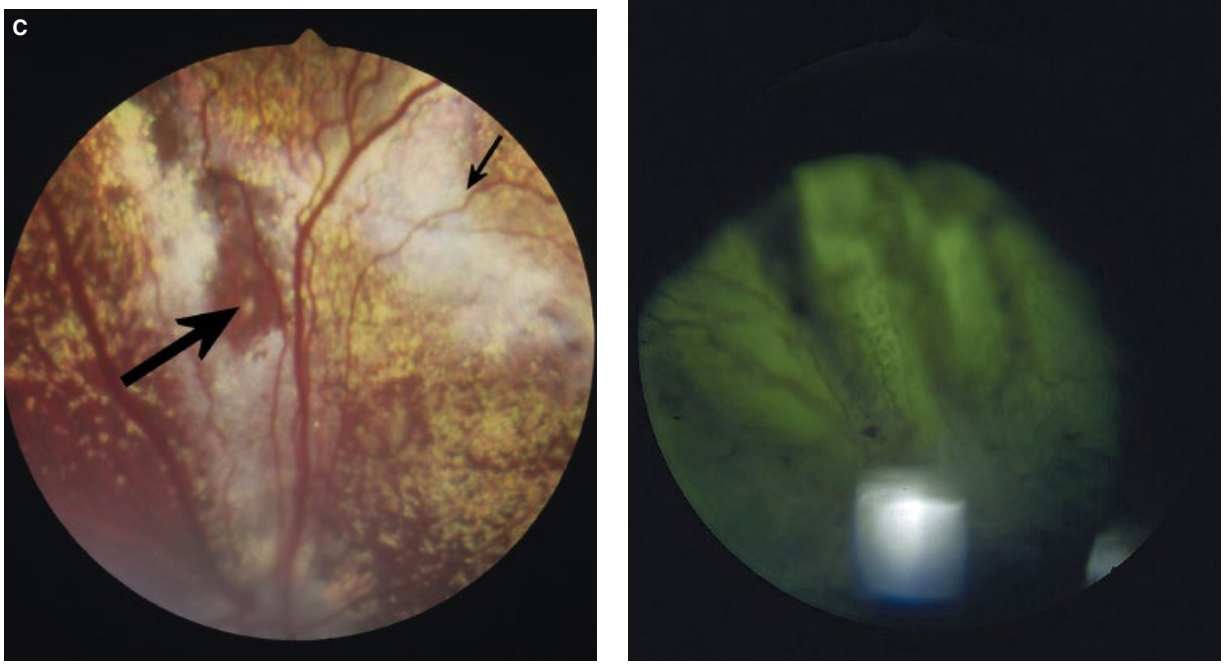


Figure 13.3 (*Continued*) (C) Geographic retinal dysplasia in an English Springer Spaniel. Located in the tapetal fundus and above the optic disc, the dysplasia has both hyperreflective (small arrow) and pigmented (large arrow) areas. Often no visual impairment can be detected. (D) Total retinal dysplasia in a Bedlington Terrier. The condition affects the entire retina, and has caused complete retinal detachment (or retinal nonattachment). Note the anterior portions of the retinal detachments are immediately behind the posterior lens. Affected dogs are blind.

Retinal Photoreceptor Dysplasia and Degeneration (Retinal Atrophy)

Progressive retinal atrophy (PRA) is a leading cause of inherited blindness in the dog. Affected dogs develop gradual visual impairment and eventual blindness. First reported in the Gordon Setter in Sweden in 1911 as retinitis pigmentosa, PRA now affects more than 60 breeds of dogs (Figure 13.4). PRA affects the photoreceptors (rods and cones) and has been divided into those conditions that occur during development (rod–cone dysplasia) and those that develop later in life (rod–cone degeneration after development of the rods and cones is complete), and those wherein the rods are predominantly affected first or those initially affecting the cones. These two distinctions, based on electroretinographic and ultrastructural studies, also help predict the time of onset. The rod–cone dysplastic types affect younger animals and the degenerative rod–cone diseases involve older dogs. Experimental studies indicate serial electroretinography can detect the disease months to years before clinical decline in vision develops.

Retinal photoreceptor dysplasia has been investigated and characterized to date in the Alaskan Malamute (cone only), Belgian Shepherd, Collie, Irish Setter, Miniature Schnauzer, and Norwegian Elkhound (two types). Rod–cone degenerations have been investigated in the Akita, American Cocker Spaniel, English Cocker Spaniel, Labrador Retriever, Miniature Long-haired Dachshund, Papillon, Portuguese Water Dog, Siberian Husky, Tibetan Spaniel, Tibetan Terrier, and the Toy and Miniature Poodles.

The clinical history for both types of PRA are similar night vision impairment and blindness that progresses to day vision impairment and eventual blindness. The rate of vision decline is somewhat variable, but in general the rod–cone dysplasia group progresses at a faster rate. Secondary cataracts can occur but appear somewhat breed-specific. Toy and Miniature Poodles with PRA often have secondary cataracts.

The ophthalmoscopic findings in PRA are reasonably consistent among the different breeds. The earliest changes involve the tapetal fundus, first affecting the peripheral fundus, and are characterized by a change in reflectivity (grayish discoloration). Slight vascular attenuation of the peripheral retinal vessels in this area also occurs. Generally, the dog has night vision problems at this stage. Drug-induced mydriasis is necessary to observe these peripheral changes.

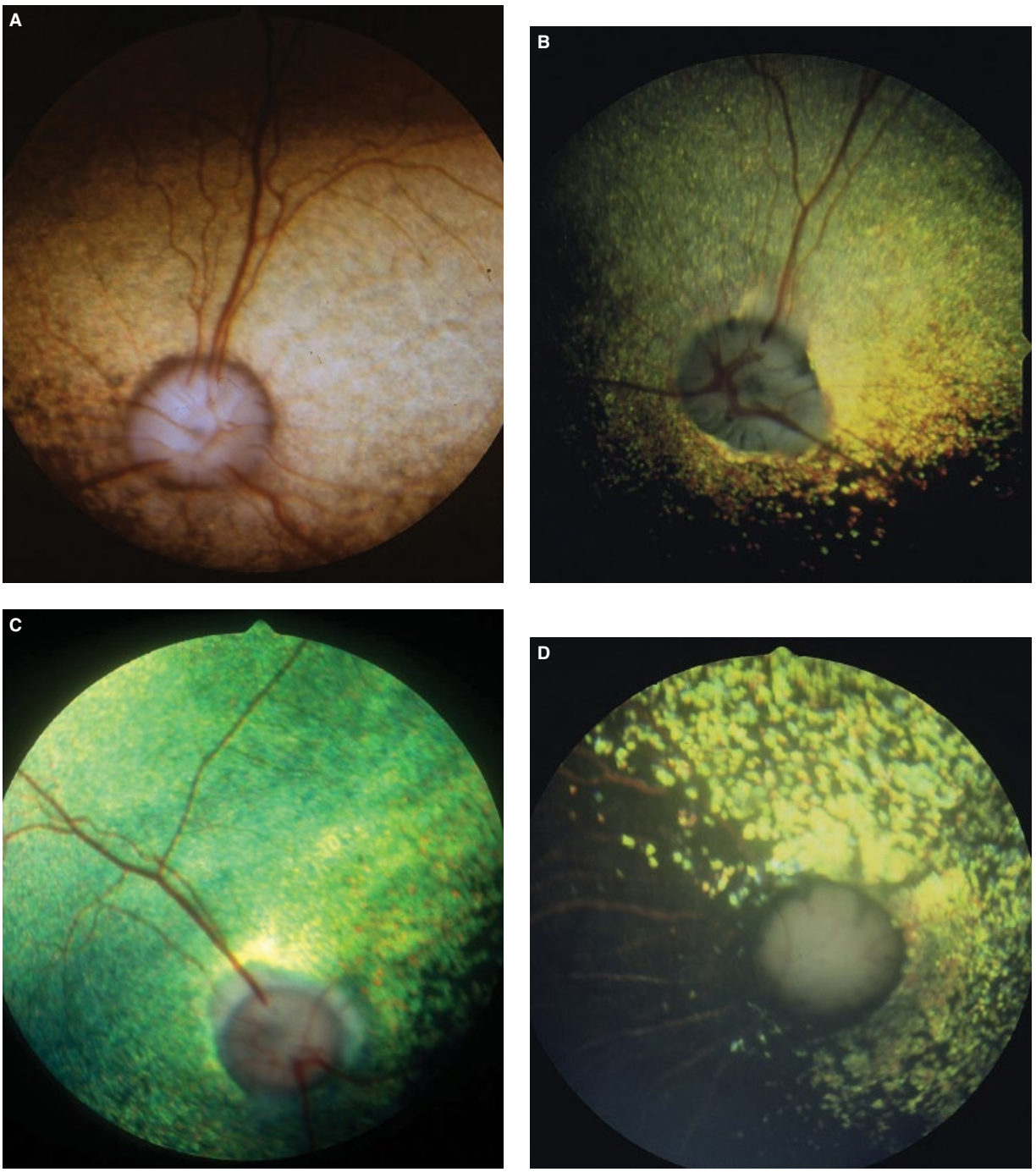


Figure 13.4 The fundus appearance of progressive retinal atrophy (PRA) is remarkably similar among the different affected breeds of dogs (even though the pathogenesis varies), but varies with the different stages of the disease. The age of onset of impaired vision varies by breed, as does the rapidity of the disease onset and its progression to blindness. (A) Early PRA in a 4-year-old Miniature Poodle. Note the reduction in the number and diameter of the retinal blood vessels and the mottling of the tapetal fundus. (B) Moderately advanced PRA in a 3-year-old American Cocker Spaniel. Note the further reduction in the retinal vasculature, increased tapetal reflectivity, and early optic disc degeneration. (C) Moderately advanced PRA. Note the diffuse hyperreflectivity of the tapetum, the vascular attenuation and the loss of myelin from the optic disc. (D) Advanced PRA in a 6-year-old Miniature Poodle. Increased tapetal reflectivity, loss of retinal vasculature, loss of pigmentation in the nontapetal fundus, and optic nerve degeneration are present. Choroidal vasculature is visible in the non-tapetal fundus.



Figure 13.4 (*Continued*) (E) Advanced PRA in a 7-year-old Miniature Poodle. Optic nerve head atrophy is also present.

In the moderate stage of the disease, the color changes in the tapetal fundus become generalized as does the vascular attenuation. The tapetal reflectivity is increased and the nontapetal fundus shows some loss of pigmentation. Changes in the optic nerve head also occur and include the loss of myelin (usually the nerve head becomes more circular in shape) and vascular attenuation (reduced numbers and diameters of retinal arterioles and veins). At this stage the dog generally shows complete night blindness as well as early daytime vision problems.

In the advanced stage of PRA, the funduscopic changes include marked increased tapetal reflectivity, severe retinal blood vessel attenuation and lack of perfusion, and reduced pigmentation of the nontapetal fundus. The optic nerve head shows advanced atrophy with reduced diameter, loss of myelin, and few blood vessels of reduced diameter on its surface.

There is no medical treatment for PRA. Affected dogs as well as parents of affected animals should be removed from the breeding pool. As PRA appears inherited as an autosomal recessive trait (except in the Siberian Husky where it is a sex-linked disease with mainly males affected), removal of affected dogs appears to be the most important step in reducing the disease frequency within certain breeds. DNA tests have been developed for a large number of breeds, and can be helpful in identifying and eliminating carrier dogs. New DNA tests are being developed (for the latest information see www.optigen.com or other sites). DNA or gene therapy has also been documented (as a model to treat similar or identical diseases in humans) in some PRA dogs.

Retinal Pigment Epithelium Dystrophy

Retinal pigment epithelium dystrophy (RPED) was formerly called central progressive retinal atrophy; however, the retina outer photoreceptors appear normal initially but the retinal pigment epithelium (RPE) is abnormal, so the condition is primarily and initially a defect in the RPE (Figure 13.5). RPED has been reported in the Labrador Retriever, Golden Retriever, Briard, Border Collie, Rough and Smooth Collies, Shetland Sheepdog, English Cocker Spaniel, English Springer Spaniel, Chesapeake Bay Retriever, among other breeds. Based on studies in the 1970s, RPED was reported as an inherited disease in the Labrador Retriever, but more recent studies suggest alterations in vitamin E levels may be involved in this disease in some breeds (Briard and English Cocker Spaniel), and dietary levels as well as metabolic disorders involving the liver may be the primary etiology. Affected Briards show hyperlipidemia, and low levels of vitamin E and taurine.

The clinical history is variable. Vision impairment varies, and in its early stages RPED may be diagnosed first by ophthalmoscopy. Working dogs can demonstrate vision impairment for stationary objects but have normal vision for moving objects. Vision can be better at night than during the day. Secondary cataracts can occur late in the disease.

Affected individuals have brown to gold color irregular size and shape spots scattered throughout the tapetal fundus, visible on ophthalmoscopy. These brown foci (thought to be hypertrophic and hyperplastic RPE filled with lipofuscin) increase in size and eventually involve the entire tapetal fundus. Some increased reflectivity occurs around each brown focus. Eventually, as considerable numbers of RPE cells are lost, a generalized reflectivity results (as in advanced PRA). Similar changes affect the RPE in the nontapetal fundus with focal variations of pigmentation. Degenerative optic nerve changes occur later in the disease.

A similar disease in Briards in Sweden has been called stationary night blindness (although some progression can occur over years). Affected puppies are night-blind and have nystagmus. By ophthalmoscopy, a subtle tapetal color change occurs with the development of gray to white spots. In the USA this disease was initially called lipid retinopathy. For additional information on available DNA tests see www.optigen.com.

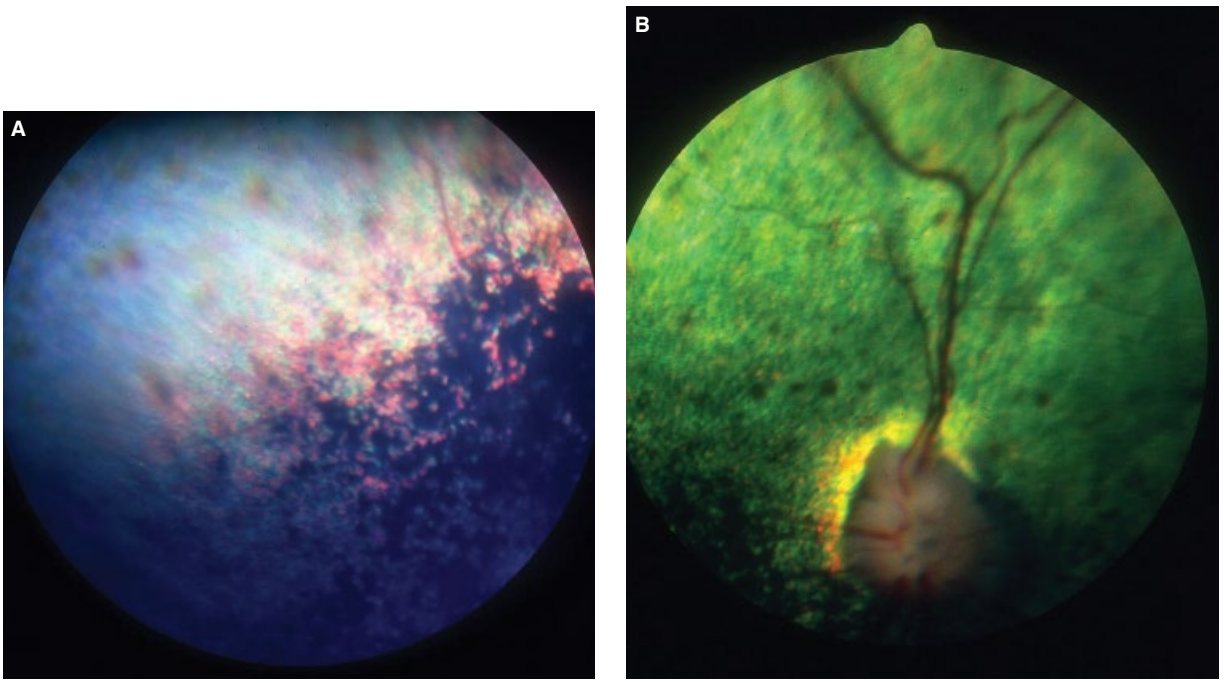


Figure 13.5 Retinal pigment epithelium dystrophy (RPED) is less common than progressive retinal atrophy, and affects primarily the retinal pigment epithelium with secondary effects on the rod and cone photoreceptors (and vision). Like PRA, the funduscopic appearance of RPED varies depending on the stage of the disease. (A) Early RPED is characterized by the development of focal brown to black areas within the tapetal fundus. At this time, the dog may not demonstrate vision impairment. (B) As RPED progresses, the pigmented spots in the tapetal fundus merge and become less obvious. Increased tapetal reflectivity, reduced numbers and diameters of retinal blood vessels, and optic nerve degeneration follow. Vision problems are common with the advanced form.

Retinitis, Chorioretinitis, and Retinochoroiditis

Inflammation of the retina and choroid is not uncommon in the dog, but often does not produce demonstrable signs of vision impairment until either the entire retina is involved or detached, or the optic nerve becomes affected (Figure 13.6). Most cases of chorioretinal inflammation are caused by systemic diseases and both eyes are often affected. A considerable amount of the retina of both eyes must be involved for visual impairment to be obvious; in contrast, relatively small lesions of the optic nerve cause pupillary and visual signs. The causes of chorioretinitis are infectious (viral, rickettsia, mycotic, algal, protozoal, parasitic), neoplastic, or immune-mediated. Often, affected dogs have additional clinical signs of ophthalmic disease (i.e., anterior uveitis, glaucoma, keratoconjunctivitis sicca) or systemic disease (see also Figures 3.20, 3.21, 5.19, and 10.7). .

Ophthalmic diagnostics vary somewhat by disease, but include ophthalmoscopy, ultrasonography (valuable when the interior of the eye cannot be visualized), and anterior chamber and vitreal paracentesis (culture/cytology), as well as a general medical workup (complete blood count, serum biochemical profile, urine analysis, and thoracic and abdominal imaging). Additional diagnostic tests are indicated depending on these baseline results.

The ophthalmoscopic appearance of chorioretinitis varies between the tapetal and nontapetal fundi, as well as by duration of disease (acute or chronic). The neurosensory or inner retina is normally completely transparent except for its vasculature, but with active or acute inflammation becomes translucent to opaque.

In acute chorioretinitis, inflamed areas in the tapetal fundus appear raised, translucent to opaque, and irregular in size and shape, often with fuzzy or indistinct margins. If the ophthalmoscope viewing light is bright, these areas can be masked and therefore missed. Hence, ophthalmoscopy with both normal illumination as well as decreased illumination of the tapetal fundus is recommended. With chronicity or inactive (healed) inflammation, retinal degeneration can appear as irregular areas with sharp edges, occasional focal vessel attenuation, focal hyperreflectivity, and variable pigmentation changes.

In acute inflammations of the nontapetal fundus, the changes of chorioretinitis are easier to detect. Contrasted against the deeper dark pigmentation, the areas of active or acute chorioretinitis appear as white to gray, raised,

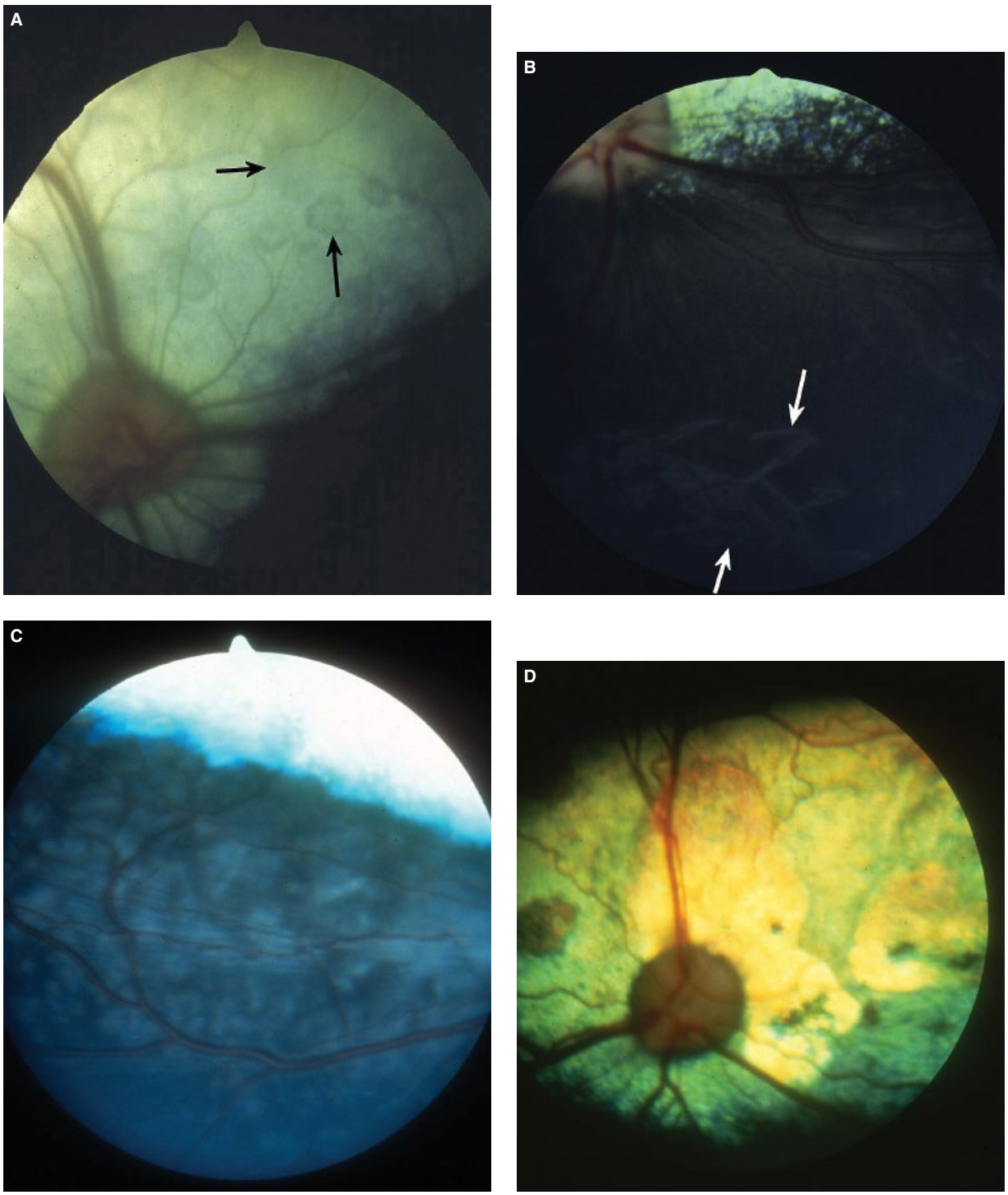


Figure 13.6 Inflammations of the retina and choroid vary by the inciting cause and the stage of the inflammatory process: acute, subacute, chronic, and end-stage (degeneration). (A) Acute retinochoroiditis in a young dog associated with canine distemper. Note the raised translucent areas in the tapetal fundus (arrows). (B) Active chorioretinitis associated with migration of fly larvae (order *Diptera*; ophthalmomyiasis posterior). Note the focal white inflamed areas suggesting migrating paths of this parasite (arrows). (C) Active chorioretinitis in a dog of unknown cause. Note how the focal inflammations alter the blood vessels' courses. (D) Chronic chorioretintis or chorioretinopathy in the tapetal fundus of a dog. Note the increased reflectivity (retinal loss/thinning) in the affected area and the pigment clumping. There is also a loss of retinal vasculature in the affected areas.

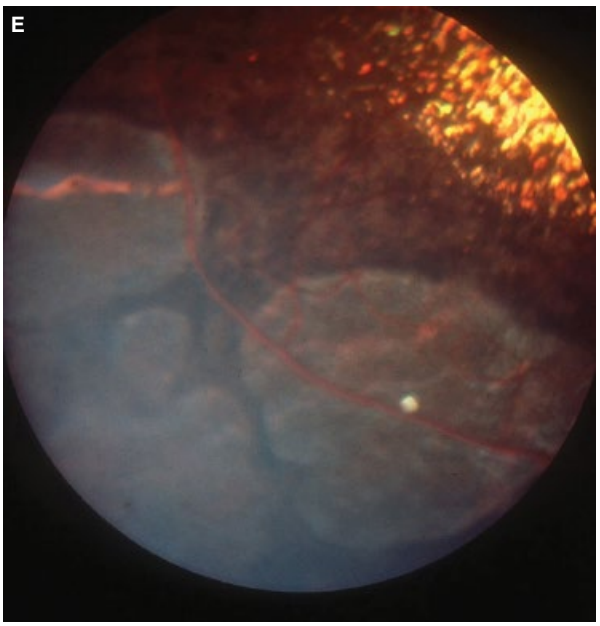


Figure 13.6 (*Continued*) (E) Inactive (healed) chorioretinitis or chorioretinopathy in the nontapetal fundus in a dog suspected of having distemper months previously. The discrete areas of reduced pigmentation and surrounding pigment proliferation have distinct margins.

irregularly sized and shaped areas with fuzzy margins. Chronic or inactive lesions show loss of or increased pigmentation with sharp margins and focal blood vessel attenuation in the same area.

Treatment is directed at the inciting cause. Often, focal chorioretinal degeneration will result in the severely inflamed areas. If exudative retinal detachments result, considerable retinal degeneration can occur and impair vision long-term.

Sudden Acquired Retinal Degeneration

Sudden acquired retinal degeneration (SARD) is an uncommon disorder in dogs, characterized by sudden and permanent loss of vision (Figure 13.7). The condition has also been referred to as toxic, metabolic, or autoimmune retinopathy (AIR). The onset is sudden, usually a few days to 1–2 weeks, with the development of dilated and nonresponsive pupils, and blindness (day and night). All breeds can become affected; however, most affected individuals are middle-aged, small to medium-sized breeds, and female. Some exhibit concurrent metabolic (elevated serum alkaline phosphatase, serum amino transferase, serum cholesterol, and serum bilirubin) and endocrine disorders. This canine retinal disease has also been compared recently to AIR in humans.

Ophthalmoscopically, the ocular fundi appear normal initially, but electroretinography indicates a generalized outer retinal (photoreceptor) disorder. Within the next 4–6 weeks, generalized retinal degeneration will be apparent on ophthalmoscopy. Treatment, including high levels of systemic corticosteroids, has been unsuccessful.

Vascular Diseases and Systemic Hypertension

Systemic hypertension is an uncommon condition in dogs, but occurs with some frequency in aged cats. The clinical history and clinical signs are similar, and frequently there are concurrent metabolic derangements (renal, endocrine, or cardiovascular disease). Doppler measurements of mean arterial pressure are critical for



Figure 13.7 Sudden acquired retinal degeneration is characterized by acute blindness, dilated and fixed pupils, initially normal appearing ocular fundi, and extinguished flash electroretinogram (flash ERG).

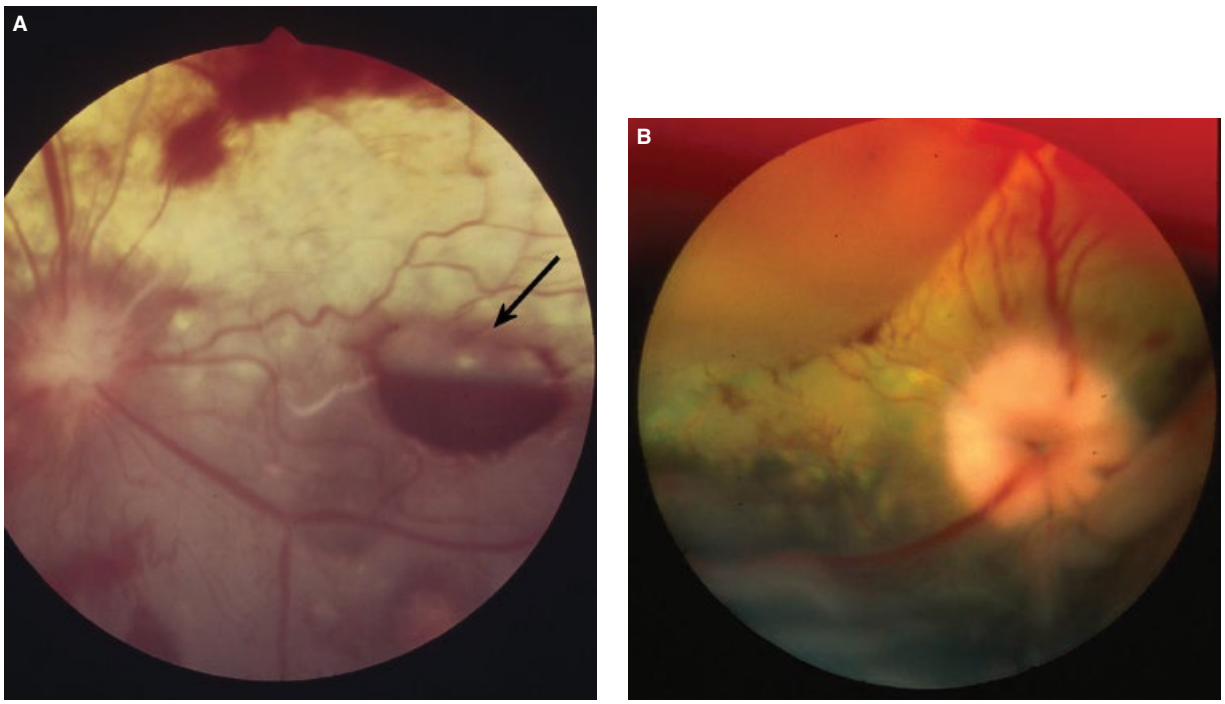


Figure 13.8 (A) Systemic hypertension in the dog is characterized by intraocular hemorrhage, retinal hemorrhages, and variable retinal detachments. Located near the optic disc is a preretinal hemorrhage (called "keelboat" hemorrhage; arrow). (B) Systemic hypertension in a dog resulting in partial and multifocal retinal detachment, focal intra- and sub-retinal hemorrhage and papilledema.

diagnosis and essential to monitor the success of therapy in lowering blood pressure long-term. Affected patients often present for hyphema or acute vision loss.

The presenting signs include anterior chamber and intravitreal hemorrhage, retinal detachment (generally from choroidal transudate), and retinal hemorrhages (Figure 13.8; see also Figure 18.22). The shape of the posterior segment hemorrhage can help localize it. Hemorrhage between the posterior vitreous and retinal inner limiting membrane (pre-retinal) are often "keelboat"-shaped. Hemorrhage within the nerve fiber layer of the retina appears flame-shaped. Intraretinal hemorrhages are discretely round and sub-retinal hemorrhage is indistinct, dull red, and diffuse. The retina is often partially to completely detached.

Lipemia Retinalis

Hyperlipidemia or hyperlipoproteinemia is associated with pancreatitis, hypothyroidism, diabetes mellitus, hyperadrenocorticism, and renal and hepatic diseases. Increased serum levels of triglycerides and cholesterol result, and the elevated triglycerides cause the blood within the conjunctival and retinal vessels to appear a milky pink color (Figure 13.9; see also Figure 18.23). With changes in the blood–aqueous barrier, this lipid material can also enter the anterior chamber (may be confused with aqueous flare and hypopyon; see also Figure 10.6E). By ophthalmoscopy, the retinal vessels appear pink, and these changes are most easily appreciated in the nontapetal fundus. As these changes often signal systemic disease, a general medical workup is in order. In young animals, lipemia retinalis can be observed normally in the immediate postprandial state.

Hyperviscosity Syndrome

In hyperviscosity syndrome, there are elevated levels of large molecules, for example immunoglobulin M (IgM) or polymerized IgA, and infrequently IgG, which increase blood viscosity and impair circulation. Most often a malignancy is involved (lymphoma, chronic lymphocytic leukemia, plasmacytoma, or multiple myeloma). The patient may present for hyphema, vision impairment or blindness (usually related to retinal detachments), or secondary glaucoma. The diseased ocular fundus can be detected by ophthalmoscopy during the general physical examination if ophthalmic clinical signs do not initiate presentation.

Ophthalmoscopy reveals impaired or poor circulation, evidenced by dilated and tortuous retinal vessels, retinal hemorrhages, "boxcar" effect of blood flow within retinal blood vessels, papilledema, and retinal detachments (Figure 13.10; see also Figure 18.24). Treatment is directed toward the underlying disease.

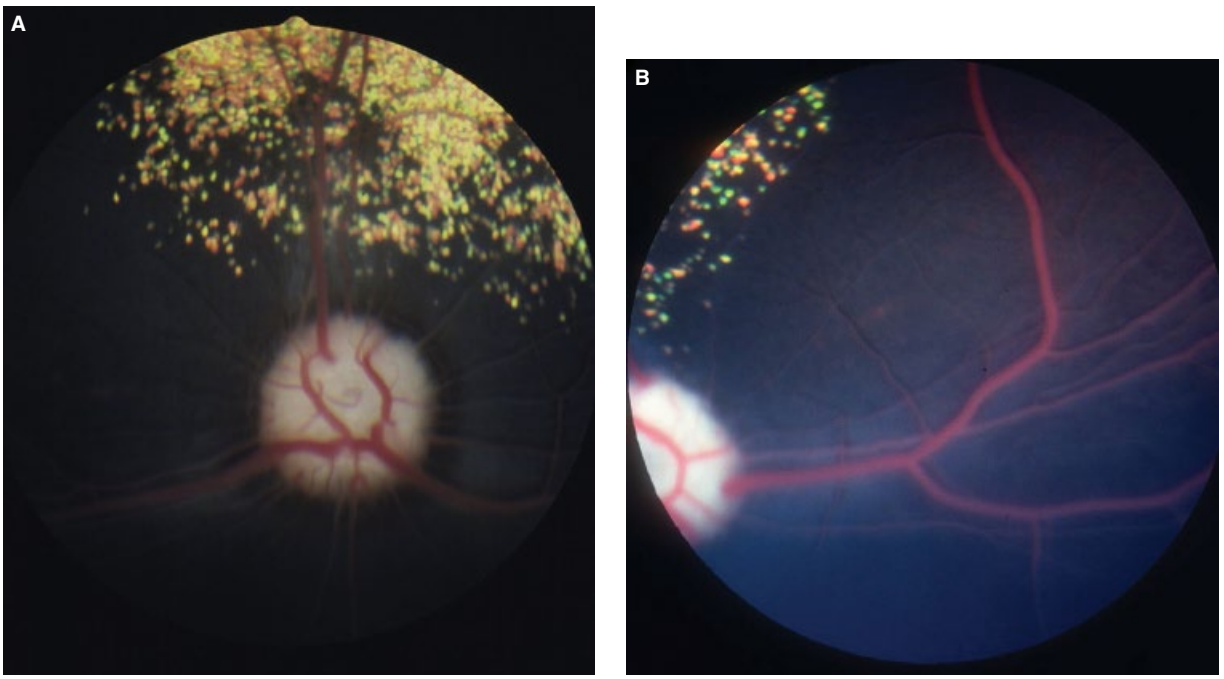


Figure 13.9 In lipemia retinalis in the dog, the retinal vessels appear pink to orange in appearance. (A) Appearance of lipemia retinalis in a dog at the optic disc and tapetal fundus. (B) Appearance of lipemia retinalis in another dog within the nontapetal fundus.

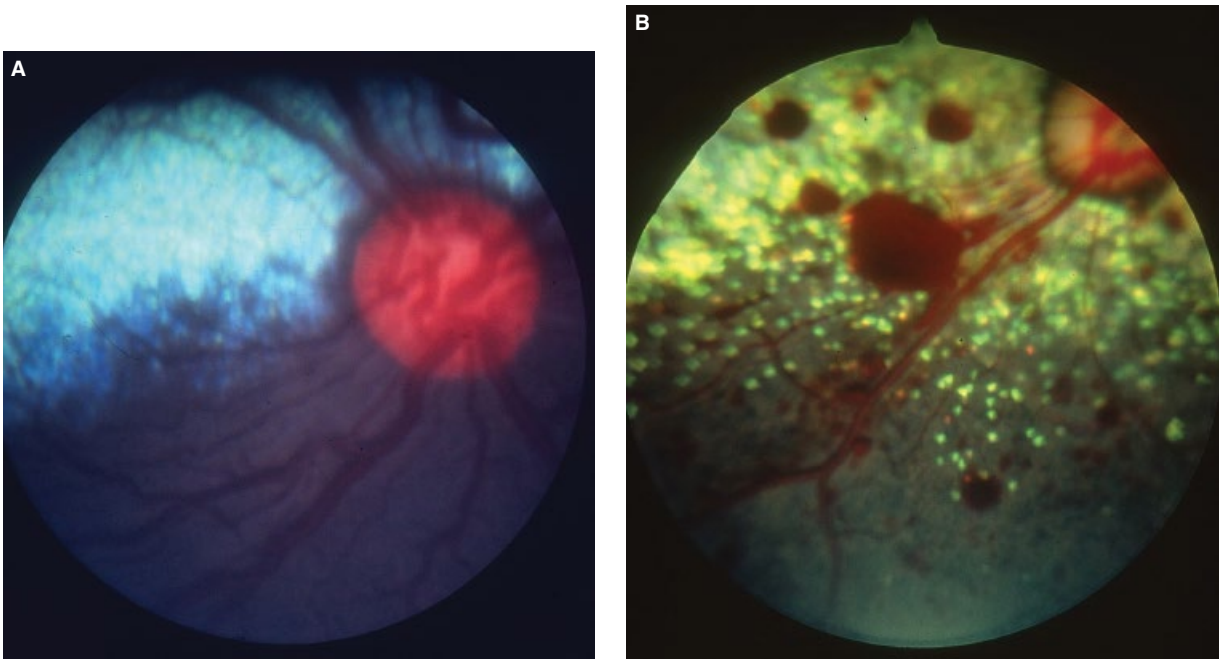
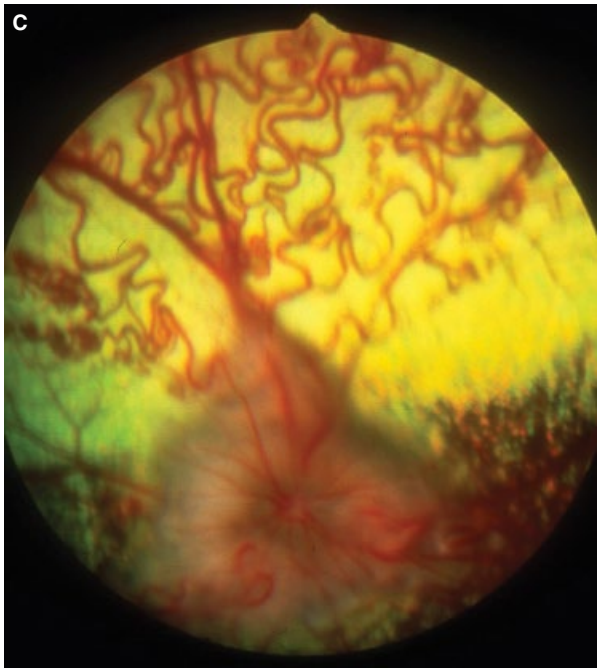
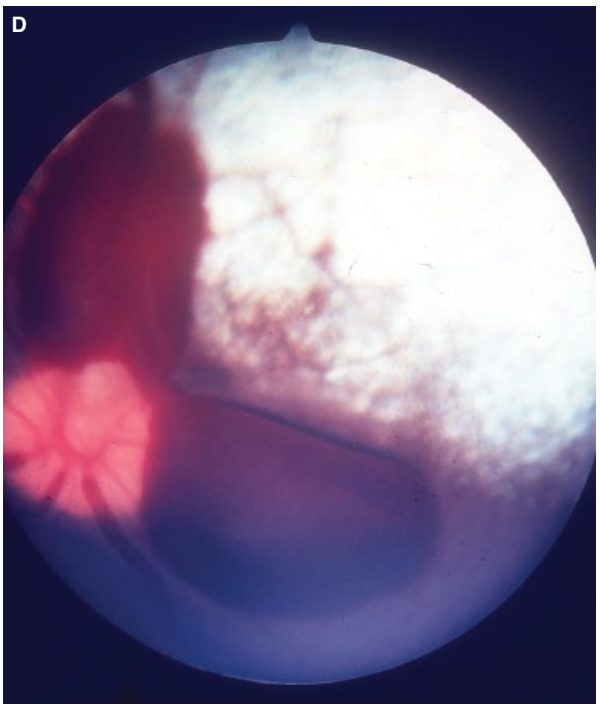


Figure 13.10 Examples of hyperviscosity conditions in dogs. (A) Hyperviscosity syndrome in a puppy affected with multiple cardiac anomalies. Note all of the retinal vasculature is dilated and congested. The optic disc is also very hyperemic. (B) Hyperviscosity syndrome in a dog affected with macroglobulinemia from multiple myeloma. Note the retinal and subretinal hemorrhages.





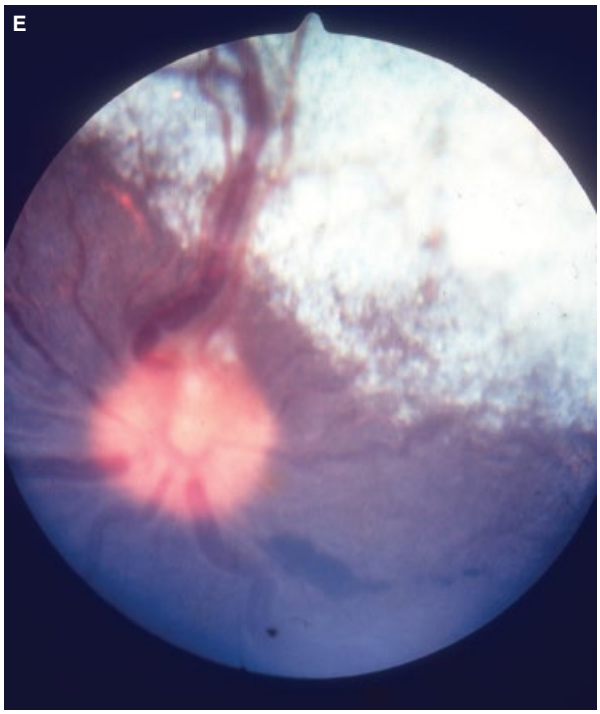


Figure 13.10 (*Continued*) (C) Tortuous retinal arteries in the tapetal fundus of dog. (D) Large pre-retinal hemorrhages near the optic disc in a dog with lymphoma. (E) Same eye as in part D 3 weeks later after treatment with systemic corticosteroids. Some remnants of the hemorrhage remain but most has disappeared.

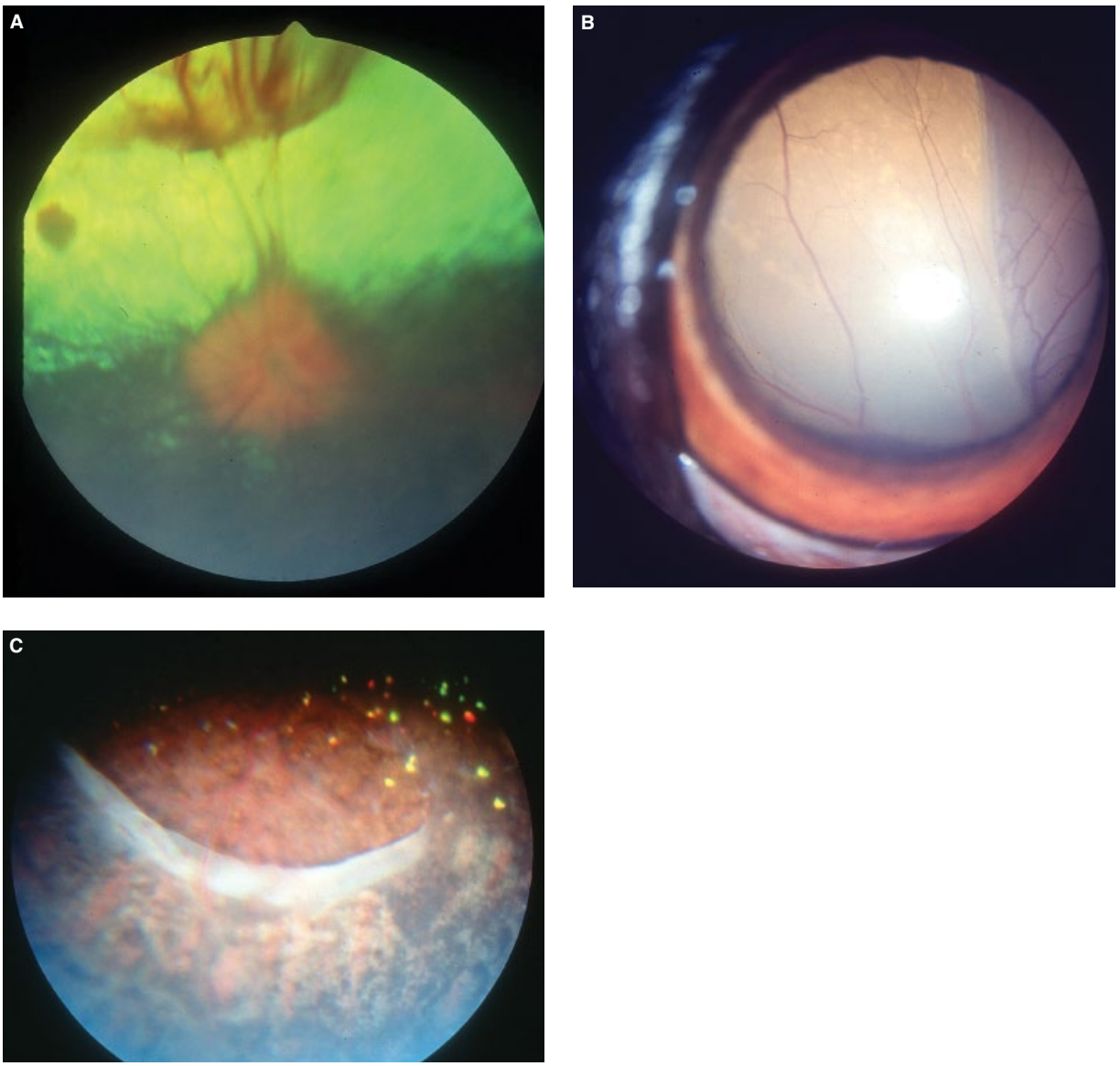


Figure 13.11 (A) Retinal detachment in a dog affected with chorioretinitis. Note the retinal detachment above the optic disc is raised and translucent. (B) Complete retinal detachment in a dog with renal failure that was the result of overhydration with IV fluids. (C) Giant retinal tear with retinal detachment a dog. The edge of the retina and underlying retinal pigment epithelium is exposed.

Retinal Detachments

Retinal detachments are characterized as either rhegmatogenous (retinal break, hole, or tear is present) or nonrhegmatogenous (no retinal break or hole). Nonrhegmatogenous detachments are usually associated with intravitreal hemorrhage and choroidal and retinal inflammation. Specific causes include congenital CEA and retinal dysplasia in many breeds (spontaneous in certain breeds such as the Shih Tzu), chorioretinitis, blunt trauma, hyperviscosity syndromes, and intraocular neoplasms (Figure 13.11).

Rhegmatogenous retinal detachments are associated with retinal breaks (holes or tears usually associated with vitreal or inflammatory membranes and traction), and giant tears (over 90°) that follow penetrating ocular trauma, cataract (phacoemulsification) surgery, and intracapsular lens extraction (for lens luxation).

Treatment of retinal detachments has made significant progress in recent years. Retinal re-attachment surgery is possible, typically consisting of tamponade with gas or silicone oil followed by laser photocoagulation. A small group of veterinary ophthalmologists have been specially trained in retinal re-attachment surgical techniques, and can be referred these patients.

Granulomatous Meningoencephalitis

Granulomatous meningoencephalitis (GME) is an idiopathic nonsuppurative disease of the central nervous system (CNS) and the eye (Figure 13.12). The condition has also been called inflammatory or granulomatous reticulosis, or neoplastic reticulosis. It is characterized histologically by the proliferation of reticuloendothelial elements and lymphoplastic infiltrates of the CNS blood vessels as well as the blood vessels of the posterior segment and anterior uvea.

Ophthalmic signs are typically consistent with inflammation of the posterior segment, accompanied occasionally by anterior uveitis. The ophthalmic signs can be the initial clinical signs initiating presentation or can concurrently accompany other neurologic abnormalities. Visual impairment to blindness usually result from exudative retinal detachments and optic neuritis. The retinitis is usually bilateral, and consists of acute, raised, irregularly shaped and sized areas with fuzzy, indistinct margins, scattered through the tapetal and nontapetal fundi. Optic neuritis can affect the optic nerve head or the retrobulbar optic nerve. When affected, the optic disc will appear swollen with fuzzy margins and may be hyperemic. Diagnosis of GME requires a CNS tap revealing increased cerebrospinal fluid (CSF) proteins levels and pleocytosis with monuclear cells and the absence of any other potential etiology.

Treatment usually consists of high levels of systemic corticosteroids or other immunosuppressant and/or immunomodulating drugs. Long-term therapy is necessary to keep clinical signs in check. The response to treatment can be monitored by ophthalmoscopy, and demonstrated by the conversion of the active retinitis into an inactive or nonprogressive state.

Neoplasms of the Ocular Fundus

Ocular fundus or posterior segment neoplasia is rare in dogs. Most primary and secondary intraocular neoplasia in the dog affects the anterior uvea rather than the retina and choroid. The most frequent primary posterior segment neoplasm is the melanoma of the choroid (Figure 13.13). Clinical signs usually occur late, when clinical vision is impaired, and/or extension into the anterior segment (development of glaucoma, hyphema, and/or uveitis) causes noticeable clinical signs.

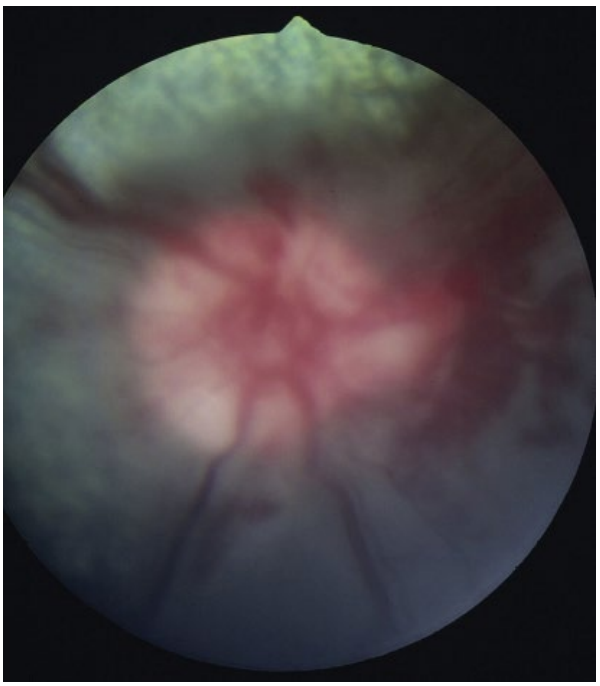


Figure 13.12 Granulomatous meningoencephalitis (GME) in a dog characterized by optic neuritis, and multiple optic disc and retinal hemorrhages.

If detected early (usually for other eye disease, part of a routine eye, or general physical examination), an irregular raised pigmented mass within the ocular fundus will be noted. Retinal blood vessels appear above the mass, and occasionally retinal detachment is evident near the margins of the tumor. Recommended treatment, after a complete medical workup, is enucleation.

Metastatic neoplasia from tumors distant to the eye can also affect the posterior segment. These lesions are usually accompanied by uveitis of both the anterior and posterior segments.

Diseases of the Optic Nerve Head (Optic Disc or Optic Papilla)

The optic nerve head is the point at which the axons of the retinal ganglion cells coalesce to become a distinct structure and exit the eye through the sclera which is modified at the lamina cribrosa to permit the axons to exit. The optic nerve

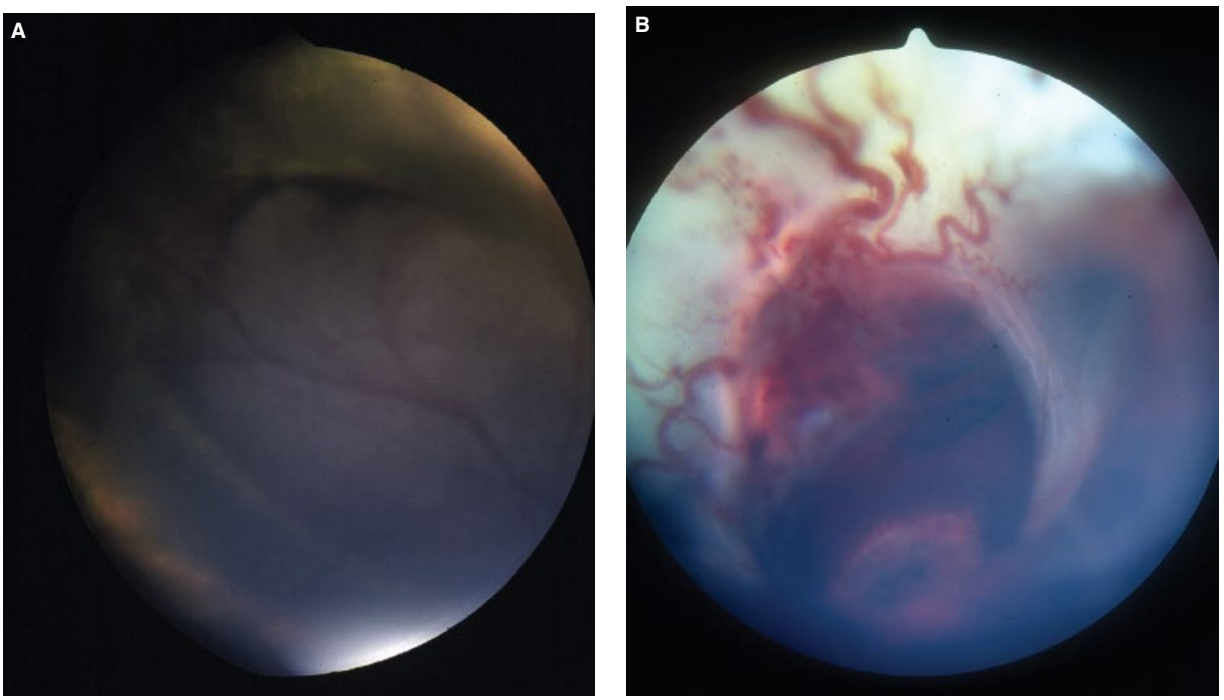


Figure 13.13 (A) Posterior primary and second neoplasms of the choroid and retina are rare. In this dog a primary choroidal melanoma was diagnosed on routine funduscopic examination. A large retinal vessel traverses the anterior surface of the mass. (B) Large choroidal melanoma infiltrating the optic nerve head. Note the adjacent retinal detachment and hemorrhages.

head (also called papilla or optic disc) is located within the tapetal fundus, the nontapetal fundus, or at the junction of the two areas (Figure 13.14). When the optic nerve head is in the tapetal fundus, it is often surrounded by a ring of pigment. Its surface is myelinated and the primary retinal arteries and veins arise from its periphery. The shape of the pink to white disc ranges from round to oval to quite irregular. There is often a central depression, the physiologic cup, which was formed by the prenatal degeneration of the hyaloid system.

Micropapilla

The term micropapilla refers to a smaller than normal optic nerve head with clinical normal vision (or at least to the extent that we can assess it) and pupillary light responses. It is not known whether the optic nerve axons or other tissues (including myelin) are reduced. The condition occurs in several breeds including Belgian Sheepdog and Belgian Tervuren (both breeds are frequently affected), Dachshund, Miniature Schnauzer, and Irish Wolfhound (Figure 13.15). In this condition, the optic nerve head is smaller than normal, but other fundus abnormalities are absent.

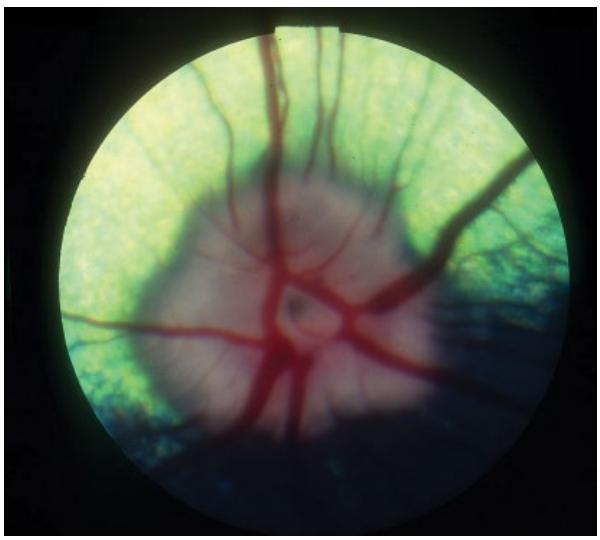


Figure 13.14 The normal optic nerve head of the dog at the junction of the tapetal and nontapetal fundi consists of a myelinated surface. Large primary retinal vessels emerge from its center, a variable central venous circle is present and small retinal arterioles and veins emerge from its periphery. A central physiologic cup and a vestige of the posterior hyaloid vessels (Bergmeister's papilla) are present.

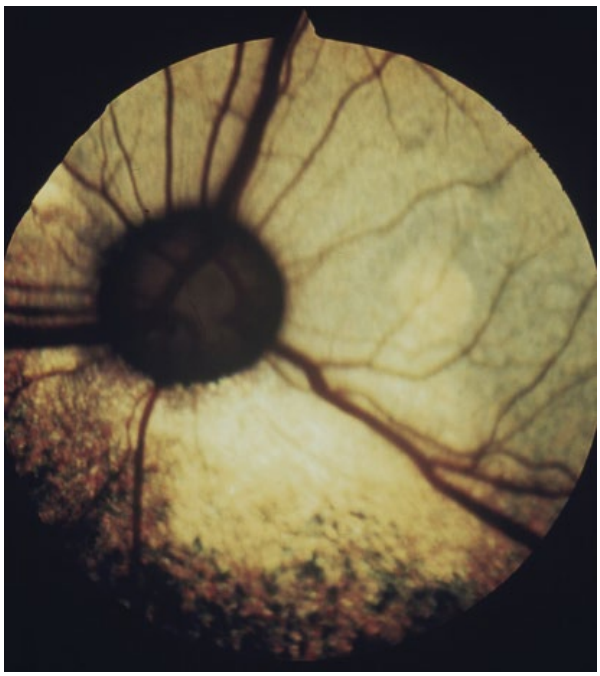


Figure 13.15 Micropapilla in a Belgian Tervuren dog which consists of a small optic papilla in an eye with apparently normal clinical vision. This disc is situated within the tapetal fundus and has a complete pigmented periphery (ring).

Optic Nerve Hypoplasia

Optic nerve hypoplasia occurs infrequently in dogs, and is unilateral or bilateral (Figure 13.16). It occurs sporadically, but can be inherited in Toy and Miniature Poodles. Affected eyes have reduced numbers of retinal ganglion cell axons. If the condition is unilateral, visual impairment or blindness may not be noticed until the opposite or fellow eye develops significant eye disease.

Resting pupil size can be normal (regulated by input from the fellow eye) or dilated (with bilateral optic nerve hypoplasia). Direct pupillary light reflexes are absent. Ophthalmoscopy reveals a small or barely detectable optic nerve head. The retinal vessels are present, but the number of arterioles and veins can be reduced. There is no treatment.

Optic Nerve Colobomas

Optic nerve colobomas are either typical (occurring at the 6 o'clock position) or atypical (any other location) (Figure 13.17). Although optic nerve colobomas can occur sporadically in any individual, they occur most often as part of the CEA syndrome. Optic nerve colobomas can also be combined with optic nerve hypoplasia.

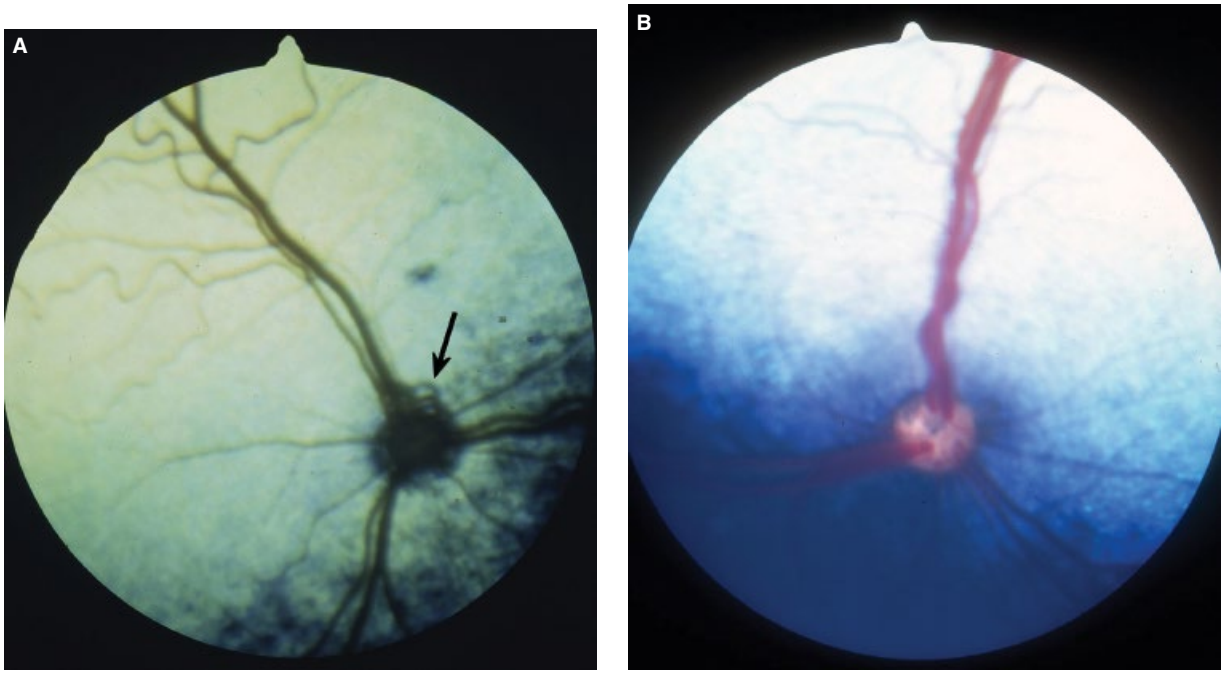


Figure 13.16 (A) Optic nerve hypoplasia in a German Shepherd puppy. The puppy has mydriasis, nonresponsive pupils, and is blind. Note the optic disc is very small and has a retinal arterial loop (arrow) on its dorsal surface. Blindness was the presenting clinical sign, because the condition was bilateral. (B) Optic nerve hypoplasia in a Miniature Poodle. The optic disc is very small. Both eyes were affected, and the young dog was blind.

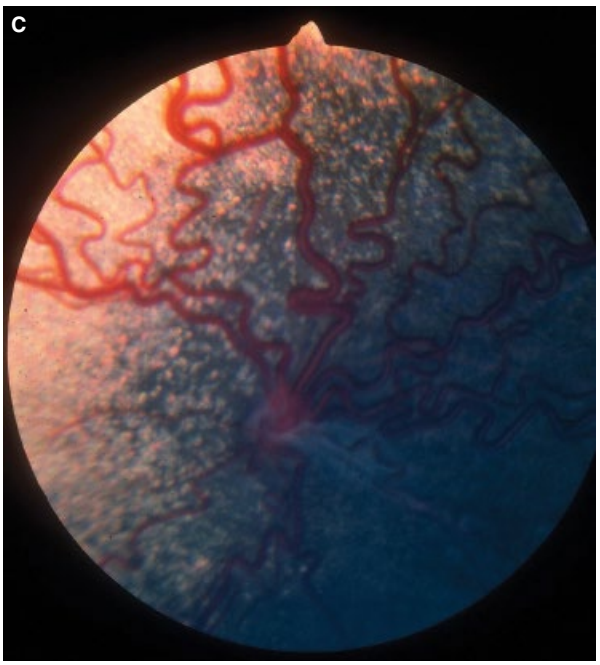


Figure 13.16 (*Continued*) (C) Unilateral optic nerve hypoplasia in a mixed-breed dog. The condition was diagnosed after the fellow eye developed cataract, and the dog became blind. The optic disc is barely visible.

They are most frequently noted in Rough and Smooth Collies, Shetland Sheepdog, Border Collie, Australian Shepherd, Nova Scotia Duck Tolling Retriever, and Lancashire Terrier.

Optic nerve colobomas appear ophthalmoscopically as excavations or "pits" within or adjacent to the optic nerve head. They vary in size, shape, and depth, and can affect nearly all of the optic disc's surface. The depth can be irregular, because of the varying glial and scleral lamina tissues which provide support for the area. In Collies, disc colobomas are associated with choroidal hypoplasia, and can communicate with the vitreous and subretinal spaces, predisposing to retinal detachment.

Papilledema

Papilledema in humans is a frequent indicator of increased CSF pressure. In dogs, papilledema has been associated with intraorbital and intracranial neoplasia, and with space-occupying lesions of the orbit, as well as elevations in CSF pressure (Figure 13.18). Excessive myelination of the optic nerve head can be confused with papilledema (pseudopapilledema). Both vision and

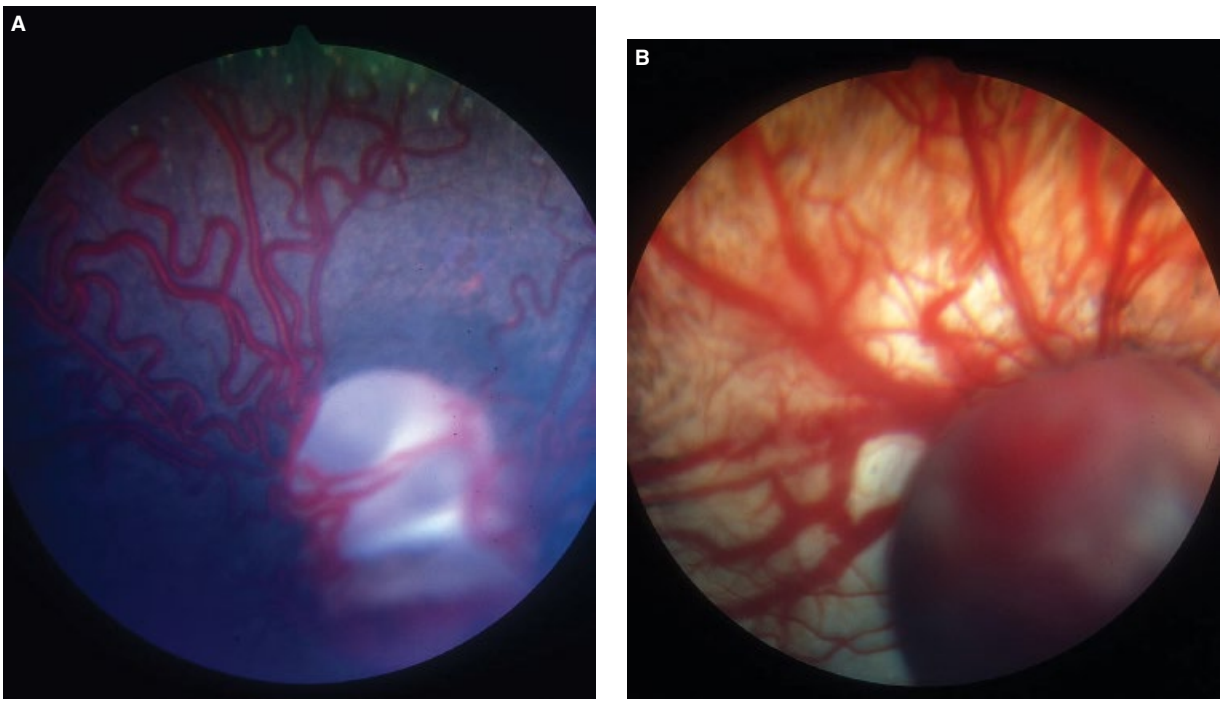


Figure 13.17 (A) Optic nerve coloboma in a Beagle. Note the large coloboma involves the entire optic disc. (B) Large coloboma in a young Collie with Collie eye anomaly. This excavation was more than 10 diopters deep.

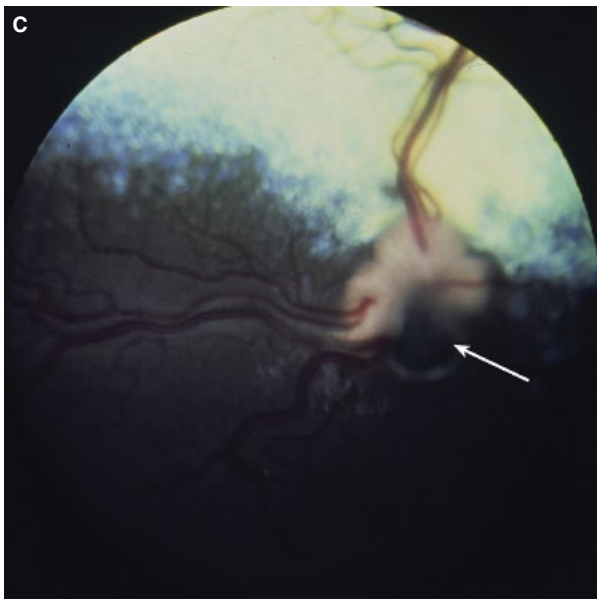


Figure 13.17 (*Continued*) (C) Focal typical optic nerve coloboma (arrow). Note its location at the 6 o'clock position in the disc.



Figure 13.18 Papilledema in a dog associated with an orbital neoplasm. The raised optic disc protrudes slightly into the posterior vitreous and above the adjacent retina.



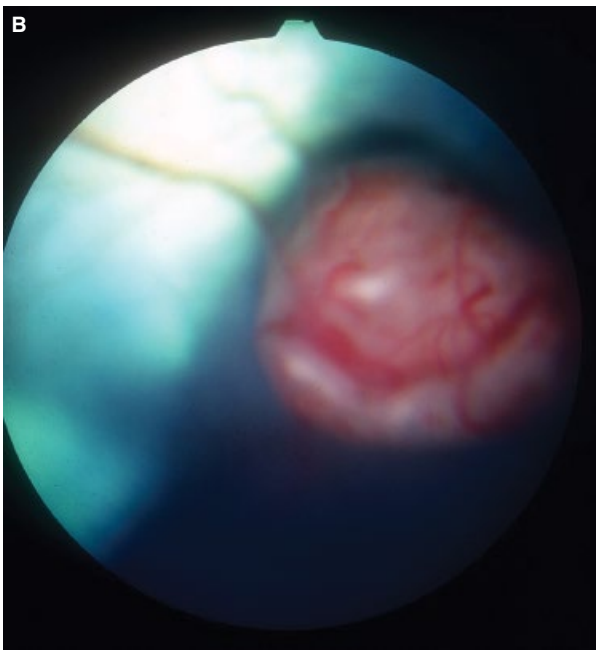


Figure 13.19 Optic neuritis in a dog. (A) Secondary to canine distemper, the inflammation also extends into the adjacent retina (neuroretinitis) and has produced multiple retinal and optic disc hemorrhages. (B) Optic neuritis with severe inflammation resulting in protrusion of the disc several diopters above the ocular fundus surface.

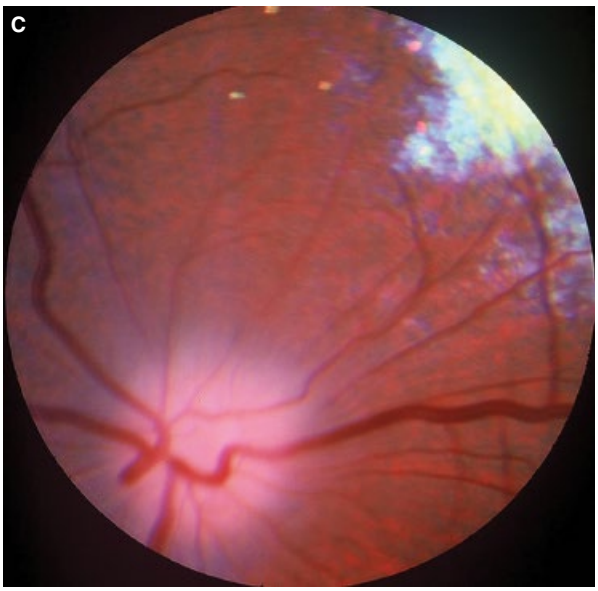


Figure 13.19 (*Continued*) (C) Optic neuritis in a young Chihuahua secondary to canine distemper. Note the hyperemia and the indistinct margins of the optic disc.

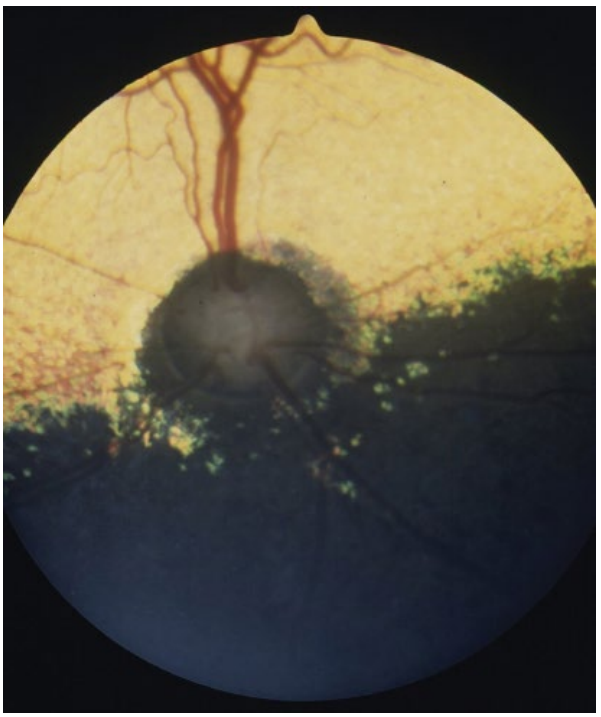


Figure 13.20 Optic nerve atrophy following repeated bouts of optic neuritis. The atrophic disc is reduced in size, depressed, and partially pigmented.

pupillary light reflexes are normal in both instances. By ophthalmoscopy, papilledema appears as an elevated optic nerve head with venous congestion, but devoid of any hemorrhages. Prolonged papilledema can result in optic nerve atrophy.

Optic Neuritis

Optic neuritis is unilateral or bilateral, and results from viral, mycotic, protozoan, and parasitic infections, as well as trauma, reticulosis, toxins, and neoplasia as well as other causes. Bilateral optic neuritis is associated with blindness and dilated and fixed pupils. Ophthalmoscopy reveals a raised edematous disc, with indistinct margins, peripapillary hemorrhages, venous congestion, and, in some instances, inflammatory cells suspended in the vitreous in front of the disc (Figure 13.19).

For patients with optic neuritis, a complete physical and medical workup is necessary. Every attempt should be made to identify the cause and provide appropriate treatment. Systemic immunosuppressant therapy to treat the inflammation of the optic nerve will be necessary. Prognosis for retention or return of vision is guarded to poor.

Optic Nerve Atrophy

Optic nerve atrophy can follow trauma, persistent and chronic inflammations, granulomatous meningoencephalitis, or primary and secondary glaucoma (Figure 13.20). Both the pupillary light reflexes and vision can be impaired. Other signs of ocular disease can also be present.

Optic nerve degeneration appears as a smaller than normal, depressed, often pigmented disc with vascular attenuation at its surface. An adjacent hyperreflective tapetal crescent as well as similar shaped loss of pigmentation in the nontapetal fundus can occur.

Treatment is not usually successful, but high levels of systemic corticosteroids can be attempted. Steroid-responsive optic neuritis can occur in the dog, and optic disc atrophy is the end result.

14

Feline Ophthalmology

Diseases of the Orbit

Microphthalmia is a rare condition in kittens, but often these globes have multiple anomalies (Figure 14.1). Breeds affected include the Domestic Shorthair and Persian. Congenital cataracts are often present. Vision is usually present unless the cataract formation is advanced. Generally there is no treatment.

Traumatic Proptosis

Proptosis or the traumatic displacement of the eye from the orbit is a serious disease in the cat (Figure 14.2). It is usually associated with considerable head trauma, and often mandibular symphysis fractures are present. Orbital hemorrhage can compound the globe luxation. The cornea undergoes rapid desiccation with malacia, and perforation is likely if not addressed promptly. Optic nerve damage (extending to involve the optic chiasm) can be a complication from the trauma with stretching and inflammation. Damage to the contralateral optic nerve can result in blindness of the fellow eye.

Replacement of the globe and short-term complete temporary tarsorrhaphy should be performed as soon as possible. The prognosis for the return of vision in cats is poor.

Orbital Cellulitis

The clinical signs and treatment of orbital inflammations in cats are similar to those in dogs. As the orbital space is more limited in cats, orbital inflammations rapidly cause protrusion of the nictitans, conjunctival hyperemia, localized orbital pain, and limited exophthalmos (Figure 14.3). Both bacteria and fungus (*Penicillium* sp.) have been isolated. Treatment is the same as for the dog.

Orbital Neoplasia

Orbital neoplasia occurs in cats, but reports are less frequent than in dogs. About 90% are malignant, and about 60% of these tumors are squamous cell carcinomas (Figure 14.4). Many of the tumors arise from the conjunctiva, nictitating membrane, or eyelids and invade the orbit from there. Others arise in the nasal passages and invade outward into the orbit. Orbital lymphosarcoma is a common orbital tumor in cats. It is unilateral or bilateral.

Diseases of the Eyelids

Diseases of the feline eyelids are similar to those that occur in the dog, but inherited defects are far less frequent. In contrast to the usual benign lid tumors in the dog, lid tumors in cats are usually highly malignant and histologic examination of the surgical margins is recommended.

Lid Agenesis

Eyelid agenesis or lid coloboma is an infrequent disorder in kittens, affecting one or both eyelids (Figure 14.5). The lid coloboma almost always involves the lateral aspects of the upper lid, less commonly the lateral canthus and lateral lower eyelid. The lid margin and the conjunctiva (palpebral and fornix) are often missing. The edge of the affected area can contact the bulbar conjunctiva and cornea and produce focal irritation and inflammation.

Eyelid agenesis can be accompanied by other anomalies of the globe including iris defects (persistent pupillary membranes, iris colobomas), cataracts, and colobomas of the optic nerve head. Clinical vision is usually normal. Both heredity and *in utero* viral infections have been suggested causes. Eyelid restoration with a myocutaneous pedicle graft from the lower eyelid or the lateral commissure of the mouth is the most common surgical therapy.

Entropion

Structural abnormalities of the eyelids (i.e., entropion and ectropion) are infrequent in cats, but entropion occurs in the Persian and other brachycephalic breeds, in tom cats of any breed that were neutered later in life, or

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Figure 14.1 (A) Microphthalmia affecting both eyes in a kitten. The microphthalmia causes the exposure of the dorsal sclera within the palpebral fissure.
(B) Symblepharon, or adhesion of the conjunctiva to the eyelids, the cornea, and to itself, in this kitten is obscuring visualization of a normal-sized globe.



Figure 14.2 (A) Marked proptosis in a cat of several hours duration. Note the marked drying and desiccation of the unprotected cornea.



Figure 14.2 (*Continued*) (B) Traumatic proptosis in another cat. The pupil is dilated and unresponsive to light. The prognosis for successful globe replacement and return of vision is very poor.





Figure 14.3 (A) Orbital cellulitis in a cat presented as exophthalmos, swollen eyelids, and secondary iridocyclitis (miosis). The signs for orbital cellulitis in cats are much more subtle than in the dog. (B) Another example of orbital cellulitis in a cat. Note the exophthalmos, conjunctival swelling and hyperemia, and the dull, dry central cornea (the result of exposure).

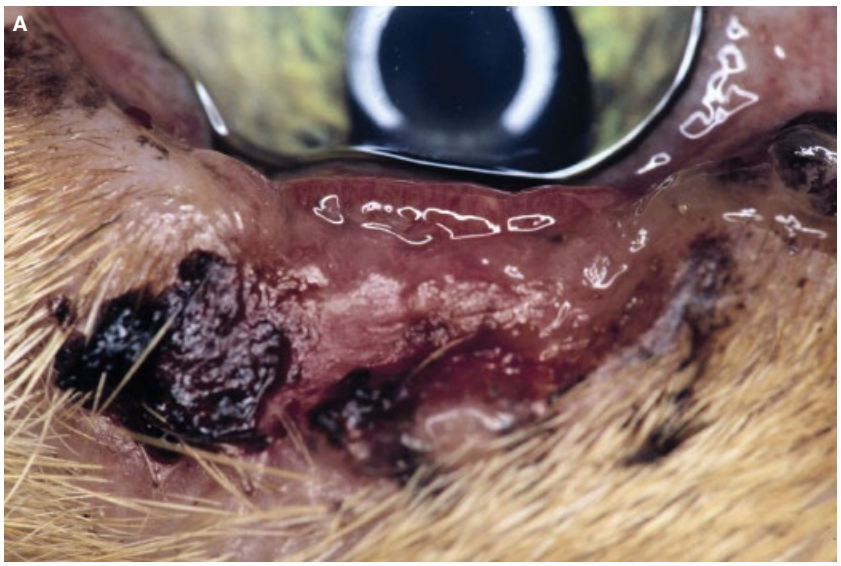






Figure 14.4 (A) Orbital neoplasms are often squamous cell carcinomas that arise from the eyelids and conjunctiva, as in this cat, and subsequently invade the orbit. (B) This cat had a neoplasm in the ventral floor of the orbit that extended from the oropharynx and resulted in dorsal deviation of the globe and elevation of the nictitans. (C) Orbital extension of squamous cell carcinoma from the nasal passages resulted in exophthalmos in this cat.



Figure 14.4 (*Continued*) (D) Orbital lymphoma in this cat resulted in exophthalmos, deviation of the globe off its axis, and exposure. Impairment of the optic nerve and the optic chiasm resulted in mydriasis and blindness.





Figure 14.5 (A) Eyelid agenesis is one of the most frequent congenital lid defects in the cat. In this young cat the eyelid agenesis affects both lateral upper eyelids. The lack of lid margin and trichiasis cause corneal and conjunctival irritation that necessitates surgical correction. (B) Same cat as in part A in a closer view of the right eye. Note the lack of lateral upper eyelid skin, margin and conjunctiva, and the trichiasis. Keratitis is present in the exposed and irritated dorsolateral cornea.

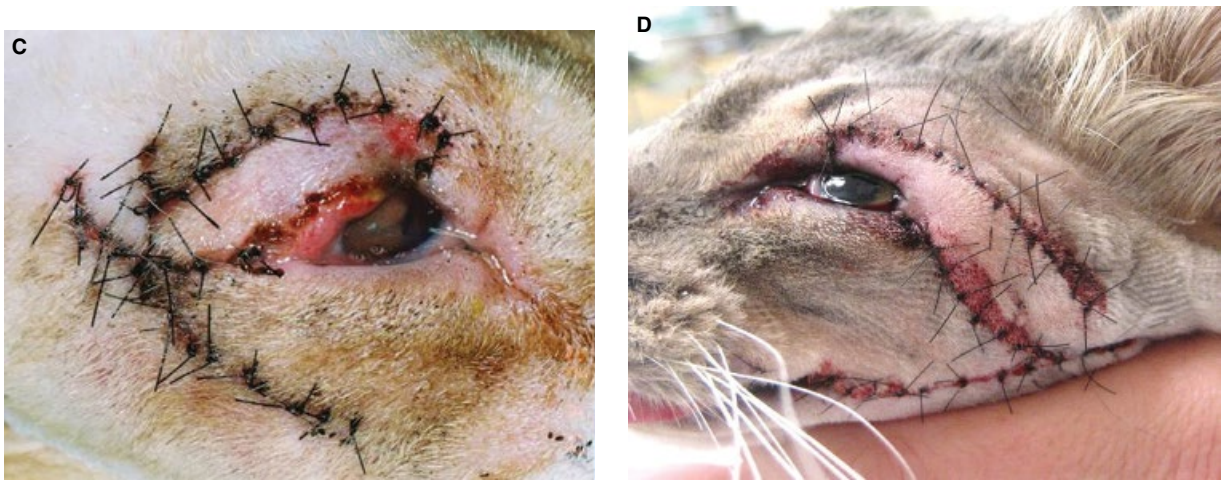


Figure 14.5 (*Continued*) (C) Same cat as in part A and B immediately postoperatively following surgical reconstruction (Dziezyc–Millichamp modification of the Roberts–Bistner technique). (D) Another cat with eyelid agenesis immediately following repair with a modified lip-to-lid procedure.

in an aged or infirm cat that has had significant weight loss or atrophy (Figure 14.6). Cicatricial entropion can follow eyelid surgery, eyelid lacerations, and prolonged blepharitis. Once the cutaneous portion of the eyelid margin contacts the cornea and/or conjunctiva, blepharospasm develops and can worsen the entropion (spastic entropion). Entropion in cats most commonly affects solely the lower eyelid.

Blepharitis

Blepharitis or inflammation of the eyelids in cats primarily involves the dermis alone, with deep involvement (i.e., meibomianitis) occurring rarely (Figure 14.7).

Lid Neoplasia

Eyelid neoplasms are not infrequent in older cats and account for 2% of all feline neoplasia. In contrast to dogs, lid tumors in cats are highly malignant, locally infiltrative, and often require extensive surgery or a combination of therapies (surgical debulking, cryotherapy, radiation, and chemotherapy). The most common tumors are squamous cell carcinoma (36–65%), fibrosarcomas (8%), lymphoma (11%), and adenocarcinomas (7–8%) (Figure 14.8).

Squamous cell carcinomas (SCC) represent about two-thirds of the eyelid tumors diagnosed in cats, and occur most commonly in older light-colored or white cats. They appear as ulcerated to proliferative lesions, most often affecting the eyelid margins and anterior nictitans. Locally invasive, SCC metastasize late.

Fibrosarcomas are frequent lid tumors, appearing as a focal nodular mass arising in the subcutaneous tissues and with an ulcerative surface. In young cats, multicentric fibrosarcomas result from the feline sarcoma virus (FeSV). Long-term prognosis is poor.

Lymphoma occurs unilaterally or bilaterally. These masses are subcutaneous or beneath the palpebral conjunctiva.

Eyelid and periocular apocrine hidrocystomas have been reported in cats, especially in the Persian and Himalayan breeds. Thought to arise from the apocrine glands along the eyelid margins, they appear as gray, often coalescing, single or multiple cystic masses. Histologically, they are adenomatous, dilated epithelial cysts, and contain brown to tan proteinaceous debris. They are benign; however, recurrence is common.

Diseases of the Tear and Nasolacrimal System

Tear and nasolacrimal disorders in cats are infrequent, but keratoconjunctivitis sicca (KCS) does occur. Nasolacrimal drainage disorders most often occur in the brachycephalic breeds.

Epiphora

Epiphora is infrequent in cats and has several causes. In the brachycephalic breeds (Persian and Himalayan), it can be associated with medial lower entropion and lacrimal punctal disorders, and is treated surgically. It is seen in cats with acute or chronic ocular surface disease, especially if there is an infectious etiology (e.g., FHV-1, *Chlamydophila*, *Mycoplasma*).



Figure 14.6 (A) Entropion or the inversion of the eyelid margin has resulted in blepharospasm and keratitis from the lid–corneal contact. (B) Same cat as in part A immediately following surgical correction using the Hotz–Celsus technique.



Figure 14.7 (A) Blepharitis associated with Demodex in a cat. Note the multiple sites of involvement (e.g., nose, forehead, and eyelids).

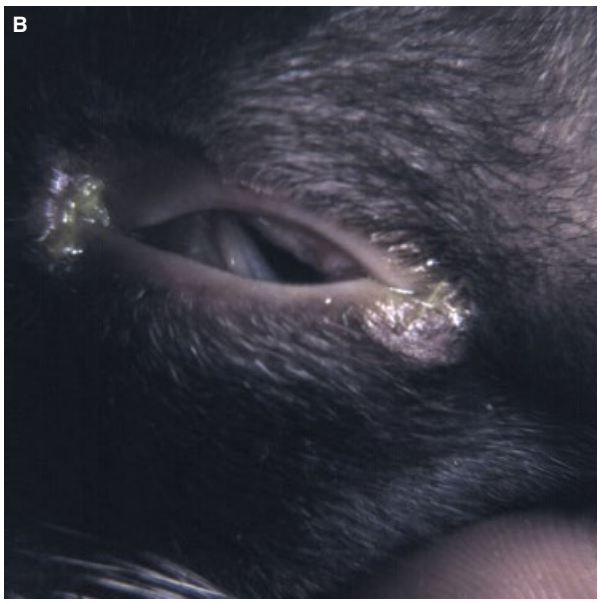




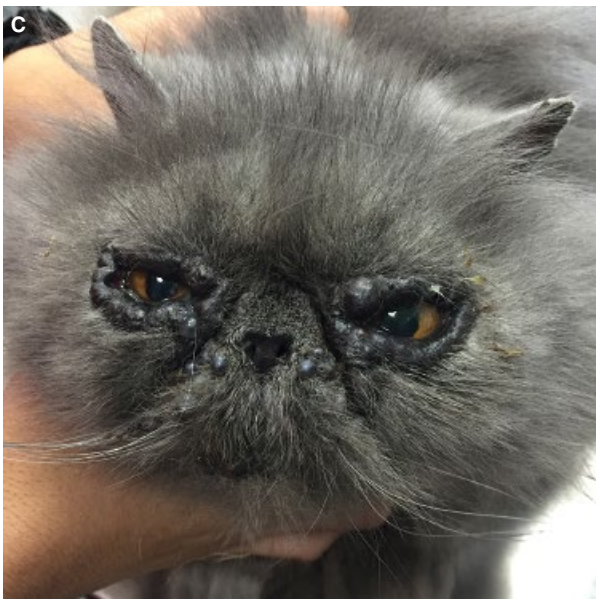
Figure 14.7 (*Continued*) (B) Blepharitis secondary to feline scabies caused by *Notoedres* in a kitten. These lesions are highly pruritic. (C) Severe bilateral autoimmune blepharitis in a cat. There is a conjunctival pedicle graft on the right cornea covering a keratectomy site where a corneal sequestrum was removed.



Figure 14.8 (A) Early squamous cell carcinoma in a white cat. The lesion initially appears resembling an ulcerated wound that fails to heal.

Figure 4.8 (*Continued*) (B) Lid squamous cell carcinoma in an aged cat. The tumor has infiltrated the entire lower eyelid and the medial canthus. Biopsy is suggested to confirm the diagnosis before attempting extensive surgery, radiation, and/or cryotherapy. (C) Apocrine hidrocystomas present as single to multiple masses often cystic and containing brown proteinaceous fluids as in this Persian cat. (D) Cutaneous mast cell tumors have been reported in the eyelids of cats. These often appear as areas of focal alopecia. They may be raised, as seen in this cat, or flat.







Keratoconjunctivitis Sicca

KCS occurs infrequently in cats and most often seems related to chronic blepharoconjunctivitis associated with feline herpesvirus (FHV-1) infections (Figure 14.9). Schirmer's tear tests values are normally a little lower in cats than dogs, but dry eyed cats will have significant reductions in aqueous tear production. Clinical signs of feline KCS are more subtle than dogs, and include conjunctival and nictitans hyperemia, mild and diffuse superficial keratitis with vascularization but little corneal or conjunctival pigmentation. There is usually no ulceration (fluorescein retention), but rose Bengal stain may be retained diffusely (minute foci of degenerating corneal epithelium).

Diseases of the Conjunctiva

Feline Herpesvirus

FHV-1 is ubiquitous among domestic and wild cats worldwide. It is estimated that over 80% of domestic cats have been exposed to FHV-1 by adulthood. Primary infection in cats results in both conjunctival and respiratory infections, often complicated by secondary bacterial infections (Figure 14.10). In kittens less than 12–14 days old, FHV-1 infections can present as neonatal ophthalmia where infection has developed under the normally closed eyelids.

In older kittens (at the time of weaning), FHV-1 can present as an acute serous to mucopurulent conjunctivitis



Figure 14.9 (A) Early keratoconjunctivitis sicca (KCS) in a cat. Brown ocular discharge is present, the result of ocular surface inflammation. KCS is commonly associated with feline herpesvirus (FHV-1) infection in cats. Feline KCS is less obvious than in the dog. (B) Chronic nasolacrimal obstruction treated by conjunctivorhinostomy in a cat. The silicone tube within the new bypass to the nose is secured to the medial canthus and nose for several weeks. The cat must wear an E-collar during the time the tubing is in place. (C) Prolapse of the nictitans gland in a cat. This is an uncommon finding in cats and is seen most often in the Burmese breed. Surgical replacement is recommended to minimize the risk of the development of KCS.



Figure 14.10 (A) Primary feline herpesvirus-1 (FHV-1) conjunctivitis in a kitten. Note the copious conjunctival exudates of both eyes. (B) Extensive symblepharon in a kitten following its initial infection with FHV-1. The palpebral conjunctiva has adhered to the cornea, the conjunctiva of the nictitans, and to itself.

with respiratory signs. The conjunctiva is hyperemic but not generally chemotic. Corneal microdendritic ulcers can be detected with topical fluorescein or rose Bengal stain. Treatment includes topical antibiotics (tetracycline, chloramphenicol, or erythromycin), and systemic supportive therapy. Symblepharon formation, or adhesion of the conjunctiva to itself or other ocular tissues, can be a complication.

After recovery from the primary FHV-1 infection, about 80% of the cats become latent carriers of FHV-1 and in about 45% of these cats the virus will spontaneously activate and result in recrudescent eye disease. Hence, in most adult cats, ophthalmic FHV-1 infections are recurrent (Figure 14.11; also Figure 18.29). Stress, the introduction of a new pet, moving, and other disease or immunocompromised states can trigger release of the virus. Some cats, instead of developing the cytolytic form of the herpetic ocular disease, which is the result of active viral replication and cell lysis, will develop an immunopathologic form that manifests with stromal keratitis.

Chlamydophila psittaci

Chlamydophila psittaci causes pneumonitis and conjunctivitis, most commonly in kittens (Figure 14.12). The respiratory infection is usually mild. The conjunctivitis is characterized by conjunctival hyperemia, chemosis, and serous to later mucopurulent conjunctival exudates. Often one eye and then both eyes are affected. Follicles form on the conjunctival surfaces in chronic infections. Cytology of acute infection can reveal intracytoplasmic inclusion bodies within the conjunctival epithelial cells.





Figure 14.11 (A) Recurrent FHV-1 conjunctivitis in an adult cat. Conjunctivitis with chemosis of the ventral conjunctiva is present. (B) More severe recurrent FHV-1 conjunctivitis in an adult cat. Marked conjunctival hyperemia and chemosis affect both ventral and dorsal conjunctivae.



Figure 14.12 *Chlamydia* conjunctivitis in a kitten. The conjunctivitis is mild, and some dried conjunctival discharge is present at the medial canthus.

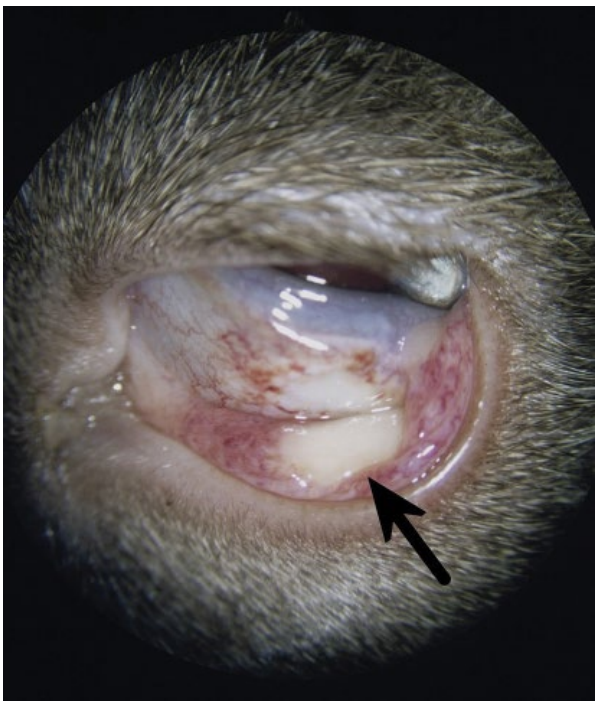


Figure 14.13 Mycoplasmal conjunctivitis in an adult cat. Note the marked conjunctival hyperemia and swelling, and the pseudomembrane (arrow) in the ventral conjunctiva.

Mycoplasmal Infections

Mycoplasma spp. (*Mycoplasma felis* and *Mycoplasma gatae*) also cause conjunctivitis in cats, which can affect one or both eyes (Figure 14.13). The conjunctivitis is characterized by epiphora, conjunctival follicles, chemosis, and formation of pseudomembranes (plaques of thick white exudates). Cytology reveals intracytoplasmic bodies.

Symblepharon

Symblepharon is the adherence of conjunctiva to itself or to the cornea. It occurs most often in young cats, and appears related to acute or recurrent FHV-1 conjunctivitis (Figure 14.14; also Figure 18.29C). Symblepharon appears as conjunctiva adhered to the cornea for varying degrees. Eyelid movements and the depth of the conjunctival fornix can also be compromised. Surgical procedures are available to treat this condition, but recurrent FHV-1 conjunctivitis can cause the condition to return.

Lipogranulomatous Conjunctivitis

Lipogranulomatous conjunctivitis is an unusual inflammatory condition in cats believed to develop from damage to meibomian glands and an inflammatory reaction to liberated glandular secretions (Figure 14.15). The lesions occur in the palpebral conjunctiva adjacent to the eyelid margins and appear as nonulcerated white nodules. They can be quite irritating, but in most cases surgical resection is curative.

Diseases of the Cornea

The feline cornea is nearly round (vertical diameter 16 mm; horizontal diameter 17 mm), and along with the nictitating membrane is the main tissue visible in the feline palpebral fissure. Very little of the bulbar conjunctiva can be seen in normal cats until the upper eyelid is manually retracted.

Feline Herpesvirus-1 and the Cornea

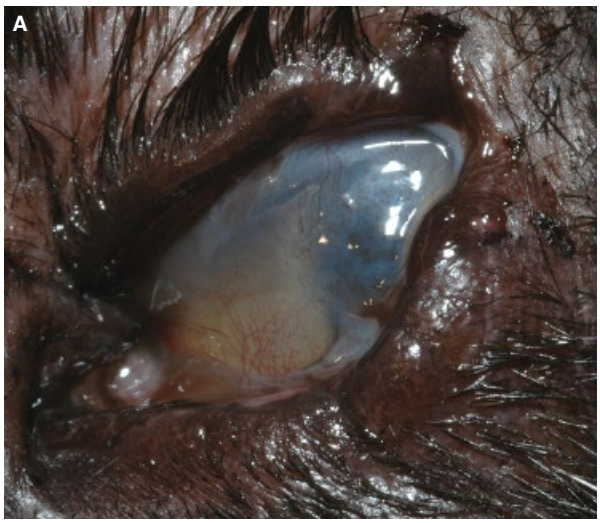
Although FHV-1 primarily affects the conjunctiva, the cornea can be involved as well (Figure 14.16). The virus can replicate in and cause lysis of the corneal epithelium. Dendritic or microdendritic corneal ulcers result, which stain poorly with topical fluorescein but fairly well with rose Bengal. Released viral particles from lysed epithelial cells infect adjacent cells and result in enlargement of the superficial ulcer.

FHV-1 keratitis can also involve the stroma (Figure 14.17). Studies suggest viral suppression of the local immune response at the time of the initial infection permits access to the stroma. Residual viral antigen in the corneal stroma can elicit a delayed inflammatory response that is not typically ulcerative. Clinical signs include corneal edema and cellular infiltrate, superficial vascularization, and fibrosis. Significant corneal scarring can eventually impair vision. Corneal ulcers are present in some cases.

Corneal Sequestration

Corneal sequestration has several synonyms including corneal black spot, corneal nigrum, corneal mummification, and focal corneal degeneration (Figure 14.18; also Figure 18.29A). It consists of a central or paracentral focal degeneration of the corneal stroma (collagen and fibroblasts), the accumulation of a brown water-soluble pigment, and a variable surrounding inflammatory response. Although it affects cats of all ages in one or both eyes, the Persian, Himalayan, and Burmese breeds seem predisposed. The cause has not established, but FHV-1 has been detected in 55–73% of sequestrum keratectomy samples.

Corneal sequestration appears as a variable shaped, size, and depth, brown to black lesion that may be so dense that slit lamp biomicroscopy cannot accurately estimate the depth of stromal involvement. High frequency ultrasonography (20–35 MHz) can be helpful to



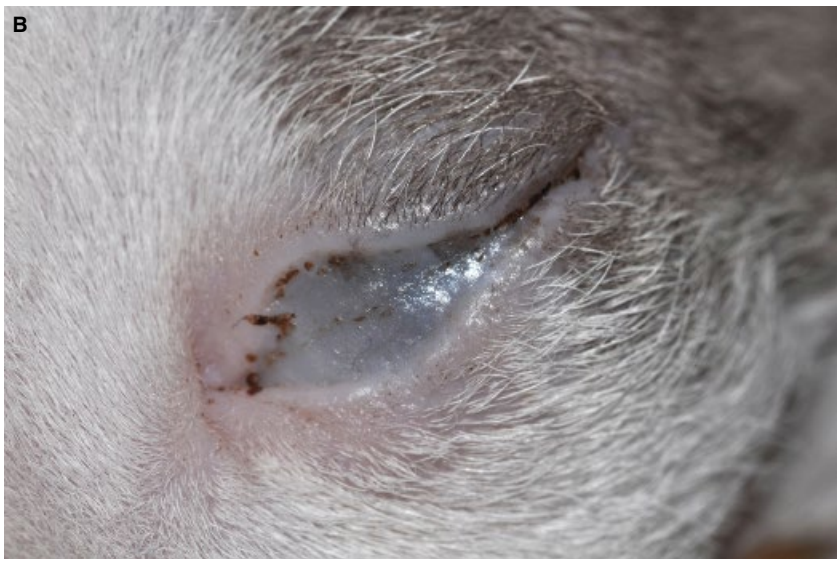


Figure 14.14 (A) Symblepharon in a cat associated with FHV-1. The adherent conjunctiva impairs this kitten's ability to blink. (B) Symblepharon in a cat involving adhesion of the upper palpebral conjunctival to the lower which obscures the entire globe.



Figure 14.15 Lipogranulomatous conjunctivitis in an aged cat.

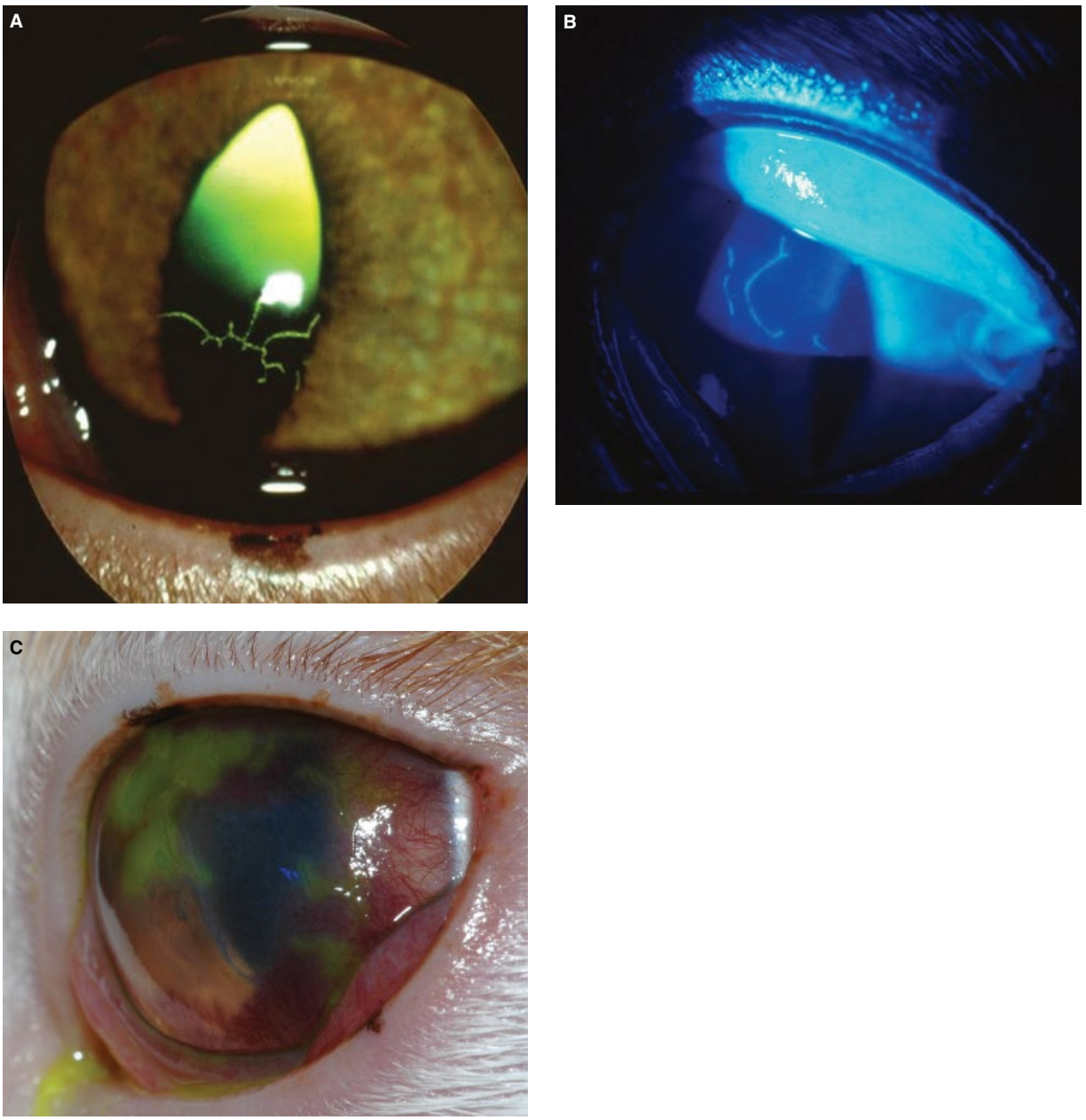


Figure 14.16 (A) Feline herpesvirus-1 can also cause corneal ulceration in the cat. Note the very fine dendritic corneal ulcers and the irregular distribution of these micro-ulcers. (B) Dendritic corneal ulcers associated with FHV-1 and stained with topical fluorescein and highlighted with a cobalt blue light filter. (C) Dendritic ulcers stained with fluorescein which are expanding into geographic ulcers in this cat.

assess depth and corneal thickness in those instances. Occasionally, corneal sequestration extends though the entire thickness of the stroma to Descemet's membrane.

Several treatment modalities have been recommended: (i) supportive medical therapy until spontaneous slough; (ii) superficial keratectomy; (iii) superficial keratectomy with palpebral conjunctival graft; and (iv) superficial keratectomy with a sliding corneoconjunctival graft. Recurrence can occur, especially if the sequestration was incompletely excised.

Proliferative Keratoconjunctivitis (Esosinophilic Keratitis)

Proliferative keratoconjunctivitis occurs most commonly in cats and is characterized by the development of single to multiple inflammatory masses and/or vasculature originating at the limbus (Figure 14.19). The conjunctiva, cornea, and the nictitating membrane are affected. Dermatologic signs of the eosinophilic complex are usually absent. Although the cause has not been resolved,

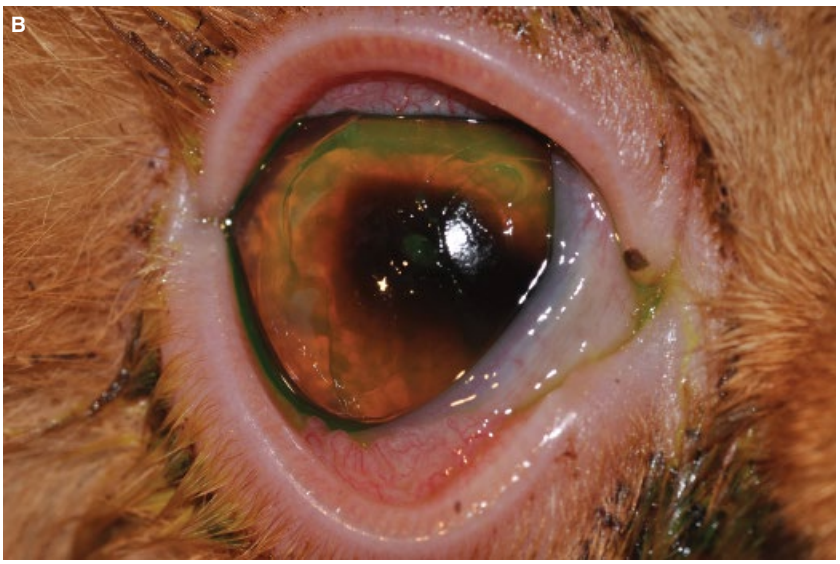


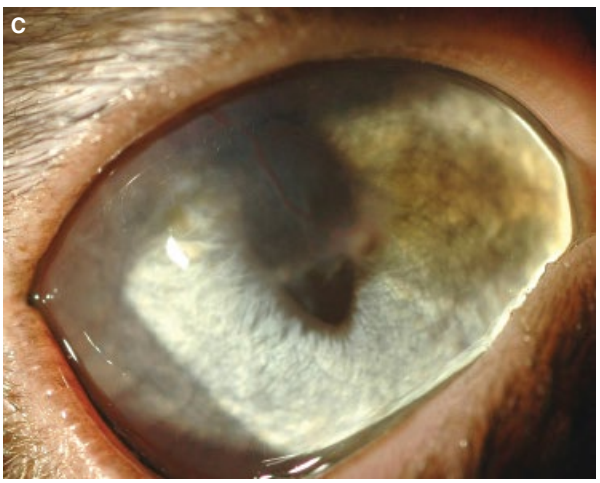


Figure 14.17 (A) Feline herpesvirus-1 stromal keratitis in a 3-year-old cat. Note the stromal edema and scarring with superficial vascularization. (B) Another example of chronic herpetic ocular disease in a cat. Note the corneal stromal vasculature, the conjunctival hyperemia and chemosis, and mild eyelid swelling.



Figure 14.18 (A) Corneal sequestration and corneal ulceration in a young cat. Note the black plaque in the base of the ulcer.





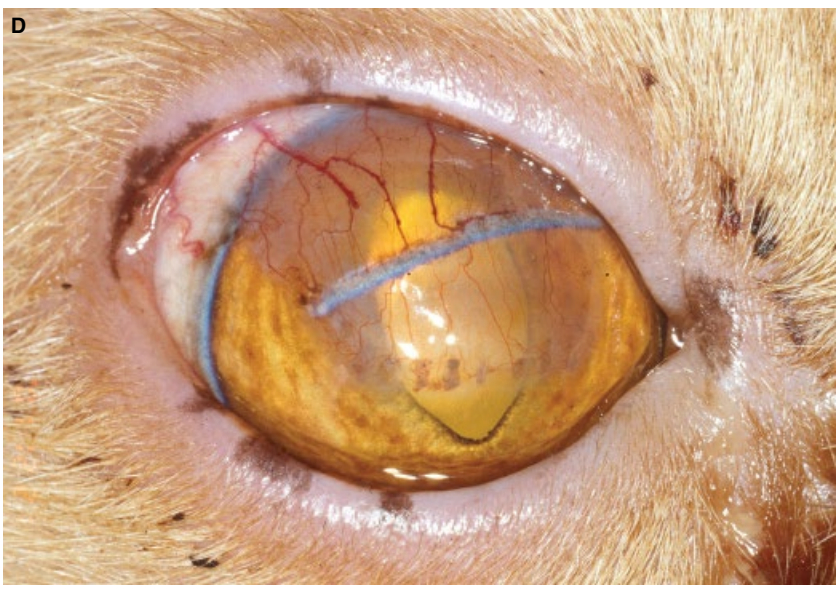


Figure 14.18 (*Continued*) (B) A larger corneal sequestration in a young cat. Note the central black sequestration and the surrounding superficial corneal ulcer. (C) Faint brown pigmentation of the cornea which is the earliest manifestation of sequestrum. (D) Postoperative appearance using a sliding corneoconjunctival graft dorsal of the surgical site.



Figure 14.19 (A) Proliferative (or eosinophilic) keratoconjunctivitis in a 4-year-old cat. Note the yellow–white masses at the limbus and within the cornea. (B) More extensive proliferative (or eosinophilic) keratoconjunctivitis. (C) Eosinophilic keratitis in a cat. Sometimes the entire cornea is affected.

polymerase chain reaction (PCR) results suggest at least 76% of the lesions are positive for FHV-1 DNA.

Clinically, there is conjunctival hyperemia, one or more gray to white limbal plaques, corneal stromal cellular infiltrates, and vascularization. The dorsolateral or lateral limbus is most often affected. Recommended treatment consists of topical corticosteroids or cyclosporine.

Florida Keratopathy

Florida keratopathy, or "Florida spots," occurs most commonly in Florida and the Caribbean region. The condition affects both cats and dogs and appears as focal gray–white opacities within the corneal stroma (Figure 14.20). Superficial corneal vascularization and inflammation are usually absent. One or both eyes are affected. Often established, the opacities are usually nonprogressive. The cause is unknown, but *Rhinosporidium* and *Mycobacterium* are suspected. Treatment is not necessary.

Acute Bullous Keratopathy

Acute bullous keratopathy has been recently reported in cats and consists of severe corneal edema and anterior uveitis, but no systemic disease (Figure 14.21). It resembles a melting ulcer, but the epithelium overlying the profoundly edematous cornea is typically intact. The cause of this disease is unknown, but it can progress to perforation. Treatment of choice is a nictitating membrane flap for 10–14 days and, if necessary, a conjunctival graft with or without a temporary tarsorrhaphy.

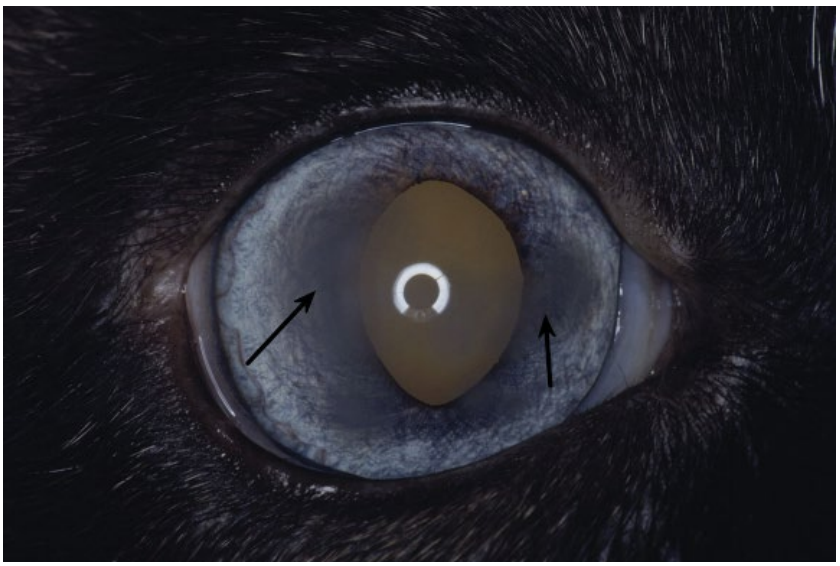


Figure 14.20 Florida keratopathy in a 3-year-old cat. Note the superficial corneal stromal opacities (arrows) have incited no inflammatory response.





Figure 14.21 (A) Acute bullous keratopathy in a young cat. The severe corneal edema is confined to the medioventral area of the cornea. (B) This cat has both a small area of bullous disease and a small sequestrum.

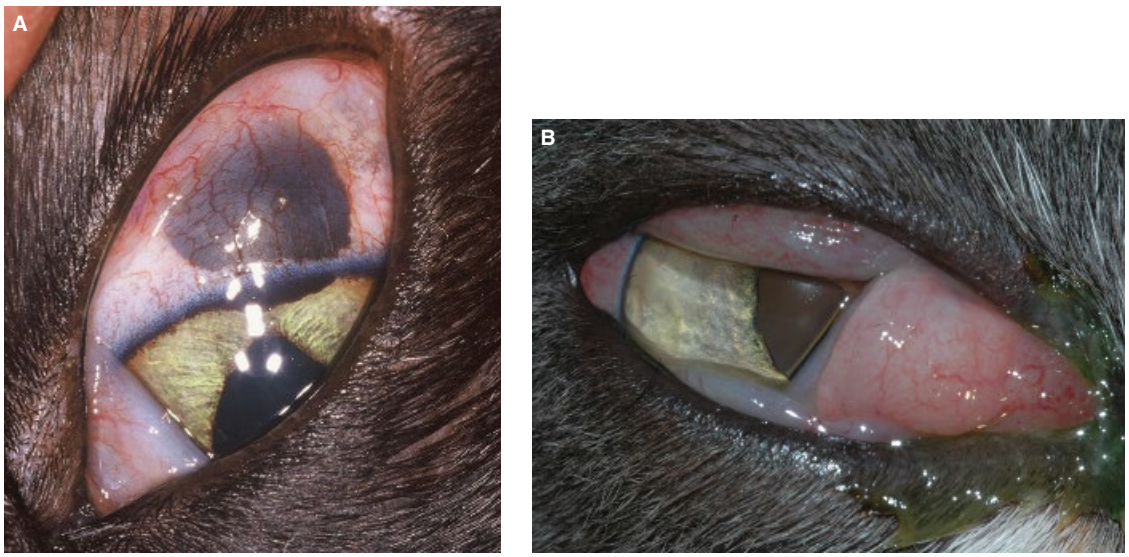


Figure 14.22 (A) Limbal melanoma in a cat. Located at the lateral canthus, the pigmented mass is confined to the dorsolateral sclera and limbus. These tumors are locally invasive, usually slow growing, and can be addressed surgically. (B) Conjunctival lymphoma in an aged cat.

Corneal and Conjunctival Neoplasia

Corneal and conjunctival neoplasms are rare in cats. Tumors of the nictitating membrane include adenocarcinoma, fibrosarcoma, and, most often, lymphoma. Limbal melanomas can occur (Figure 14.22). Squamous cell carcinomas and lymphomas are the most frequent tumors of the conjunctiva.

Diseases of the Iris and Ciliary Body

The domestic cat often develops iridocyclitis, frequently with life-threatening systemic diseases. As a result, patients with feline anterior uveitis are often subjected to comprehensive clinical pathology analyses and antibody titers.

Heterochromia Iridis

Heterochromia iridis primarily affects the Siamese and Himalayan, and white cats (either uni- or bilaterally) (Figure 14.23). Heterochromia iridis in the Siamese breed is associated with esotropia, decreased stereopsis, and nystagmus. In white cats, heterochromia iridis has been associated with deafness, causing bony alterations in the modiolus and membranous changes in the labyrinth.

In the Siamese breed, the major retinal pathways travel an anomalous route, with nearly all projections crossing to the pretectum and superior colliculus. The esotopia is thought to be caused by the limited ability to process spatially coordinated binocular information. The esotropia can also result from the suppression of the extensive crossing of the temporal fibers at the optic chiasm, and a conscious attempt to position the functional temporal fields into a more frontal position. Intermittant nystagmus is common, and easily detected. There is no treatment.



Figure 14.23 Heterochromia iridis in a white cat. Cats with both irides blue are often deaf.

Persistent Pupillary Membranes

Persistent pupillary membranes (PPMs) are rare in cats but can affect one or both eyes (Figure 14.24). As in other species, these arise from the collarette, and extend to other areas of the iris, the posterior cornea, or the anterior lens capsule. Breeds of cats predisposed to PPMs have not been reported. Generally, there is no treatment.

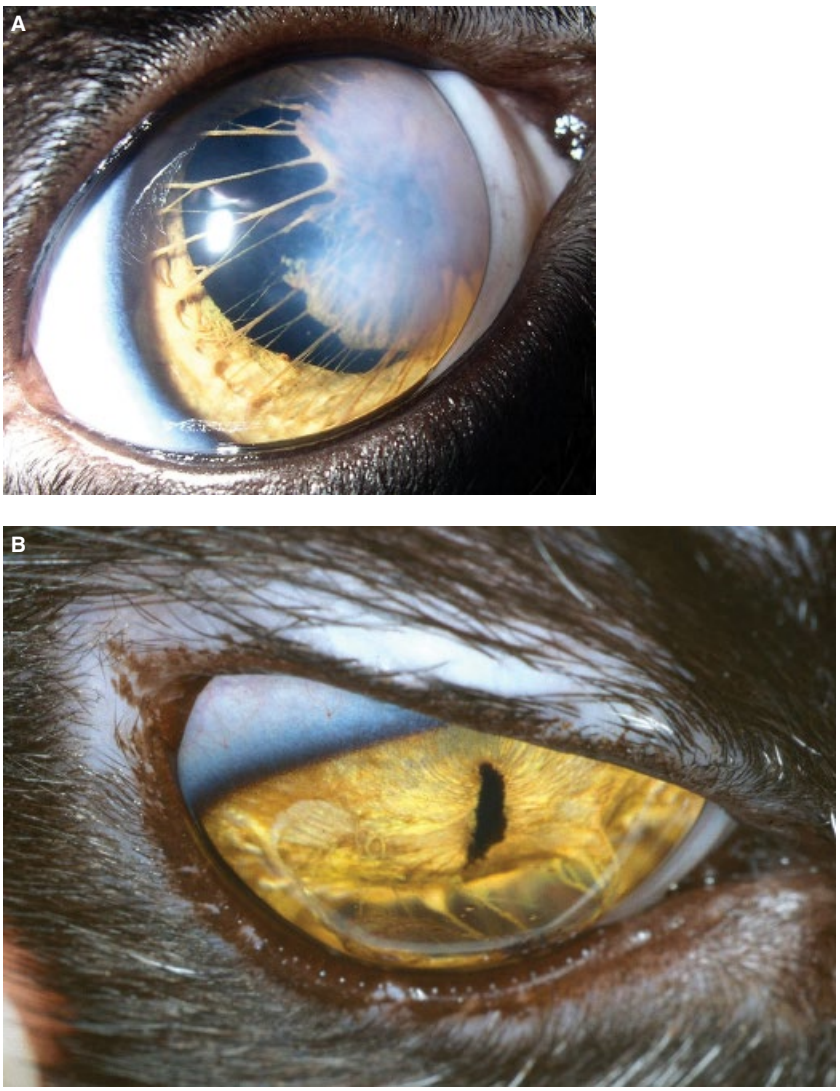


Figure 14.24 (A) Persistent pupillary membranes (PPMs) in a kitten. Note the PPM has caused a posterior corneal opacity. (B) Iris to cornea PPMs. Note the strands traversing the anterior chamber.

Iridocyclitis or Anterior Uveitis

A large percentage (30–70%) of young cats with anterior uveitis or iridocyclitis have systemic disease, and of these diseases, feline infectious peritonitis (FIP), feline leukemia (FeVL), and feline immunodeficiency (FIV) infections are the most significant (Figure 14.25). These iridocyclitides can be the first clinical signs noticed by the owner, and the primary reason for initial presentation to the veterinarian. Hence, for the feline iridocyclitides, a complete physical examination and serology of at least FeVL and FIV is recommended.

The clinical signs that occur with anterior uveitis in the cat are similar to those that occur in the dog. However, cats are not often as overtly or acutely in pain as dogs with uveitis. Blepharospasm occurs as well as ciliary flush and episcleral injection. The cornea can become edematous. Aqueous flare and cell, fibrin, hypopyon and hyphema can fill the anterior chamber and synechia can develop with time and severity. Iris color change and vessel proliferation develops with chronicity. Many cases of anterior uveitis in cats are idiopathic and chronic. These eyes should be monitored regularly because glaucoma is a common sequela to chronic uveitis.

Feline Infectious Peritonitis

FIP caused by coronavirus produces a chronic and progressive anorexia, depression, weight loss, fluctuating fever, with variable peritoneal and thoracic involvement and debility (Figure 14.26; also Figure 18.31). The ocular signs can develop initially or later in the course of the disease.

FIP produces a pyogranulomatous infection of both the anterior and posterior segments of the eye. The iridocyclitis is often initially mild but highly exudative. Both the keratic precipitates and hypopyon that develop contain large amounts of fibrin, blood cells, and inflammatory cells. Posterior synechiae develop rapidly and can be



Figure 14.25 (A) Rubeosis iridis in a young cat with systemic disease. (B) Anterior uveitis in this cat resulted in dyscoria, entropion uvea (inversion of the pupil margin), posterior synechia, inflammatory debris in the anterior chamber across the surface of the iris and attached to the corneal endothelium and lens capsule. (C) Uveitis in this cat has resulted in posterior synechia and iris bombé (iris stroma bulges into anterior chamber). There is aqueous flare and hyphema as well. (D) Chronic uveitis in this cat has resulted in iris color change (previously iris stroma was blue in color) and vessel proliferation.

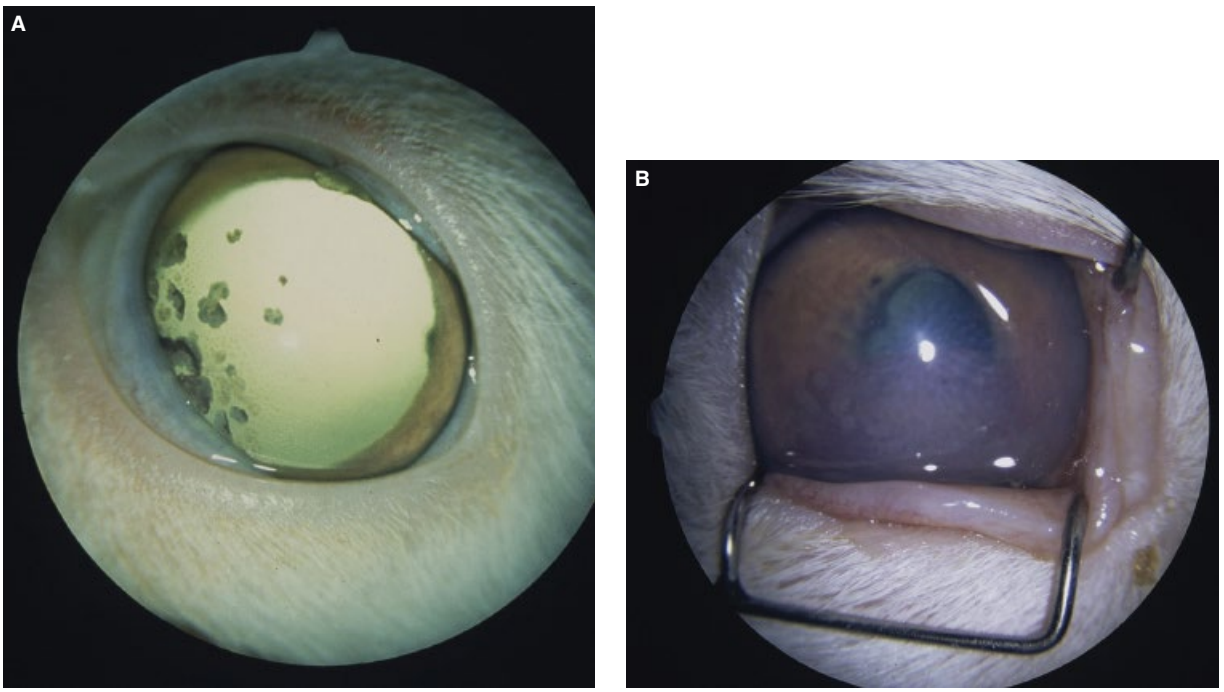


Figure 14.26 (A) Anterior uveitis with keratic precipitates in a young cat with feline infectious peritonitis. (B) The same cat as in Figure 14.26A several weeks later. The panuveitis has progressed to hypopyon and endophthalmitis, and examination of the ocular fundus is not possible.

followed by secondary glaucoma. The chorioretinitis is often overshadowed by the anterior segment inflammation, and may not be visualized through miotic pupils. The chorioretinitis is focal or generalized inflammation. Perivascular cuffing and occasional hemorrhages occur along the retinal vessels. As the uveal inflammation intensifies, endophthalmitis or panophthalmitis results.

As the coronavirus tends to cross-react with other viruses, the FIP test is used only as a guide. Hyperproteinemia (polyclonal gammopathy) is present in 50–70% of affected cats. Aqueous humor cytology reveals fibrin, blood cells, neutrophils, and monocytes. Survival after development of the systemic clinical signs is usually less than 12 months. Response to topical anti-inflammatory therapy for uveitis is poor.

Feline Leukemia

FeLV can affect all of the orbital and ocular tissues, and its clinical presentation (chronic and persistent inflammation to overt masses) is highly variable. Clinical diseases associated with FeLV include orbital masses, eyelid subcutaneous and subconjunctival masses, anterior uveitis, anterior uveal masses, secondary glaucoma, chorioretinitis, and retinal detachments (Figure 14.27).

The iridocyclitis is characterized by iris swelling, aqueous flare to keratic precipitates to hypopyon, prominent blood vessels on the iridal surface, and gray iridal nodules. As the iridocyclitis progresses, posterior synechiae, cataract formation, and secondary glaucoma develop. Masses secondary to FeLV appear as variable sized, white–pink masses within the iris and anterior chamber.

See also Figure 18.34A,B; also, Figure 19.7.

Feline Immunodeficiency

FIV affects the feline uvea causing a mild chronic anterior uveitis (Figure 14.28; also Figure 18.32). Clinical signs include iris swelling, aqueous flare, gray iridal nodules, inflammatory cells on the anterior lens capsule and anterior vitreous (pars planitis), posterior synechiae, and development of secondary cataract formation and glaucoma.

Toxoplasmosis

Toxoplasmosis is a frequent cause of anterior and posterior uveitis with retinal vasculitis in cats (Figure 14.29; also Figure 18.33). The anterior uveitis is variable in severity, and, like other chronic infectious uveitis, gray iridal nodules occur. Chorioretinitis and retinal hemorrhages along with retinal detachments frequently occur. Both secondary glaucoma and cataract formation can occur in chronic cases.

Diagnosis of toxoplasmosis is by paired serum samples; titers greater than 1:256 are considered positive. PCR tests (B1 gene) can be used for *Toxoplasma gondii*-specific immunoglobulins M and G (serum and aqueous humor

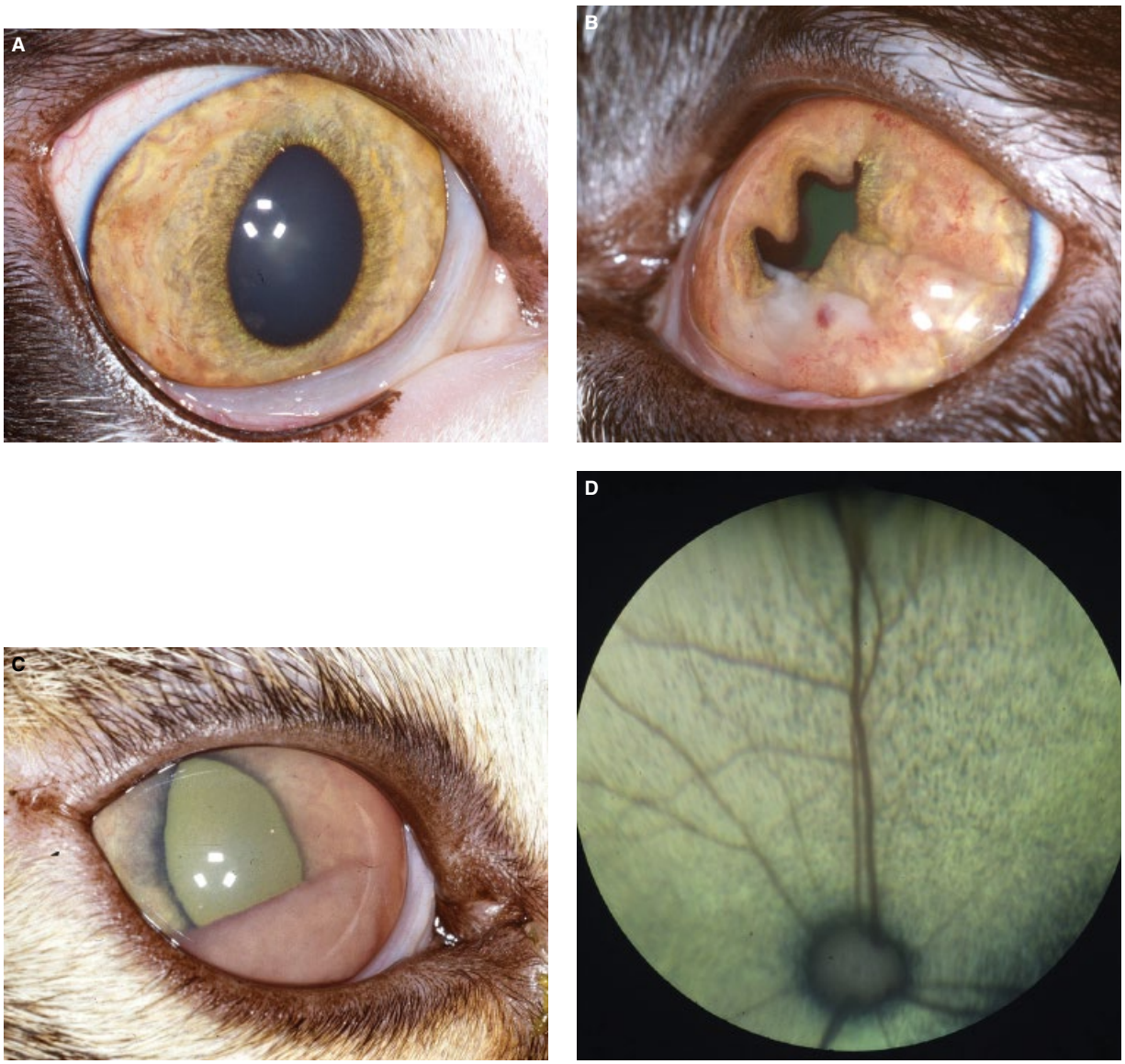


Figure 14.27 (A) Anterior uveitis in a cat with feline leukemia (FeLV). Note the dull character of the iris stroma and the neovascularization of the iris surface. (B) FeLV can affect all of the orbital and ocular tissues, and can appear as inflammation, discrete mass(es), or a combination of both. Change in iris coloration (blue to brown) or tan in a cat affected with FeLV. (C) Anterior uveitis, iridal thickening, and an anterior chamber mass with FeLV. This cat has developed lymphoma. (D) Retinopathy in a cat affected with FeLV. The brown spots in the tapetal fundus are either chorioretinitis or neoplastic infiltrates.

samples). Treatment usually includes topical mydriatics (usually 1% atropine ointment), systemic clindamycin (25 mg/kg/day in divided oral doses), topical corticosteroids, and occasional systemic corticosteroids.

Ophthalmic Trauma

Both blunt and penetrating trauma can produce anterior uveitis, often complicated with intraocular hemorrhage (hyphema and vitreal hemorrhages) (Figure 14.30). Penetrating cat scratch can lacerate the cornea and sometimes the anterior lens capsule. If lens material is extruded through the anterior lens capsular tear, lens removal may be necessary to prevent chronic lens-induced uveitis, and phthisis bulbus or secondary glaucoma.

Penetrating foreign bodies, such as cactus thorns, airgun or shotgun pellets, can traverse the cornea, traumatize the anterior lens capsule, iris, and deeper ocular tissues. If the iris is lacerated, hemorrhage and inflammation can result. Organic and iron foreign bodies are usually retrieved whereas those of lead or glass may be

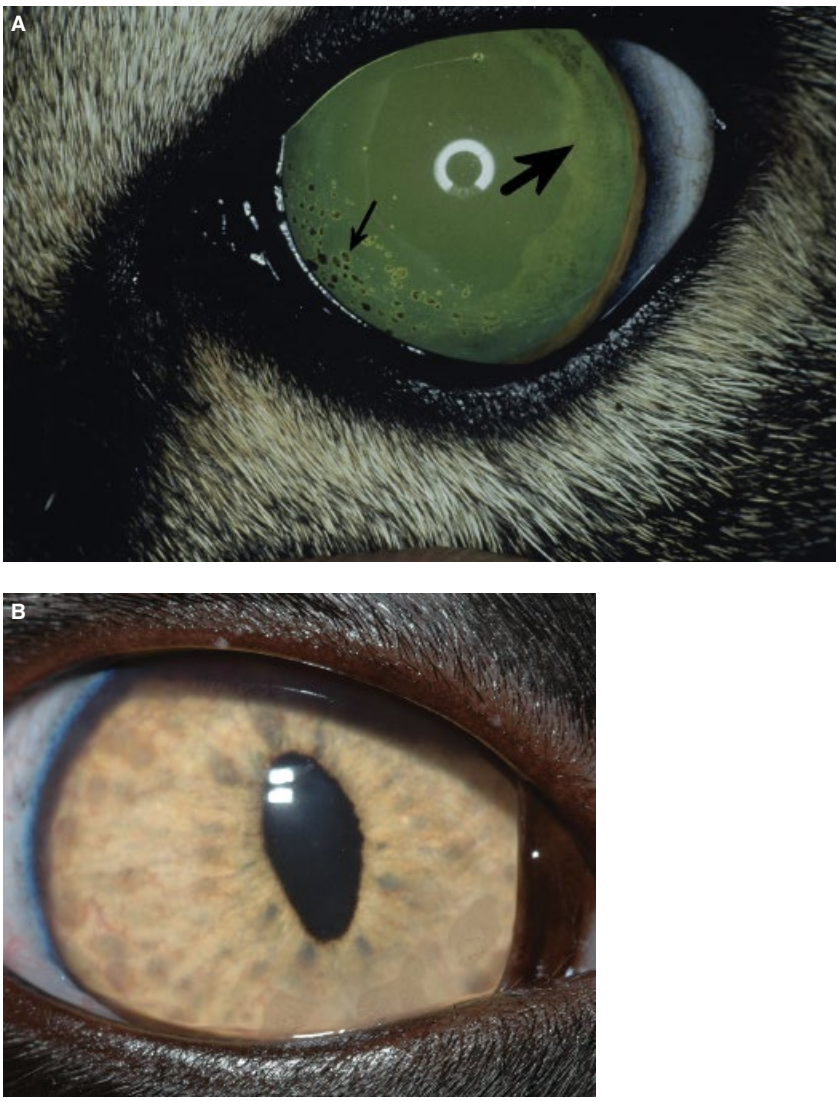


Figure 14.28 (A) In this 5-year-old cat the panuveitis was caused by feline immunodeficiency virus (FIV). Note pigment deposits on the anterior lens capsule (small arrow) that signal chronic iridocyclitis, and inflammatory cellular deposits on the posterior lens capsule (large arrow) that suggests pars planitis (inflammation of the caudal ciliary body). (B) Another cat with FIV. Note the iridal nodules on the its surface. These nodules are a darker color. Some iridocyclitis is also present. These patients are more apt to develop secondary glaucoma, the result of peripheral anterior synechiae and iridocorneal angle closure.

tolerated by the ocular tissues. Prognosis varies according to the extent of the trauma and response to therapy. Therapy consists of possible surgical removal, mydriatics (usually 1% atropine ointment), topical and systemic antibiotics, and corticosteroids.

Ocular Neoplasia

There are differences between the clinical characteristics of anterior uvea masses of the dog and cat. In the cat, the diffuse iridal melanoma which seems to arise from the anterior iridal surface is the most common manifestation of intraocular melanoma. In dogs, this tumor is more commonly nodular. Benign iridal melanosis in cats can precede the development of melanoma. It can be sometimes difficult to differentiate these two conditions. Intraocular lymphoma, often as a manifestation of generalized disease, is also common in cats.

Diffuse Iridal Melanomas

Diffuse iridal melanomas are the most frequent pigmented primary intraocular neoplasms in the cat (Figure 14.31). This tumor is characterized by progressive pigmentation on the iris surface in middle-aged and older cats. The time for this pigmentation to progress is quite variable and ranges from months to years. The pigmentation can eventually become invasive into the stroma and appear thickened. At this stage, it is defined clinically as a neoplasm and can impede pupillary shape, size, and/or movements. Glaucoma results from tumor growth into the outflow channels, and posterior segment involvement from tumor growth into the ciliary body.

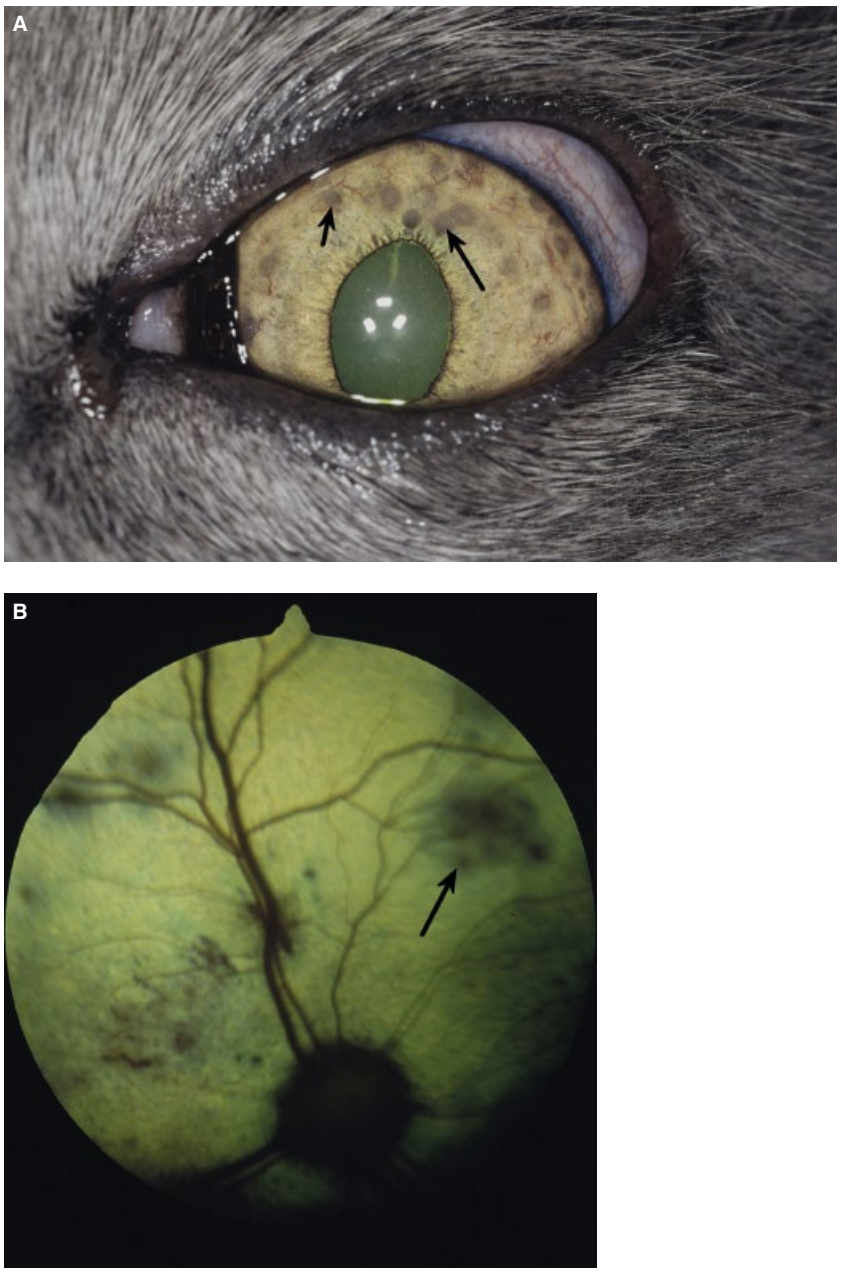


Figure 14.29 (A) Toxoplasmosis in a young cat has caused chronic panuveitis. Note the distinct gray nodules within the anterior iridal stroma (arrows). (B) Focal granulomatous chorioretinitis (arrow) in a cat caused by *Toxoplasma gondii*.

Metastasis occurs more readily than in dogs (5% in dogs and as high as 60% in cats), affecting the cat's liver and lungs (occurring as late as 1–3 years post-enucleation).

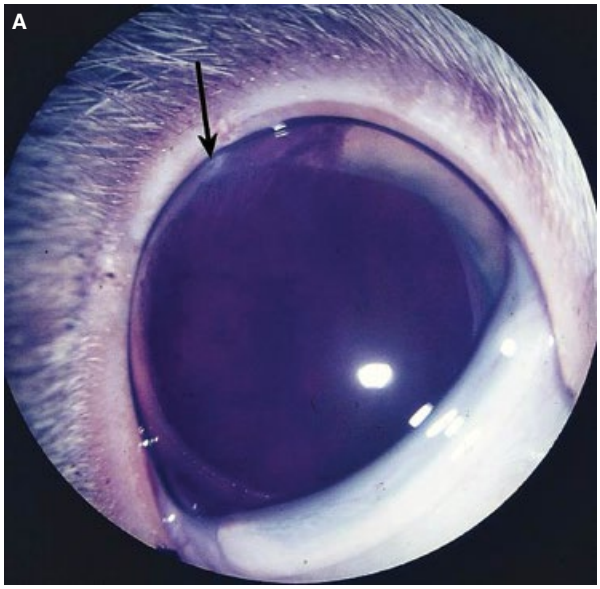
The variable progression of diffuse iridal melanoma presents a dilemma when determining the best time for enucleation of a visual eye. In general, once iridal thickening and pupillary abnormalities occur, enucleation is recommended.

Anterior Uveal Melanomas

A small group of anterior uveal melanomas appear similar clinically and histopathologically to those in the dog. These tumors present as gray to black vascular masses (amelanotic melanomas have been reported) protruding though the pupil, iris, and into the iridocorneal angle and ciliary cleft (Figure 14.32). The presenting signs are a color change in the eye (the appearance of the tumor), iridocyclitis, hyphema, enlarged globe (secondary glaucoma), and/or blindness. Treatment is enucleation.

Ciliary Body Adenoma and Adenocarcinomas

Primary ciliary body adenoma and adenocarcinomas in cats are rare (Figure 14.33). Most are nonpigmented tumors that originate from the ciliary pars plicata.





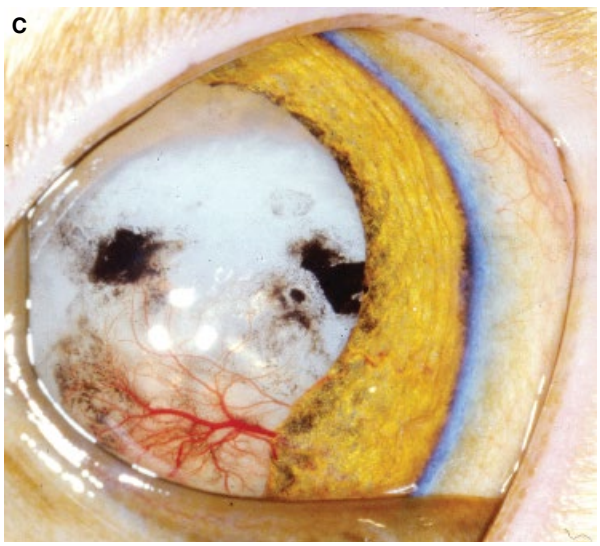


Figure 14.30 (A) Traumatic iridocyclitis and hyphema in a Domestic Shorthair male cat. The globe had been penetrated by a BB shot at the limbus (arrow). (B) Traumatic iridodialysis in a cat. Note the base of the iris had been torn from its base in the dorsolateral quadrant and part of it is adhered to the anterior lens capsule. (C) Traumatically induced cataract in a cat. Note the pigment deposition on the anterior lens capsule and the vessel migration onto the lens. This eye should be monitored for uveitis and the development of traumatic sarcoma.





Figure 14.30 (*Continued*) (D) Limbal laceration in a cat. The iris has prolapsed through the rent and is underneath the conjunctiva. (E) Penetrating laceration to the cornea in a cat following an encounter with another cat. The hemorrhage and fibrin in the anterior chamber signal full-thickness penetration. They have sealed the wound. The pupil has been pharmacologically dilated.



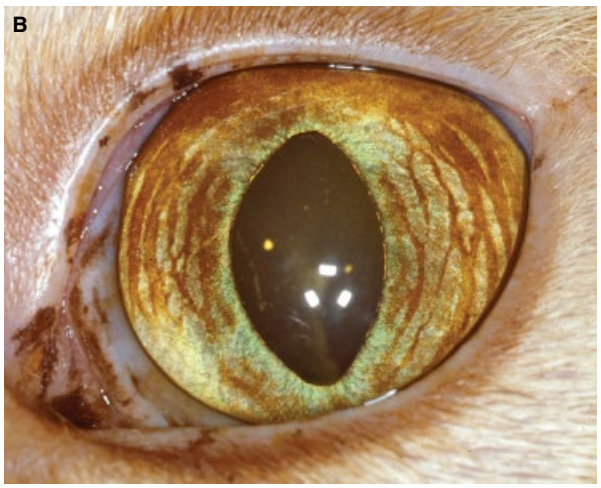
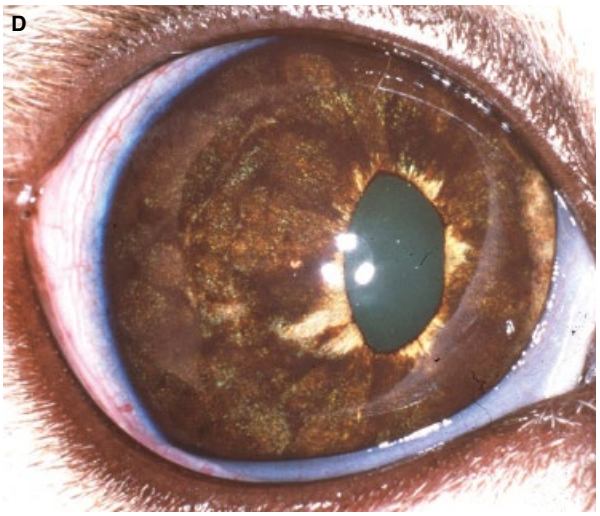


Figure 14.31 Diffuse iridal melanoma is unique to the cat and arises from the anterior surface of the iris. (A) Early diffuse iridal melanosis in a cat. Note one iris has become partially (about 50%) pigmented while the opposite iris is normal. Monitor at 1–2 month intervals. (B) Diffuse melanosis in a cat. It is very difficult to appreciate clinically when this benign lesion transforms into the malignant form.

Figure 14.31 (*Continued*) (C) Diffuse iridal melanoma in a cat. One iris is nearly completely pigmented while the fellow iris has few suspicious pigmented foci. Monitor at 1–2 month intervals. (D) Advanced diffuse iridal melanoma in a 10-year-old cat. The tumor has involved most of the iris and has distorted the pupil shape. Enucleation is recommended. (E) Another example of diffuse iris melanoma in a cat. This cat had elevated intraocular pressures (mild relative mydriasis) even though the eye does not look overtly glaucomatous. Enucleation is recommended.







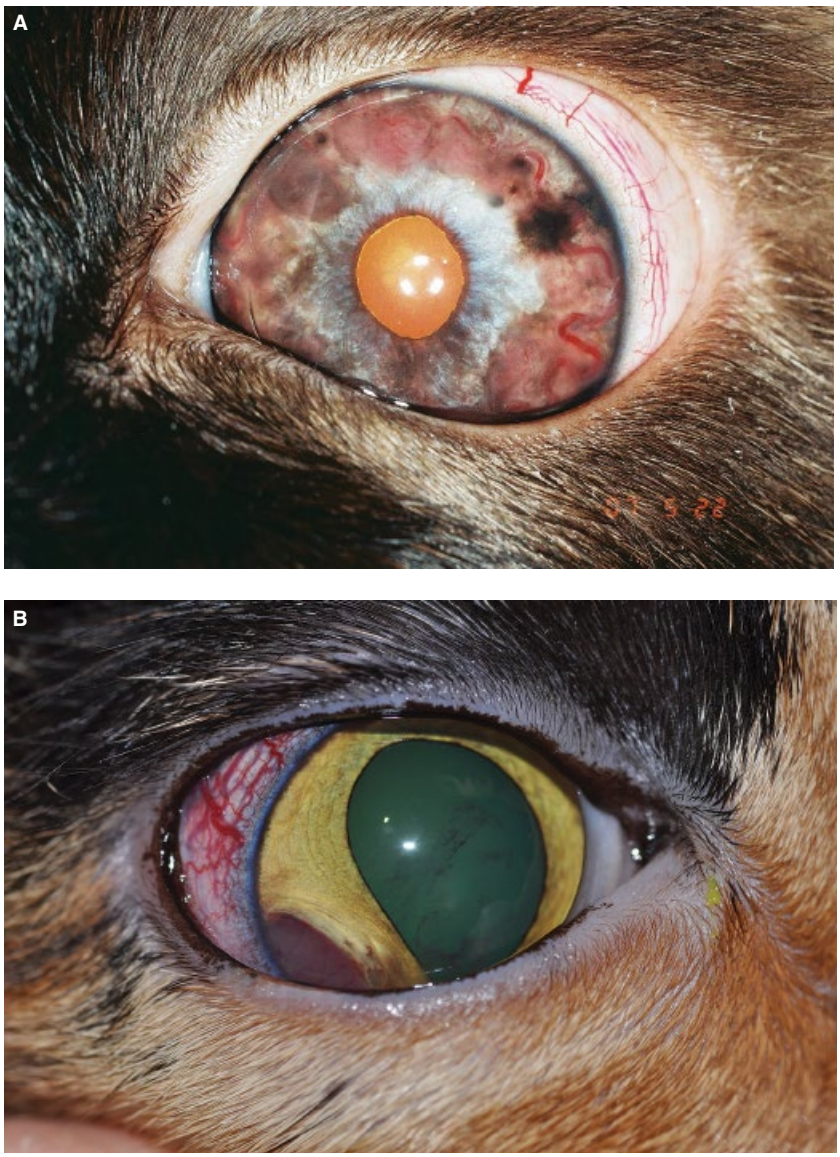


Figure 14.32 (A) An amelanotic uveal melanoma in a cat. (B) Another example of a nodular minimally pigmented melanoma in a cat originating at the base of the iris. There is intraocular hemorrhage and fibrin suggesting inflammation. The episcleral injection and mydriasis signal the development of secondary glaucoma.

These tumors present as a mass within the pupil, grow slowly, and often cause secondary glaucoma. Recommended treatment is enucleation.

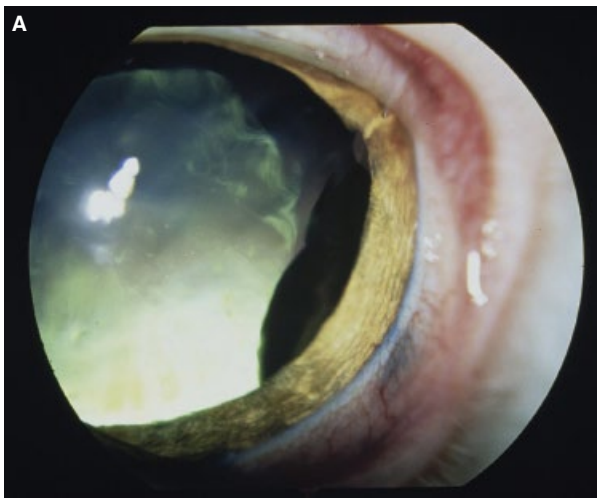
Trauma-associated Sarcomas

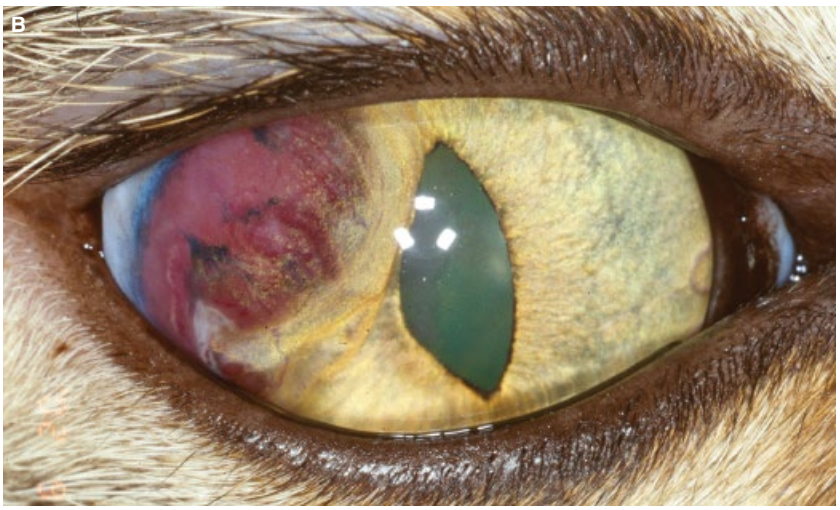
Trauma-associated sarcomas are the second most common of the feline intraocular tumors and appear unique to the cat (Figure 14.34). These masses occur in cats with a previous history of ocular trauma (average of 5 years post-trauma) and in middle-aged cats. Risk factors include trauma of the lens, chronic uveitis, intraocular surgery, and gentamicin destruction of the ciliary body for the treatment of absolute glaucoma. Clinical presentation is usually with chronic uveitis, intraocular hemorrhage, glaucoma, and a single to multiple white to pink mass in the anterior chamber or vitreous. As cartilage and bone formation can develop in this tumor, radiology and ultrasonography are useful diagnostic modalities. Early enucleation and exenteration of the orbit are recommended. Prognosis for long-term survival is guarded.

Systemic Lymphoma

Systemic lymphoma is the most frequent secondary tumor in cats, appearing as intraocular inflammation or masses, secondary glaucoma, and retinal detachment (Figure 14.35). Metastatic tumors in cats appear less frequently than in dogs, and squamous cell carcinomas are the most frequently reported tumor type.

In some cats, the presenting clinical signs are limited to the eye, and range from a color change to an abnormal pupil shape ("D" or "reverse D" pupil). In other cats,





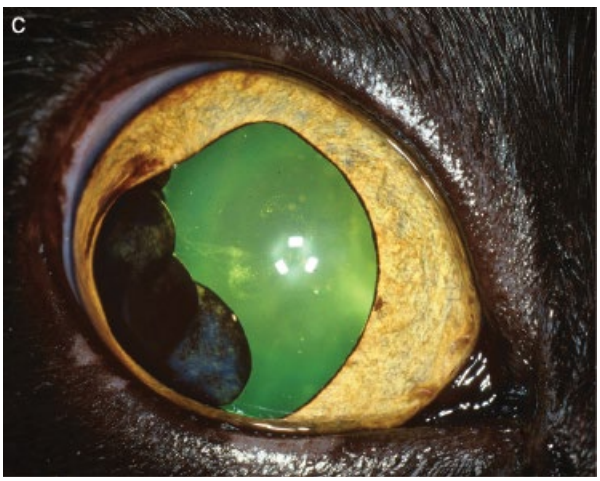
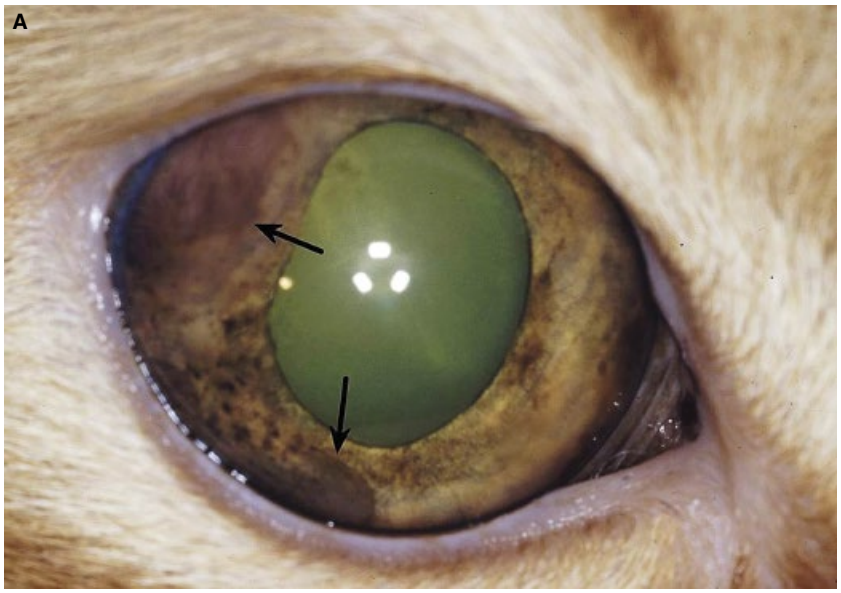


Figure 14.33 (A) Pigmented ciliary body adenocarcinoma of the ciliary body. The black tumor has protruded through the pupil permitting its detection. (B) Ciliary body adenocarcinoma protruding through the base of the iris into the anterior chamber. (C) Take care not to mistake a pigmented solid-tissue mass associated with the uveal tissue with a ciliary body or iris cyst. In most instances, cysts can be transilluminated. If they are heavily pigmented, ultrasound examination can assist with the differentiation. This is a benign uveal cyst in a cat.



Figure 14.34 Trauma-associated sarcoma in an older cat. The mass has infiltrated the majority of the globe and caused intraocular hemorrhage.



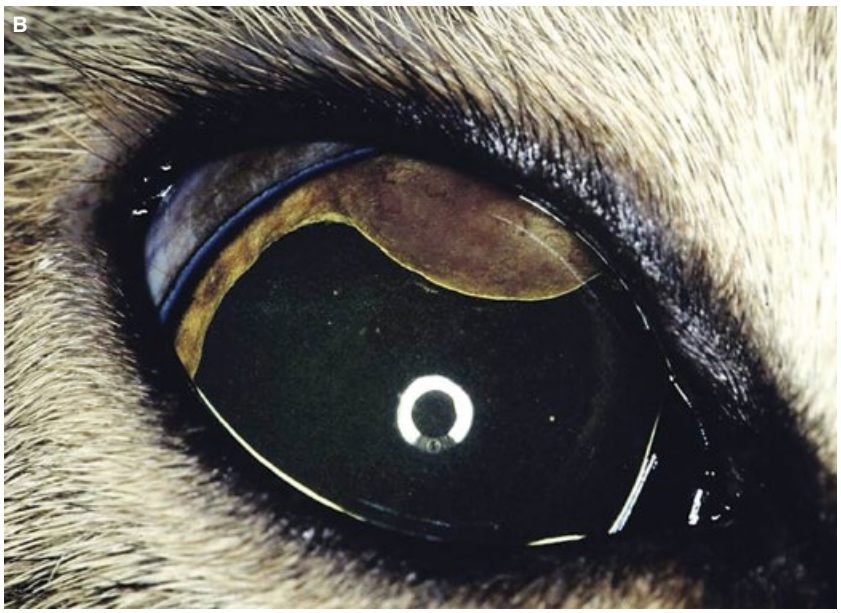


Figure 14.35 (A) Early anterior uveal lymphoma in a cat. Note the base of the lateral iris (arrow at 10 o'clock position), and small mass of blood vessels and cells (arrow at 6 o'clock position). (B) Iridal lymphoma in a 4-year-old cat. The raised pink mass has involved the dorsal iris.

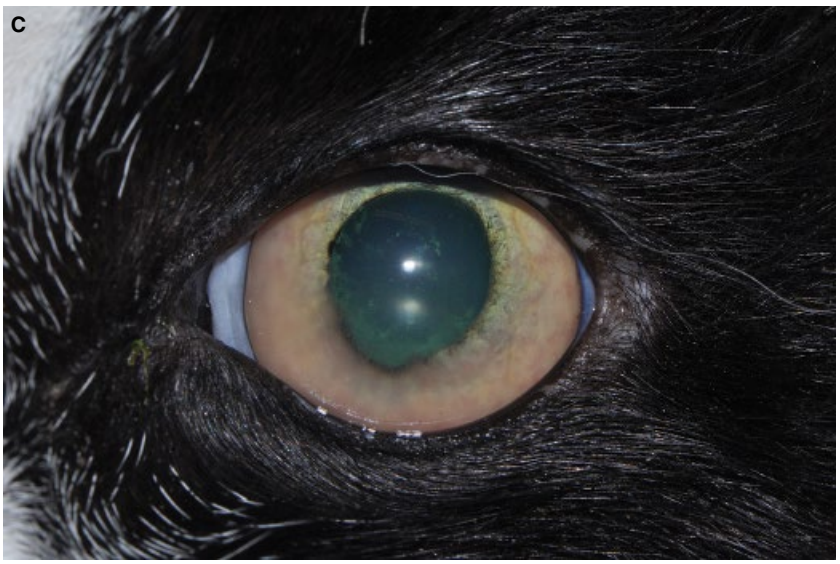




Figure 14.35 (*Continued*) (C) Lymphoma masquerading as uveitis. Note the distorted pupil with posterior synechiae and the swollen hyporeflective iris.
(D) Lymphosarcoma in a young cat. Note a large nonpigmented mass distorting the entire iris and filling the anterior chamber. The globe is buphthalmic and has developed an axial ulcer from exposure and the lids' inability to blink over this enlarged eye.

anterior uveitis with aqueous flare, keratic precipitates, hypopyon, or pink to white masses originating in the iris are present in the anterior chamber. Secondary glaucoma is a frequent complication, with obstruction of the aqueous outflow pathways with tumor and/or inflammatory cells. Treatment with topical corticosteroids can temporarily reduce the intensity of the anterior uveitis or the size of the anterior segment mass.

Feline Glaucoma

Although different forms of glaucomas occur in the cat, information about feline glaucomas is more limited than in dogs. Congenital glaucomas secondary to outflow anomalies are rare in kittens (Figure 14.36). One eye is usually affected, and a rapid enlargement of the globe



Figure 14.36 Bilateral congenital glaucoma in a kitten. Note the enlarged globe.

is the usual presenting sign. Treatment is usually not successful. As exposure of the cornea rapidly develops, enucleation is the usual treatment.

Primary glaucomas appear rare in cats, but the Siamese breed may be predisposed (Figure 14.37). They are affected with an inherited form of congenital glaucoma. Most cases of glaucoma in cats, however, are secondary to anterior uveitis or intraocular neoplasia. Clinical signs are similar to those in dogs and include mydriasis, corneal edema, conjunctival hyperemia and venous congestion (often hidden by the eyelids), globe enlargement, and lens luxation; however, generally signs are considerably more subtle and insidious. Atrophy of the iridal stroma can also develop. Retinal and optic nerve degeneration, and blindness eventually result. Cats present quite late in the course of the disease so prognosis for both control of intraocular pressure and long-term maintenance of vision is poor. Treatment is aimed at the primary disease (if one can be identified) and at lowering the intraocular pressure.

Aqueous misdirection glaucoma is an unusual form of glaucoma occurring occasionally in the cat (rarely in the dog) (Figure 14.38). In this condition, the entire lens–iris diaphragm is anteriorly displaced resulting in a very shallow anterior chamber and, thus, obstruction to aqueous outflow. In these cases the aqueous humor, which should normally flow between the iris and the lens into the anterior chamber, is directed posteriorly into the





Figure 14.37 (A) Buphthalmos and mydriasis in a cat with primary glaucoma. Note the corneal striae in the lateral cornea suggesting globe stretch. (B) Glaucoma and anterior uveitis in a cat secondary to FIV. Note the slight enlargement of the globe, episcleral congestion, corneal edema, and iridal swelling.





Figure 14.37 (*Continued*) (C) Iris bombé secondary to chronic anterior uveitis in a cat. No pupil is visible, and the forward displacement of the iris has nearly filled the anterior chamber. (D) Profound buphthalmos in a young cat with chronic uveitis related to FeLV. Note the significant recession of the iridocorneal angle evident dorsally (dark band along limbus), the dyscoria, and the cataract. Young animals have greater capacity for globe enlargement than do adults because of the larger percentage of elastic fibers in young sclera.



Figure 14.38 Aqueous misdirection in a cat. The lens and iris are forward resulting in a very shallow anterior chamber.

vitreal chamber and pushes the lens forward. Surgical removal of the lens and medical therapy for the elevated intraocular pressure is recommended.

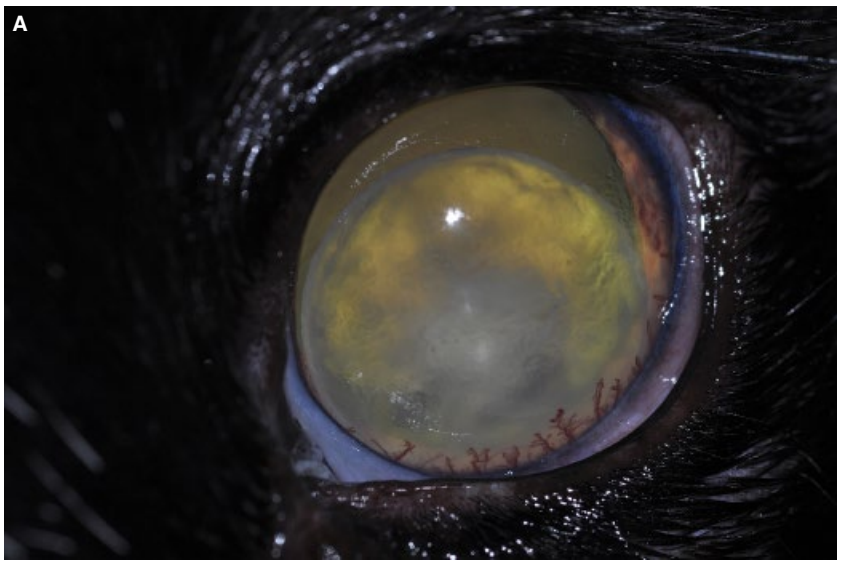
Cataracts and Luxations

Like in the dog, lens luxation or displacements usually occur secondary to uveitis or glaucoma in cats.

Primary and Secondary Lens Luxations

Primary lens luxation, in the absence of glaucoma and anterior uveitis, occurs occasionally in older cats (usually over 10 years of age) (Figure 14.39). The usual clinical history is that of a change in the iris color (the lens is luxated anteriorly masking the iris and pupil), or an enlarged pupil (better visualization of the tapetal reflection). Secondary glaucoma can result over time, presumably from anterior uveitis and damage to the aqueous humor outflow pathways. With anterior lens luxation, corneal edema can develop over time, from damage to the corneal endothelium from lens contact. Cats with primary lens luxation are candidates for lens removal which are most successful when removed early before extensive damage results.

Secondary lens luxations from globe enlargement (megaloglobus) are frequent sequelae from different forms of glaucomas. The loss of the zonulary attachments is believed to result from stretching and tearing of the zonules near their insertion on the lens equator. Chronic uveitis can also damage the lens zonules. The result is lens displacement, often as subluxation or into the anterior chamber. Tilting of the lens and an aphakic crescent are the usual clinical signs which accompany iridodonesis and phacodonesis. As most feline glaucomas are secondary to uveitis, posterior synechiae, iris tissue deposits on the anterior lens capsule, and cortical cataract formation are other findings. In visual eyes, removal of the luxated lens can prolong vision and facilitate medical control of the anterior uveitis and intraocular pressure.



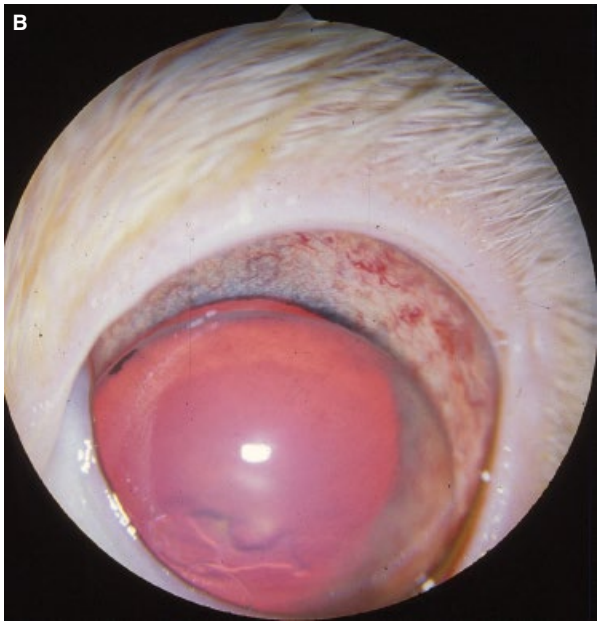




Figure 14.39 (A) Anterior lens luxation in a cat. Note the lens within the anterior chamber has caused some corneal edema. Early cataract is developing. (B) Anterior luxation of a minimally cataractous lens in a cat. Note the rubeosis iridis that signals anterior uveitis. (C) Anteriorly luxated hypermature cataract in an old cat. Note the cataract is in front of the pupil and iris.

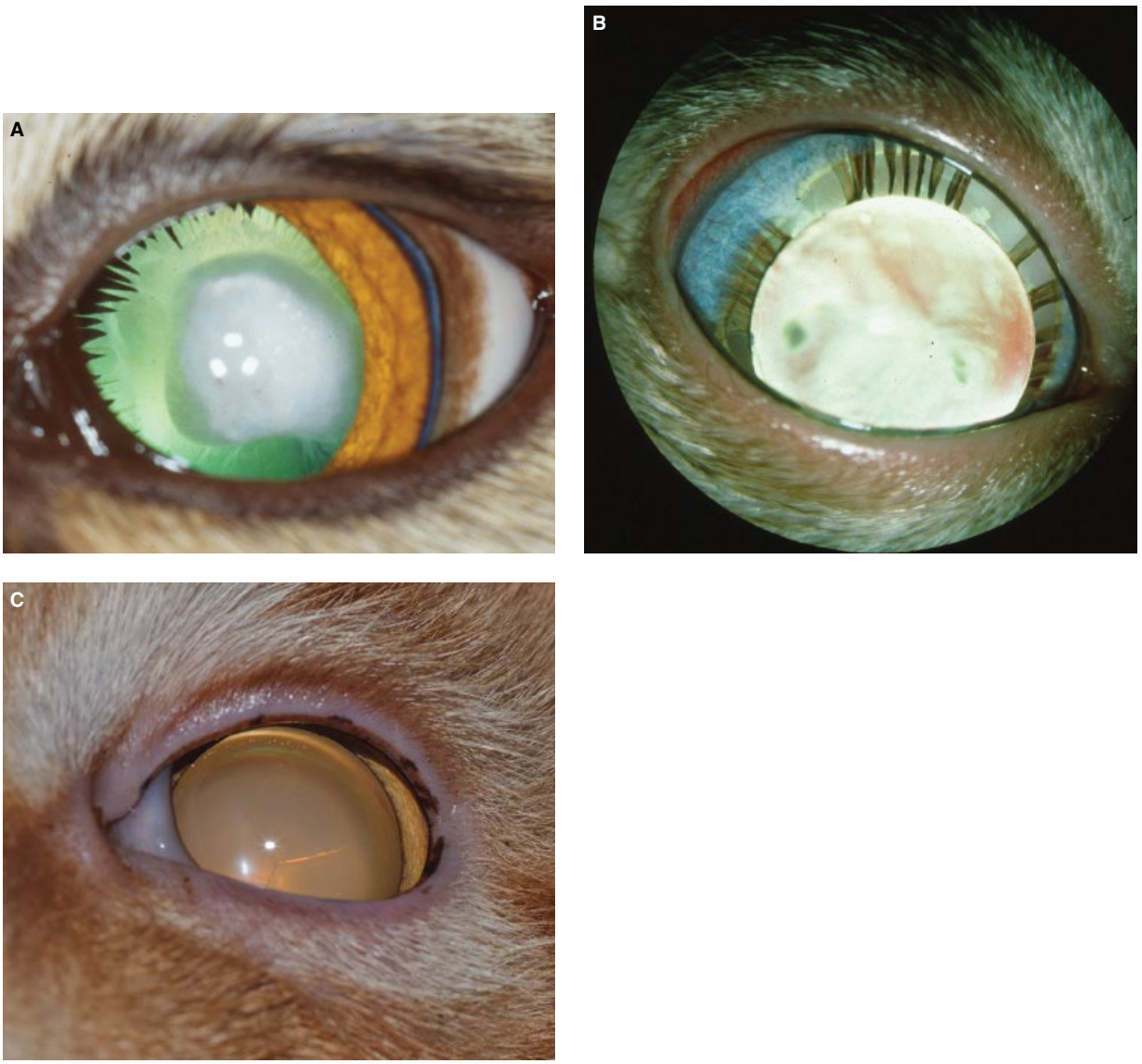


Figure 14.40 (A) Resorbing hypermature cataract in a kitten. The condition was congenital and bilateral. (B) Microphakia and early cataract in a cat. Elongated ciliary processes are observed at the lens equator. (C) Immature cortical cataracts in a young cat. Note the prominent suture lines.

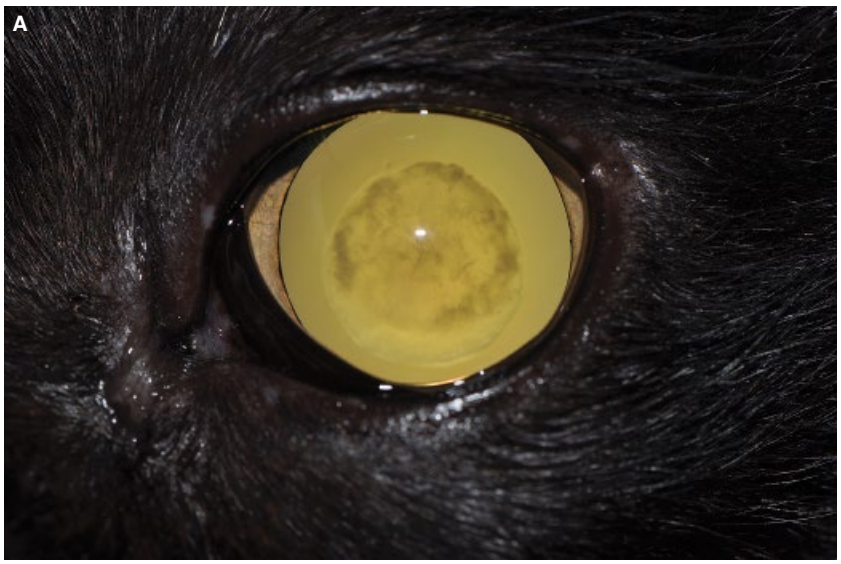
Primary Cataracts

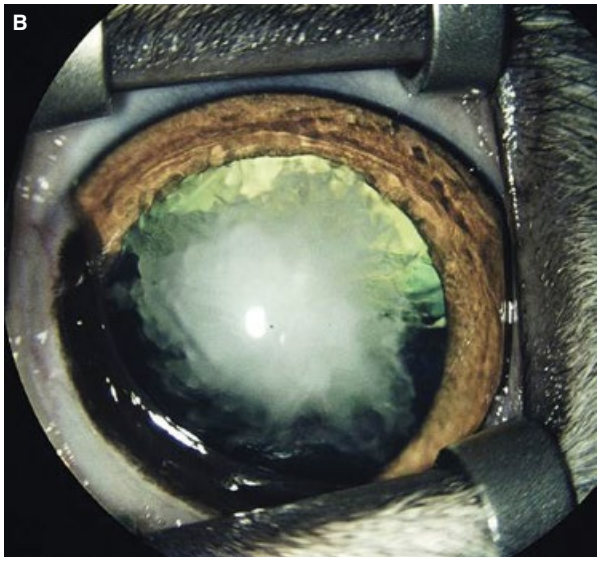
Congenital cataracts occur infrequently in kittens, and many are associated with multiple ocular defects (Figure 14.40). Breeds affected include the Persian, Birman, and Domestic Shorthair. Treatment is removal of the cataract by phacoemulsification.

Primary or inherited cataracts are rare in cats (in contrast to the dog) and currently all those reported are congenital forms (Figure 14.41). Even when bilaterally affected, cats are infrequently presented for cataract removal surgery.

Secondary Cataracts

Cataracts secondary to anterior uveitis and synechiae formation are the most common type encountered clinically, and occur most often with chronic uveitis (Figure 14.42). Hence, the signs of uveitis are present: variable corneal edema, irregular pupil (usually from posterior synechiae), iris tissue deposits on the anterior lens capsule, inflammatory membranes on the iridal surface, pupil and anterior lens capsule, and accompanying cortical cataract formation. The anterior uveitis can also weaken the zonulary attachments to the lens, resulting in





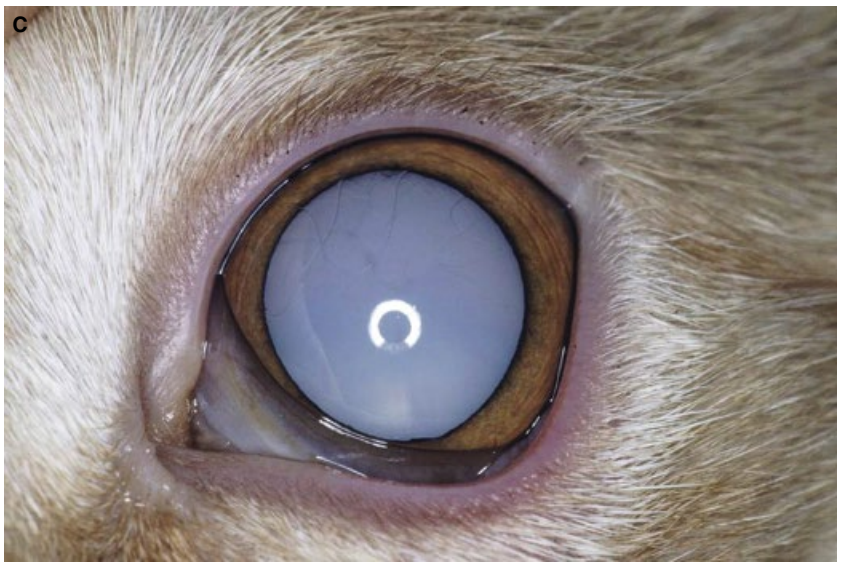


Figure 14.41 (A) Nuclear cataract in a 2-year-old cat. (B) Nuclear cataract that is resorbing in a 1-year-old cat. Lens-induced secondary anterior uveitis is also present. (C) Primary mature cataract in a cat. Both lenses were involved.

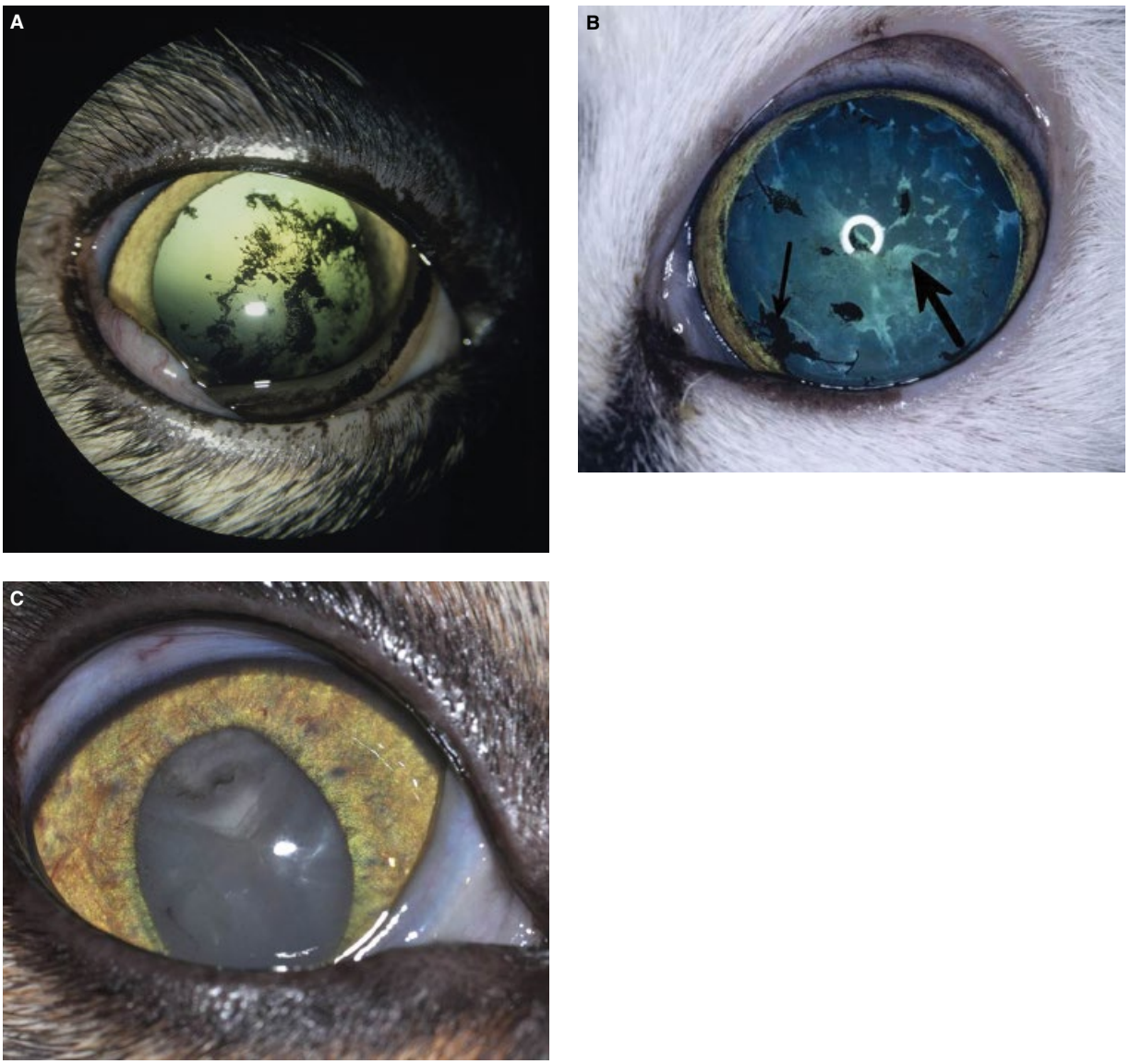


Figure 14.42 (A) Cataract secondary to chronic iridocyclitis in a cat. Note pigmented remnants of the posterior iris on the anterior lens capsule. (B) Cataract formation and chronic iridocyclitis in a cat. Note the pigmented tissue (small arrow) on the anterior lens capsule and cortical areas of cataract formation (large arrow). (C) Mature cataract in a cat secondary to chronic anterior uveitis.

subluxation or complete luxation. Cats with these cataracts may be candidates for lens removal, provided the anterior uveitis is under medical control and the possibility for the return of vision is reasonable.

Diseases of the Ocular Fundus

"Normal" ocular fundus

The normal feline ocular fundus has both a tapetal fundus (perhaps a little more reflective than the dog, and either orange or green–yellow) and a nontapetal fundus (usually brown to black), as well as retinal vasculature (usually three pairs of primary retinal arteries and veins), and the small optic nerve head (located in the tapetal fundus) (Figure 14.43). The color of the tapetal fundus can vary as kittens develop; it obtains adult coloration by about 16 weeks of age. Cats with blue irides may be atapetal, and the entire ocular fundus is usually subalbinoid. About 3 mm lateral of the optic disc is the area centralis, a cone-rich area.

The nontapetal fundus color results from the pigment within the retinal pigment epithelium, and is usually brown to black. In cats with white and lightly pigmented hair coat, the minimal pigmentation of the nontapetal fundus sometimes reveals the deeper choroidal blood vessels ("tigroid" nontapetal fundus). The optic disc is round, slightly depressed, relatively smooth, nonmyelinated, and often surrounded by a pigmented rim. The feline



Figure 14.43 (A) Normal feline ocular fundus in a Shorthair Domestic cat at higher magnification to view the optic nerve head. Note the round optic disc within the tapetal fundus, surrounded by pigment, and several primary retinal arterioles and veins. (B) Normal feline ocular fundus in a cat with blue irides. The ocular fundus contains no tapetal zone and has limited pigmentation (sub-albinoid fundus). (C) Normal feline ocular fundus in a cat. Note the nontapetal fundus has reduced pigmentation permitting visualization of the gradual transition of the tapetal and nontapetal fundi. (D) Normal feline ocular fundus. Note the nontapetal fundus has a focal area devoid of pigmentation of the retinal pigment epithelium and choroid which permits visualization of the choroidal blood vessels. (E) Normal feline ocular fundus.

vasculature is holangiotic and consists of three or more pairs of primary cilioretinal arteries and veins that emerge at the optic disc's periphery.

Retinal Dysplasia

Retinal dysplasia is rare in kittens (Figure 14.44). Retinal dysplasia and optic nerve head colobomas occur in association with eyelid agenesis. Retinal dysplasia can also occur in the developing eye with intrauterine infections with panleukopenia and FeLV viruses. Retinal dysplasia most often affects the central fundus directly above the optic disc. Retinal dysplasia appears as focal areas of variable pigment proliferation and tapetal hyperreflectivity.

Nutritional (Taurine) Retinal Degeneration

Retinal degeneration caused by taurine deficiency has been investigated for about five decades in the cat and it has been established that taurine is an essential amino acid for the cat (Figure 14.45). It was first described as feline central retinal degeneration (believed to be inherited), but was later recognized as dietary in origin. The first affected cats had poor quality, dry dog food diets. As cats have limited ability to synthesize taurine from cysteine, commercial cat foods were changed to contain about 500–750 ppm taurine, and the disease has now nearly disappeared. However, this retinopathy is still being diagnosed, usually in feral and zoo cats.

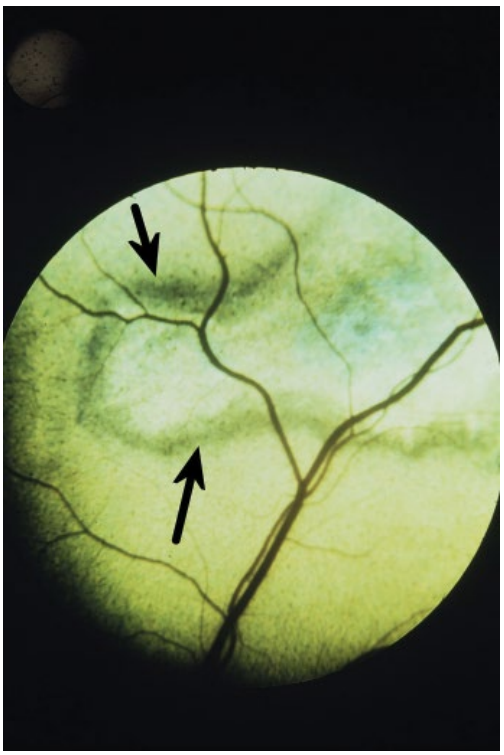


Figure 14.44 Retinal dysplasia in a kitten secondary to intrauterine infection with feline panleukopenia. The dysplastic area is dorsal of the optic disc (arrows), within the tapetal fundus, and consists of focal hyperreflectivity and pigmentation. Courtesy of Alan MacMillan.

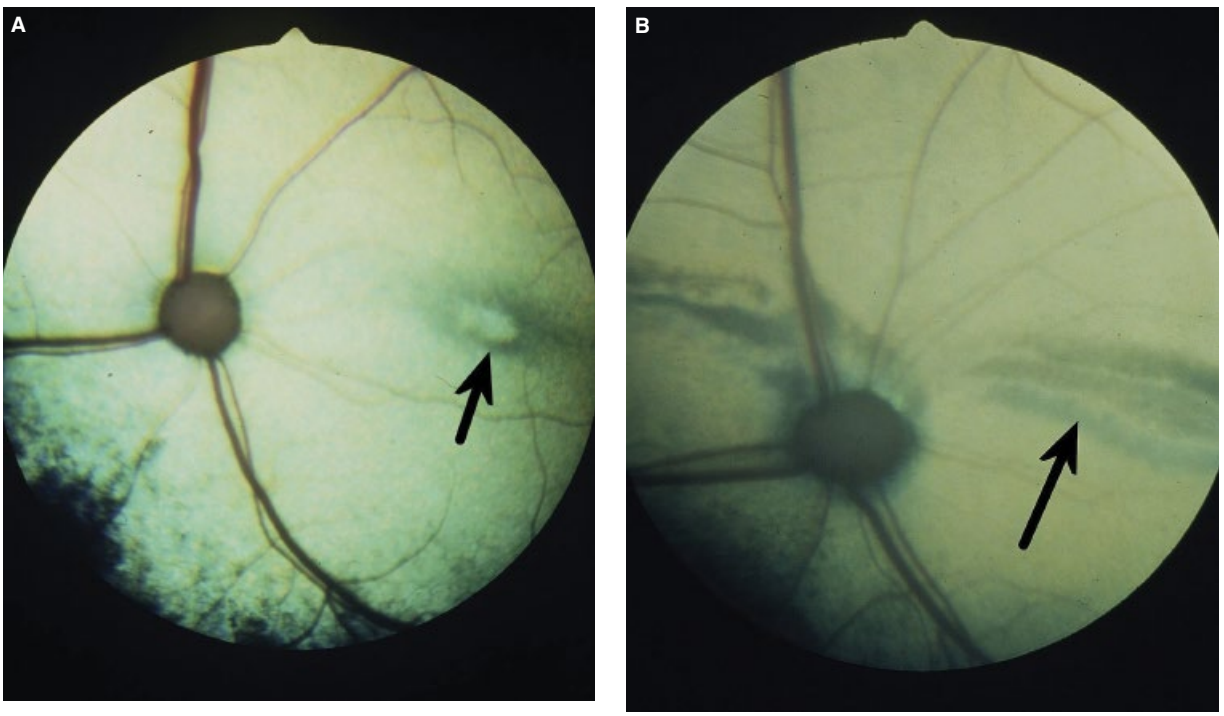


Figure 14.45 (A) Early taurine retinopathy in a young cat. Note a focal area of increased reflectivity (arrow) surrounded by a ring of pigmentation dorsal of the area centralis. (B) Intermediate taurine retinopathy in a cat. Note the area of increased reflectivity (arrow) and ring of pigmentation has enlarged and extended over the optic disc and into the area centralis.

Diets deficient in taurine can cause low plasma and retinal amino acid levels within 5 weeks. By 10 weeks the electroretinogram indicates both rod and cone abnormalities, which are potentially reversible. However, the ophthalmoscopic changes seem to persist even if dietary taurine levels are returned to normal. Low taurine levels have also been associated with cardiomyopathy. The ophthalmoscopic disease has been divided into five stages and the early phases (1A and 2B) are considered pathognomonic for taurine deficiency.

Inherited Retinal Photoreceptor Disorders

Two forms of retinal degenerations are inherited in the cat and both occur in the Abyssinian breed (Figure 14.46). Rod–cone dysplasia affects kittens as young as 4 weeks of age (mydriasis and nystagmus) and is inherited as an autosomal dominant trait. The first ophthalmoscopic changes occur in affected 8- to 12-week-old kittens and appear as increased tapetal reflectivity, reduced pigmentation of the nontapetal fundus, and loss and attenuation of the retinal blood vessels.

Another form of rod–cone degeneration is inherited as an autosomal recessive trait in the Abyssinian breed. Cats are affected at 1–2 years of age, and the condition is inherited as an autosomal recessive trait. The clinical and ophthalmic signs are similar to rod–cone dysplasia.

Chorioretinitis and Retinochoroiditis

Inflammation of the feline choroid and retina are not infrequent and occur most often with infectious diseases.

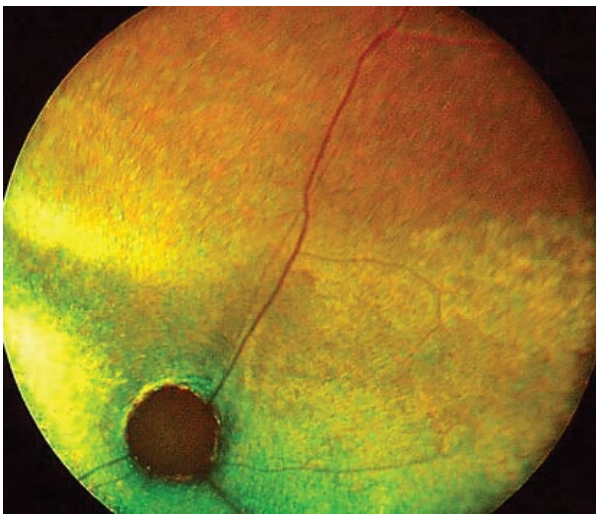


Figure 14.46 The two inherited retinal photoreceptor dysplasia and degeneration in an Abyssinian cat appear ophthalmoscopically similar. Note the retinal degeneration is advanced with generalized increased tapetal reflectivity and loss of the retinal vasculature.

FIP causes a pyogranulomatous panuveitis, retinitis, and optic neuritis. The clinical presentation is usually because of the anterior uveitis; however, ophthalmoscopic examination can assist in the diagnosis. In its early stages, cats with FIP present with perivascular cuffing, focal chorioretinitis, and then eventually diffuse chorioretinitis (Figure 14.47; also Figure 18.31). The retinochoroiditis exudates may be extensive, resulting in exudative retinal detachments.

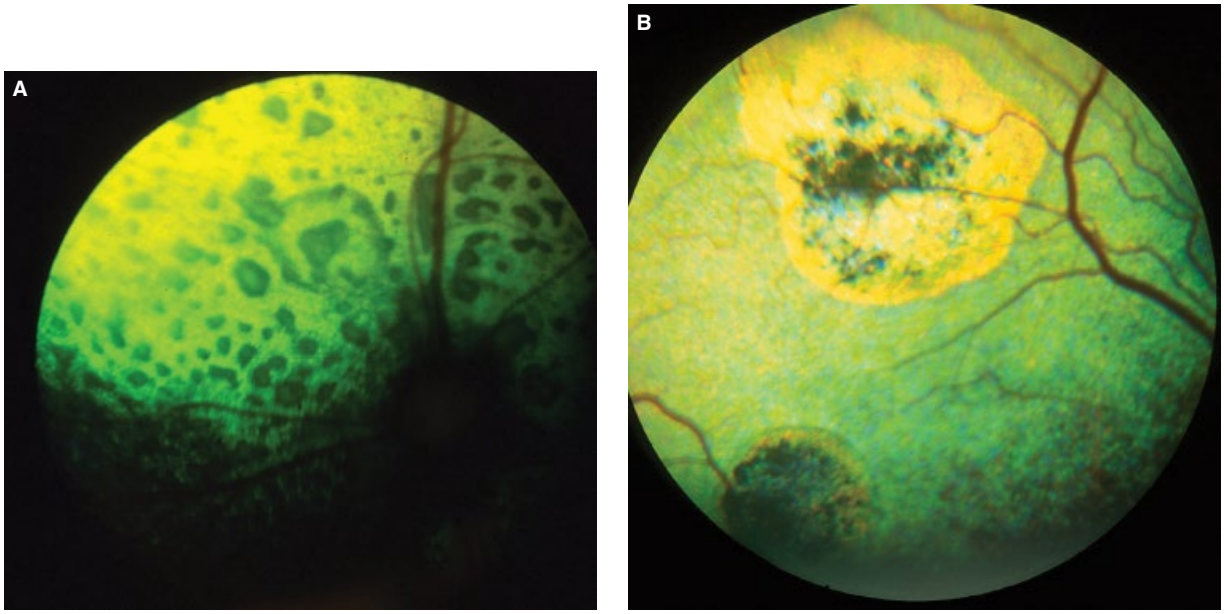


Figure 14.47 (A) Multifocal chorioretinitis in a cat with small bullous elevations of the retina and hyporeflective foci in the tapetal fundus. (B) Inactive chorioretinitis in a cat. Note the distinctly marginated foci of hyperreflectivity and pigment deposition.

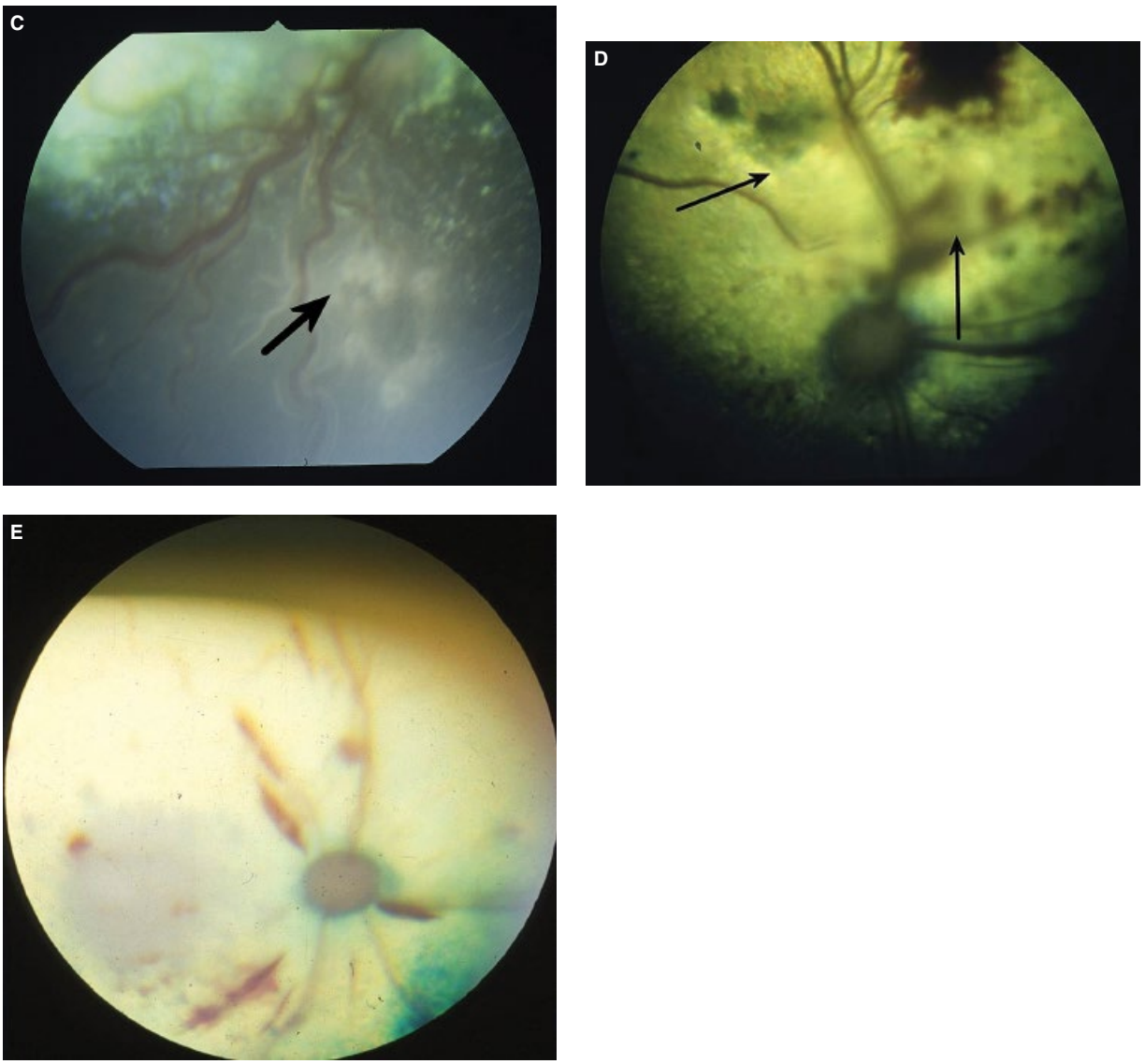


Figure 14.47 (*Continued*) (C) Chorioretinitis in a cat secondary to FIP. Note the chorioretinitis (arrow) within the nontapetal fundus, and perivascular areas of inflammation. (D) Chorioretinitis in a cat with FeLV. Note the round areas of intraretinal exudates in the tapetal fundus (top arrow). The dorsal retinal artery is displaced immediately above the optic nerve head, suggesting exudative retinal detachment (bottom arrow). (E) Chorioretinitis in a cat with FeLV. This cat was anemic and systemically compromised.

Ocular Fundus Mycosis

Cryptococcus neoformans in our experience may be the most frequent mycosis in cats, and is most often associated with nasal and sinus cavity granulomas. It also affects the feline optic nerve and retina, and is apparently an extension of CNS cryptococcal infections (Figure 14.48; see also Figure 18.35). Blindness and mydriasis can be the presenting clinical signs and can precede any CNS signs. Ophthalmoscopically, multifocal chorioretinitis, large retinal granulomas, and exudative retinal detachments occur. Treatment is costly, and the prognosis is guarded because with optic nerve involvement the possibility of the return of vision is low.

Hypertensive Retinopathy

Hypertensive retinopathy is not infrequent in older cats (>10 years). Most cats present to the veterinarian because of intraocular hemorrhage (hyphema and vitreal hemorrhage), mydriasis, or blindness (usually from retinal detachments) (Figure 14.49; see also Figure 18.37). The hypertension is usually secondary to some other

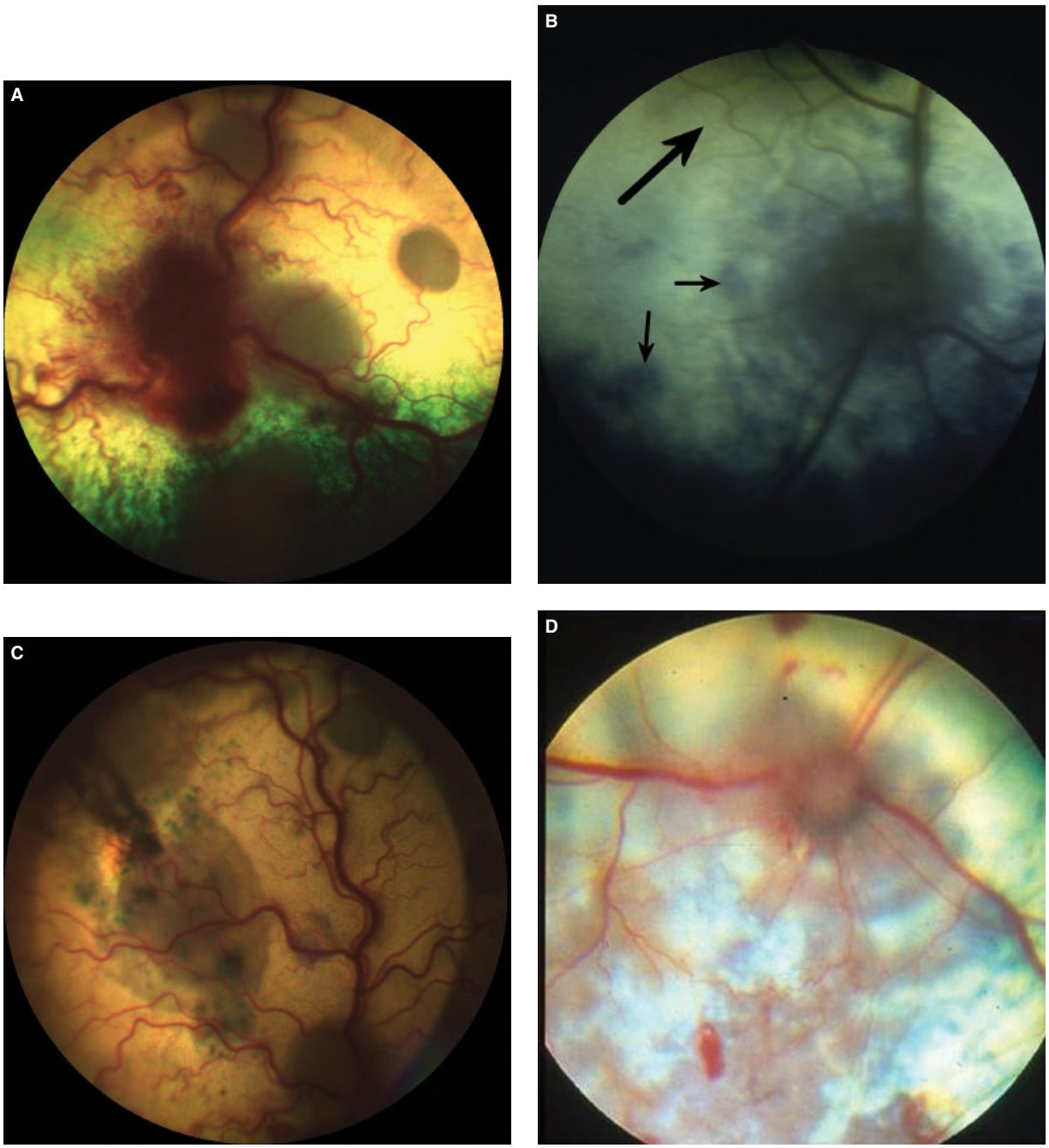


Figure 14.48 (A) Chorioretinitis secondary to cryptococcosis in a cat. Note several areas of granulomatous chorioretinitis within the tapetal and nontapetal fundi. There is also significant subretinal hemorrhage. (B) Chorioretinitis secondary to cryptococcosis in a cat. Note the peripapillary granulomas (arrows) that surround the entire optic nerve head. (C) Chorioretinitis secondary to cryptococcosis in a cat. This represents a more chronic inflammatory response to cryptococcosis. There are retinal elevations, multifocal granulomas, and some intra- and sub-retinal hemorrhages. (D) Chorioretinitis in a cat secondary to histoplasmosis. Note the diffuse changes to tapetal reflectivity, the peripapillary elevation of the retina, optic nerve swelling, and sub-retinal hemorrhages.

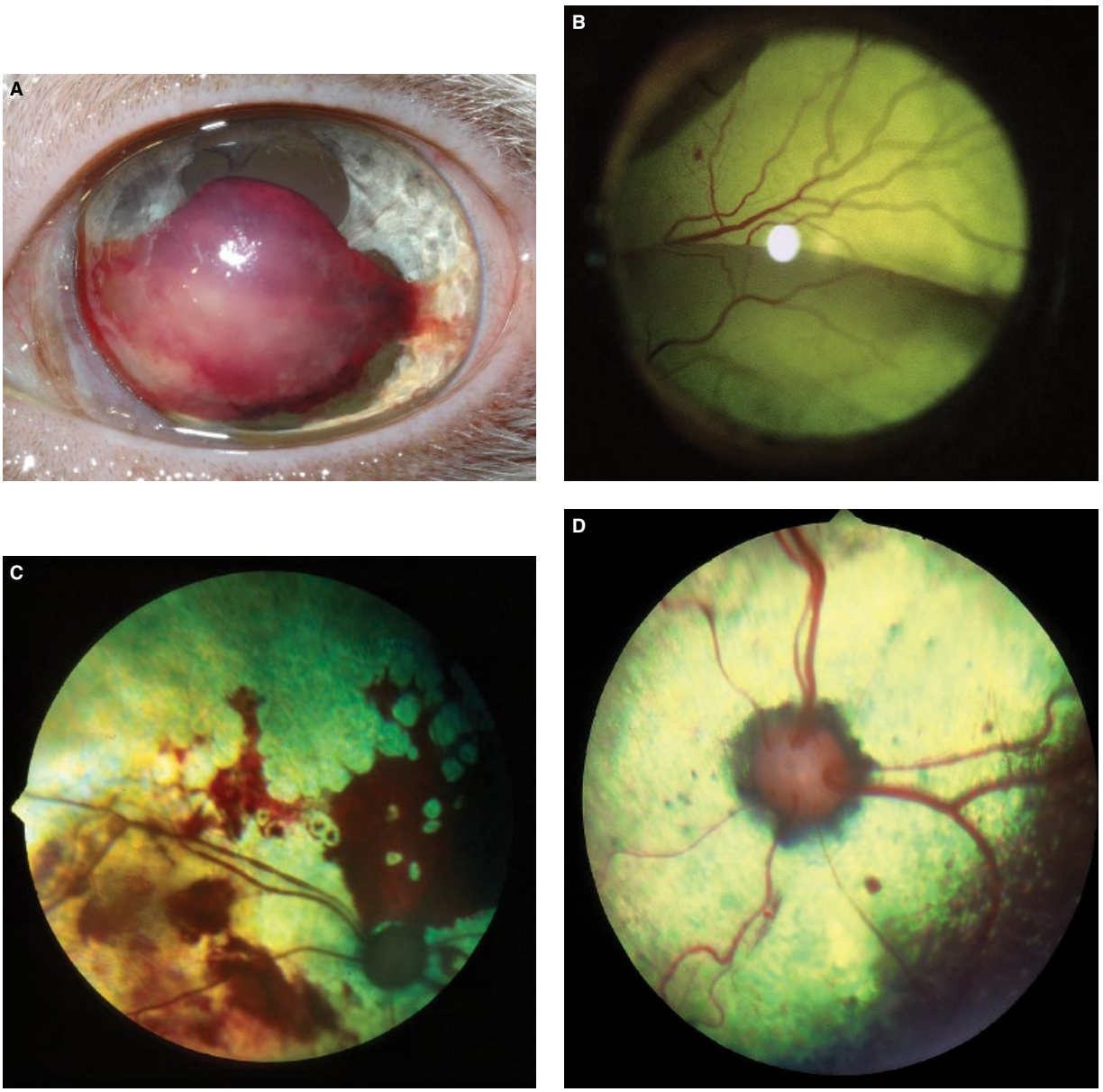


Figure 14.49 (A) Hypertensive retinopathy in the cat is frequently presented as hyphema, dilated pupils, and/or blindness. Note clotted blood in the anterior chamber in the absence of anterior uveal inflammation. (B) Retinal detachments are common with uncontrolled systemic hypertension. This external photograph shows a bullous detachment and a small intraretinal hemorrhage. (C) Areas of intra- and sub-retinal hemorrhages, and an increased tapetal reflectivity in a cat with systemic hypertension. (D) Multiple intraretinal hemorrhages and tapetal hyperreflectivity in a cat with systemic hypertension.

systemic disease state, such as cardiovascular disease, renal disease, or an endocrinopathy. The fundus lesions suggest both retinal and choroidal vascular abnormalities. The mean arterial blood pressure is usually well over 160–170 mmHg.

Ophthalmic abnormalities include hyphema, glaucoma secondary to hemorrhage, intravitreal hemorrhage, retinal vascular tortuosity, sub- and intraretinal hemorrhages, and retinal detachments (focal, multiple, and complete). Prognosis for restoration of vision is based on the duration of the disease, the ophthalmic abnormalities (the duration and extent of the retinal detachment (s), and number and extent of the retinal hemorrhages), and the rapidity of response to medical therapy. The drug of choice for the treatment of feline hypertension is amlodipine (a calcium channel blocker, administered orally (0.625–1.25 mg once daily).

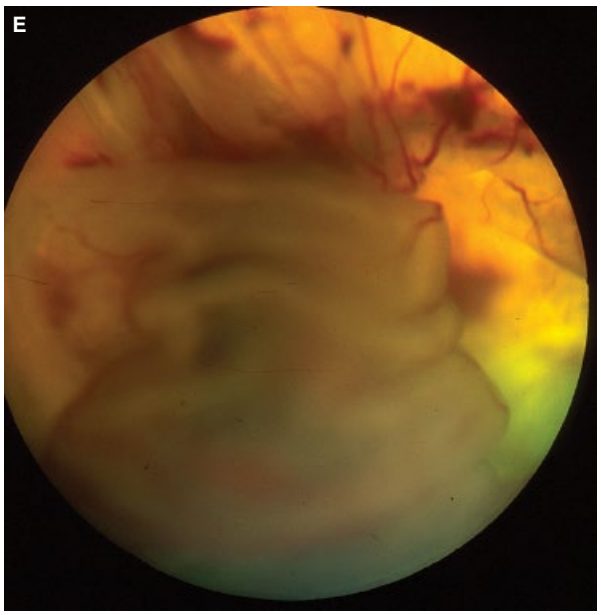
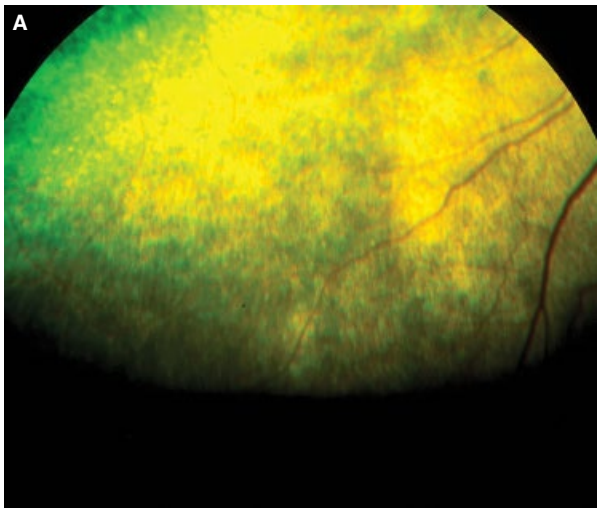


Figure 14.49 (*Continued*) (E) Retinal detachment with sub-retinal hemorrhage in a cat with systemic hypertension. The severe disorganization of the retina makes return to function highly unlikely in this case.

Ocular Fundus and Enrofloxacin

Acute retinal degeneration was associated with enrofloxacin toxicity several years ago (1997) when the enrofloxacin label dosing was changed from 2.5 mg/kg every 12 hours to a flexible dose ranging from 5 to 20 mg/kg as a split or single dose. Acute and severe retinal degeneration resulted. In the earliest patients examined, there was blindness, bilateral dilated pupils that were nonresponsive to light (usually the observation that prompted clinical presentation), and a retina that appeared gray with very constricted retinal blood vessels (Figure 14.50). Within days, there is an increase in tapetal reflectivity and the appearance of large dark yellow to brown foci in the tapetal fundus and reduced pigmentation of the nontapetal fundus. Most often cats remain blind, although a few recover some vision. A functional defect in the transport protein ABCG2 has been associated with this toxicity which allows photoreactive fluoroquinolones to accumulate in the retina leading to reactive oxygen species and retinal degeneration.



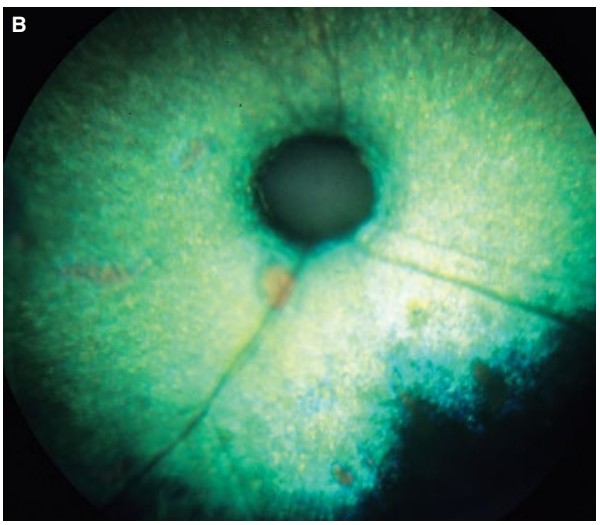


Figure 14.50 (A) Enrofloxacin-associated retinal degeneration in a cat, about 5 days after onset of blindness. Note the tan–brown spots in the tapetal fundus (may be debris from necrosis of the photoreceptors) and increased tapetal reflectivity. (B) More advanced retinal degeneration. Note the increased tapetal reflectivity and very small brown spots in the tapetal fundus.

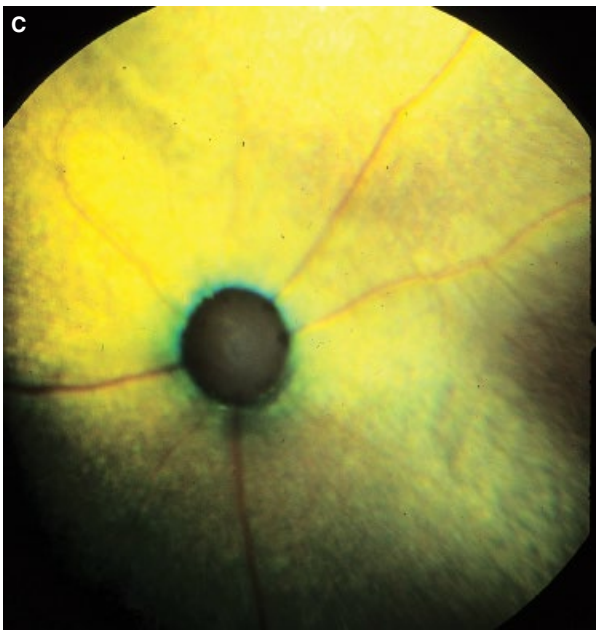


Figure 14.50 (*Continued*) (C) Advanced retinal degeneration with increased tapetal reflectivity, attenuation retinal blood vessels, and optic nerve degeneration.

Ophthalmomyiasis Interna

Ophthalmomyiasis interna affects the anterior segment as well as the posterior segment, and is caused by the aberrant migration of a *Diptera* larva. Affected animals are usually asymptomatic, and the lesions are detected by ophthalmoscopy.

Ophthalmoscopy reveals multiple criss-crossing, curvilinear tracks by the migrating larva within the tapetal and nontapetal fundi (Figure 14.51). Variable retinal and vitreal hemorrhages occur. Healing is evidenced as pigment loss and deposition in the nontapetal fundus, and in tapetal fundus hyperflectivity and pigmentation deposition. Treatment includes systemic corticosteroids for the reactive retinitis or chorioretinitis, but killing the parasite within the posterior segment should be avoided as this incites a significant inflammatory response. Surgical removal of the larva is indicated if it is visible.

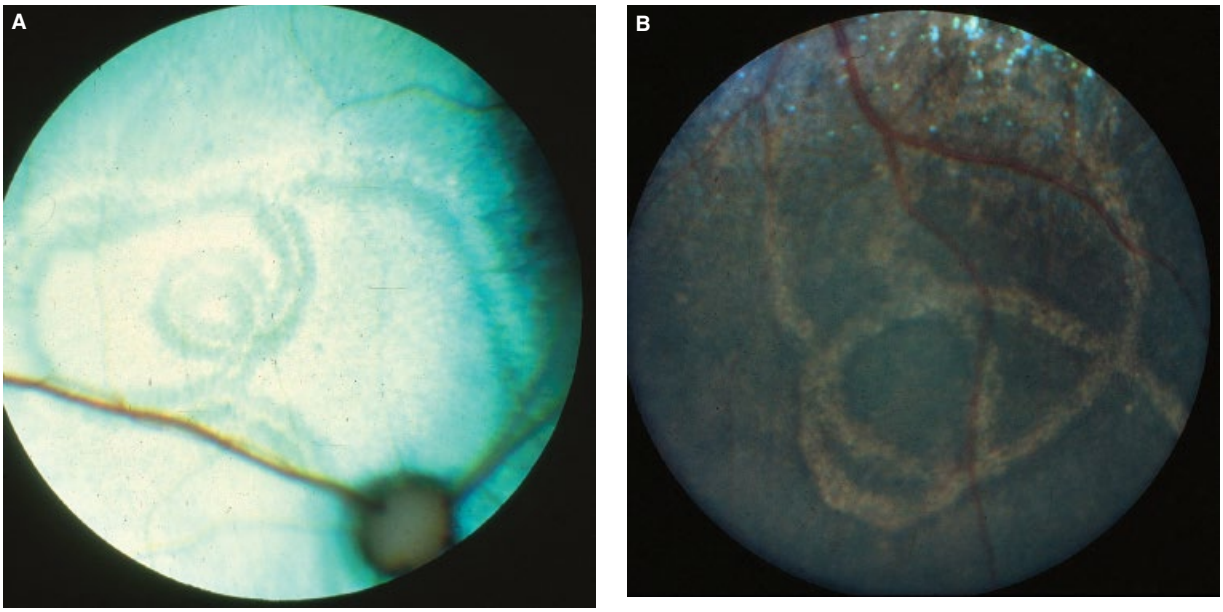


Figure 14.51 (A) Ocular ophthalmomyiasis affecting the tapetal fundus of a cat. The parasite's track is clearly defined. (B) Ocular ophthalmomyiasis affecting the nontapetal fundus in the same cat as in part A. Note the depigmented parasitic migratory tracts within the retina.

Retinal Detachments

Retinal detachments are either nonrhegmatogenous or rhegmatogenous (detachments related to breaks or holes). In cats, most retinal detachments appear nonrhegmatogenous, and most are exudative associated with chorioretinitis (mycotic infections, toxoplasmosis, histoplasmosis, and FIP), lymphoma (FeLV), systemic hypertension, hyperviscosity syndromes, ethylene glycol toxicosis, and intraocular neoplasms (Figure 14.52).

As most exudative retinal detachments are frequently associated with systemic diseases in cats, affected patients require a thorough physical examination and accompanying diagnostic tests. Complete blood count, clinical chemistry, urine analysis, FeLV and FIV tests, and thoracic and abdominal imaging should be considered. Resolution of the inciting inflammatory disease will generally allow retinal re-attachment. Long durations and extensive retinal detachments usually result in retinal degeneration.

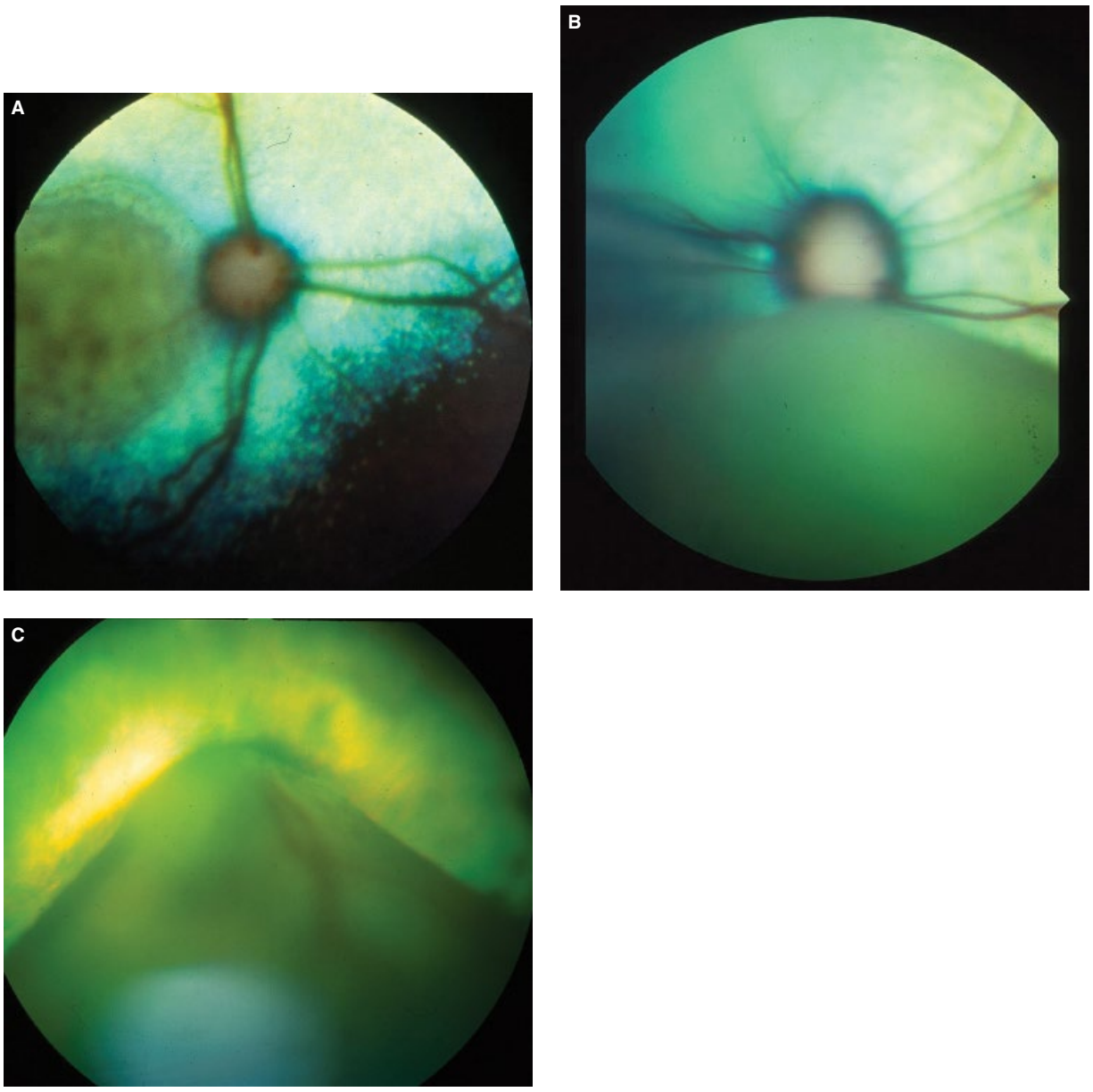


Figure 14.52 (A) Focal retinal detachment in a cat secondary to lymphoma. (B) Large ventral complete retinal detachment of a cat with systemic hypertension. The optic nerve head can be seen above the retinal detachment. (C) Rhegmatogenous retinal detachment in a cat. Note the retina has torn off its dorsal attachment and is hanging over the optic nerve like a veil. The cause could not be determined.

15

Equine Ophthalmology

This chapter is divided into ophthalmic diseases in foals and adult horses. Manual restraint in foals is usually possible while in the adult horse some sedation and eyelid nerve blocks are necessary for a complete ophthalmic examination. Topical medical therapy for foals is quite easy (especially with two adults), but intensive topical therapy in the adult horse usually requires some type of subpalpebral delivery system.

Ophthalmic Diseases in Foals

Microphthalmia

Microphthalmia is one of the most frequent congenital defects in foals, and is often associated with congenital cataracts (Figure 15.1). The condition is unilateral or bilateral, and the Thoroughbred appears to be predisposed. The presenting signs are reduced palpebral fissure size, prominent nictitating membrane, and a smaller than normal eye. Vision is normal or impaired, depending on the size of the eye, and other abnormalities. There is no treatment for microphthalmia.

Strabismus

Strabismus is a deviation of the globe from its normal position. In foals, this condition occurs more commonly in the Appaloosa breed and can accompany stationary night blindness (STNB) (Figure 15.2). The globe is usually rotated upward (hypertropia), or dorsomedial or medial (estropia). If both eyes are affected, the foal may position its head as if to compensate for the strabismus. Stumbling is a common complaint. STNB should be excluded when a young animal presents with strabismus. In this condition the ocular fundus appears normal by ophthalmoscopy and the animal has clinically normal vision in daylight but night vision is profoundly impaired. This is easily noted by behavior changes when the ambient illumination is reduced. STNB has a very distinct pattern on electroretinography. Primary strabismus in foals has been treated by surgery of the involved rectus muscles (either recession or resection).

Congenital Entropion

Congenital entropion or the inversion of the eyelid margin is an infrequent abnormality in foals (Figure 15.3). The lower eyelid is most often affected. Microphthalmia, dehydration, prematurity/dysmaturity, and eyelid trauma are contributing disorders. Presenting signs are increased lacrimation, blepharospasm, inverted lid margin, conjunctivitis, and occasionally corneal ulceration. Temporary eversion of the lid until the underlying disease state has resolved or the foal's growth alleviates the entropion is indicated. Temporary surgical repair should be delayed until the foal has approximated its adult size. Permanent entropion repair is rarely necessary in the horse unless the condition has resulted from cicatrix formation.

Lid and Conjunctivae Dermoids

Dermoids or choristomas can affect the foal's eyelids, conjunctiva, nictitating membrane, or cornea (Figure 15.4). Most often the cornea and limbal bulbar conjunctiva are affected. The dermoid presents as a raised, pigmented, hair-covered mass (which can be confused with a limbal-based melanoma). Treatment is surgical removal.

Nasolacrimal Duct Atresia

The development of the nasolacrimal system starts as two buds that eventually become the lacrimal puncta and canaliculi, and extend through the developing facial bones and nasal cavity to exit at the muctocutaneous junction on the floor of the external nares (easily observed). In nasolacrimal duct atresia, the distal end of the nasolacrimal duct stops prematurely and ends in a blind pouch (Figure 15.5). Clinical signs include a persistent conjunctivitis (which can respond temporarily to topical antibiotics) and epiphora, but these may not be readily apparent until the foal is several months of age.

Color Atlas of Veterinary Ophthalmology, Second Edition. Kirk N. Gelatt and Caryn E. Plummer.
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Figure 15.1 Severe microphthalmia in a foal. Histologically, all ocular tissues were present in this globe, but were reduced in size and disorganized.



Figure 15.2 Strabismus in an Appaloosa yearling. Note the strabismus is dorsomedial. It affects clinical vision, producing frequent stumbling.



Figure 15.3 Entropion in a Thoroughbred foal. Secondary blepharospasm has worsened the lid defect.

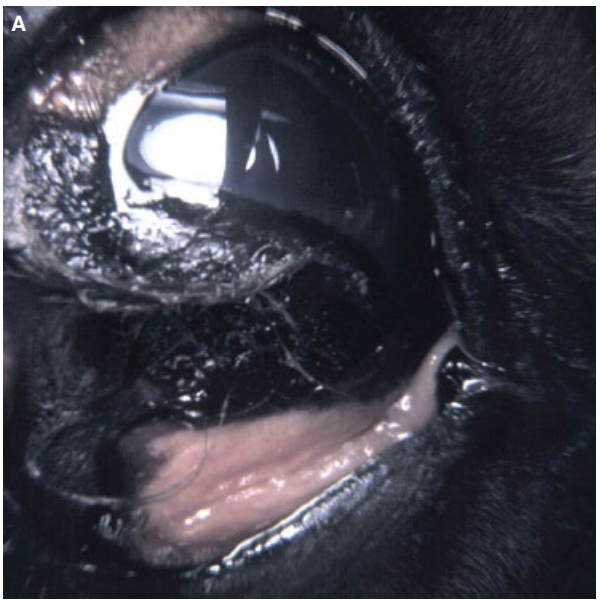
Dacryocystitis with mucoid or mucopurulent discharge develops with time. Fluorescein applied on the eye does not exit at the external nares and close examination of the external nares reveals absence of the nasal punctum. Nasolacrimal flush through the upper or lower lacrimal punctum results in the accumulation of the irrigation solution in the enlarged blind end. Dacryorhinocystography facilitates the diagnosis and localizes the endpoint of the atretic duct. Restoration of nasolacrimal duct patency is by surgery.

Irial Anomalies

Congenital disorders of the iris in the foal are rare, but include aniridia (in Thoroughbreds, Belgians, and Quarter Horses) and persistent pupillary membranes. In heterochromia iridis, the color of the iris is blue, blue and white, or some combination of brown, blue, and white, and affects one or both eyes (Figure 15.6). The term "wall eye" refers to a blue–white iris with a black corpora nigrum (or granula iridica), while "china eye" refers to a white iris with a black corpora nigrum. Heterochromia iridis is related to hair coat color and occurs in Appaloosa, gray, white, palomino, spotted, and chestnut horses. It can be accompanied by hypoplasia or colobomatous malformations of the iris.

Congenital Glaucoma

Congenital glaucoma is rare in foals and usually signals anomalous development of the anterior chamber angle and aqueous humor outflow pathways (Figure 15.7). Presenting signs include buphthalmia (an enlarged globe), mydriasis, variable corneal edema, lens luxation, and



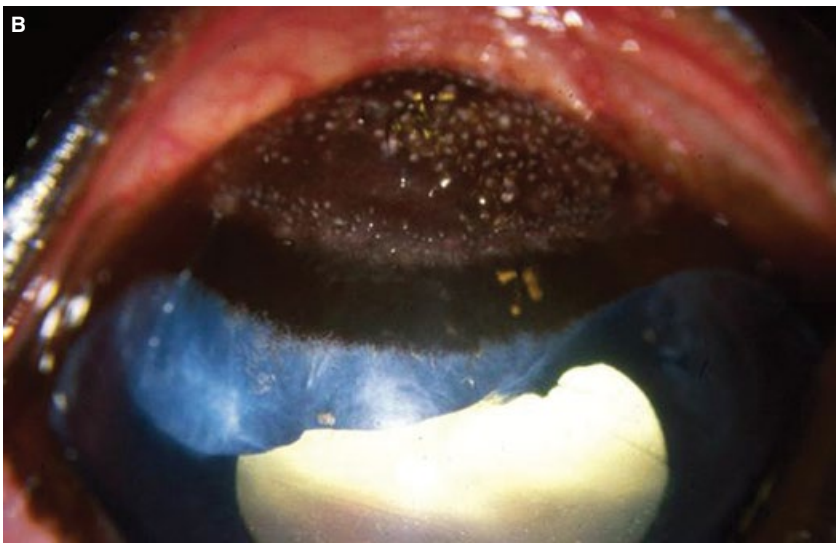


Figure 15.4 (A) A pigmented dermoid affecting the ventral conjunctiva and cornea in a foal. Note the long coarse hair emerging from its surface. (B) Another example of a corneal dermoid in a horse. The pigmented lesion along the dorsal cornea is skin with small, fine hairs.

variable degeneration of the optic nerve head and retina. Tonometry indicates elevated intraocular pressure (IOP). Treatment is not usually attempted, but includes topical drugs to lower IOP and laser cyclophotocoagulation.

Iridocyclitis

Iridocyclitis in foals can be secondary to other ocular diseases (usually corneal ulcers) or associated with systemic diseases such as *Salmonella* sp., *Rhodococcus equi*, *Escherichia coli*, *Streptococcus equi*, *Actinobacillus equuli*, adenovirus, and equine viral arteritis as well as any other cause of septicemia, pneumonia, or any systemic inflammatory condition (Figure 15.8). The iridocyclitis is often bilateral and the foal will exhibit lacrimation, blepharospasm, and photophobia. Ophthalmic findings include eyelid swelling, corneal edema, conjunctival hyperemia, ciliary injection, aqueous flare, hypopyon and/or hyphema, and miosis. Fibrin and contact of the iris and lens can result in posterior synechiae formation and secondary cataracts. A complete general physical examination as well as a complete blood count, clinical chemistry, and thoracic radiographs should be performed.

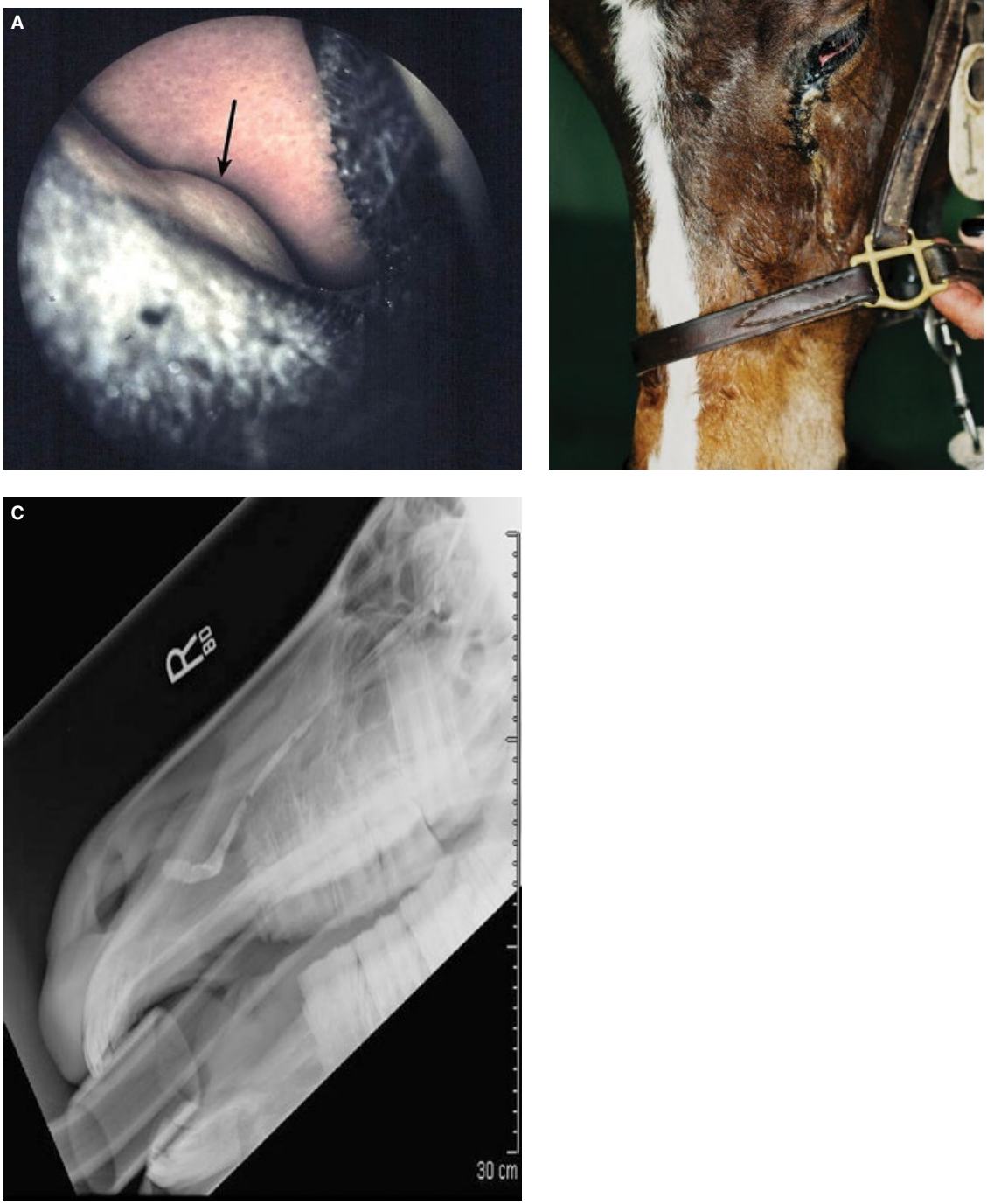


Figure 15.5 (A) Nasolacrimal duct atresia in a foal. The lower, blind end of the nasolacrimal duct appears as a swelling (arrow) on the floor of the nasal cavity just caudal of the nares following orthograde instillation of fluid. (B) An 8-month-old Thoroughbred colt with a 4-month history of mucopurulent ocular discharge had nasolacrimal duct atresia. (C) Dacryorhinocystography study of the same horse as in part B. The contrast material does not exit the distal nasal punctum, confirming the location of the distal end of the duct and the reason for the lack of patency.

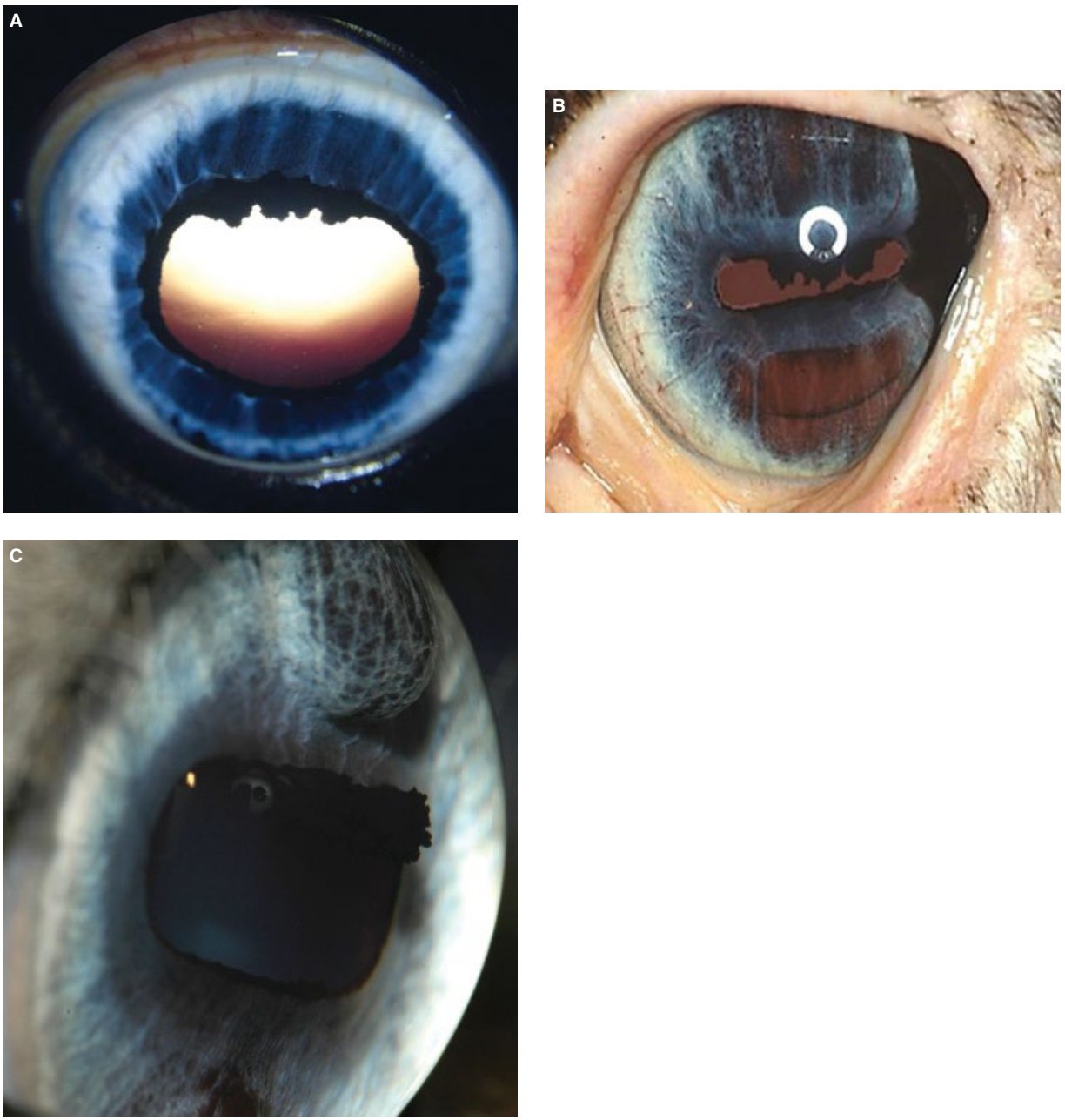


Figure 15.6 (A) Heterochromia iridis in an Appaloosa foal. Note the iridal periphery is white and its central portion blue. (B) Iris hypoplasia (nearly a coloboma) in the typical position in a blue-eyed horse. The ventral equator of the lens is visible behind the thin iris. (C) Iris hypoplasia in a blue-eyed horse. Note the dorsal bulge of the iris. This is commonly mistaken for a mass lesion. Instead, the iris is bulging as a result of thinning or weakness of the hypoplastic tissue.

Treatment should be aimed at the underlying disease but should include symptomatic therapy for the iridocyclitis (mydriatics, and topical and/or systemic anti-inflammatory medications).

Congenital Cataracts

Congenital cataracts and microphthalmia are the two most frequent congenital ophthalmic anomalies in foals (Figure 15.9). Inherited cataracts occur in the Belgian, Thoroughbred, Morgan, and Rocky Mountain Horse breeds. The cataracts in the Belgian may be related to aniridia (actually hypoiridia) and are inherited as a dominant trait. Cataracts are also thought inherited in the Thoroughbred breed as a dominant trait. The Morgan Horse has inherited and bilateral nonprogressive nuclear cataracts that do not progress and interfere with vision.

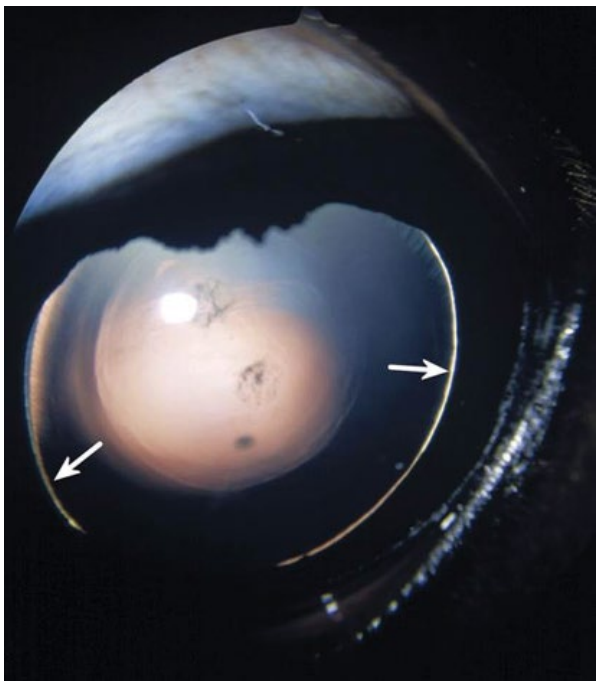


Figure 15.7 Congenital glaucoma and lens subluxation in a foal. The periphery of the lens (arrows) can be seen within the pupil.

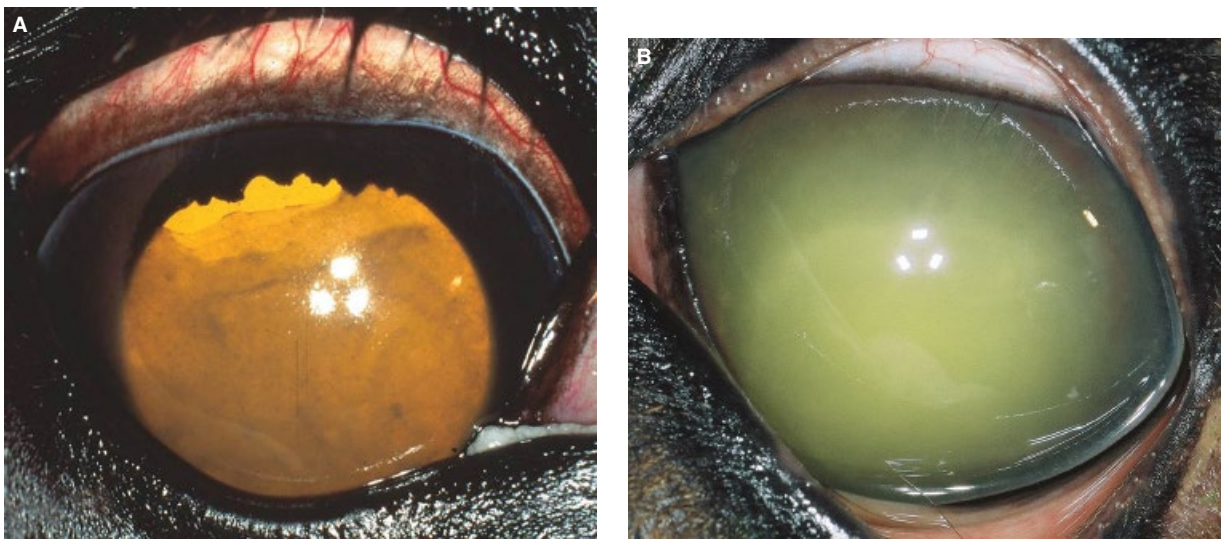


Figure 15.8 (A) Iridocyclitis in a foal secondary to bacteremia associated with *Salmonella* infection. Fibrin filled the anterior chamber. The pupil has been pharmacologically dilated. *Salmonella* organisms were cultured from the anterior chamber. (B) Fibrinous uveitis in a foal with *Rhodococcus* pneumonia. The large fibrin clot in the anterior chamber is obscuring view of the iris.

Congenital cataracts in the Rocky Mountain Horse are associated with multiple ocular anomalies such as megalocornea, goniosynechiae, cysts of the iris, ciliary body and peripheral retina, and retinal dysplasia.

Often, a foal from a normal-eyed mare and stallion is presented to the veterinarian because of either white opacities within the eye or vision impairment to a variable degree. Ophthalmic findings include normal or slightly smaller globes, normal pupillary light response, and mature and complete cataract formation. Ultrasonography and electroretinography are usually normal. Cataract surgery is performed most commonly in the foal for congenital cataracts (adult horses usually have uveitis cataracts) and phacoemulsification is the

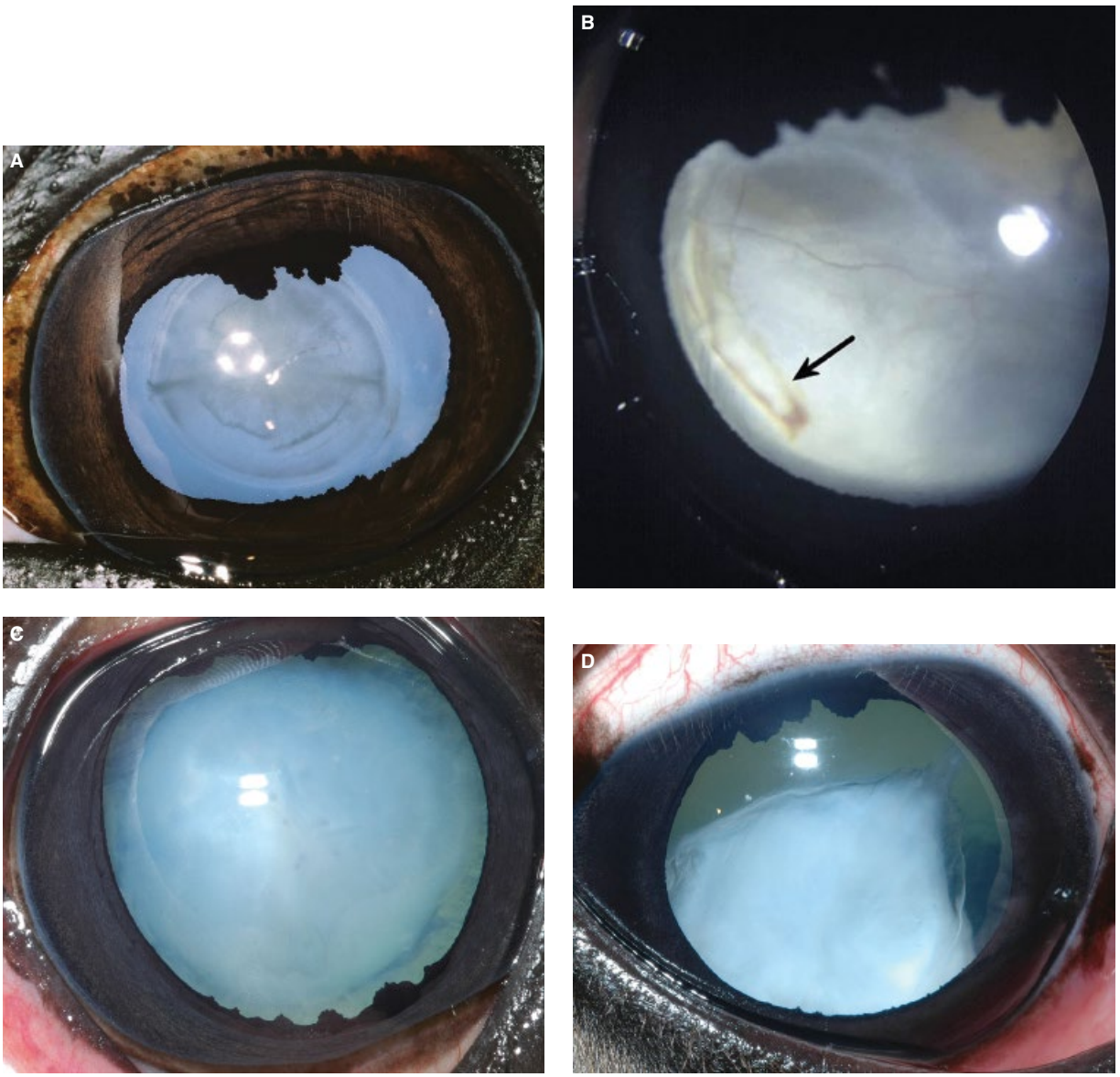


Figure 15.9 (A) Congenital cataract in a foal affecting the nucleus (center) of the lens. Progression of the cataract is unlikely. (B) Congenital cataract secondary to persistent hyaloid remnants in a foal. Note the red hyaloid vessels (arrow) on the posterior lens surface. (C) Late immature cataract in a Quarter Horse weanling filly. (D) Hypermature, resorbing cataract in a yearling Thoroughbred.

technique of choice. Young foals used to handling have surgery at young ages (at 1–2 months before lens-induced uveitis occurs and the foal is more difficult to manage); success rates are over 80%.

Optic Nerve Hypoplasia

Optic nerve hypoplasia is a rare cause of congenital blindness in foals (Figure 15.10). The condition is unilateral or bilateral. Diagnosis is made with ophthalmoscopy. The optic nerve head is smaller than normal, white, and contains few retinal blood vessels. There is no treatment.

Equine Ophthalmology in Adult Horses

Orbital Diseases

Orbital Inflammations

Orbital cellulitis occurs occasionally in horses, and can be associated with trauma, foreign bodies, and the extension of infections from the adjacent nasal and sinus cavities as well as tooth root infections (Figure 15.11). The presenting clinical signs are acute swelling of the eyelids and supra-orbital fossa, blepharospasm, exophthalmia, orbital pain,

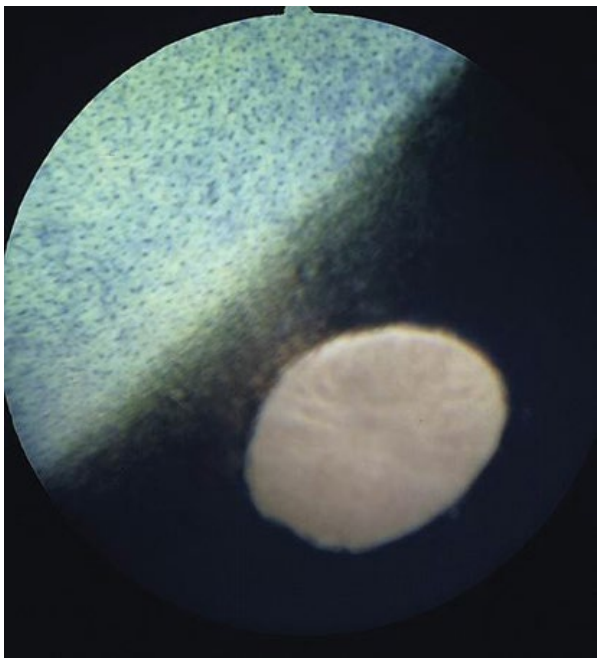


Figure 15.10 Optic nerve hypoplasia in a Quarter Horse foal. The optic disc is tilted, smaller than normal, and pale white.



Figure 15.11 Orbit cellulitis in a Standardbred horse. Note the marked swelling of the eyelids and supraorbital fossa, blepharospasm (from pain), and copious mucopurulent discharge.

epiphora, conjunctival hyperemia, and protrusion of the nictitating membrane. Ultrasonography can assist with characterization and localization of disease as well as sample acquisition.

Orbital Trauma and Fractures

Orbital trauma is more frequent in young horses maintained in groups. Blunt as well as penetrating trauma occurs. Orbital swelling produces a prominent or partially luxated globe, nictitans protrusion, conjunctival hyperemia and chemosis, variable corneal edema, miosis, and hyphema. An impaired blink reflex can occur, and corneal exposure with ulceration rapidly develops. Treatment consists of placement of the subpalpebral system, a complete temporary tarsorrhaphy, and topical and systemic antibiotics, mydriatics, and systemic nonsteroidal anti-inflammatory drugs (NSAIDs).

Orbital trauma can also produce fractures of the orbital bones (Figure 15.12). Clinical signs include asymmetry of the orbits, epistasis, exophthalmos, conjunctival and lid swelling, crepitus, and pain on orbital palpation. Ultrasonography and radiology are essential diagnostic modalities. Surgical treatment includes bone plates or wires for the orbital fractures with displacement, subpalpebral lavage system, and complete temporary tarsorrhaphy to protect the cornea.

Orbital Neoplasia

Orbital neoplasms are rare in horses, but include most histopathological types (Figure 15.13). The most frequent are squamous cell carcinoma (usually extending from the nictitating membrane and conjunctiva) and lymphoma (often bilateral). Clinical signs are progressive exophthalmia, strabismus, orbital and supraorbital fossa swelling, and blindness. Prognosis is usually poor.

Atrophy of the Globe or Phthisis Bulbus

Phthisis bulbus or atrophy of the globe is a not uncommon condition in older horses (Figure 15.14). It results from severe trauma, chronic anterior uveitis, and glaucoma that eventually impair aqueous humor formation. Reduced IOP results (usually less than 5 mmHg) which can cause corneal decompensation (corneal edema), cataract formation, and retinal detachment. The condition does not respond to therapy.

Diseases of the Eyelids

Eyelid Lacerations

Eyelid lacerations are common in horses. Either the upper or lower eyelid can be affected; however, because of its size, mobility, and prominence, the upper lid is most often affected (Figure 15.15). The laceration often involves the lid margin, and must be repaired carefully to avoid a postoperative notch or defect. The cornea should be carefully examined as corneal lacerations may be also present (especially if hyphema is present). Treatment consists of two-layer closure starting with the eyelid margin. A temporary complete tarsorrhaphy is recommended



Figure 15.12 (A) Orbital trauma and fracture of the supraorbital process in a horse. Note the upper lid swelling, exophthalmia, ventral strabismus, and dorsal subconjunctival hemorrhage. (B) Orbital trauma resulted in a displaced fracture of the dorsal orbital bones which resulted in exophthalmos and severe conjunctival swelling as well as an impaired blink and exposure keratitis. (C) Computed tomography image of the same horse as in part B revealing displaced fracture and globe compromise.

to protect the cornea during lid healing and when the blink reflex is impaired.

Eyelid Neoplasia

Squamous cell carcinoma is a common ophthalmic neoplasm that affects the eyelids, conjunctiva, nictitating membrane, limbus, and cornea of older horses (Figure 15.16). It is the most frequent ocular and periocular neoplasm. Age, exposure to ultraviolet radiation, and reduced pigmentation of the eyelid margins and conjunctiva seem predisposing factors. There is a greater incidence of periocular squamous cell carcinoma in draft breeds such as the Belgian and Clydesdale and in Paints and Appaloosas.

Squamous cell carcinomas appear as either proliferative or ulcerative lesions. They are locally invasive but





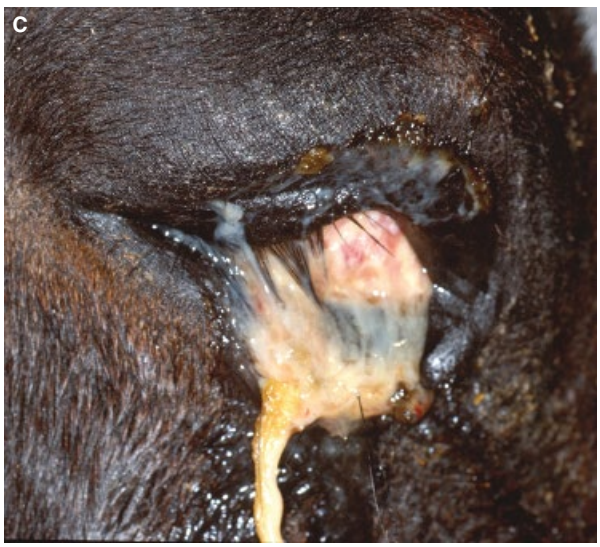


Figure 15.13 (A) Orbital lymphosarcoma in a horse with protrusion of the nictitating membrane and hyperemia of the ventral conjunctiva. The mass affected the ventral orbit and lower eyelid. (B) Orbital lymphoma in an aged Quarter Horse gelding. This horse presented several years previously with a unilateral focal conjunctival swelling that was excised and diagnosed as lymphoma. (C) Orbital squamous cell carcinoma. The tumor originated on the third eyelid. Excision of the nictitans was incomplete and the tumor spread throughout the orbit.

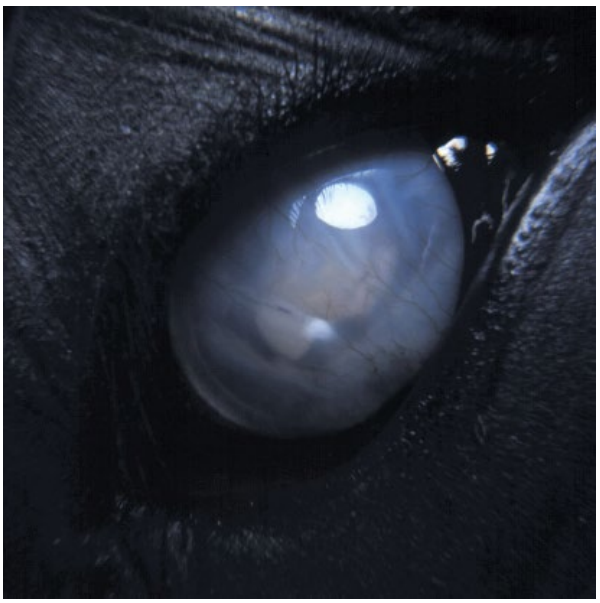


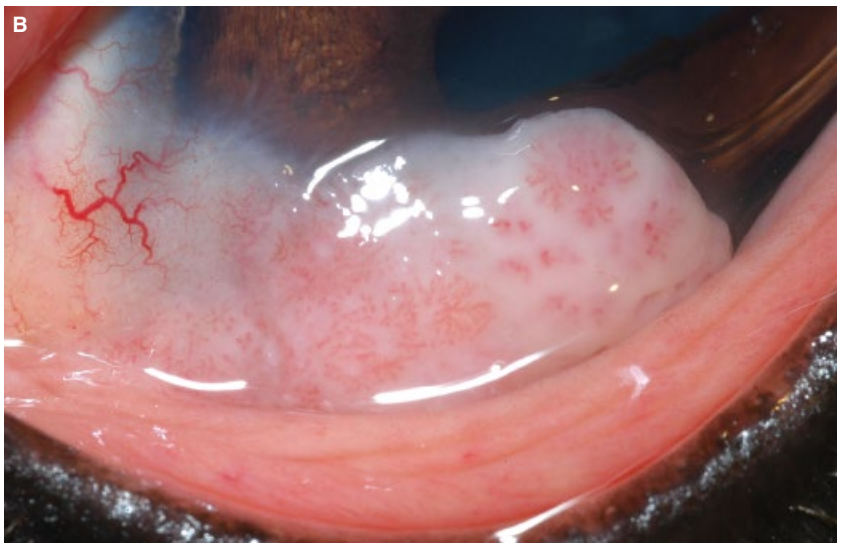
Figure 15.14 Phthisis bulbus of a 10-year-old horse. Note the reduction in the globe size, diffuse corneal edema and superficial vascularization, and mature cataract. The globe has very low intraocular pressure and the horse is blind.



Figure 15.15 Eyelid laceration in a young horse. The lateral upper eyelid has been partially torn and remains attached medially. Surgical apposition with no debridement is recommended.



Figure 15.16 (A) Squamous cell carcinoma in a Paint horse involving the lateral lower eyelid and lateral canthus.



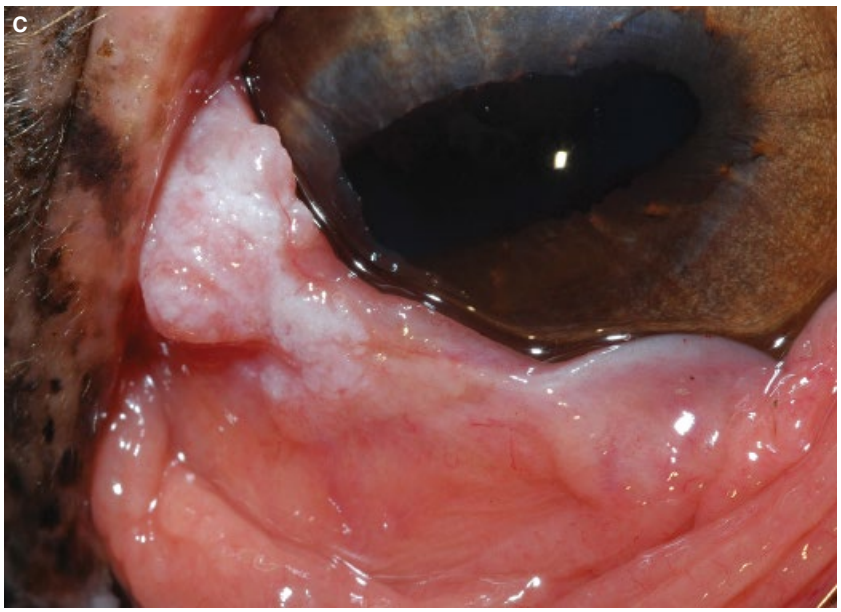


Figure 15.16 (*Continued*) (B) Squamous cell carcinoma of the limbus in an older draft horse. Note the raised pink mass involves both the lateral bulbar conjunctiva and cornea. (C) Large squamous cell carcinoma involving the nictitating membrane in a Belgian draft horse. The deep aspects of the neoplasm should be carefully examined as orbital extension can occur.

fortunately metastasize late (regional lymph nodes, salivary glands, and thorax). Treatment varies according to the size and position of the tumor, age of the horse, and other factors. Combinations of therapy are often used and include surgical excision and radiation or cryotherapy, intralesional chemotherapy, or photodynamic therapy. Prognosis is most favorable when diagnosis and treatment are performed early.

Sarcoids represent the second largest group of eyelid neoplasms, tend to affect younger horses, and often involve multiple sites (Figure 15.17). Clinical types include occult, verrucous, nodular (types A and B), fibroblastic, malignant, or mixed forms. These neoplasms show variable and sometimes rapid growth, are locally infiltrative, and frequently recur after surgical excision.

Melanocytic tumors occur most commonly in gray horses, although they have been reported in animals with other coat colors. They occur throughout the body and have been noted to occur in eyelid conjunctiva and within the globe associated with the uveal tract (Figure 15.18). Adnexal melanomas can affect the health and comfort of the globe when they are in contact with the eye or affect eyelid function. Intraocular melanomas are seen most frequently affecting the iris and can be solitary or multiple. Progression is variable, and the development of glaucoma is possible if the neoplastic tissue compromises aqueous humor drainage.







Figure 15.17 Sarcoid is the second most frequent eyelid neoplasm in horses, tending to affect younger horses. In some countries with limited ultraviolet radiation exposure, sarcoids are the most common lid tumor. (A) Mixed sarcoid of the lateral two-thirds of the upper eyelid in a Thoroughbred horse. (B) Nodular type A sarcoid of the upper eyelid in a 2-year-old horse. The neoplasm at the lateral canthus is extending into both the upper and lower eyelids. (C) Nodular type B sarcoid of the upper eyelid in a 4-year-old Tennessee Walking Horse. This lesion rapidly progressed following biopsy trauma.

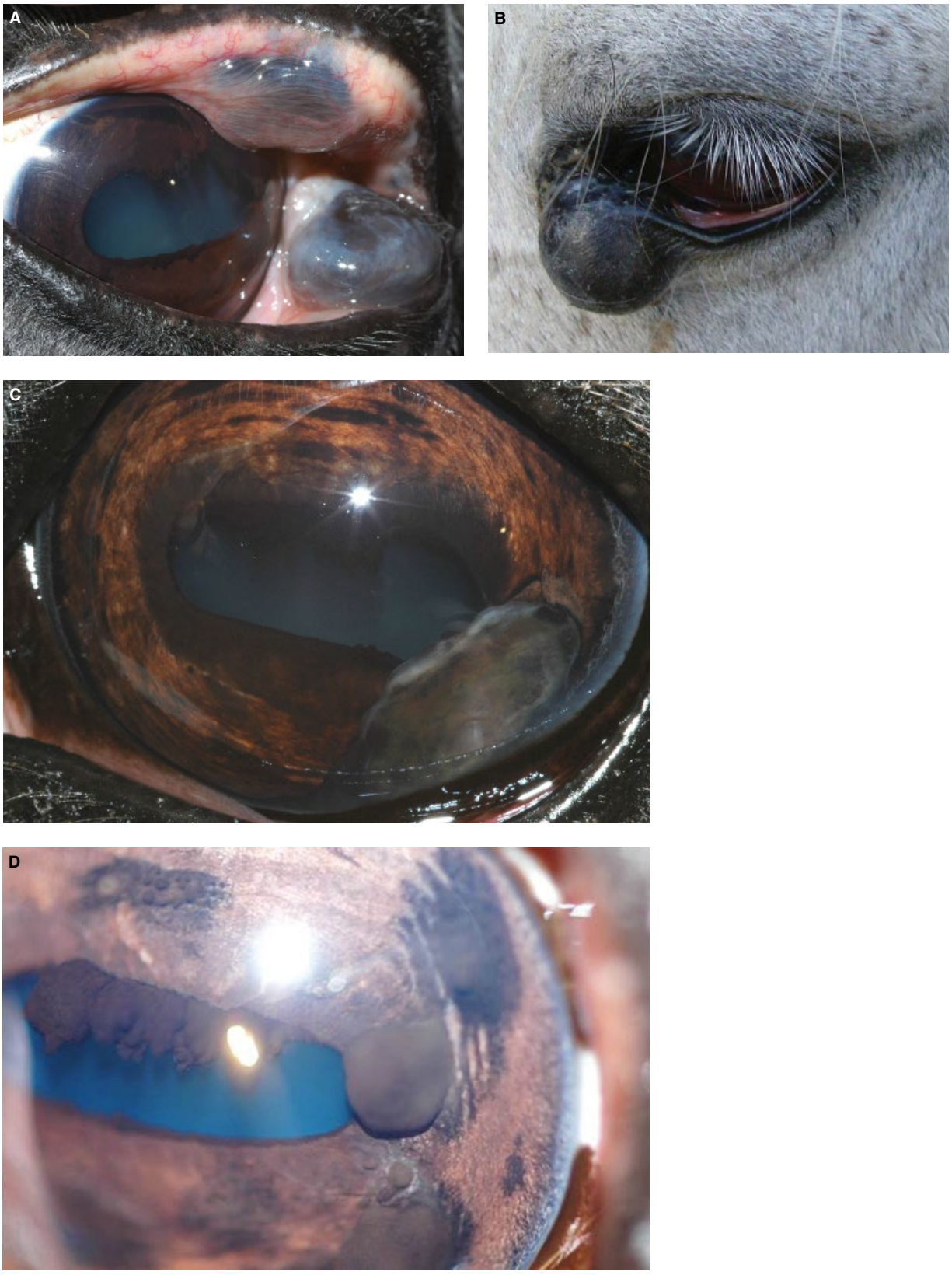


Figure 15.18 (A) Conjunctival melanoma in a gray horse. There are several distinct foci of melanoma in this example. These masses can cause significant irritation for the globe. (B) Eyelid melanoma in an aged gray Thoroughbred mare. (C) Uveal melanoma in a horse. Note the raised mass originating in the ventrolateral iris that is contacting the cornea. (D) Multifocal areas raised areas of melanoma in the iris of a horse.



Figure 15.18 (*Continued*) (E) Iris melanoma in a blue-eyed horse. Note the corneal opacity where the mass is in contact with the corneal endothelium.

Cysts of the Corpora Nigra

Cysts of the corpora nigra or granula iridica affect the upper, lower, or a combination of both structures (Figure 15.19). Sometimes the cystic corpora nigra become large, involve the pupil, and can cause behavioral effects and visual interference.

Nasolacrimal Diseases

Nasolacrimal sac and duct obstructions are not infrequent in adult horses (Figure 15.20). The history is recurrent conjunctivitis that responds temporarily to topical antibiotics, and limited epiphora. As part of routine ophthalmic examination, fluorescein is applied to the eye to check the cornea and nasolacrimal system. Failure of the stain to traverse the system and appear at the distal nasal punctum of the nasolacrimal duct confirms obstruction (Figure 15.21). Patency is re-established by retrograde nasolacrimal flush (with 5–10 mL of saline or sterile water) with the catheter positioned in the distal nasolacrimal duct. Topical antibiotics and corticosteroids are instilled for several days thereafter. Microbiologic culture and susceptibility testing of the exudate removed from the duct is recommended.

Conjunctival Disease

Conjunctival inflammations in adult horses can be part of other ocular diseases as well as associated with systemic diseases. As a result, complete ophthalmic and general physical examinations may be warranted to try to establish the cause. Treatment is directed at the primary condition; topical antibiotics are instilled for the conjunctivitis.

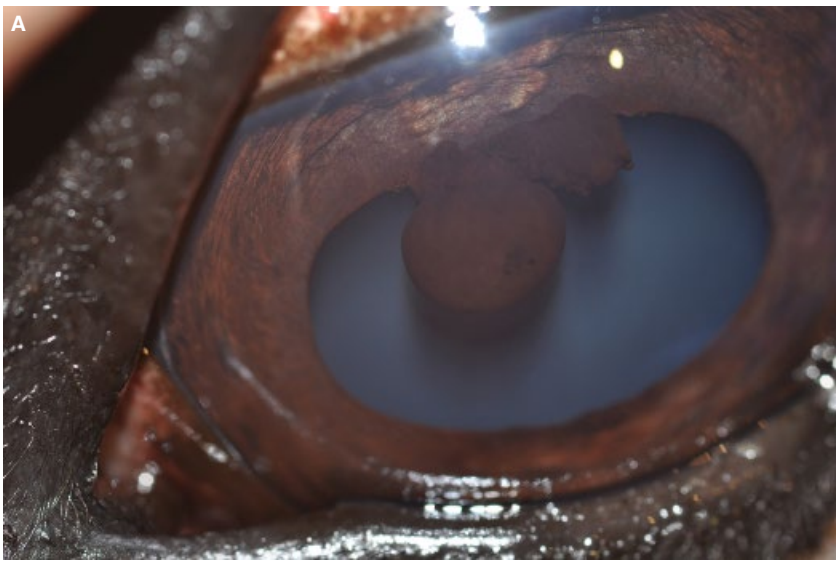


Figure 15.19 (A) Corpora nigra cyst along the dorsal pupil margin. These benign lesions can be confused with melanomas of the iris. Ultrasonography can help differentiate between a solid tissue mass and a fluid-filled cyst if the cyst is not easily transilluminable.



Figure 15.19 (*Continued*) (B) Corpora nigra cyst along the ventral pupil margin. Particularly large cysts can obscure the pupil and affect vision. The treatment of choice is noninvasive laser deflation of the cyst.



Figure 15.20 Duct obstruction in a horse. Note the mucoid discharge at the medial canthus.



Figure 15.21 Dacryocystitis and secondary conjunctivitis in a horse. The conjunctivitis results from the failure of tear drainage as well as the reflux of tears from the obstructed and inflamed nasolacrimal sac.

Habronemiasis is a common cause for granulomas of the eyelids, base of the nictitans (caruncle), lacrimal canaliculi, and the conjunctiva (Figure 15.22; also Figure 18.39). These masses appear as raised, nonhealing, and ulcerated masses with occasional fistulous tracts and a yellow caseous exudate. The granulomas develop in response to dying microfilariae and cytology usually reveals eosinophils, mast cells, polymorphonuclear cells, and plasma cells. Most granulomas are treated topically with antibiotics and corticosteroids, and systemically with antiparasitic agents to kill the microfilariae.

Corneal Ulcerations

Corneal ulcers are a common eye disease of horses (Figure 15.23). Trauma, proliferation, and invasion of opportunistic bacteria and fungi normally present in the conjunctival fornix have important roles. Once the corneal epithelial barrier is lost, degradation of the stroma by infectious agents and the host's inflammatory cells result. Bacteria isolated most frequently from corneal ulcers in horses include Gram-negative *Pseudomonas* and *Enterobacter* spp., and Gram-positive *Staphylococcus* and *Streptococcus* spp. Fungal pathogens are a common cause or complicate corneal ulcers in horses worldwide, but most commonly in hot and humid climates.

Corneal ulcers produce pain as evidenced by blepharospasm, eyelid swelling, conjunctival hyperemia and chemosis, variable corneal edema, miosis, aqueous flare to hypopyon, and eventually superficial and deep corneal vascularization. Topical fluorescein is retained by the corneal ulcer (absence of epithelium) and is instilled repeatedly to monitor the progress of healing.



Figure 15.22 Habronemiasis in a yearling horse. Note the raised yellow granulomas at the base of the nictitating membrane.

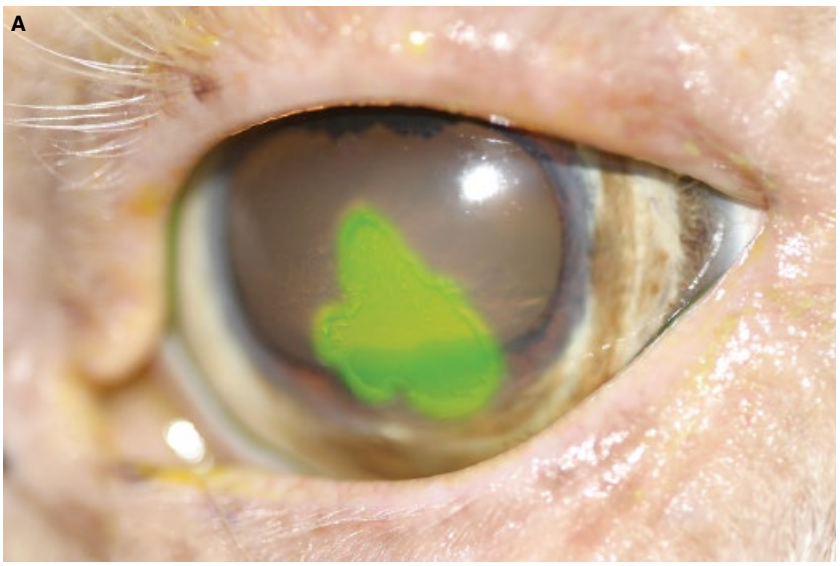
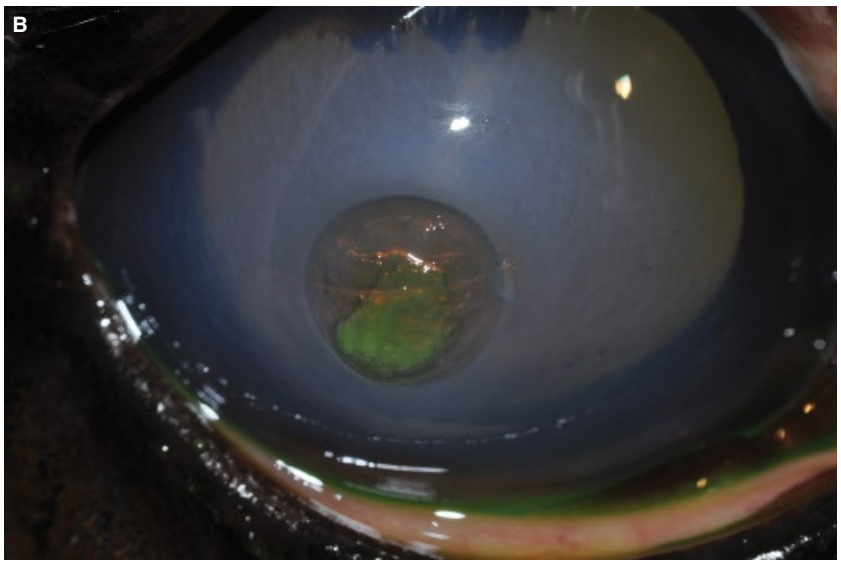
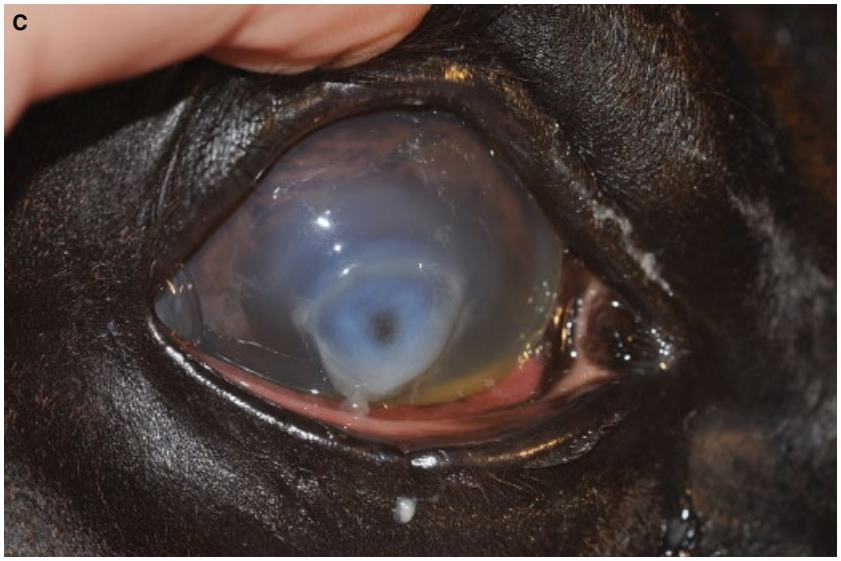


Figure 15.23 (A) Superficial corneal ulcer in a young horse. Fluorescein has been applied to the ocular surface and is delineating the wound margins.





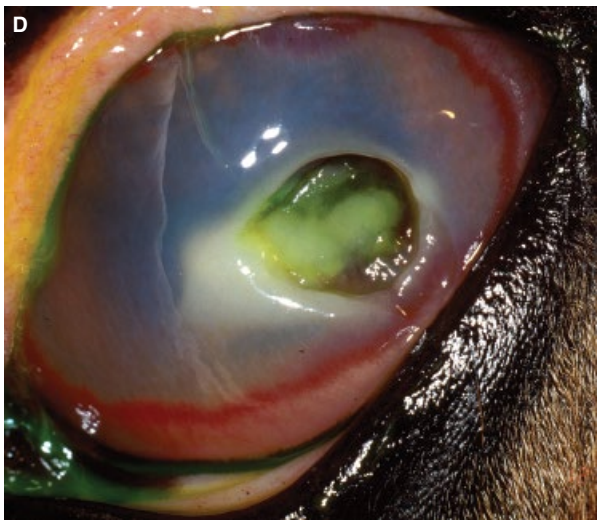


Figure 15.23 (*Continued*) (B) Stromal ulcer in a horse. Both the epithelium and the anterior stroma are absent. (C) Corneal ulceration secondary to *Pseudomonas* sp. in a horse. The gray friable corneal stroma suggests malacia. The dark center is a thin area in the cornea. Hypopyon is present in the ventral anterior chamber. (D) Deep, infected corneal ulcer in a horse. This lesion is near descemetocele in spots. Cellular infiltrate in the wound bed and the surrounding stroma indicated an infection is present.



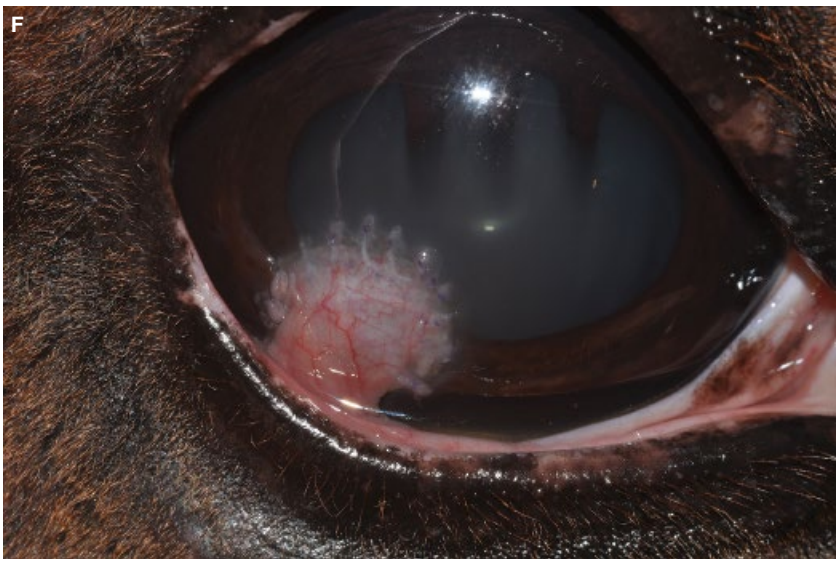




Figure 15.23 (*Continued*) (E) Melting corneal ulceration with iris prolapse in a horse. The iris prolapse is covered by fibrin and contains areas of hemorrhage. (F) Treatment of a deep corneal ulcer with a pedicle conjunctival graft. Appearance 4 weeks postoperatively. (G) Fungal keratitis in a horse forming a plaque on the surface that will attempt to slough.

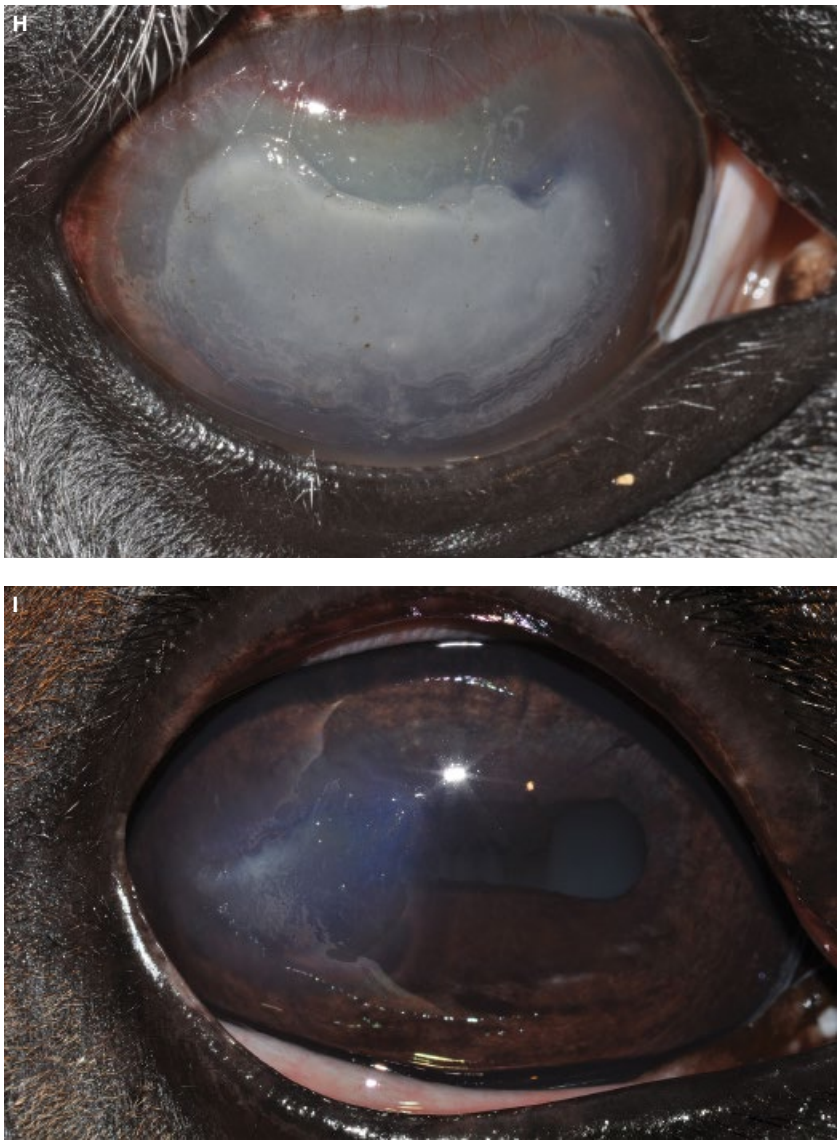


Figure 15.23 (*Continued*) (H) Fungal ulcer in a horse. Note the cream-colored cellular infiltrate in the cornea as well as the soft, irregular margins. (I) Fungal keratitis in a horse. Note the gritty surface of the corneal wound. This is a common finding in fungal infections of the cornea.

Fungal keratitis and corneal ulcers occur more frequently in certain parts of the USA, and appear frequently in the southeastern states. The fungi involved most frequently are *Fusarium*, *Aspergillus*, and *Penicillium* spp. These ulcers can appear clinically similar to bacterial ulcers, but fail to respond to antibiotic therapy or they may have distinctly different manifestations.

Diagnostic procedures for corneal ulceration should include culture and sensitivity tests and cytology to guide antimicrobial selection. Medical treatment usually includes topical antibiotics (and antifungals if indicated), anticollagenase agents such as autologous serum and 1% atropine for mydriasis and cycloplegia. Systemic NSAIDs are helpful to address the reflex anterior uveitis that is always present when a corneal ulcer develops. If the corneal ulcer progresses in spite of aggressive medical therapy, surgical stabilization is indicated.

Corneal Stromal Abscesses

Corneal stromal abscess formation results when corneal re-epithelialization entraps septic material beneath it and above the corneal stroma (Figure 15.24). The abscess can contain bacteria, fungi, a combination, or be sterile. Most cases of stromal abscess are thought to be related to a fungal infection.

Clinical history includes a persistent white to yellow corneal opacity surrounded by vascularization, and a

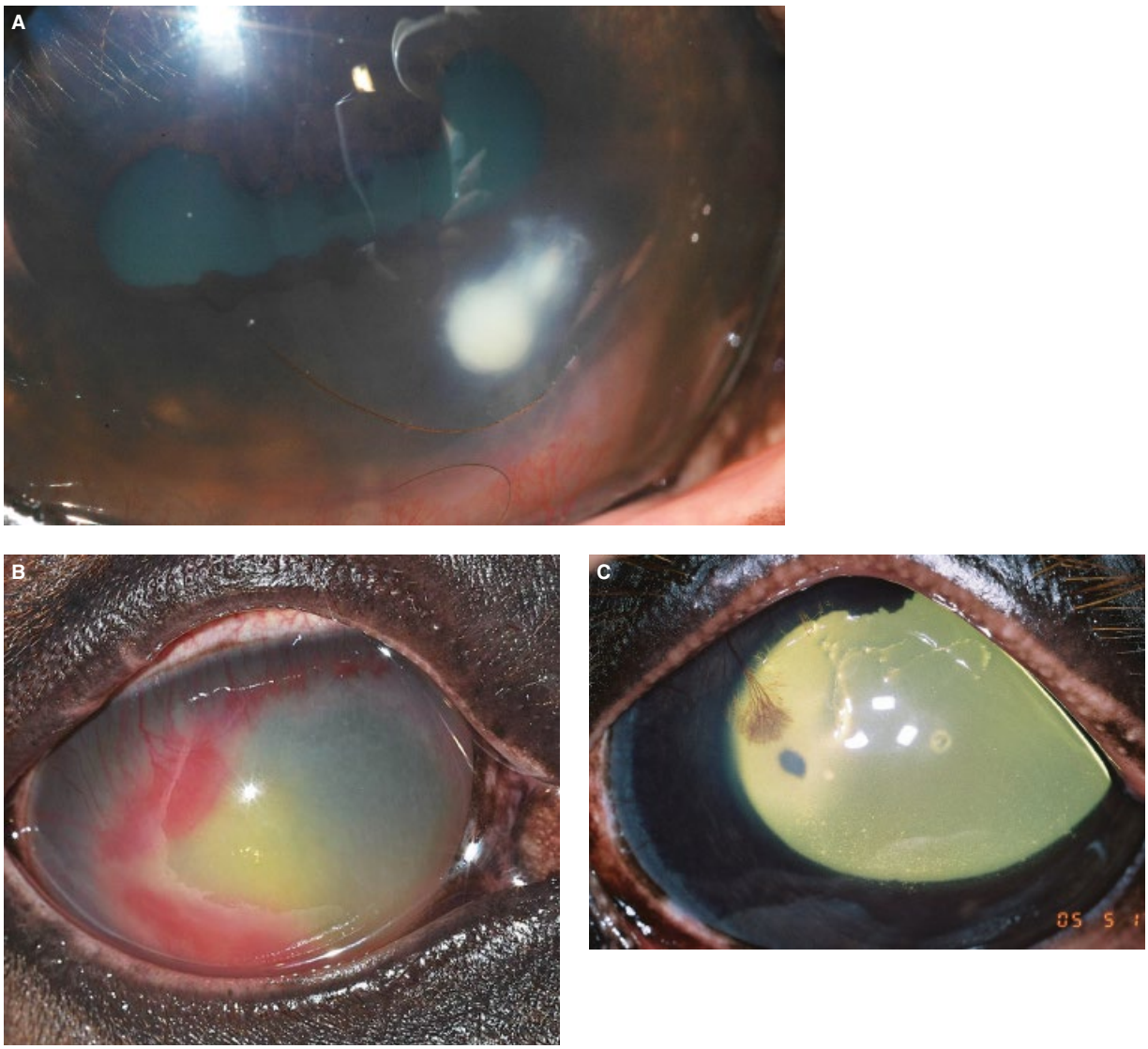


Figure 15.24 (A) Corneal stromal abscess formation in the ventrotemporal cornea. The cornea did not retain fluorescein stain. (B) Another example of a stromal abscess. In this instance, the corneal opacity makes it difficult to localize and delineate the abscess. There is considerable secondary uveitis present as well. (C) Stromal abscess in a horse. The abscess is deeper in the cornea stroma than the approaching vessel.

secondary, sometimes intense, iridocyclitis. The stromal abscess is usually quite dense and its depth in the cornea difficult to establish accurately.

Treatment often combines medications and surgery. If medical therapy does not result in resolution of the abscess or if the iridocyclitis worsens despite treatment, surgical removal of the abscess is indicated.

Viral Keratitis

Herpesvirus-2 affects the corneal epithelium and stroma (Figure 15.25). Clinical signs include variable pain (photophobia, lacrimation, and eyelid swelling), conjunctival hyperemia, multiple focal areas of keratitis and edema, and a mild secondary iridocyclitis (miosis, aqueous flare, and decreased IOP). The punctate areas may not retain fluorescein but usually stain with rose Bengal (rose Bengal is also retained by early mycotic keratitis). Superficial corneal vascularization can be present in chronic cases. Treatment is with topical antivirals and NSAIDs but recurrences are frequent.

Corneal Lacerations

Corneal lacerations are often full-thickness and can result in iris prolapse. They occur frequently in young horses and stallions, and are unilateral. The central cornea is most often involved (more exposed and thinner

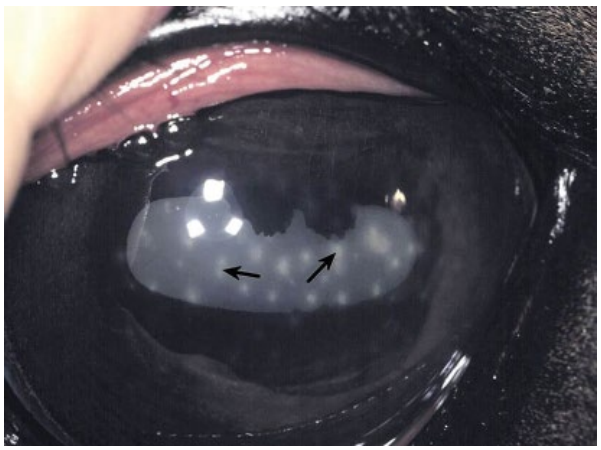


Figure 15.25 Herpes viral keratitis in a horse affecting the central superficial cornea. Note the punctate areas of keratitis (arrows).

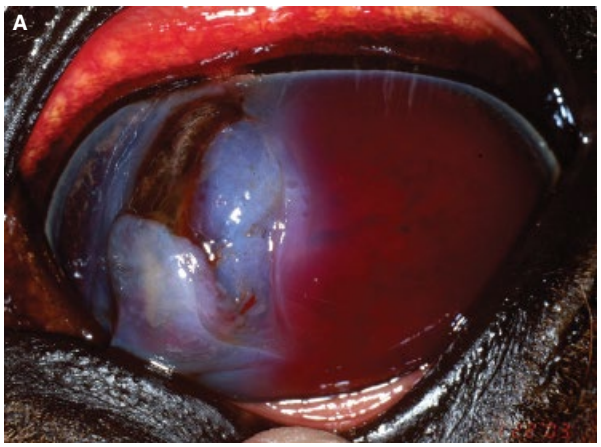






Figure 15.26 (A) Large corneal lacerations, iris prolapse, and intraocular hemorrhage in a horse following head trauma. The iris is protruding through the corneal wound. (B) Iris prolapse through a traumatic laceration in a horse. Corneal edema surrounds the iris prolapse, but the remainder of the cornea is clear. (C) Partial-thickness laceration in the ventromedial cornea. A flap of tissue has been elevated off the corneal surface that has become edematous and begun to retract.

than the periphery), but peripheral lacerations can occur with lid lacerations (Figure 15.26). Dependent on the size and position of the full-thickness corneal laceration, other ocular tissues, such as the sclera, iris, ciliary lens, and vitreous can be involved. Iris prolapsed through lacerated cornea quickly enlarges from vascular congestion, inflammation, and escape of fibrin. Secondary iridocyclitis and considerable hyphema usually accompany

corneal lacerations. Often, horses with full-thickness corneal lacerations are in considerable pain and will not tolerate a complete ophthalmic examination. As a result, after the induction of general anesthesia and before surgical repair of the corneal wound, the ophthalmic examination is performed and accompanied by ultrasonography (as the hyphema can obscure deeper ocular structures). Prognosis depends on the duration and characteristics of the corneal laceration, other ocular tissue involvement, temperament of the horse, and other factors.

Immune-mediated Keratitis

Immune-mediated keratitis is not infrequent in the horse. Its clinical appearance varies depending upon the location of the inflammation (superficial, stromal, or endothelial), the severity, and the chronicity. Eosinophilic keratitis is a form of superficial immune-mediated keratitis that appears as raised vascular plaques, foci of white necrotic tissue, or ordinary-appearing corneal ulcers that fail to heal appropriately (Figure 15.27). Topical immunosuppressants or immunomodulators are usually effective. Recurrence is possible. Superficial forms are

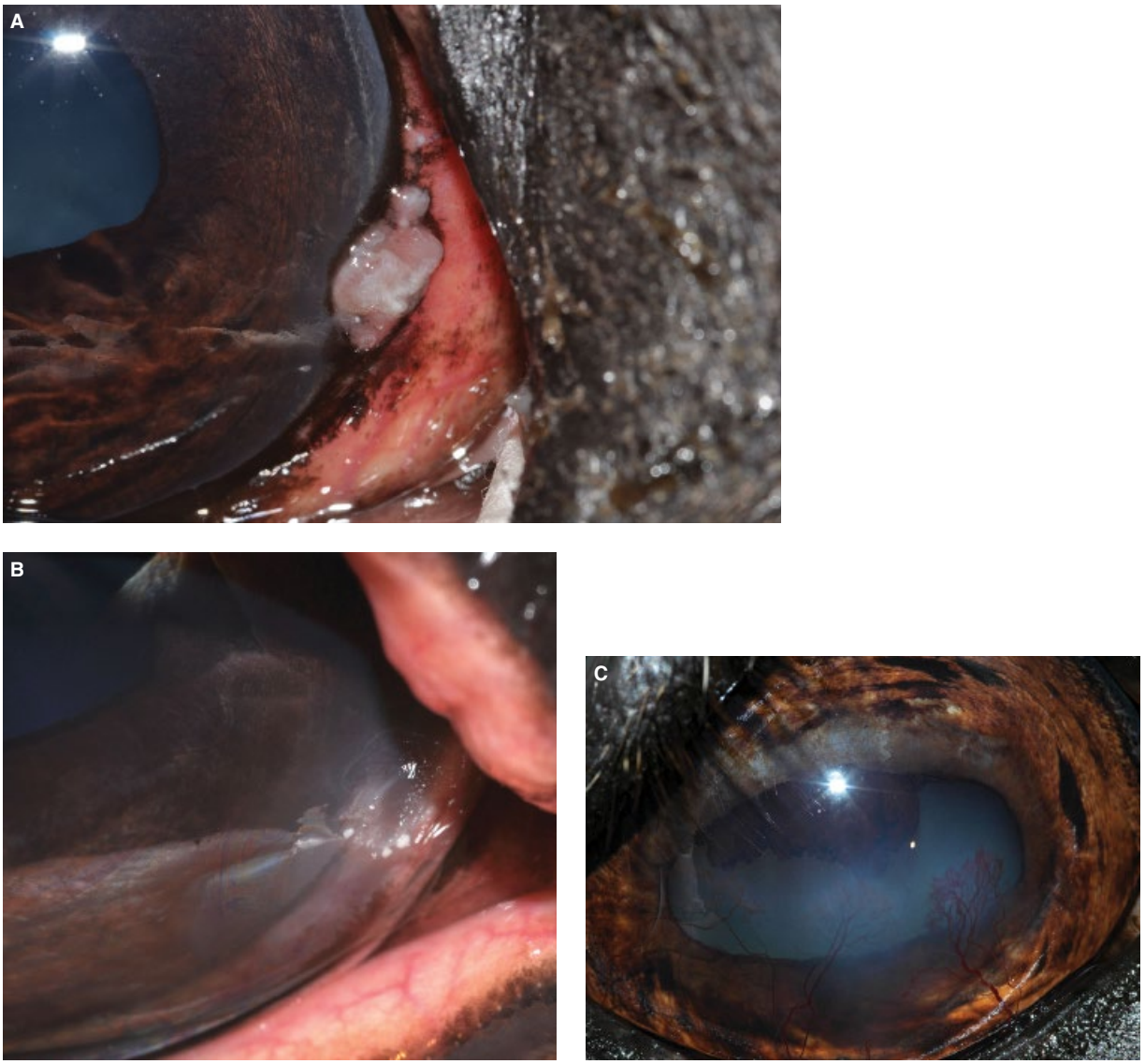


Figure 15.27 (A) Eosinophilic keratitis is this horse has caused raised pink vascular lesions near the limbus. Eosinophilic keratitis lesions are often found beneath the third eyelid. (B) Eosinophilic keratitis in this horse has resulted in nonhealing ulcers and necrotic plaques. Cytology revealed eosinophils in the wound bed. (C) Superficial immune-mediated keratitis may have superficial punctate or diffuse opacities associated with the epithelium, subepithelial space, or the anterior stroma. Superficial vasculature is usually present but to variable degrees.

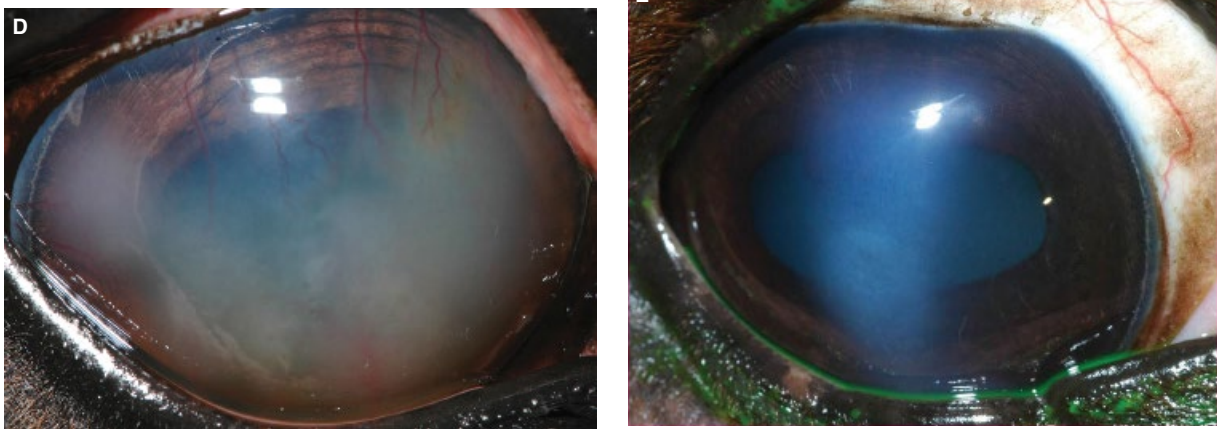


Figure 15.27 (*Continued*) (D) Stromal keratitis often occurs with mid-stromal vessels and stromal edema and discoloration. Green, fluid-filled lacunae may be present. (E) Endotheliitis begins with a focal area of quite dense corneal edema, usually centrally or in a vertical orientation. Keratic precipitates are usually present on the endothelium in the edematous area.

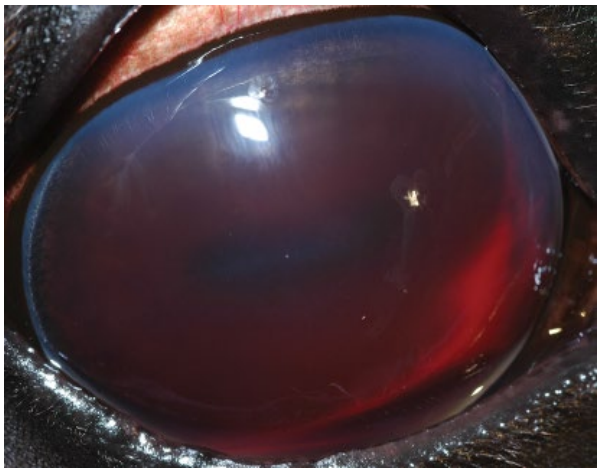


Figure 15.28 Traumatic hyphema from blunt injury in a horse. Note the hemorrhage within the anterior chamber has clotted, but considerable fibrin is present suggesting an intense iridocyclitis.

usually accompanied by mild blepharospasm. The deeper forms are associated with less discomfort, but have more significant impacts on corneal clarity. Endothelial inflammation has a generally poor prognosis as it is difficult to treat and can have concurrent intraocular sequelae, such as uveitis and glaucoma.

Diseases of the Anterior Uvea

Traumatic Iridocyclitis and Hyphema

Traumatic hyphema and iridocyclitis can follow blunt trauma to the horse eye (Figure 15.28). The trauma may be a stick or board, whip, or other object. The history includes eyelid swelling (either from the original trauma or self-induced from rubbing), focal corneal opacity(s), pain, conjunctival hyperemia, nictitating membrane protrusion, and "blood" in the eye.

Ophthalmic examination reveals an intact cornea with focal areas of corneal edema probably from the initial trauma. The anterior chamber contains variable amounts of hemorrhage, mixed with fibrin. The hyphema is usually clotted. The iris and pupil may not be visible, depending on the amount and position of the hyphema. Repeated instillations of mydriatics may be necessary to dilate the pupil, and examine the lens and ocular fundus.

Treatment is directed at the iridocyclitis (mydriatic, topical corticosteroids, and systemic corticosteroids or NSAIDs), and, as these medications can cause local vascular changes, the eye is carefully re-examined daily for bleeds. Hyphema that persists after 7 days can serve as the scaffolding for an iridal or pupillary fibrous membrane that obscures the pupil and produces traction on focal areas of the posterior cornea, iris, anterior chamber angle, and anterior lens capsule. If this complication develops, general anesthesia and intracameral (anterior chamber) injection of 50 µg tissue plasminogen activator (tPA) may dissolve the fibrin matrix and allow the red blood cells to exit the anterior chamber.

Recurrent Uveitis

Recurrent or chronic iridocyclitis or uveitis (periodic ophthalmia) is the most common intraocular disease of adult horses, and the major cause of blindness in the horse worldwide. It affects all breeds, all ages – although most horses are middle-aged (8–10 years old) – and one or both eyes. The cause of equine recurrent uveitis (ERU) has been the focus of considerable study, and leptospiral organisms and an immune-mediated uveitis (iris, ciliary body, and choroid) have been found to be the most likely etiologies. The clinical history is usually repeated bouts of anterior uveitis of variable duration and intensity, development of cataracts, glaucoma, and eventual retinal

detachment, phthisis bulbus, and blindness. Histopathologically, affected eyes show evidence of chronic inflammation, suggesting the disease is likely persistent even in quiescent periods and that chronic treatment of eyes is usually indicated.

Clinical signs of ERU include photophobia, eyelid swelling, lacrimation, conjunctival hyperemia, protrusion of the nictitans, and variable corneal edema. Other ophthalmic findings include aqueous flare to hypopyon, miosis, posterior synechia formation, iris deposits of the anterior lens capsule and peripapillary chorioretinitis (most commonly in the peripapillary region surrounding the optic nerve). In chronically affected eyes, cortical cataract formation with multiple posterior synechiae formation, atrophy of the granula iridica or corpora nigra, increased pigmentation of the iris, secondary glaucoma, vitreal syneresis and floaters, advanced peripapillary chorioretinal degeneration, optic atrophy, retinal detachments, and phthisi bulbus can develop.

Treatment of acute ERU consists of topical mydriatics, corticosteroids and/or NSAIDs, and systemic corticosteroids and/or NSAIDs (Figure 15.29). The topical drugs are administered to effect and tapered as the inflammation subsides. Long-term intermittent systemic corticosteroids and NSAIDs have been used to treat the chronic phases of the disease. Suprachoroidal cyclosporine implants are available and have had favorable results when implanted early in the course of the disease.

In chronically affected eyes, cortical cataract formation with band keratopathy, multiple posterior synechiae formation, atrophy of the granula iridica or corpora nigra, secondary glaucoma, vitreal syneresis and floaters, advanced peripapillary chorioretinal degeneration, optic atrophy, retinal detachments, and phthisis bulbus can develop (Figure 15.30).

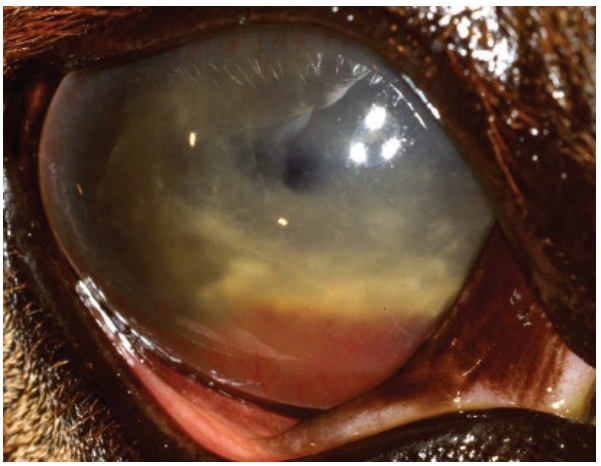


Figure 15.29 Acute equine recurrent uveitis (ERU) affects both anterior and posterior uvea. Note the miosis, fibrin, hypopyon, corneal edema, and corneal vascularization in this horse.

One of the more frequent complications of ERU is secondary cataract formation (Figure 15.31). The cataracts probably develop from multiple posterior synechiae as well as changes within the aqueous humor. The earliest evidence of cataract formation is in the equatorial lens with eventual involvement of the anterior and posterior

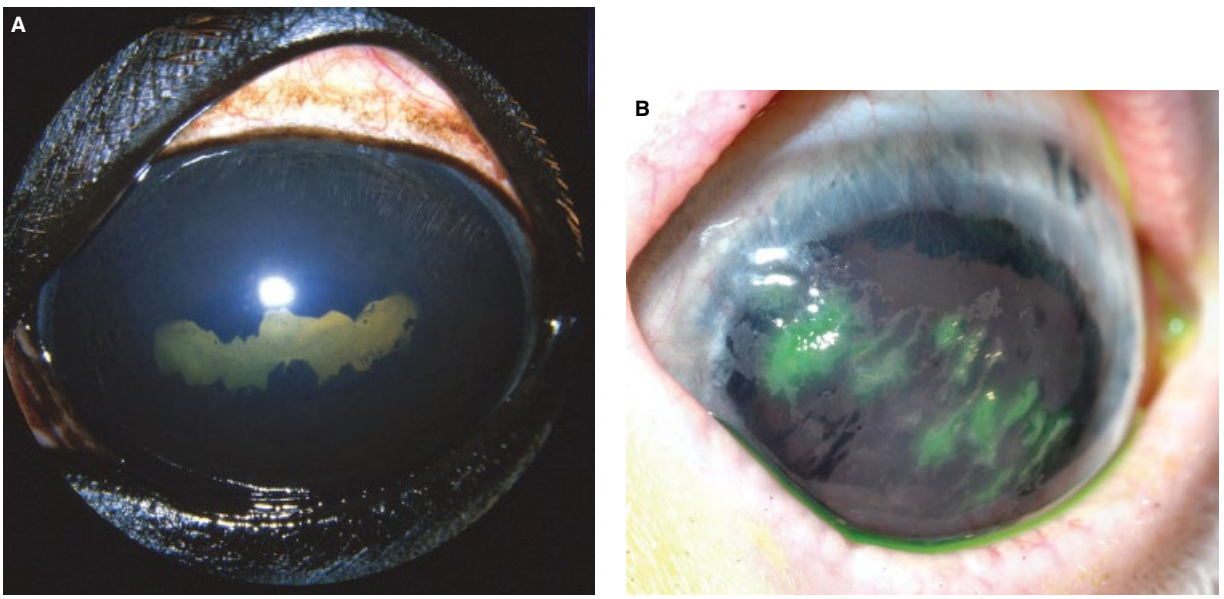


Figure 15.30 (A) Chronic ERU can eventually cause considerable damage to the eye. In this horse, the recurrent bouts of panuveitis have caused corneal edema and superficial vascularization, increased iris pigmentation, corpora nigra atrophy, extensive posterior synechia, and cataract formation. (B) Chronic ERU in this horse has resulted in band keratopathy (note the white deposits in the superficial cornea), secondary corneal ulcers, and vascularization.

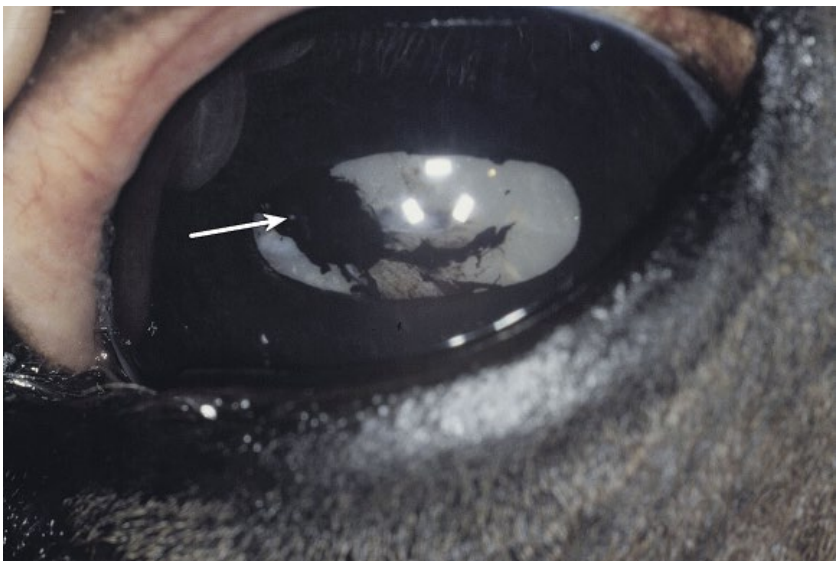


Figure 15.31 Chronic ERU often results in secondary cataract formation, as seen in this horse. The multiple posterior synechiae formation have caused partial dislodgement of the dorsal granula iridica (arrow).

cortices. Cataract progression seems related to the intensity and duration of the uveitis "attacks." Cataract removal has been attempted experimentally to investigate the role of the lens in ERU, and can temporarily restore vision. However, the ERU continues and eventually results in blindness.

Glaucomas

Equine glaucomas were previously thought rare, but appear more frequent (0.07%) with the routine use of tonometry to measure IOP in horses (Figure 15.32). At least three risk factors have been identified for the equine glaucomas: (i) age (horses over 15 years of age); (ii) the Appaloosa breed; and (iii) uveitis (usually ERU). The clinical history often includes one or more of these risk factors, as well as eye enlargement and variable corneal edema. Ophthalmic findings include deep linear band opacities (corneal striae or breaks in Descemet's membrane), variable corneal edema, mydriasis, and episcleral injection. Tonometry indicates either normal or elevated IOP, but repeated tonometry over a few days usually reveals periods of elevated IOP. Lens subluxation and optic nerve head degeneration may be present. Topical medical treatment with the usual glaucoma drugs has not been predictable or particularly successful in horses. Long-term control of IOP and maintenance of vision in equine glaucomas is difficult, but laser cyclophotocoagulation yields the most successful results.

Diseases of the Lens

Acquired cataracts are the most frequent type encountered in adult horses (Figure 15.33). They are primary (no demonstrable cause), secondary to trauma, secondary to anterior uveitis and ERU, glaucoma, and lens luxation. In horses older than 20 years, nuclear sclerosis (gray to blue reflection of the central or nuclear lens) can be fairly dense and must be distinguished from cataract formation. Unilateral cataracts can be secondary



Figure 15.32 (A) Glaucoma in horses is not rare. Note the slightly enlarged globe, diffuse corneal edema, corneal striae (breaks or thinning of Descemet's membrane), and moderately dilated pupil in this horse.

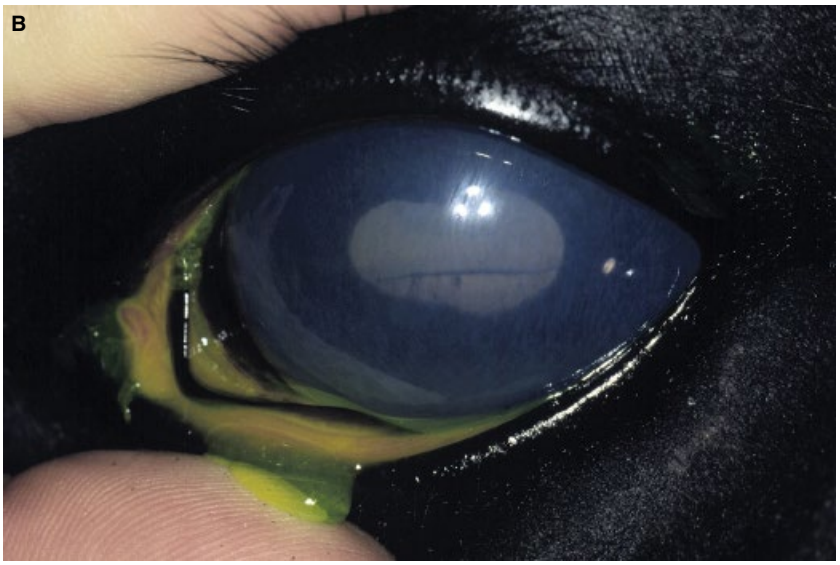
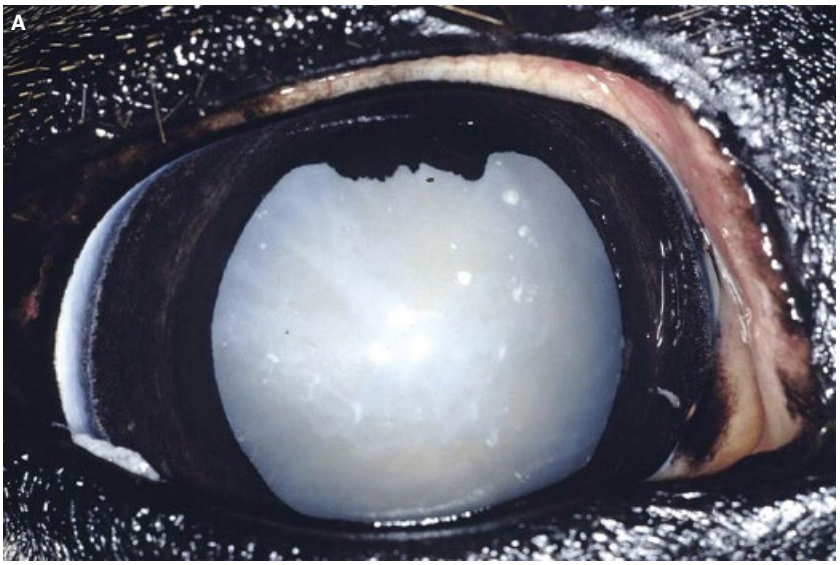


Figure 15.32 (*Continued*) (B) An example of glaucoma secondary to ERU. Note the increased pigmentation of the iris and the posterior synechiae. The corneal edema is diffuse.



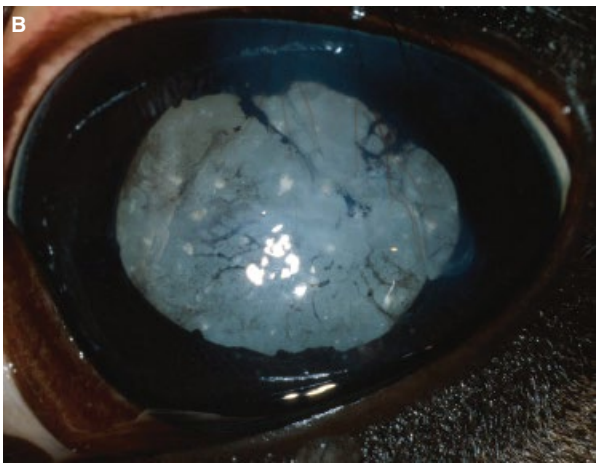


Figure 15.33 (A) Acquired cataracts are not uncommon in horses. Note the complete mature cataract in this horse. An etiology was not determined. (B) In this horse complete mature cataract appears secondary to anterior uveitis. Note the numerous deposits of iridal tissue (from previous posterior synechiae) on the anterior lens capsule.

to trauma or anterior uveitis. Bilateral cataracts with anterior uveitis should be presumed to be ERU until proven otherwise. Both ultrasonography and electroretinography are recommended prior to cataract surgery. Adult horses with chronic uveitis and inflammatory cataracts have poorer success rates with cataract surgery than horses with other types of acquired cataracts. The preferred surgical technique for cataract removal in the horse is phacoemulsification.

Lens luxations in adult horses can result from trauma, advanced cataract maturity, glaucoma and globe enlargement, and chronic anterior uveitis (Figure 15.34). Lens luxations can cause vision impairment but rarely blindness. If the lens luxation is related to glaucoma or ERU, removal of the lens may have limited effect. However, if the lens luxation is producing vision impairment, and no other intraocular disease is present, lens removal can be considered. Lens removal by phacoemulsification is preferred (Figure 15.35).

Diseases of the Ocular Fundus

The normal equine ocular fundus consists of the tapetal fundus, the nontapetal fundus, the retinal vasculature, and the optic nerve head (optic disc or optic papilla). Pupillary dilation, and direct (about 8× magnification) and/or indirect ophthalmoscopy (20 diopter lens 0.8 magnification) are used to examine the ocular fundus.

The tapetal fundus is a roughly triangular area in the upper ocular fundus, immediately above the optic nerve head. Its color varies depending on the horse's coat color; in most horses it is usually yellow–green; in color-dilute horses it may be a combination of yellow–green with a small area of yellow (subalbinism) or totally yellow in white horses (Figure 15.36). Scattered in a regular pattern throughout the tapetal fundus are small dark spots called "stars of Winslow" that represent the penetration of the tapetal fibrous layer by the choriocapillaries.

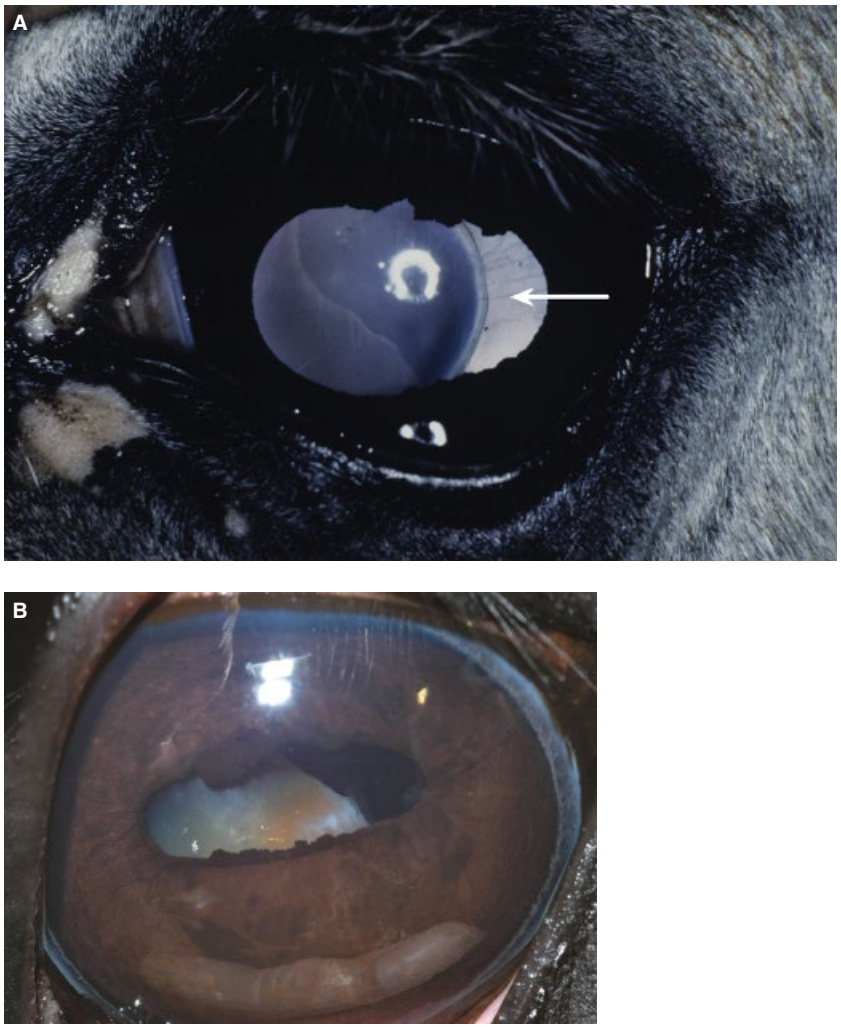


Figure 15.34 (A) Lens subluxation in this horse is secondary to globe enlargement from glaucoma. An aphakic crescent (arrow) can be seen in the lateral pupil. (B) Lens subluxation in a horse with chronic uveitis and a hypermature cataract. Note the irregular and wrinkled capsule and the lens equator and the brunescence of the lens.



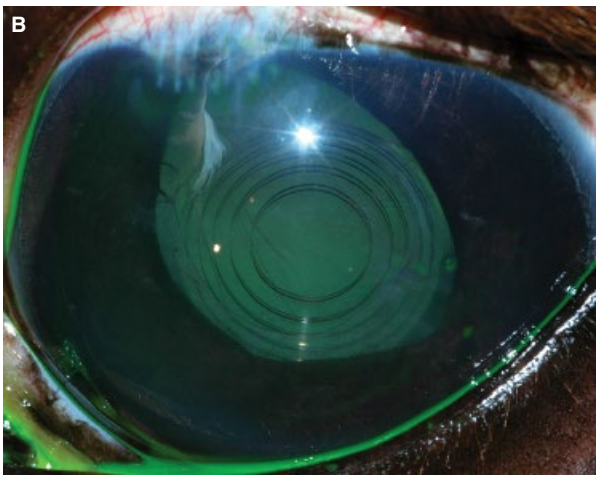


Figure 15.35 (A) In this adult horse after phacoemulsification, minimal postoperative capsular fibrosis (arrows) is present and the horse is visual. This horse is now aphakic as an artificial intraocular lens has not been placed. Courtesy of Dennis Brooks. (B) This adult horse had phacoemulsification cataract surgery 6 weeks previously. Note the corneal fibrosis at the location of the incision, dyscoria with the pupil margin drawn up toward the incision. This horse has had an intraocular lens placed and is therefore pseudophakic.

The larger nontapetal fundus completely surrounds the tapetal fundus, and is usually a fairly uniform brown–black color. In Appaloosas, chestnuts, grays, paints, and palominos, the nontapetal fundus contains less pigmentation, allowing the direct visualization of the deeper (and larger) choroidal blood vessels. The transition from nontapetal to tapetal fundus is an irregular junction rather than a distinct demarcation.

The equine retinal vasculature is classified as paurangiotic, and about 50–80 retinal vessels consists of both arterioles and veins that cannot be differentiated except by fluorescein angiography. They emerge about the periphery of the optic nerve head and extend only 2–3 disc diameters into the retina's periphery. Therefore, the equine retina is very dependent on choroidal blood flow (in contrast to dogs, cats, and most food animals).

The adult optic nerve head is an oval pink to orange structure within the nontapetal fundus, just below the junction of the tapetal and nontapetal fundi, and somewhat temporal. The tapetal region immediately above the disc can be variable in appearance because of this variable colored junction. The scleral lamina cribrosa becomes more prominent as the horse ages.

Chorioretinal Inflammations

Chorioretinitis is the most frequent disease of the ocular fundus in the adult horse (Figure 15.37). Not surprisingly, the majority of the chorioretinal diseases are clustered around the optic nerve head. Bullet hole chorioretinitis, however, occurs ventral to the disc in a multifocal punctate pattern. Rarely, one may appreciate diffuse chorioretinitis or nontapetal horizontal chorioretinitis. Causes of chorioretinitis are varied but include equine herpesvirus-1 (EHV-1), influenza, vascular infarcts, and ERU.

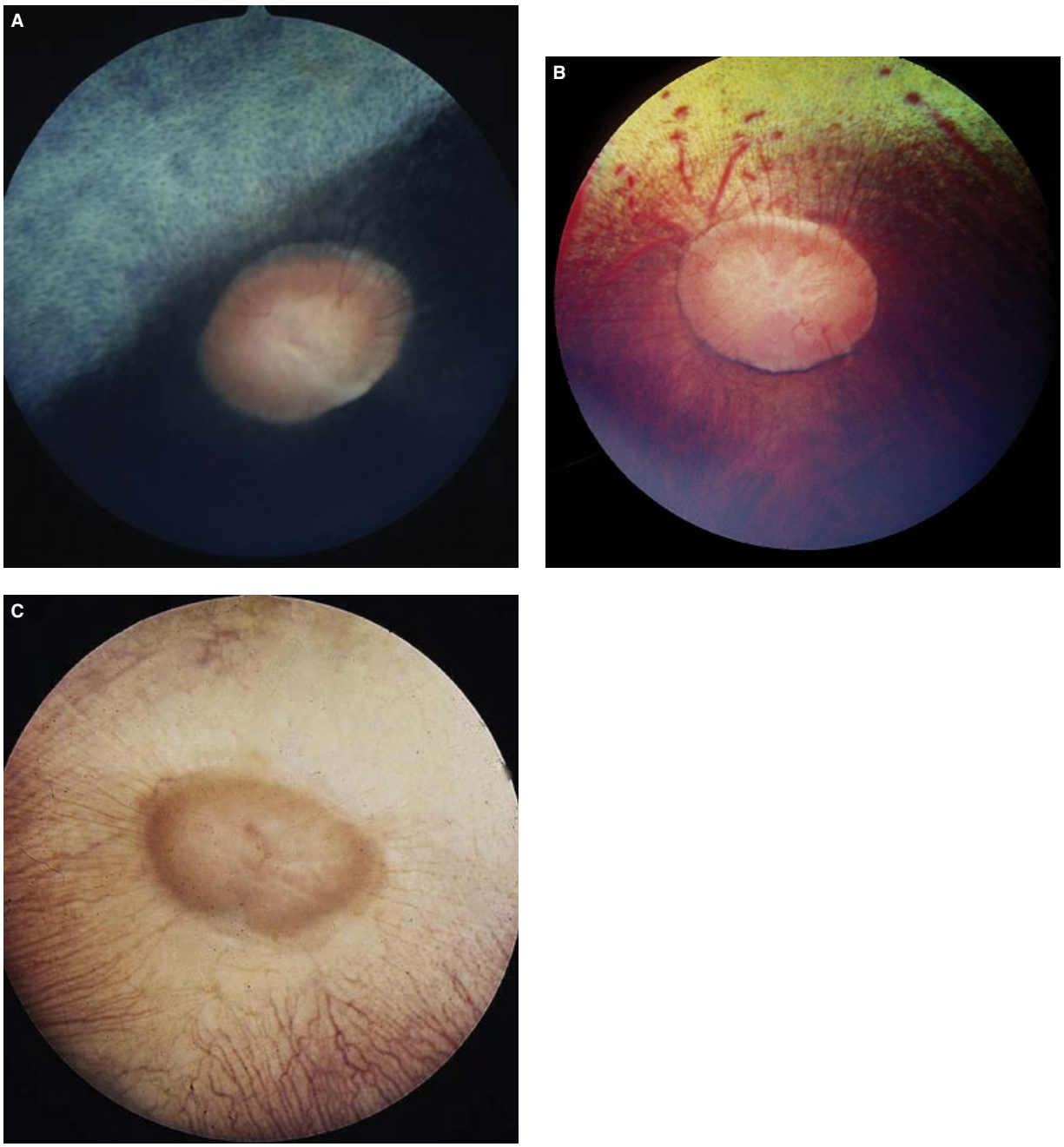


Figure 15.36 The normal ocular fundus of the horse has several variations, and most are related to iris and hair coat color changes. (A) Normal ocular fundus in a bay Thoroughbred horse. Note the optic disc is within the nontapetal fundus, and 50–80 retinal blood vessels emerge from its periphery. The retinal arterioles cannot be distinguished from the retinal veins. (B) Focal tapetal fundus albinism in a Palomino horse. The nontapetal fundus is not as densely pigmented in this animal, so it is possible to visualize some of the choroidal vasculature. (C) An ocular fundus in a white horse. Note the retinal pigment epithelium and choroidal pigmentation is absent, and the tapetal fundus is yellow.

Peripapillary chorioretinitis has been associated with, but is not pathognomonic for ERU. Visual impairment is variable depending on the severity and how much of the fundus is affected. The ophthalmoscopic appearance of an active lesion usually includes multiple areas of focal retinal edema adjacent to the optic disc which make that area appear fuzzy and indistinct. Chronic lesions appear as focal round to oval areas of mottled pigmentation affecting primarily the sides and beneath the optic disc.

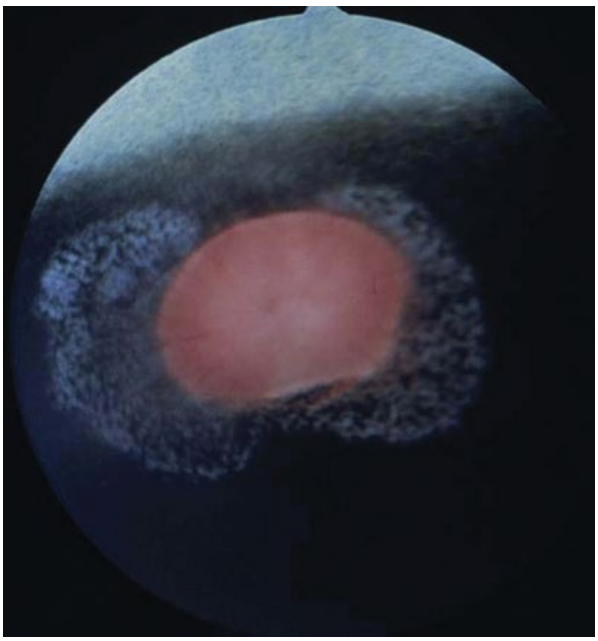


Figure 15.37 Chorioretinitis is acute or chronic, and can affect any region of the equine ocular fundus. Most often the area immediately around the optic disc is involved. In this horse with recurrent uveitis, the chronic uveitis has produced what is sometimes referred to as a "butterfly" chorioretinitis.

Retinal Detachments

Retinal detachments are infrequent in adult horses but result from trauma, vitreal degeneration, glaucoma, chorioretinitis, and ERU (Figure 15.38). Affected eyes show a slightly dilated pupil (the opposite eye provide the resultant pupil) and blindness. Ophthalmoscopic examination reveals retinal detachment with the retina attached at the optic disc and the ora ciliaris retinae. The retina appears as a gray to white membrane devoid of blood vessels. Retinal detachment surgery has not been successful in horses.

Optic Nerve Diseases

Optic disc atrophy or degeneration has been associated with chorioretinitis, ERU, trauma, blood loss, and glaucoma (Figure 15.39). Ophthalmoscopy reveals a small depressed white optic nerve head, usually devoid of retinal blood vessels. Traumatic optic neuropathy occurs in horses following head trauma, usually after kicks, sharp blows, or falling over backwards after rearing, especially if poll injury is sustained. The optic nerve damage may be the primary insult or associated with other brain lesions. Intracranial portions of the optic nerve can be traumatized, compressed, and/or displaced with cranial fractures and subsequent degeneration and blindness. One or both eyes are involved. Affected horses show impaired pupil light reflexes, loss of the dazzle reflex, and menace response. By ophthalmoscopy, optic nerve degeneration is first detected 1–4 weeks post-trauma.

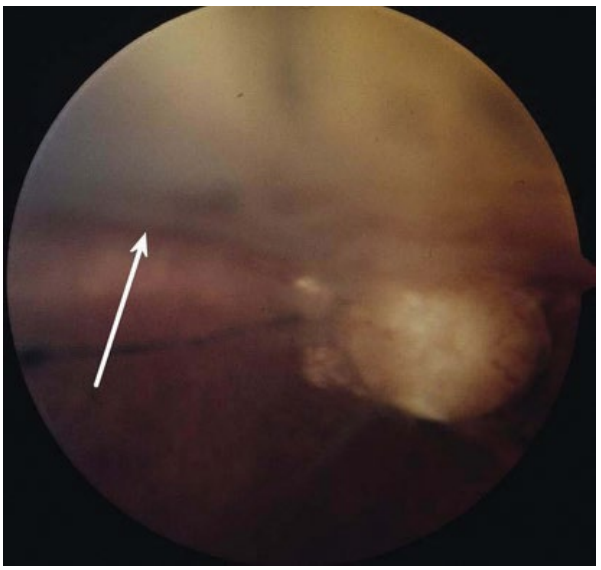


Figure 15.38 Retinal detachment in a foal following head trauma. Note the edematous and detached retina (arrow) immediately dorsal of the optic disc.

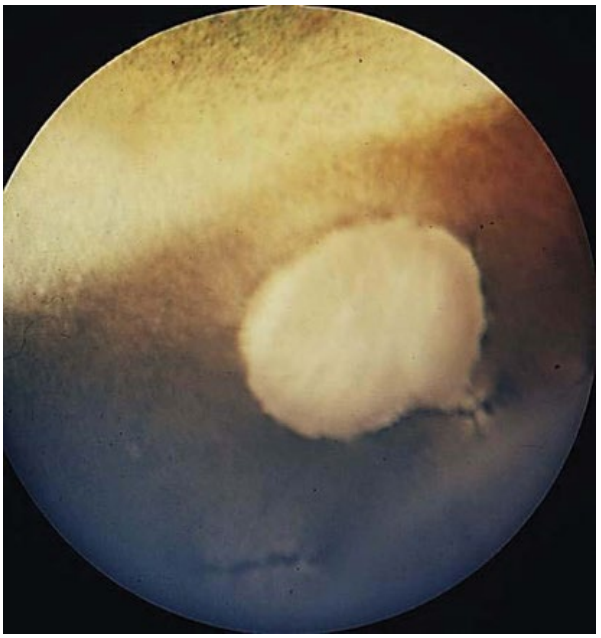


Figure 15.39 Optic disc degeneration can follow trauma, inflammation, glaucoma, and other causes in the horse. In this horse, head trauma has produced optic nerve degeneration, as evidenced by a white optic disc, devoid of retinal blood vessels, and pigmentation proliferation in the nontapetal fundus.

Proliferative neuropathy occurs in older horses and is characterized by a focal or multiple nodular yellow–white mass protruding from the optic disc's surface (Figure 15.40). There is no demonstrable effect on clinical vision. Histopathology has characterized these lesions as astrocytoma, schwannoma, or xanthoma.



Figure 15.40 Proliferative neuropathy in the horse is characterized by one or more cream-colored to yellow smooth protrusions from the optic disc margin. These masses are not associated with any changes in clinical vision, and occur most often in older horses.

Ischemic neuroretinopathy occurs in the horse in, at least, two situations. The first is marked blood loss following trauma and surgery and the second is after surgical ligation of the internal and external carotid arteries, or the greater palatine arteries for the treatment of epistasis secondary to guttural pouch mycosis (Figure 15.41).

Following massive hemorrhage, patients are often weak and unable to stand for a few days. Once standing and mobile, affected horses demonstrate variable mydriasis and blindness. Ophthalmic lesions in patients several weeks following the insult are limited to the ocular fundus and consist of multiple areas of chorioretinal degeneration and optic disc atrophy. The chorioretinal degeneration occurs throughout the tapetal and nontapetal fundi, and is characterized by pigment mottling (both loss and increase of pigmentation), and in the tapetal fundi increased reflectivity. The blindness appears related to the optic nerve degeneration.

The ophthalmoscopic appearance of the optic nerve head damage following the external and internal carotid and greater palatine artery ligations is somewhat different. The optic disc is initially swollen, contains focal hemorrhages, and has multiple white exudates protruding from the disc and extending into the vitreous (believed to be myelin and axoplasmic material). The classic appearance of optic nerve degeneration (disc pallor and loss of vessels) follows some time later.

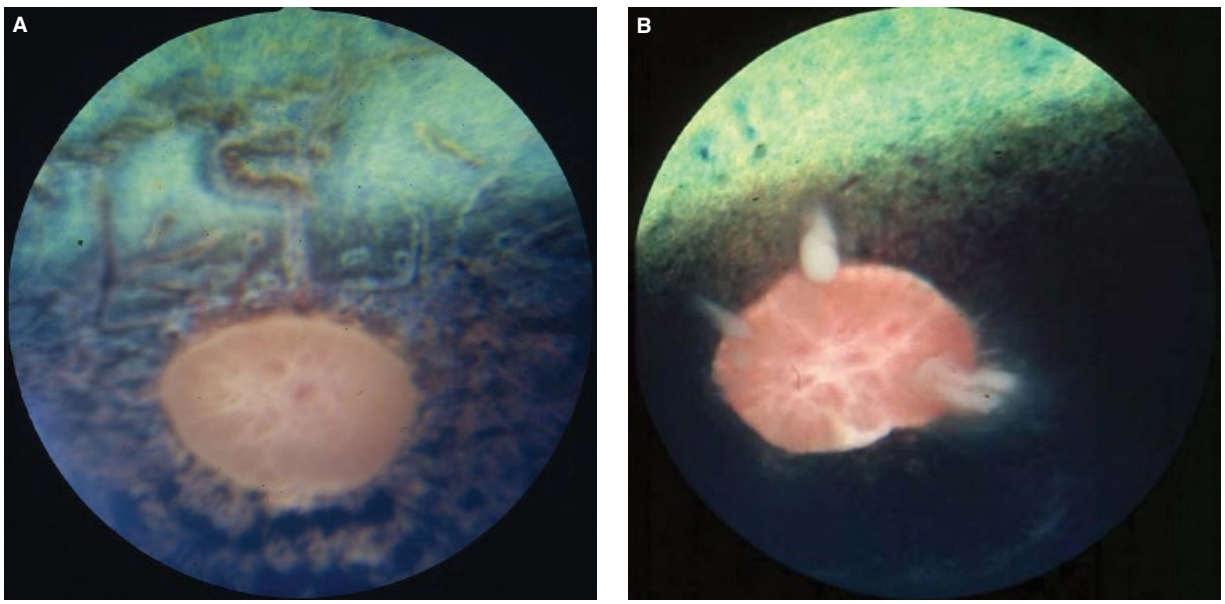


Figure 15.41 (A) Ischemic neuroretinopathy in a young Quarter Horse following castration complicated by massive and uncontrolled hemorrhage. Note the peripapillary areas of retinal degeneration, pigment proliferation, and loss of the retinal blood vessels. (B) Ischemic neuroretinopathy in a horse following carotid artery ligation. Note the hyperemia of the disc and the white material protruding from the disc.

16

Food and Fiber Animal Ophthalmology

Diseases of the Orbit

Microphthalmia, or a congenitally small eye, is one of the more frequent ophthalmic anomalies in food animals (Figure 16.1). Congenital eye anomalies occur in 3.5% of lambs, 5.5% of pigs, and 18.7% of calves. In calves, microphthalmia is the most frequent eye defect. Microphthalmia is inherited, viral (calves – bovine viral diarrhea virus; lambs – bluetongue virus), or nutritional (pigs – vitamin A deficiency), as well as combined with other ocular and systemic anomalies.

Clinically, microphthalmia occurs in one or both eyes and is characterized by a smaller than normal palpebral fissure, nictitans protrusion, and a small globe. Pupillary abnormalities, congenital cataracts, and retinal dysplasia and detachments can also occur.

Strabismus, or the deviation of the eye, occurs most frequently in cattle (Figure 16.2). Esotropia, or convergent strabismus, is inherited in the Jersey, German Brown Swiss, Holstein, Ayrshire, and Shorthorn breeds. It can be transmitted as a recessive (Jersey and Shorthorn cattle) or dominant trait (German Brown Swiss cattle). These eyes can also appear to be exophthalmic. Acquired strabismus, usually esotropia, also develops as a sign of polioencephalomalacia (PEM) and listeriosis in cattle.

Orbital neoplasms produce slow and progressive exophthalmia, strabismus, conjunctival hyperemia, and nictitans protrusion, and with advanced exophthalmia can also cause impaired blink, exposure keratitis, vision impairment, and even blindness. Orbital neoplasms in cattle are usually unilateral as with squamous cell carcinomas, but can be bilateral in lymphoma (Figure 16.3).

Diseases of the Eyelids

Dermoids affect the eyelids, conjunctiva, nictitating membrane, or cornea (Figure 16.4). In food animals, calves are most frequently affected, and dermoids are inherited in the Hereford breed. One or both eyes are affected. A white, black, or colored mass with long coarse hair extending from its surface will be present upon examination of the irritated eye. Recommended treatment, when pain and disfigurement are present, is superficial keratectomy. Limited scarring results after surgery.

Entropion in food animals occurs most frequently in lambs, and a considerable percentage (up to 80%) of the flock can be affected. The entropion affects one or both eyes, and either the upper or lower eyelids, or both (Figure 16.5). Clinical signs range from mild blepharospasm, lacrimation, nictitating membrane protrusion, and conjunctivitis with serous exudates to severe blepharospasm with mucopurulent conjunctivitis and corneal ulceration. Persistent entropion with corneal ulceration can lead to corneal perforation and iris prolapse.

To treat entropion in immature animals, the eyelid margins are everted to the normal position (away from the cornea) using vertical mattress sutures or skin staples. Permanent surgical correction with the Hotz–Celsus procedure used in small animals should be delayed until the animal has approximated its adult size. The eyes of lambs have excellent healing ability, and a completely opaque but nonperforated cornea can clear within several weeks.

Diseases of the Conjunctiva and Cornea

Chlamydophila Infectious Keratoconjunctivitis in Sheep and Goats

Sheep and goats present with similar forms of infectious keratoconjunctivitis (Figure 16.6). *Chlamydophila psittaci* and the *Mycoplasma* spp. (*M. conjunctivae*, *M. mycoides* var. *capri*, and *M. conjunctivae* var. *ovis*) are the most commonly implicated pathogens. Other microorganisms have been isolated, such as *Branhamella ovis, Escherichia coli,* and *Staphylococcus aureus,* but these are often found

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Figure 16.1 (A) Microphthalmia in a goat. Note the elevated nictitans, enophthalmia, and mydriasis. (B) Marked microphthalmia in a white Shorthorn calf with the palpebral fissures less than 50% of normal size.



Figure 16.2 (A) Inherited strabismus in a Holstein cow. Note the exposed lateral bulbar conjunctiva and esotropia (convergent strabismus).



Figure 16.2 (*Continued*) (B) Congenital strabismus and microphthalmia in a Hereford.

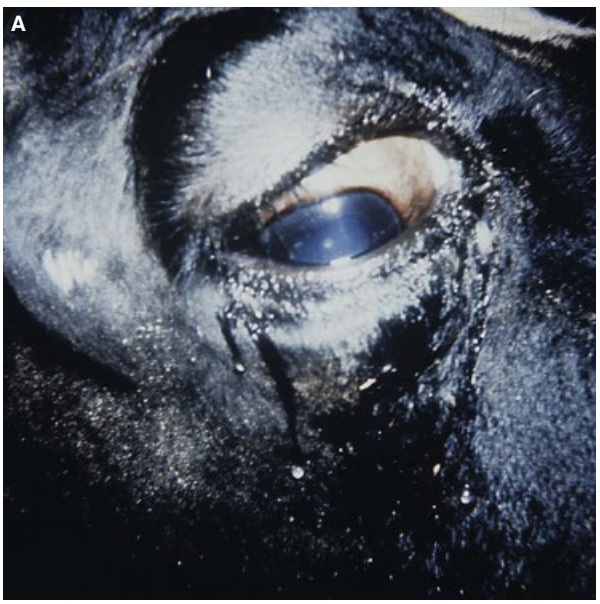




Figure 16.3 (A) Lymphoma and squamous cell carcinoma are the most common forms of orbital neoplasia in cattle. In this Holstein cow, systemic lymphoma has involved the retrobulbar tissues and resulted in exophthalmia. (B) Extensive size and invasion of the orbital bones with squamous cell carcinoma in this Hereford cow.



Figure 16.4 Corneoconjunctival dermoid in Jersey calf involving the ventromedial bulbar conjunctiva and cornea. Note the brown mass and the long coarse hair protruding from its surface.



Figure 16.5 Entropion in the most common ophthalmic disease in sheep. In this lamb entropion affects the entire lower eyelid and had caused a ventral corneal ulcer. Note the corneal vascularization.

in mixed infections or concurrent with the main pathogens. *Chlamydophila* is most prevalent during lambing and when lambs are present, and adults can be affected repeatedly (over years) suggesting limited immunity.

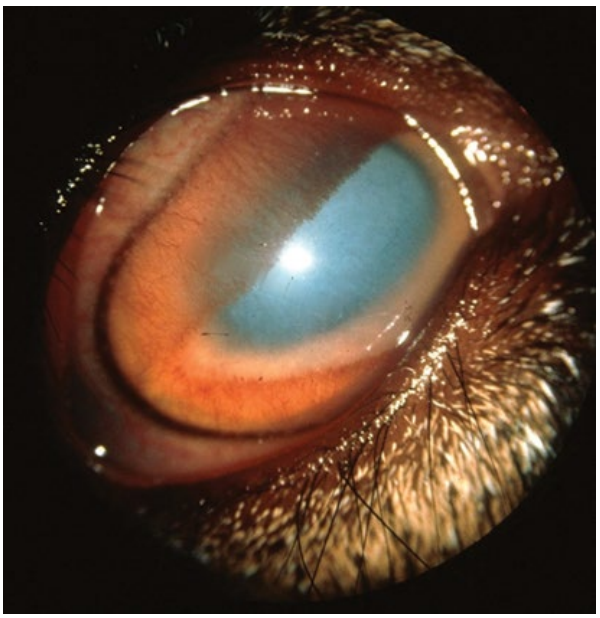


Figure 16.6 *Chlamydophila* infectious keratoconjunctivitis in a ram. Note the interstitial keratitis and corneal edema.

Polyarthritis can also occur with the keratoconjunctivitis. The disease affects predominately the conjunctiva (epiphora, chemosis, and conjunctival hyperemia), and serous progressing to mucopurulent conjunctival exudates. Conjunctival lymphoid follicles can develop in the third week of infection when clinical signs are regressing. The corneal disease does not affect all sheep and consists of interstitial (deep) keratitis, corneal vascularization, and pigmentation. Diagnosis can be made using conjunctival scrapings and cytology. The disease is generally self-eliminating, but systemic tetracyclines can shorten the clinical signs.

Mycoplasma Infectious Keratoconjunctivitis

Both sheep and goats are also affected with mycoplasmal infectious keratoconjunctivitis, and this disease is generally more severe than that caused by *Chlamydophila*. The disease starts as a conjunctivitis that then extends to the peripheral cornea as microabscess formation. Initial clinical signs include eyelid swelling, conjunctival hyperemia, epiphora, and blepharospasm (Figure 16.7). Soon, keratitis with both superficial and deep vascularization develops with the formation of microabscesses. The conjunctival exudates range from serous (early) to mucopurulent (late). Infrequently, and in severely affected eyes, corneal ulceration, iridocyclitis with hypopyon, and panophthalmitis develop.



Figure 16.7 (A) Early mycoplasmal infectious keratoconjunctivitis in a young goat. Note the circumferential corneal neovascularization and corneal infection. (B) Mycoplasmal infectious keratoconjunctivitis in a goat. Note the conjunctival exudates, marked corneal vascularization, corneal edema, and corneal microabscesses below the dorsal blood vessels. A secondary iridocyclitis is also present. (C) More advanced mycoplasmal keratoconjunctivitis. The entire cornea is opaque and surrounded by corneal vascularization. The disease has progressed to panophthalmitis.

Diagnosis is made by means of clinical signs and cytology (intracytoplasmic coccobacillary to ring-shaped bodies). For treatment, topical and intramuscular tetracyclines are recommended. These mycoplasmal organisms have also been associated with mastitis, arthritis, and pleuropneumonia.

Infectious Bovine Keratoconjunctivitis

Infectious bovine keratoconjunctivitis (IBK) occurs worldwide, and affects all breeds of cattle (Figure 16.8). The infection rates vary by season, but can be as high as 63% in cows and 75% in calves. Older, previously exposed cattle generally develop a milder form of the disease, suggesting some immunity develops and persists. The *Bos indicus* are more resistant than the *Bos taurus* breeds. Although many different bacteria have been implicated, the hemolytic and piliated form of *Moraxella bovis* is known to cause the disease. Transmission occurs via flies, animal handlers' hands, or direct contact. The nonhemolytic and nonpiliated forms of *M. bovis* appear to be







Figure 16.8 The clinical appearance of infectious bovine keratoconjunctivitis (IBK) varies depending on the stage of the disease. (A) Early IBK with intense conjunctivitis, and a central large corneal ulcer and edema with corneal vascularization. (B) Another case of IBK. Note the abscessed cornea, corneal edema, and peripheral vascularization. (C) More advanced IBK with corneal ulceration perforation and iris prolapse. The pigmented iris is protruding within the necrotic corneal ulcer. Considerable edema and vascularization involve the surrounding cornea.

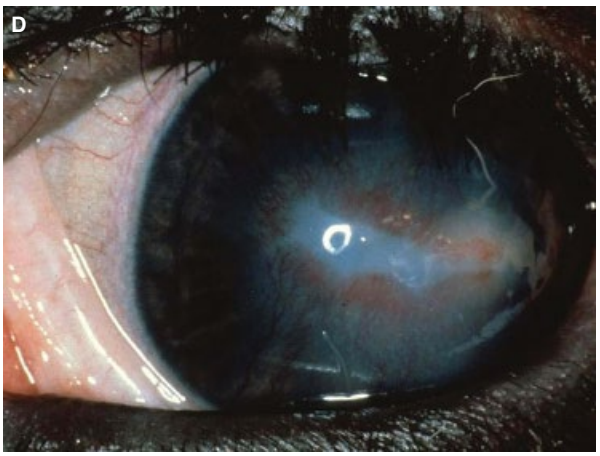


Figure 16.8 (*Continued*) (D) IBK with a healing corneal ulcer. There is faint fluorescein retention in the central ulcer. A faint to small corneal opacity will result.

normal inhabitants of the bovine conjunctival sac. The nonpathogenic *M. bovis* appears to undergo conversion to the pathogenic form quickly when exposed to greater ultraviolet radiation during the early summer months.

The initial early clinical signs include marked lacrimation, blepharospasm, photophobia, conjunctival hyperemia, and chemosis. Within 24–48 hours a central small pale yellow corneal abscess forms that quickly sloughs leaving a shallow corneal ulcer. The corneal ulcer can expand in size and depth, and a secondary iridocyclitis develops. Superficial corneal vascularization starts within a few days, and within 5–7 days the entire cornea is vascularized leading to healing of the corneal ulcer. Within 2 weeks the corneal ulcer is usually healed, and by 1–2 months only a faint central corneal scar remains. Infrequent complications of IBK include corneal perforation and iris prolapse, panophthalmitis, and secondary glaucoma.

Fortunately, *M. bovis* is usually sensitive to most topical antibiotics, and systemic antibacterial medications are not usually necessary (drug residues in meat and milk). Several vaccines have been developed for IBK, but offer limited protection. The most effective vaccination schedule is when the vaccine is administered and boostered prior to the beginning of the summer season.

Neoplasia

Periocular and ocular squamous cell carcinomas (OSCC) are the most frequent neoplasms in cattle but are rare in other food animal species. Age, increased levels of solar radiation, and lack of eyelid pigmentation are predisposing factors. The Hereford breed seems to be the most frequently affected (Figure 16.9).

OSCC affect the bulbar conjunctiva and cornea (most often the dorsolateral limbus), the palpebral

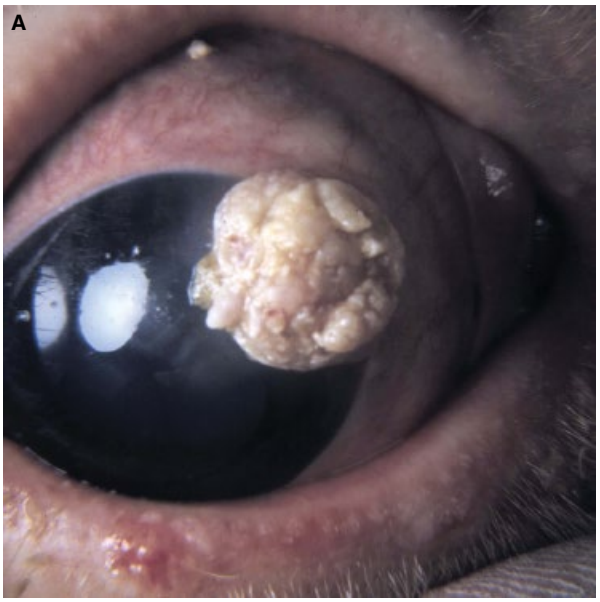


Figure 16.9 (A) Limbal squamous cell carcinoma in an 8-year-old Hereford cow. The mass involves the dorsomedial limbus.



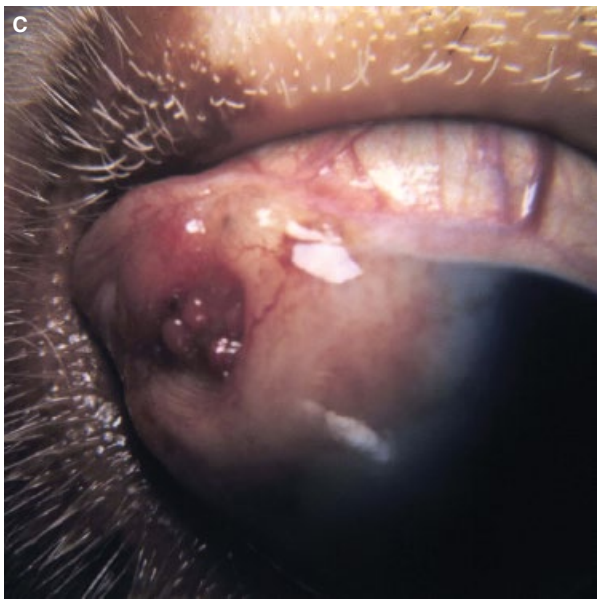




Figure 16.9 (*Continued*) (B) An ulcerative squamous cell carcinoma involving the medial lower eyelid in a Hereford. Involvement of the lower lacrimal punctum also occurred. (C) Squamous cell carcinoma of the lateral bulbar conjunctiva and peripheral cornea in a Hereford cow. Intraocular infiltration by the neoplasm has already occurred. (D) Advanced squamous cell carcinoma of the entire entire lower lid and both canthi. Complete excision of this size of mass would be very difficult.

conjunctiva, the nictitating membrane, or the eyelid skin. Limbal OSCC progress through four stages: plaque (hyperplastic epithelium); papilloma (core of connective tissue); noninvasive carcinoma (does not penetrate the lamina propria of the epithelium); and, lastly, carcinoma. Secondary ulceration, necrosis, inflammation, and hemorrhage occur. Systemic metastases appear late, usually to the regional lymph nodes, orbital bones (maxilla and frontal), and parotid salivary gland and are most common with eyelid and nictitans lesions. Distant metastatic sites include lungs, heart, pleura, liver, kidney, and thoracic lymph nodes.

Treatment of OSCC is most successful when the masses are small and localized, and large surgical defects can be avoided. Treatment modalities include surgical excision, cryotherapy, hyperthermy, radiation, or a combination of these therapies.

Diseases of the Anterior Uvea

Persistent pupillary membranes (PPMs) are rare congenital iridal anomalies in food animals (Figure 16.10). Most often, iridal attachments to either the lens or cornea are post-inflammatory. As in other species, PPMs originate from the iridal collarette area, a few millimeters from the pupil margin. PPMs can extend to other areas of the iris, the anterior lens capsule (causing focal cataracts), and/or to the posterior cornea (causing deep corneal opacities). Treatment is not usually necessary.



Figure 16.10 Persistent pupillary membranes (PPMs) and a pigmented anterior capsular cataract in a cow. Note the iridal attachments originate from its anterior surface.

Heterochromia Iridis

Heterochromia iridis is a change of color within a single iris or between opposite irides. The heterochromic iris is white, blue and white, or blue, white, and brown. The granula iridica or corpora nigra of the upper and lower pupillary margins are usually black. Heterochromia iridis is the most frequent anterior uveal condition in cattle, and in one report represented 10% of the total ocular congenital variations.

Heterochromia iridis in cattle is often inherited and commonly affected breeds include Hereford, Ayrshire, Holstein, Angus, Brown Swiss, and Guernsey (Figure 16.11). In the Hereford breed, iridal heterochromia is also associated with tapetal fibrosum hypoplasia and colobomas of the optic nerve head, and the condition is inherited as an autosomal dominant trait. In albino Guernesy cattle, the iris is pink to white with a non-pigmented granula iridica, and photophobia (increased sensitivity to light) and nystagmus are present.

Heterochromia Iridis in Pigs

Heterochromia iridis also occurs in swine, and appears to be more often associated with white hair (38%) than pigmented coats (16%) (Figure 16.12). The irides appear blue to a combination of blue and black, and the ocular fundus also has reduced pigmentation. Heterochromia iridis is inherited in pigs as an autosomal recessive trait.

Inflammation of the Anterior Uvea

In food animals, iridocyclitis is usually secondary to corneal or systemic diseases (Figure 16.13). Clinical signs include photophobia, blepharospasm, lacrimation, corneal edema, deep corneal vascularization, aqueous flare to hypopyon, swollen irides, miosis, and posterior synechiae formation and cataracts. With systemic diseases, both eyes are usually involved.

Causes in cattle include neonatal infections, bacterial septicemia associated with severe mastitis, metritis, or traumatic reticuloperitonitis, malignant catarrhal fever, tuberculosis, IBK, thromboembolic meningoencephalitis, leptospirosis, toxoplasmosis, listeriosis, and lymphoma. In sheep and goats, etiologies include neonatal pyosepticemia with various bacterial agents, listeriosis, mycoplasmal disease, toxoplasmosis, thiamine deficiency, trypanosomiasis, blunt trauma, and various toxemias. Treatment depends on the cause; however, both topical and systemic antibiotics, and topical mydriatics are usually indicated.

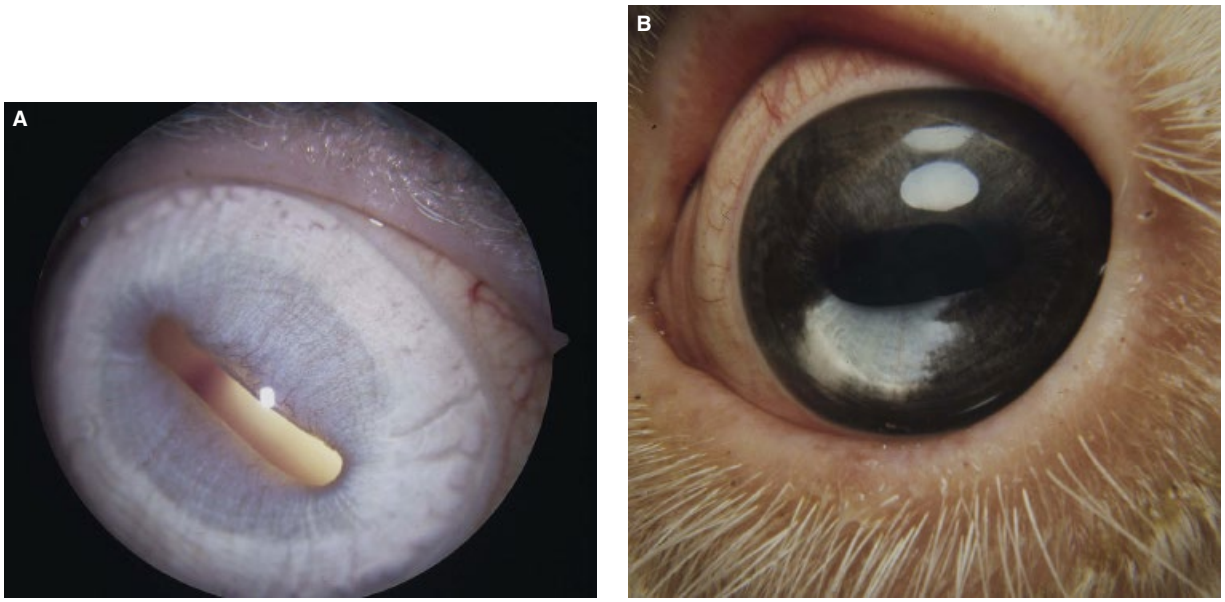


Figure 16.11 (A) Heterochromia iridis in an albino Guernsey calf. Note the complete absence of iridal pigmentation, and the iridal colors are white and pink. Nystagmus and photophobia are also present. (B) Incomplete albinism and heterochromia iridis in a Hereford cow. The iris has brown, blue, and white zones. The granula iridica is absent or poorly developed dorsally and ventrally.

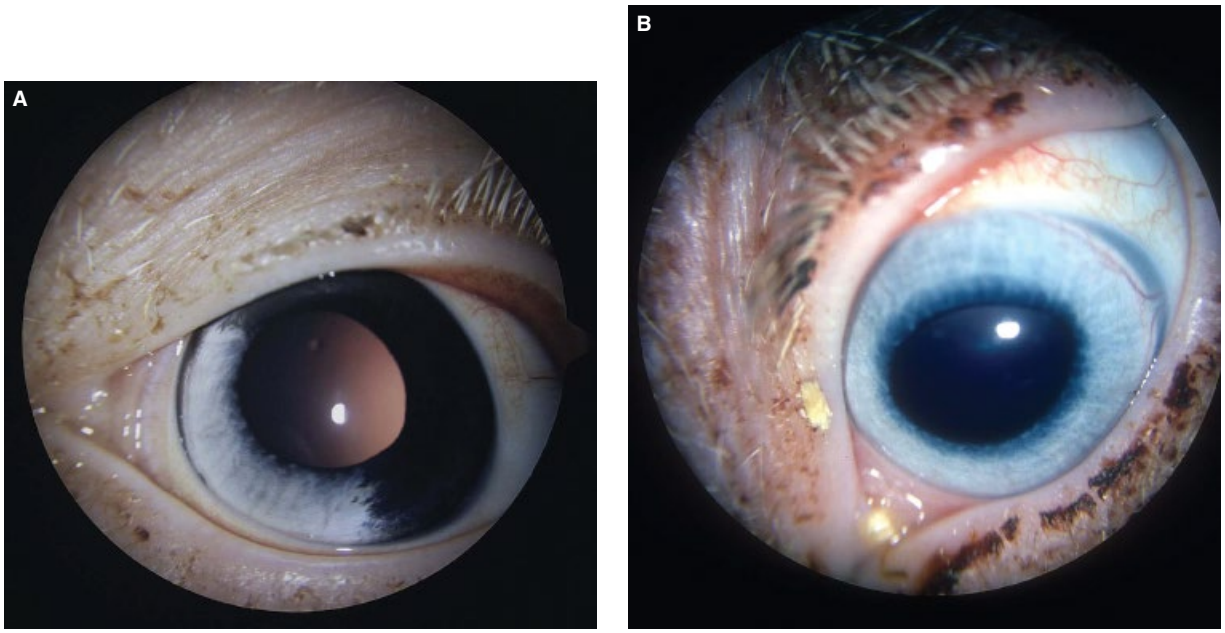


Figure 16.12 (A) In this partly pigmented pig, the iris is only a mix of brown, blue, and white. (B) In this white pig the iris is mostly blue and white.

Glaucomas

The different forms of glaucoma are rare in food animals. In cattle, secondary angle closure glaucoma is most frequent, following severe IBK (Figure 16.14). In the Holstein breed in New Zealand, glaucoma (with enlargement of the globe or buphthalmia), cataract formation, and lens luxation have been reported. Treatment is possible but rarely pursued.

Diseases of the Lens

Cataracts occur fairly frequently in cattle; however, rarely do the cattle behave as if they are significantly impaired even with advanced cataract (Figure 16.15). Cattle seem to behave in illuminated obstacle courses as visual, even though a corneal or lens opacity is dense enough to prevent indirect ophthalmoscopy. This may



Figure 16.13 Severe iridocyclitis in a cow secondary to infectious bovine rhinotracheitis. The cornea is very edematous.

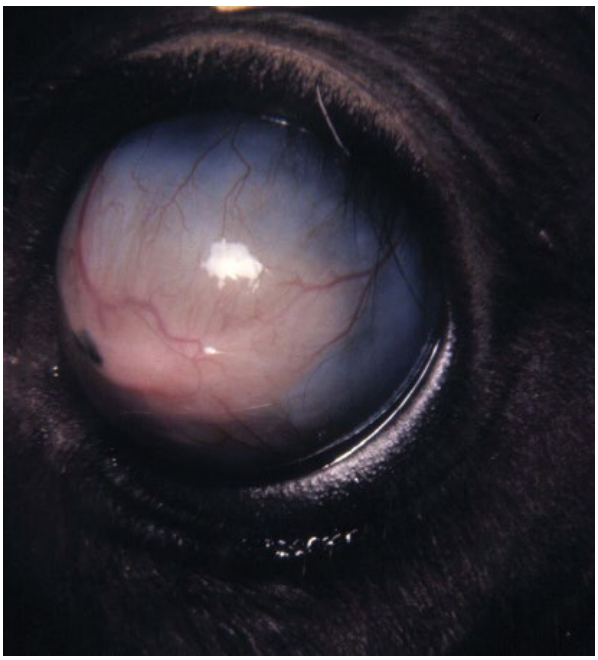


Figure 16.14 Secondary glaucoma in a Holstein calf secondary to infectious bovine keratoconjunctivitis. The central corneal ulcer has perforated and iridocorneal angle closure resulted in the elevation in intraocular pressure and globe enlargement. The protruding cornea is inflamed and vascularized.

be an adaptation based on their status as a prey species. Congenital cataracts occur in calves, lambs, goats, and piglets, but appear most frequent in calves. Autosomal recessive congenital cataracts affect the Jersey, Hereford,

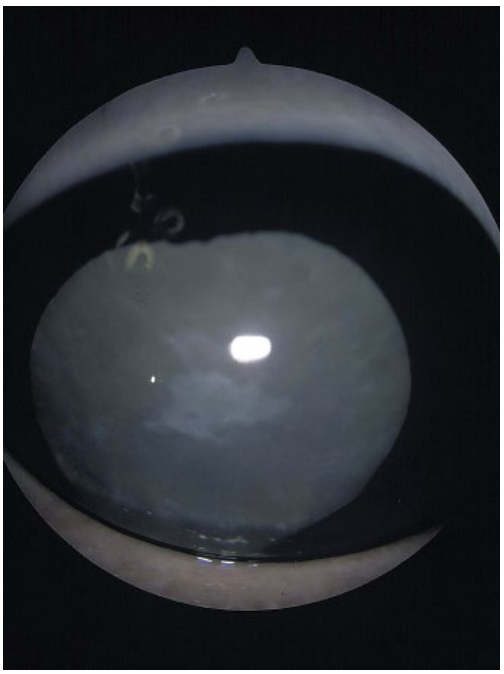


Figure 16.15 Congenital cataracts are uncommon in calves, and can be inherited. In this Hereford calf the cataracts were bilateral. The calf's dam also had bilateral cataracts.

and Holstein breeds, and in these breeds as well as the Shorthorn breed can also occur with other ocular anomalies (microphthalmia, persistent pupillary membrane, retinal dysplasia and detachments).

Congenital cataracts with other ocular anomalies in calves and lambs can also be secondary to intrauterine viral infections (calves – bovine viral diarrhea virus; lambs – bluetongue virus). Successful cataract surgery (usually phacoemulsification) has been performed in both calves and lambs.

Cataracts secondary to anterior uveitis are not infrequent in cattle, because anterior uveal inflammation occurs commonly and is rarely treated promptly or aggressively, if it is treated at all (Figure 16.16). With the lack of mydriatics for iridocyclitis, the inflamed iris readily adheres to the anterior lens capsule (posterior synechiae formation), and pieces of torn iris appear on the anterior lens capsule (suggesting previous inflammatory attachments). The clinical history is often signs of anterior uveitis followed by resolution of the inflammation but progressive vision impairment and eventual blindness. If blindness is present, usually only cataract surgery can restore vision. With inhalation general anesthesia, cataract surgery (phacoemulsification) can be performed (similar to the horse, dog, and human). Postoperative treatment should include topical and systemic antibiotics, mydriatics (usually atropine), and topical corticosteroids.



Figure 16.16 Cataracts secondary to anterior uveitis are more frequently encountered in food animals. In this goat multiple posterior synechiae and advanced cataract formation are present.

Diseases of the Ocular Fundus

Familiarization with the normal bovine, ovine, caprine, and porcine ocular fundi is essential for the diagnosis and monitoring posterior segment diseases (Figure 16.17). In cattle, the tapetal fundus is usually yellow–green with prominent "stars of Winslow" (penetration of the tapetal fibrosum by the choriocapillaries); the nontapetal fundus is brown–black but may show less pigmentation in cows with lightly pigmented hair coats; the optic disc is at the junction of the tapetal and nontapetal fundus, and large prominent retinal arteries and veins emerge from its center and margin and traverse the superficial retina.

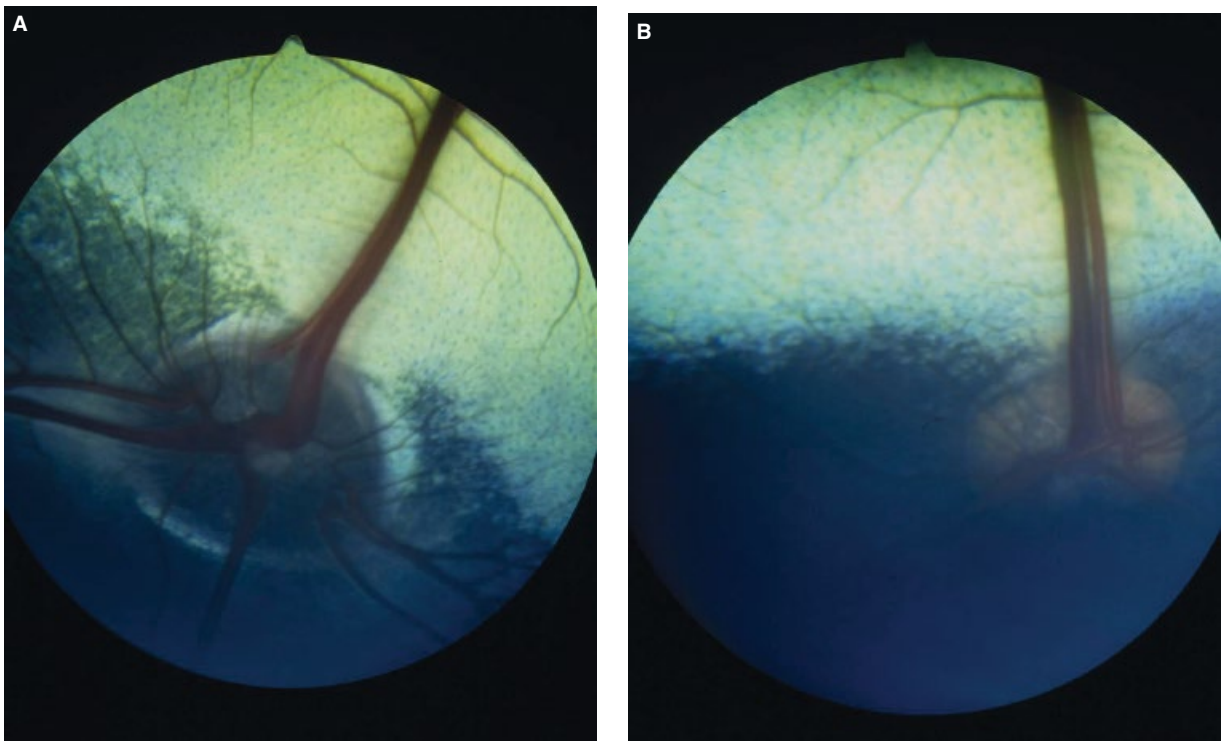
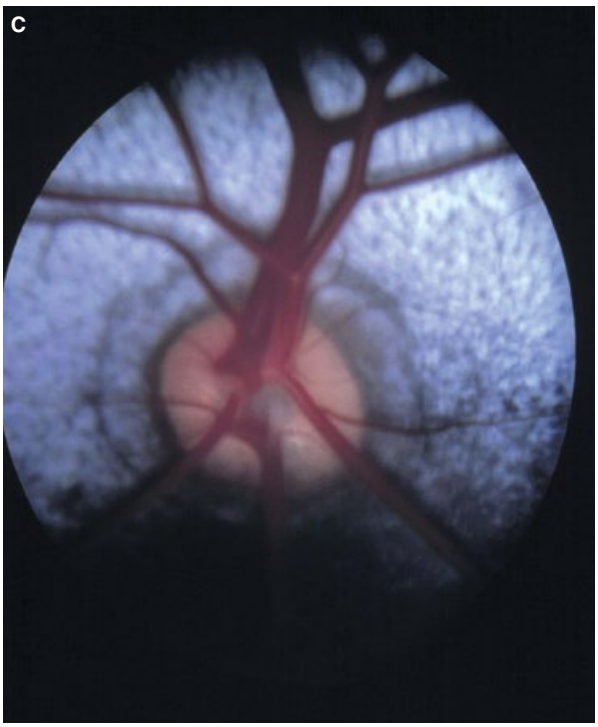


Figure 16.17 (A) Normal ocular fundus of the cow. The triangular and dorsal tapetal fundus is usually yellow–green; the nontapetal fundus is dark brown to black; the optic disc is located at the junction of the tapetal and nontapetal fundus; and the primary retinal blood vessels are large and protruding somewhat into the vitreous. (B) The normal ocular fundus of sheep is similar to the cow, but the optic disc has a notch in the ventral margin (sometimes referred to as "kidney bean" disc).



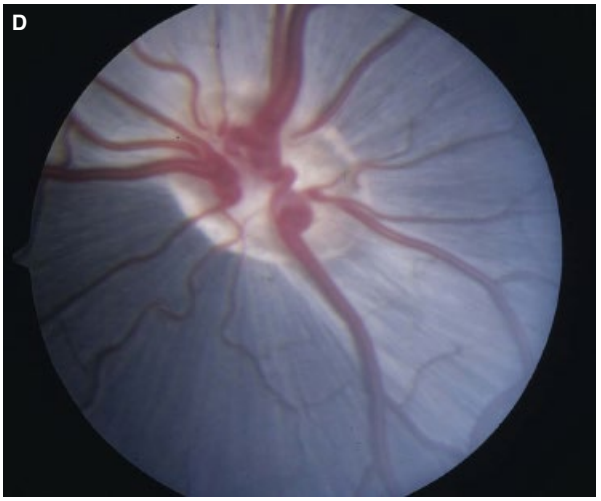


Figure 16.17 (*Continued*) (C) The normal ocular fundus of goats differs from sheep and cattle. The round optic disc is more distinct (due to the presence of a larger amount of myelin), and located within the tapetal fundus. The primary retinal vessels are quite prominent. (D) The normal ocular fundus of pigs lacks a tapetal zone, and the amount of pigmentation of the entire fundus varies depending on the amount of white hair coat of the animal. The optic disc is oval and has prominent primary retinal blood vessels.

In sheep, the ocular fundus is similar to that in cattle, but the optic disc has a distinct "kidney bean" shape with a noticeable notch at the 6 o'clock position. In goats, the optic disc is distinct, round, and more prominent.

In swine, there is no tapetum, and the entire ocular fundus is variably pigmented (associated with iris and hair color). Retinal arteries and veins emerge from the optic disc's center and periphery, and traverse the retina.

Colobomas of the Ocular Fundus

Typical optic nerve colobomas or defects of the ventral (6 o'clock position) optic disc are not infrequent in cattle, can be inherited, and affect the Charolais and Hereford breeds most often (Figure 16.18). Typical optic nerve colobomas usually affect both eyes but are not usually symmetrical. In Herefords, optic nerve colobomas are larger and inherited as an autosomal dominant trait, and are associated with mostly white hair coat, iridal heterochromia, and tapetal hypoplasia. In the Charolais breed, these smaller optic disc colobomas appear inherited in either a dominant or polygenic mode. Breeding of affected animals is not recommended.

Inflammation of the Ocular Fundus

Chorioretinitis occurs in all food animals, usually secondary to systemic or infectious diseases (Figure 16.19). Posterior segment inflammation can be masked by concurrent anterior uveitis. Clinical signs are usually those associated with anterior uveitis (blepharospasm, discharge, and anterior segment opacities), or vision impairment or blindness secondary to generalized chorioretinitis, exudative retinal detachments, and/or optic neuritis.

Common causes of chorioretinitis in cattle include neonatal bacterial (often *Escherichia* and *Pasteurella* sp.) septicemia, thromboembolic meningoencephalitis (*Haemophilus somnus*), rabies, viral diarrhea, toxoplasmosis, tuberculosis, and listeria. In sheep and goats, frequent causes are mycoplasmal disease, listeria, elaeophorosis, trypanosomiasis, toxoplasmosis, scrapie, and bluetongue virus.

Retinal Degenerations

Retinal degenerations in food animals are usually not inherited (reported only in the Toggenburg goat), in contrast to companion species. Often, retinal degenerations occur secondary to various plants and toxins. The ingestion

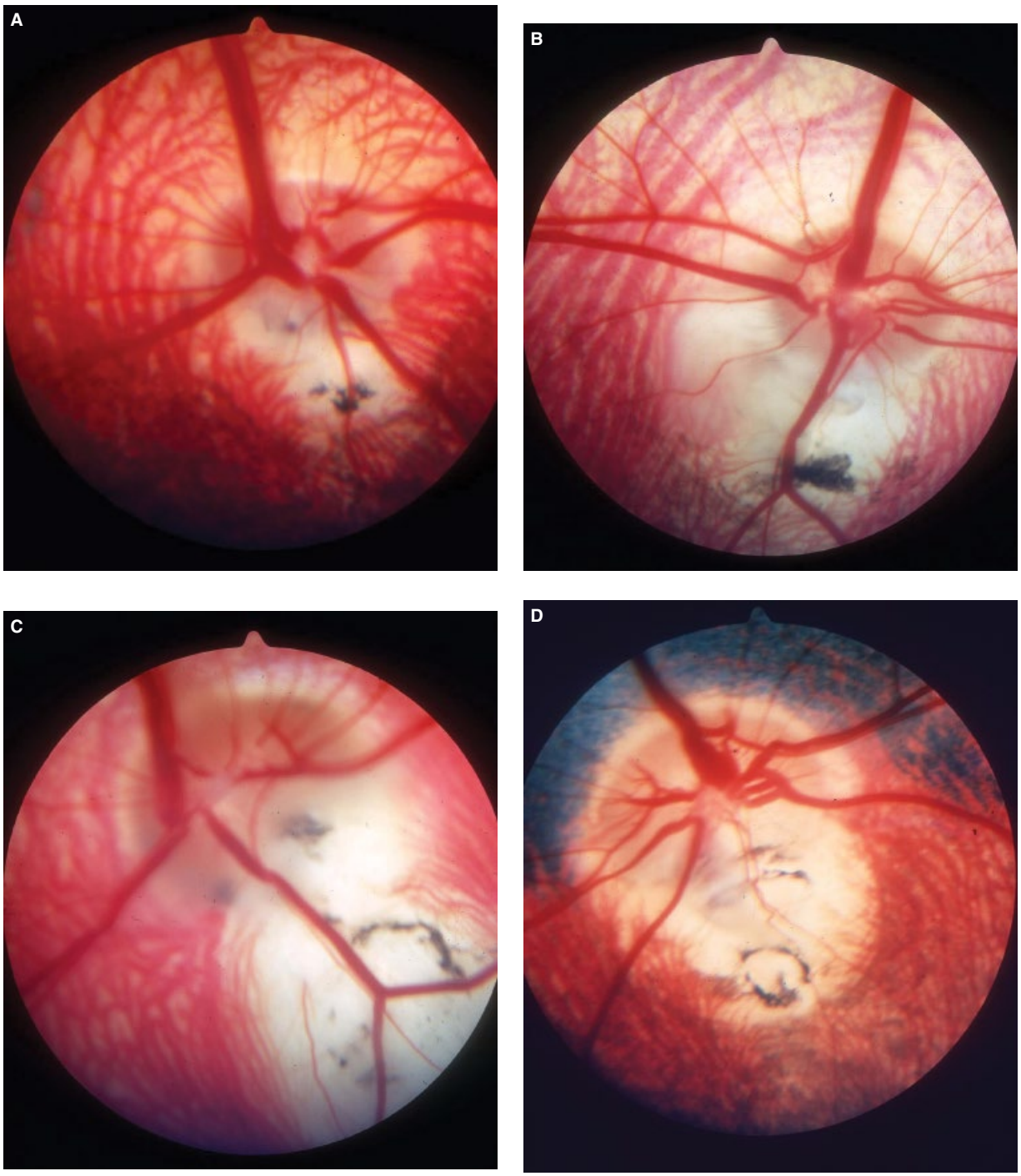


Figure 16.18 (A) Typical or ventral optic nerve head coloboma in a white Hereford cow with heterochromia iridis. The very small coloboma is devoid of choriodal blood vessels and has a patch of pigmentation. (B) White Hereford with larger typical optic nerve coloboma. The white and depressed coloboma is immediately beneath the optic disc. Within these colobomas, the retina is very thin and glial-like. (C) White Hereford with a large typical coloboma. Large retinal blood vessels traverse the coloboma's surface. There is no choroidal blood vessel in the coloboma which allows the sclera to be visualized. (D) White Hereford cow with some pigmentation and a typical optic nerve head coloboma.

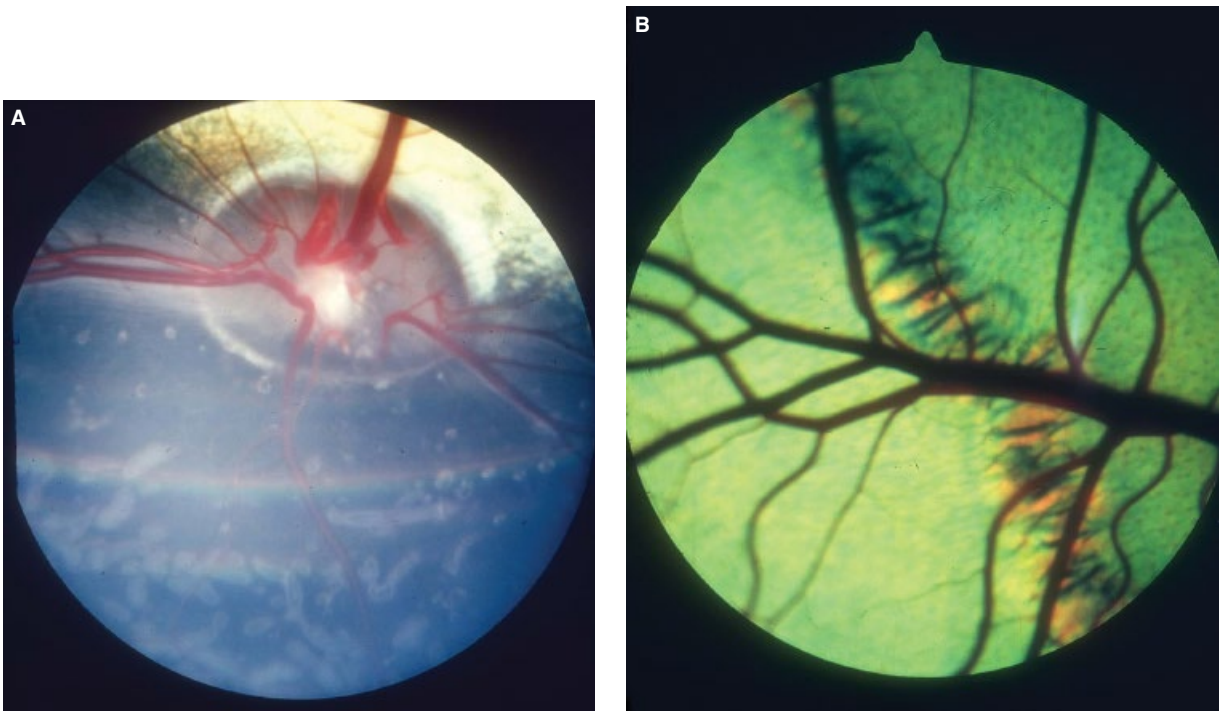


Figure 16.19 Ocular fundus inflammations in cattle can be associated with systemic infectious diseases. (A) Chorioretinitis in a young heifer affecting the nontapetal fundus. Note the inflamed and edematous areas have indistinct margins and some appear raised. (B) Chronic chorioretinitis or chorioretinopathy in an adult Guernsey cow after streptococcal pyosepticemia. Note the change of color and loss of the tapetal fibrosum and increased pigmentation.

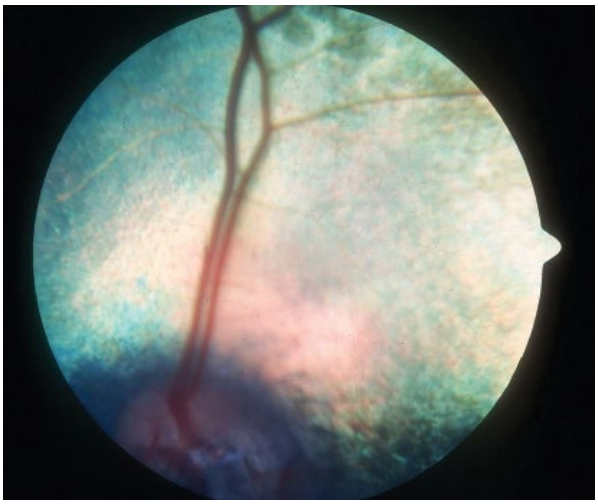


Figure 16.20 Advanced nutritional retinal degeneration in a sheep. Note the increased tapetal reflectivity and paucity of retinal vasculature. Courtesy of Keith C. Barnett.

of bracken fern (*Pteris aquilina)* in sheep produces degeneration of the outer retinal layers, resulting in variable mydriasis, vision impairment, and blindness (Figure 16.20). The ophthalmoscopic signs are primarily increased tapetal reflectivity, loss and attenuation of the retinal blood vessels, and optic disc atrophy.

Avitaminosis in Cattle

Vitamin A deficiency in cattle can have two manifestations, depending on the age of animal when exposed to the lack of adequate vitamin A (Figure 16.21). Affected calves develop bilateral optic nerve hypoplasia while affected adult cattle exhibit papilledema and retinal degeneration. Microphthalmia and other ocular anomalies occur in affected piglets with hypovitaminosis A. Low maternal vitamin A levels cause abnormal bone development and a reduced diameter of the optic nerve canal, optic nerve hyperplasia, and blindness in calves.

In growing and adult cattle, low vitamin A levels cause increased cerebrospinal fluid pressure, papilledema, and generalized retinal degeneration. As viewed with ophthalmoscopy, the optic nerve head is raised and swollen with congestion of the retinal vessels. The retinal degeneration causes increased tapetal reflectivity, and loss of pigmentation of the nontapetal fundus. Plasma blood levels of vitamin A less than 20 mg/dL are usually diagnostic. Therapy with parenteral vitamin A (440 IU/kg) can be successful in adult cattle if the retinal degeneration is not advanced.

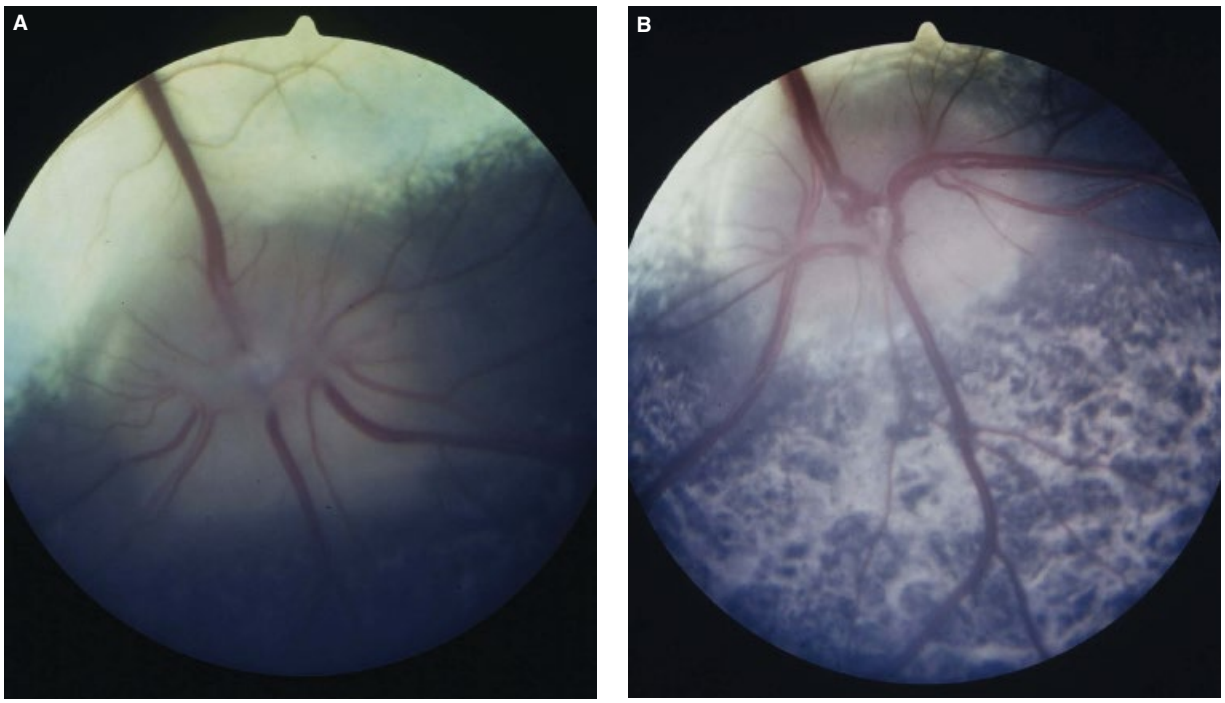


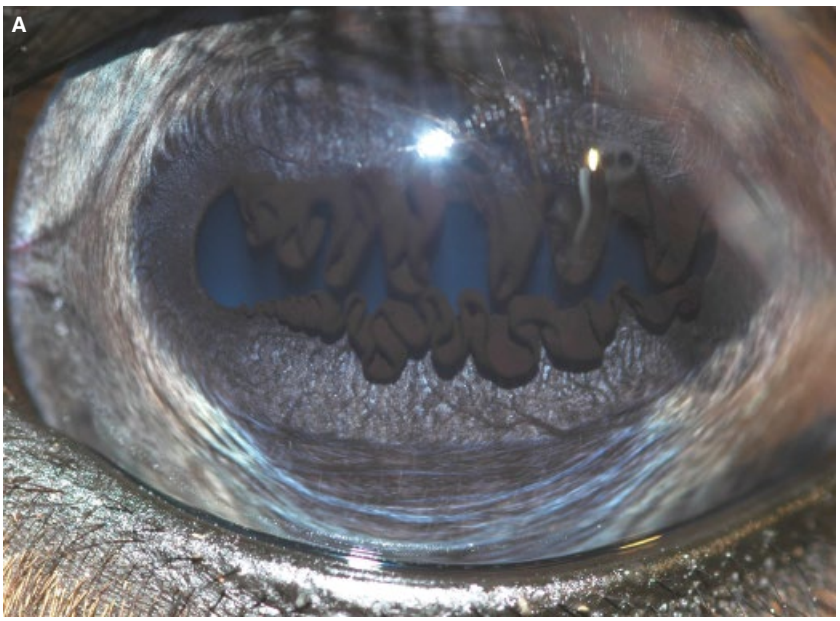
Figure 16.21 (A) Vitamin A deficiency in a heifer with papilledema. The enlarged and protruding optic disc has irregular or "fuzzy" margins. The retinal vasculature is normal. (B) Chronic vitamin A deficiency in a young steer. Both papilledema with some optic disc degeneration, and focal loss of pigmentation on the nontapetal fundus (signaling retinal degeneration) are present.

Ophthalmic Diseases in New World Camelids

Of the New World Camelids, the llama and alpaca species are the most common domesticated species, and the most likely to be presented for ophthalmic disease. Maintained in environments such as farms, zoos, ranches and woodlands, these animals function as food and fiber sources, pack animals, and as guard animals for sheep and goats. Their distinct and prominent eyes are most vulnerable to trauma and the resultant inflammation, healing, and scarring. Survey of llamas and alpacas in Chile during the 1990s indicated 10–20% or more of the llama and alpaca populations had evidence of ophthalmic trauma with significant corneal scarring and focal cataract formation. Often parts of the "pupillary ruff" were permanently adhered to either the posterior cornea (anterior synechiae) or anterior lens capsule (posterior synechiae).

Llamas (*Lama glama*) and alpacas (*Lama pacos*) are easily examined standing, in stocks, or lying down ("cushed" position). The normal eye of these New World camelids has some unusual characteristics (Figure 16.22A). Their overall globe size is slightly smaller than cattle but larger in relative proportion to the size of the head. Cilia (eyelashes) are present on both eyelids. The exposed cornea is large and prominent and, as such, is at risk for injury and a common site for ophthalmic disease. The horizontal pupil is surrounded by a "pupillary ruff," – an exaggerated corpora nigrum or granular iridica with distinct multifocal projections. The pupillary ruff can enhance the effect of pupillary closure for their horizontally elongated pupils. The ocular fundus is different from that in cattle, and contains a highly vascularized optic nerve head, and pigmented (denser in dark coated animals) ocular fundus. A tapetum is not present, but in the dorsal fundus it is not unusual to observe areas devoid of pigment which result in a red reflex. The large dorsal primary retinal arteries often spiral around each other.

The most frequent ophthalmic disorders include blepharitis (often with generalized dermatologic diseases), nasolacrimal diseases (including distal nasolacrimal duct atresia), trauma and corneal ulcerations, corneal edema and endothelial disease, iridocyclitis with and without systemic disease, anterior and posterior uveitis, and congenital and secondary glaucomas (Figure 16.22 B–J). Congenital cataracts and cataract surgery have been reported in camelids. Neoplasia is uncommon. Systemic disease resulting from equine herpesvirus-1 can result in anterior and posterior ocular inflammation and a high mortality rate.



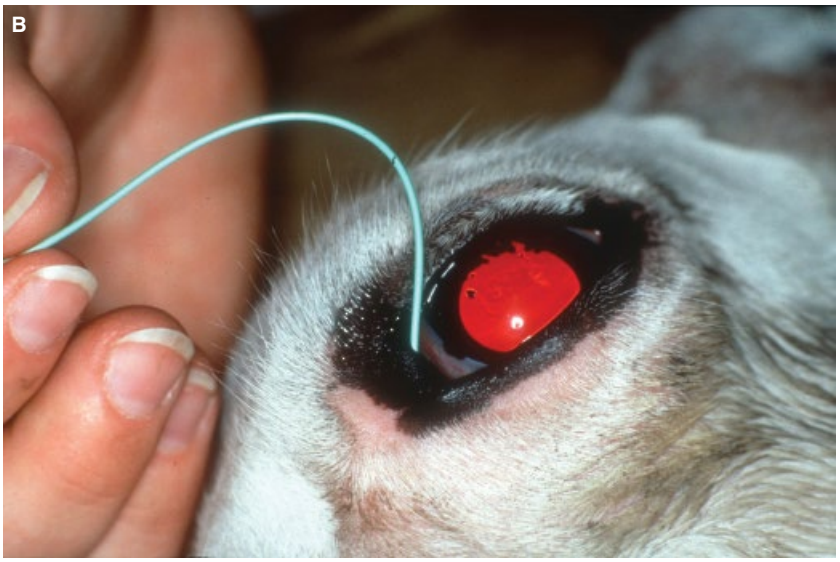
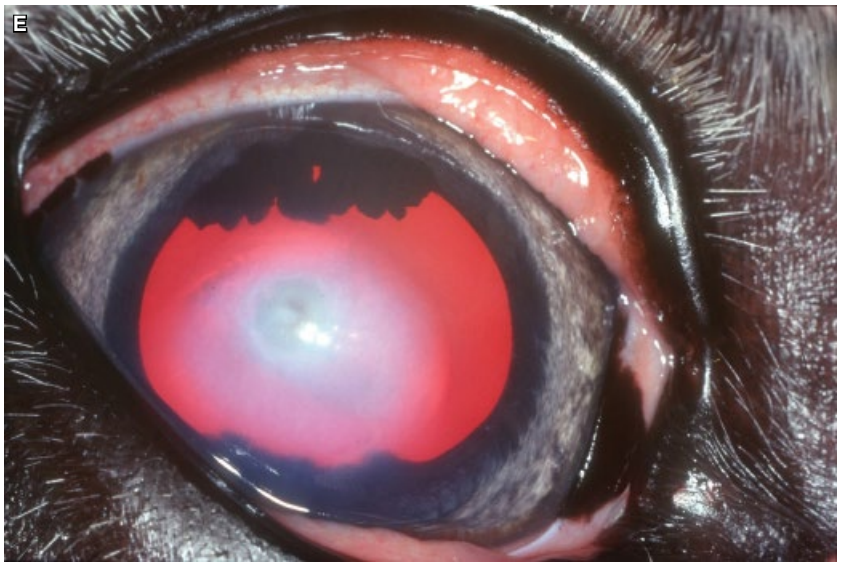




Figure 16.22 (A) Normal eye of an alpaca. Note the prominent granula iridica, or "pupillary ruff." The pupillary ruff, consisting of roughly triangular nodules attached to both upper and lower edges of the pupil, are thought to assist in restricting further light when the pupil is constricted. (B) Cannulation of a llama with nasolacrimal duct atresia. The llama has both upper and lower lacrimal puncta. (C) Infected corneal ulcer in a llama. Note the cellular infiltrate within the corneal stroma and the invading corneal vasculature and secondary iridocyclitis. Treatment of ocular disease in camelids can be facilitated by the use of a subpalpebral lavage, as in a horse.





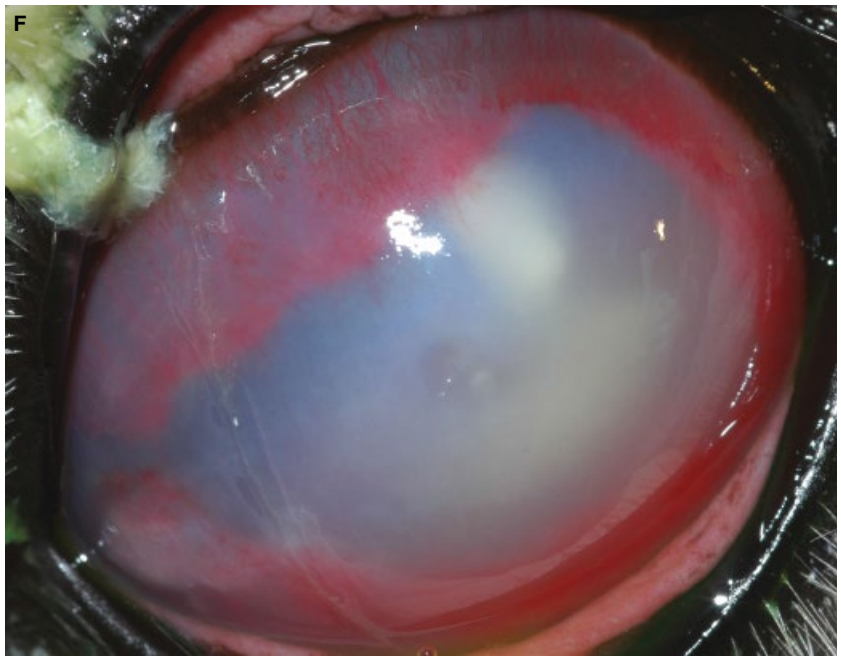
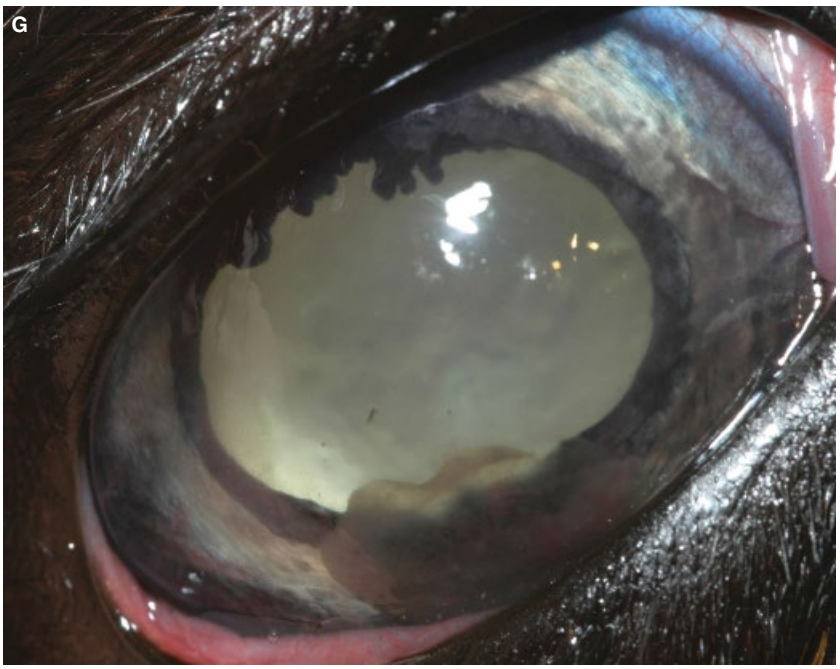


Figure 16.22 (*Continued*) (D) Same eye as in Figure 16.22C after treatment with a bulbar conjunctival pedicle graft. (E) Central keratopathy from previous corneal trauma and corneal ulceration in a llama. (F) Deep stromal corneal abscess in a llama, very similar to the condition in horses. The causative etiologic agent in this case was fungal.



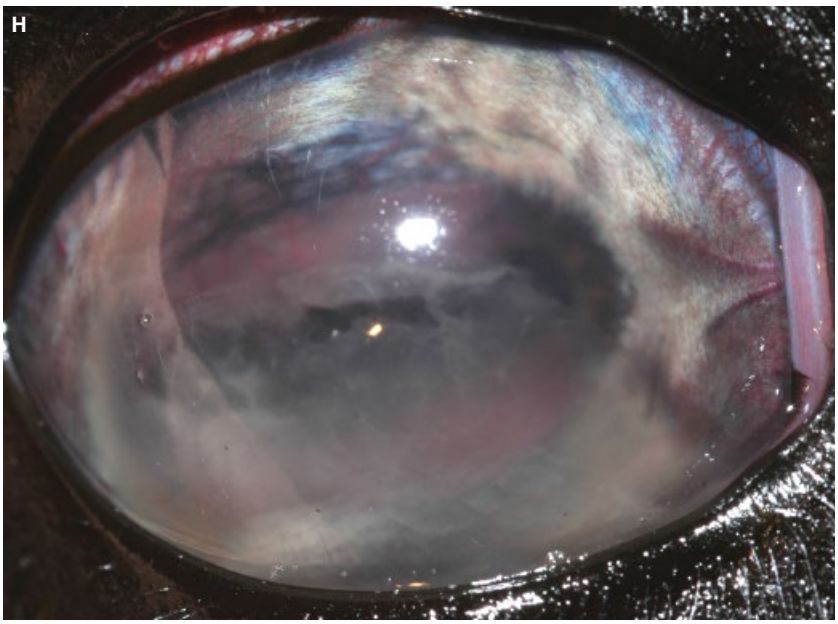
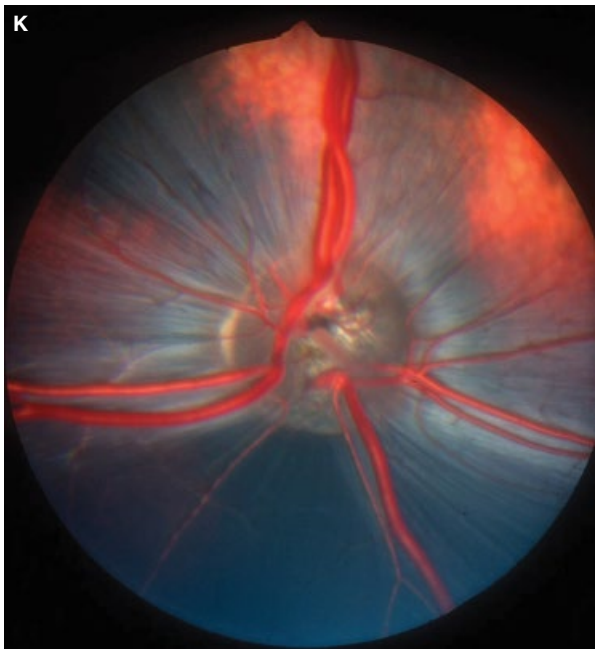




Figure 16.22 (*Continued*) (G) Anterior uveitis in an alpaca. Note the fibrin clot in the ventral anterior chamber. (H) Anterior uveitis in an alpaca, secondary to systemic illness. Note the miosis and the extensive fibrin formation that covers the majority of the iris surface. Corneal vascularization has begun and there is considerable iris neovascularization as well. (I) Unilateral secondary glaucoma and buphthalmia in an older llama.





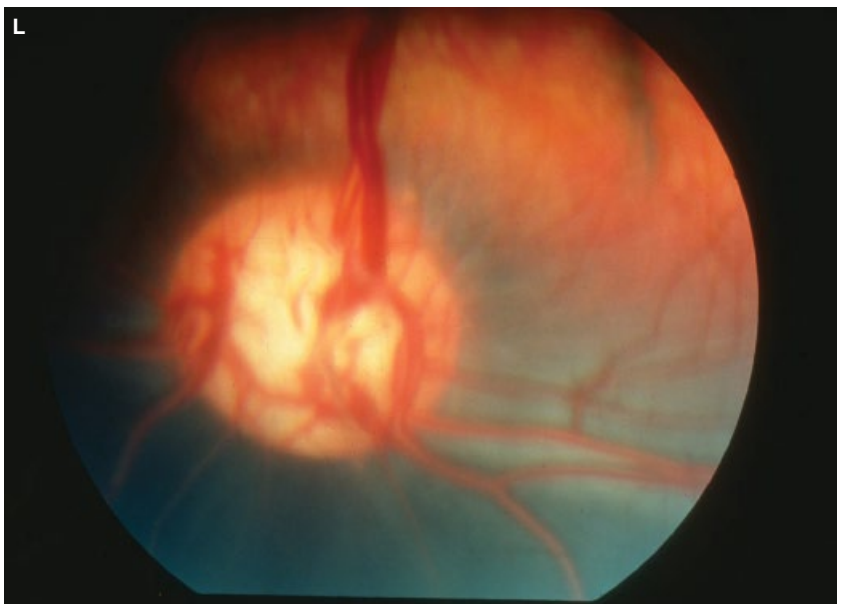


Figure 16.22 (*Continued*) (J) Traumatic cataract following anterior uveitis in an alpaca. Note the cataractous lens is subluxated. (K) Example of the llama ocular fundus (animal was black and white). There is no tapetum. The optic nerve head is round and pigmented. The primary blood arteries and veins are quite large and twist around each other. (L) Another example of a normal ocular fundus in a black llama. This round white optic nerve head has a prominent and large central physiologic cup, and appears myelinated.

17

Ophthalmology in Exotic Pets

This chapter focuses on small exotic pets rather the large zoo species. Among exotic pets, the domestic rabbit seems the most popular; however, rodents and reptiles are frequently encountered. While there are many ophthalmic diseases in exotic pets, this chapter is devoted to those ophthalmic diseases that are the most often presented to the veterinarian.

Diseases of the Snake Spectacle

Snakes lack mobile eyelids, having instead evolved with a clear spectacle that covers the corneas for their protection. The outer aspects of the spectacle of snakes are normally shed during each ecdysis, as eyelid skin would be. It becomes cloudy with the thickening of its layers and accumulation of fluid between the old and new layers just before ecdysis. Retained spectacles are associated with dry environments, generalized integumentary diseases, local injury of the spectacle, systemic illnesses, and mite and tick infestations (Figure 17.1). Semi-translucent to opaque spectacle can cause a snake to become irritable, aggressive, and unable or unwilling to eat.

Conservative treatment is recommended initially, and consists of misting or soaking the snake and facilitating a natural shed for the next cycle. Topical acetylcysteine may soften and loosen the spectacle and permit careful removal with thumb forceps. Damage to the deeper aspects of the spectacle and the cornea should be avoided.

Closely related but more serious, subspectacular infections and abscesses can potentially damage the cornea. Infection can enter the subspectacular space from penetrating injuries, systemic infections, or ascending through the nasolacrimal duct. Clinical signs include an enlarged bulging spectacle (often confused with glaucoma, i.e., pseudobuphthalmia) with subspectacular exudate. Bacteria recovered include *Pseudomonas* and *Proteus* spp., and *Providencia rettgeri*. Treatment typically includes excision of a 30° wedge of the spectacle, taking care not to damage the underlying cornea, irrigation of the subspectacular space, and antibiotics.

Ophthalmic Disease in Raptors

Trauma

Trauma is the most common cause of ophthalmic disease in raptors and other birds of prey (Figure 17.2). Damage is to the eyelids, nictitating membrane, cornea, lens, or posterior segment. Intraocular hemorrhage, retinal tears and detachments are often present. Prognosis and treatment depend on the amount of ocular damage present and the secondary inflammation. Treatment is generally directed at controlling the corneal and intraocular inflammation, and preventing or treating infection.

Ophthalmic Disease in Rabbits

The rabbit is among the most common exotic pets and can suffer from a wide variety of ophthalmic diseases. The rabbit has a very prominent eye that is positioned laterally which permits a nearly 360° visual field. The retrobulbar space in rabbits has a very large venous plexus, which presents challenges to orbital surgery and enucleation procedures because there is considerable risk of hemorrhage. The nasolacrimal system has only one lacrimal punctum (lower) which is close to the molar and incisor teeth roots. Because of this close proximity, abscesses of the tooth roots can affect the nasolacrimal system. In these instances, dacryocystorhinography can be valuable in outlying the entire nasolacrimal system. The rabbit's pupil is round, and in albino rabbits the iris is pink. The rabbit has an atapetal fundus that is variably pigmented. The optic nerve head is oriented horizontally with myelinated nerve fibers and retinal blood vessels extending both laterally and medially from the disc. The rabbit's ocular fundus has a visual streak and the

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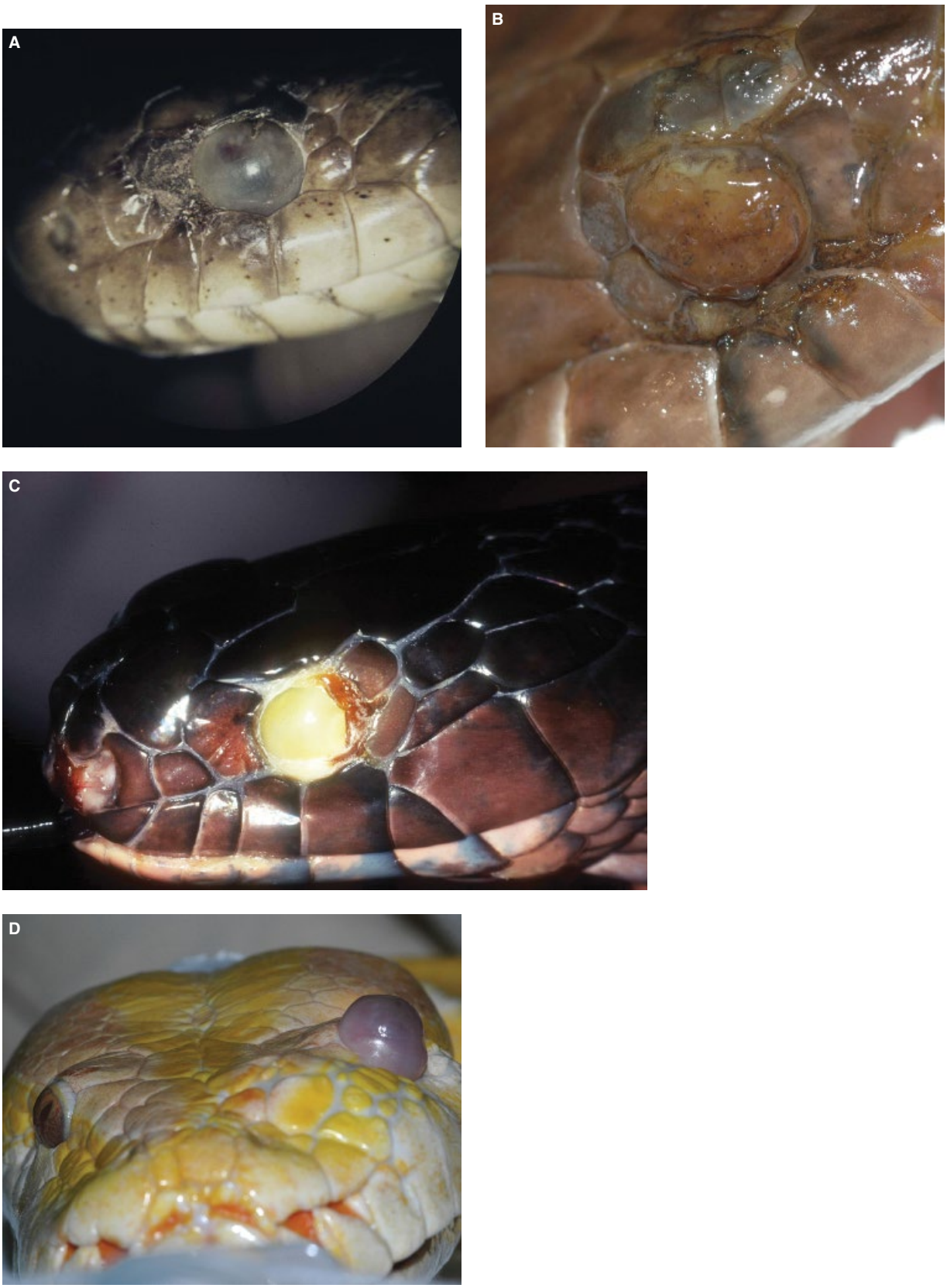
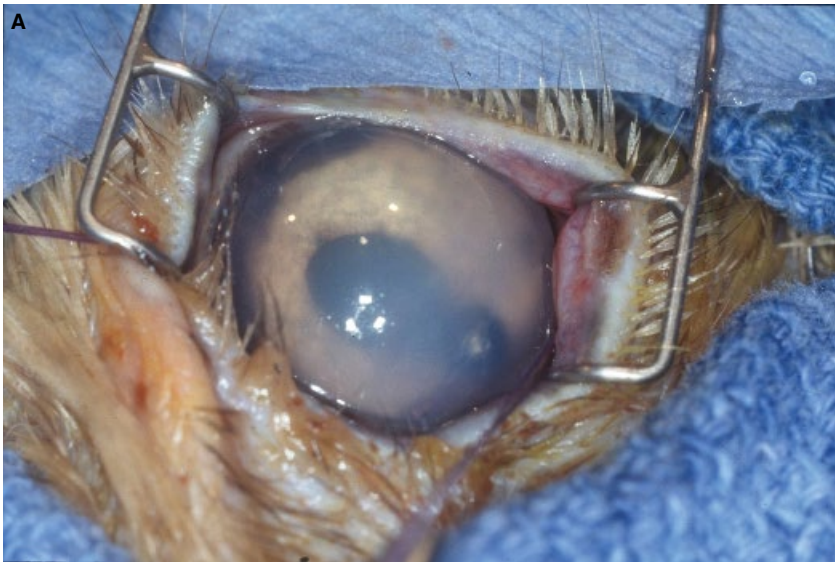
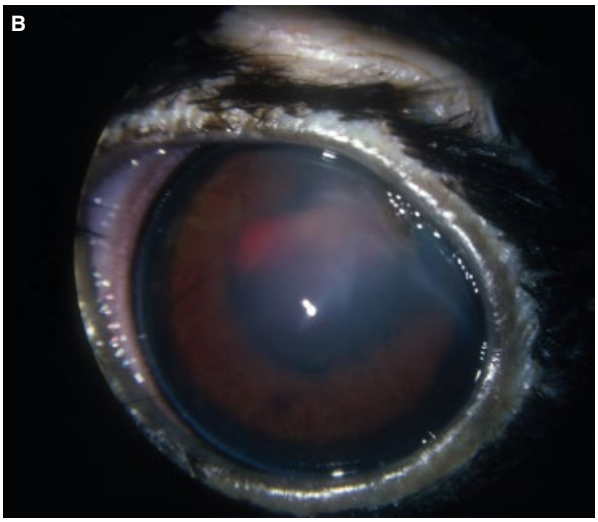


Figure 17.1 (A) Retained/opaque spectacle in a snake. Note the spectacle nearly obstructs the cornea, pupil, and vision. (B) Retained spectacle in another snake. (C) Subspectacular infection in an Indigo snake. Note the infection has affected the entire spectacle. (D) Exophthalmos, spectacular inflammation, and opacity and uveitis in a septic albino Burmese python.





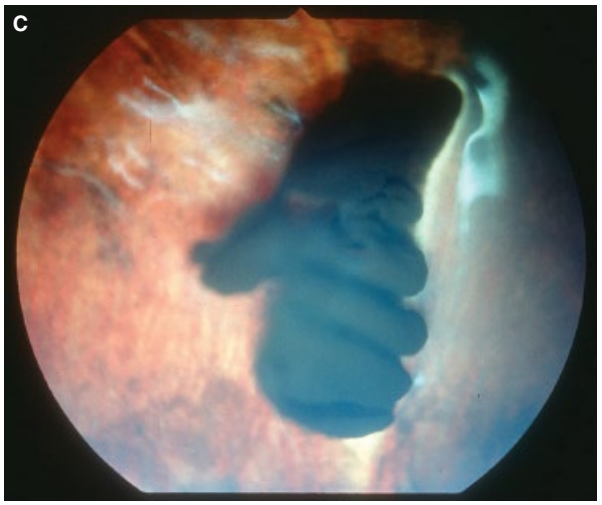
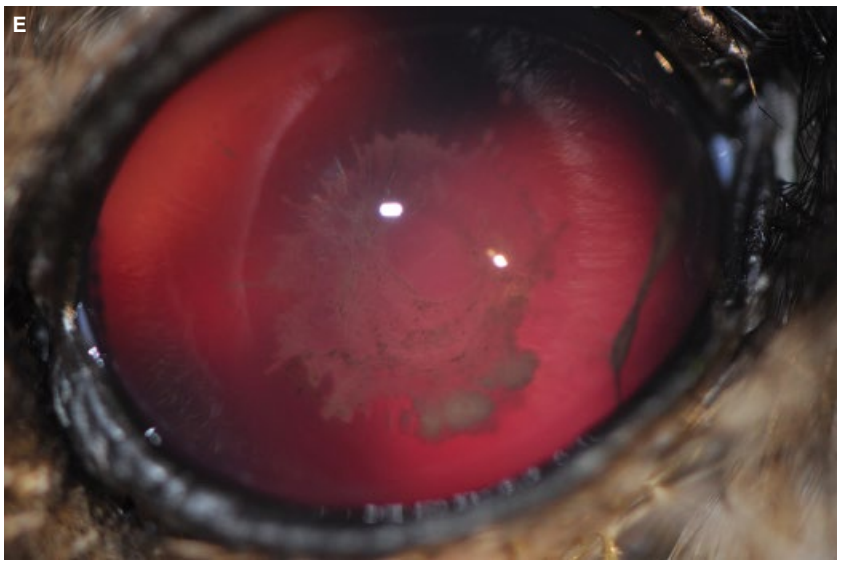




Figure 17.2 Raptors are frequently presented with ophthalmic trauma (blunt and penetrating). (A) In this Bald Eagle a lead pellet has penetrated the cornea and lodged in the anterior chamber. Secondary iridocyclitis with considerable iridal swelling and fibrin are present. (B) Trauma to the eye of a Golden Eagle. Behind a corneal defect, fibrin, clotted hyphema, and secondary iridocyclitis can be appreciated. (C) Chorioretinal scars and damaged pectin with hemorrhage in a Barred Owl secondary to trauma and inflammation. (D) Traumatic globe rupture in a Screech Owl resulting in globe hypotension, loss of the anterior chamber, and deflation.



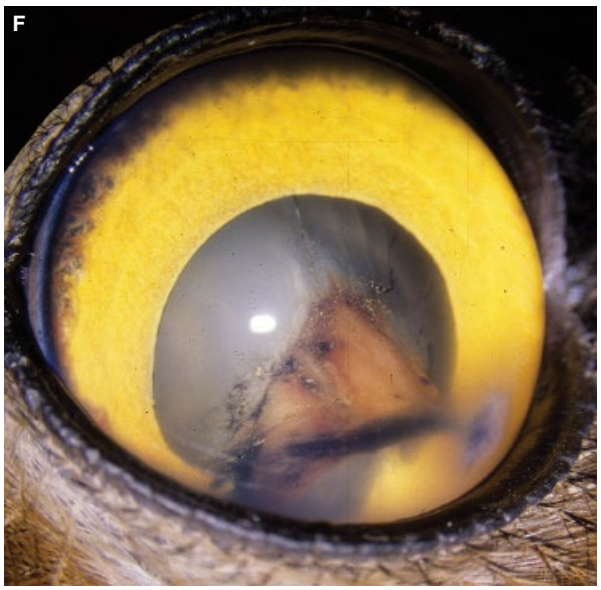


Figure 17.2 (*Continued*) (E) Traumatic uveitis in a Barred Owl has resulted in posterior segment hemorrhage, anterior chamber fibrin, and an axial cataract. (F) Sealed corneal perforation in a Great Horned Owl with anterior synechia, anterior lens capsule rupture, and cataract.

estimated visual acuity is 22/200 (based on pattern-evoked cortical potentials). Color vision appears limited to the blue (425 nm) and green (520 nm) wavelengths.

Orbital Abscessation

Orbital abscessation is the most common cause of exophthalmia in rabbits and usually results from an infection associated with a tooth root (Figure 17.3). The exophthalmia is often rapidly progressive with resultant exposure keratitis and strabismus. Orbital imaging is valuable to demonstrate the site and extent of the abscess and image the orbital tissue contents. Enucleation can be attempted but postoperative infections of the surgical site are common.

Entropion

Entropion occurring in rabbits can be treated surgically as in dogs and cats. In very young rabbits (kits), tacking of the eyelids can be used to prevent secondary severe corneal damage until adult size is reached (Figure 17.4).



Figure 17.3 (A) Exophthalmos in a rabbit with orbital and tooth root abscess. Culture revealed *Pasteurella* sp. If enucleation is performed, the retrobulbar infection may remain. Prognosis is guarded. (B) Advanced exophthalmos and retrobulbar abscess.



Figure 17.4 Entropion in a rabbit with a secondary corneal ulcer. The upper and lower eyelids are also inflamed (blepharitis). The eyelids were excessively long and when released, passively inverted so that the facial hair contacted the cornea.



Figure 17.5 This rabbit presented with dacryocystitis and an obstructed nasolacrimal duct. Note the swelling of the medial canthus and conjunctivitis.

Dacyocystitis

The rabbit nasolacrimal system is unusual in that it has only the lower lacrimal punctum and canaliculus. Hence, nasolacrimal lavage and dacryocystorhinography must be performed using the lower lacrimal punctum for entry into the entire system. The signs of dacyocystitis include epiphora, persistent or chronic conjunctivitis, and severe nasofacial dermatitis (Figure 17.5). Abnormalities of the incisor and molar dental arcades, tooth root infections, and maxillary osseous changes secondary to nutritional hyperparathyroidism are contributing disorders.

Nasolacrimal lavage is essential to re-establish (and often maintain) nasolacrimal patency, as are topical and systemic antibiotics to resolve secondary bacterial infection. If the nasolacrimal system becomes repeatedly occluded, a nylon suture can be positioned within the duct to permit drainage and re-establish a healthy ductal epithelium and is left in place for several weeks.

Blepharoconjunctivitis

Blepharitis in rabbits is often secondary to bacterial conjunctivitis and nasolacrimal duct obstructions (Figure 17.6). It can also be associated with *Treponema*



Figure 17.6 (A) Severe bacterial blepharoconjunctivitis in a rabbit. Note the marked inflammation of the lids, mucopurulent exudate, and secondary keratitis.



Figure 17.6 (*Continued*)
(B) Blepharoconjunctivitis in a dwarf rabbit. Note the chalazia (subconjunctival accumulation of meibomian secretions) in the upper eyelids. (C) Blepharitis and meibomianitis in a dwarf rabbit. Note the swellings along the eyelid margin and the trichiasis at the upper eyelid that has occurred secondary to the eyelid swelling.

cuniculi (rabbit syphilis). Topical antibiotics are usually recommended rather than parenteral initially to avoid dysenteribiosis. If topical antibiotics are ineffective, systemic antibiotics may become necessary. In the viral disease myxomatosis, blepharoconjunctivitis often proceeds death in unvaccinated rabbits.

Conjunctivitis

Conjunctivitis is a common and often persistent disorder in rabbits, and often occurs with dacryocystitis and blepharitis. Bacteria frequently isolated include *Staphylococcus* sp. (42%), and *Pasteurella* sp. (12%) as well as *Haemophilus* sp. The amount of mucopurulent discharge can be considerable (Figure 17.7). In stubborn or chronic cases, bacterial culture and susceptibility testing are recommended.

Conjunctival Overgrowth

Conjunctival overgrowth occurs more frequently in the dwarf breeds of rabbit, but has been documented in a variety of individuals (Figure 17.8). A fold of conjunctiva attached at the limbus slowly progresses 360° across the cornea, but, unlike pterygium in humans, is nonadherent to the corneal surface. Treatment consists of surgical excision followed by topical cyclosporine. With surgical resection alone, recurrence of the adherent conjunctiva is common.

Prolapse of the Nictitans Glands

Prolapse of the rabbit's nictitans glands differs from cherry eye in dogs and cats, in that the rabbit has four nictitans glands (deep and superficial nictitans glands,



Figure 17.7 Severe *Pasteurella* sp. conjunctivitis in a rabbit. The cornea is covered by mucopurulent exudate.



Figure 17.8 Conjunctival overgrowth in a rabbit. The proliferative tissue originates at the limbus and extends 360° over the cornea, but does not attach to the cornea.

Harderian gland, and the deep orbital gland) (Figure 17.9). Hence, nictitans gland prolapse in the rabbit appears as small, round, raised, focal mass to a very large prolapse that nearly fills the palpebral fissure. Because of the very extensive venous plex in the retrobulbar space in rabbits, anchoring procedures are avoided and a pocket procedure recommended.

Corneal Ulceration

The different types of corneal ulcerations in rabbits are similar to those encountered in dogs and cats, as is their medical and surgical treatment. The rabbit cornea is very prominent, so trauma and foreign bodies are the most frequent causes of corneal ulceration. Most superficial corneal ulcers heal within 3–5 days. Stubborn central superficial corneal ulcers, similar to refractory ulcers in other domestic species, occur mainly in dwarf rabbits (Figure 17.10). Healing can be stimulated with debridement, keratotomy, or keratectomy. Superficial keratectomy specimens of these ulcers reveals abnormalities of the anterior stroma and failure of the union of corneal epithelium and the underlying stroma, similar to canine indolent ulcers. Self-trauma can also contribute to ulcer formation and persistence, and is prevented by the use of an Elizabethan collar.

Bacterial Iridocyclitis

Staphylococcus and *Pasteurella* spp. are the most likely causes of bacterial iridocyclitis in rabbits. Clinical signs range from diffuse iritis with miosis, posterior synechiae, aqueous flare, and hypopyon to an abscess in the ventral anterior chamber. Both bacterial species enter the eye hematogenously. Topical and systemic antibiotics as well as mydriatics are indicated. Some rabbits are resistant to the effects of topical atropine because of endogenous systemic atropinase.

Congenital Glaucoma

Congenital glaucoma occurs in both New Zealand White and pigmented rabbits (Figure 17.11). It is transmitted as an autosomal recessive trait with semi-lethal factors in New Zealand Whites. Normal intraocular pressure in rabbits is about 20 mmHg, and the diurnal cycle differs from that in humans and dogs with higher pressures in the evening and lower pressures in the morning. The increase in intraocular pressure starts in affected rabbits at 3–6 months of age, and is demonstrated by an enlarging globe, corneal edema, mydriasis, and episcleral congestion. As viewed by gonioscopy and histology, the pectinate ligaments and trabecular meshwork are absent or poorly formed, and the ciliary cleft collapsed. Lens luxation secondary to buphthalmia often occurs. Over time, apparently from atrophy of the ciliary body, intraocular pressure can return to normal levels.

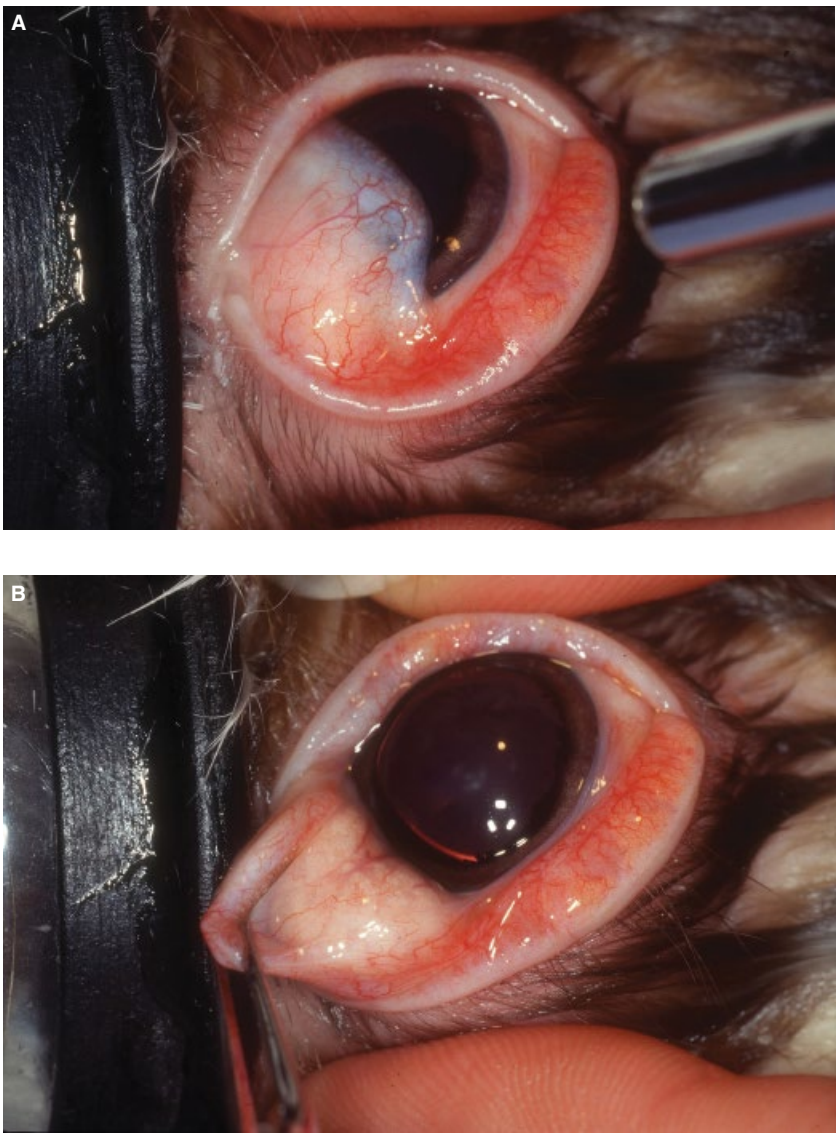


Figure 17.9 (A) Prolapse of the nictitans and its glands in a rabbit. The enlarged nictitans covers a large amount of the cornea. (B) In the same rabbit as in part A, eversion of the nictitans reveals prolapse of the yellow tear glands.

Medical therapy is successful in lowering intraocular pressure. A large number of topical ophthalmic drugs have been studied in normal rabbits. Anterior chamber shunts have also been attempted.

Cataracts

Cataracts are infrequent in rabbits, and most reports are of congenital nuclear cataract with and without other ocular anomalies (Figure 17.12). Microphthalmia and persistent pupillary membranes can be present. Inherited cataracts appear rare. Cataract surgery using phacoemulsification has been performed in rabbits. Postoperative capsular fibrosis is often extensive in rabbits, and the capsular opacities after surgery can produce significant visual deficits within several months.

Encephalitozoon cuniculi, Iridocyclitis, and Cataract Formation

The microsporidal protozoal parasite, *Encephalitozoon cuniculi*, is transmitted vertically, and presumably enters the lens during development (Figure 17.13). This intralenticular parasite not only produces cataract formation, but the leakage of lens material into the aqueous humor causes a variable but sometimes intense lens-induced uveitis. Often, focal granulomas are present on the surface of the anterior lens capsule and/or within the iris stroma.

Ocular Fundus

The ocular fundus of the rabbit varies depending on the coat color (Figure 17.14). In albino rabbits, limited to no pigment occurs within the retinal pigment epithelium

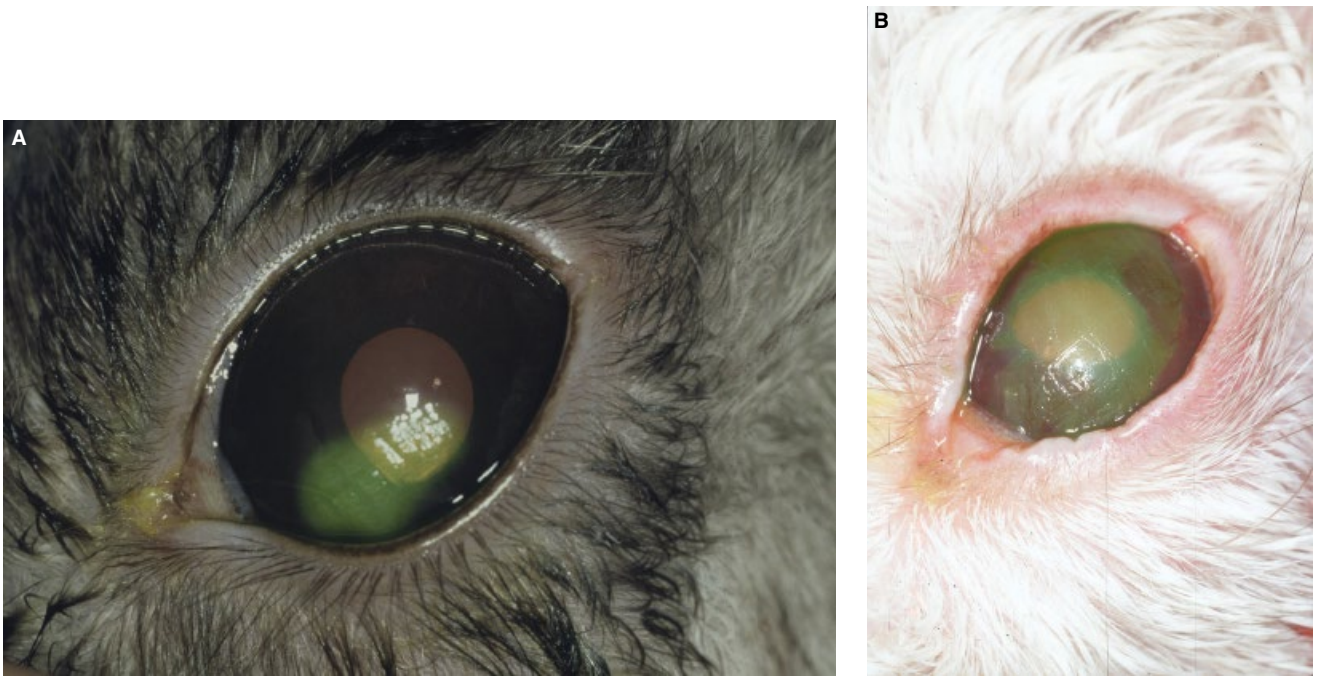


Figure 17.10 (A) Superficial corneal ulcer, stained with topical fluorescein, in a dwarf rabbit, post grid keratotomy. (B) Indolent superficial ulcer covering about 80% of the cornea (stained with fluorescein) immediately following grid keratotomy. Note the superficial linear defects in the anterior stroma of the wound bed.

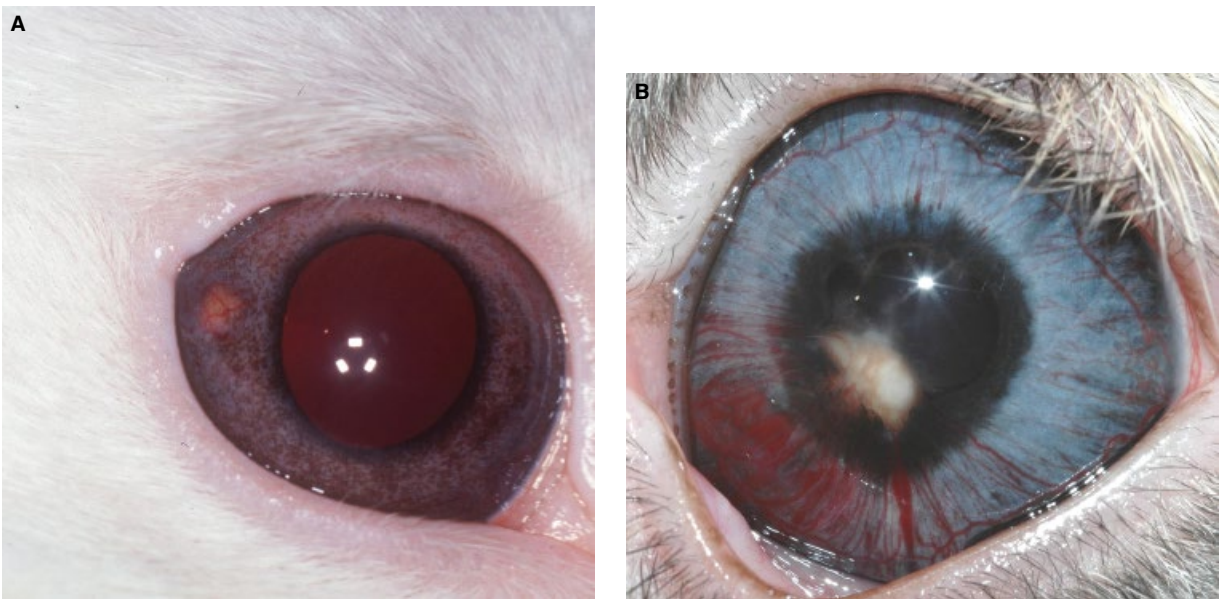
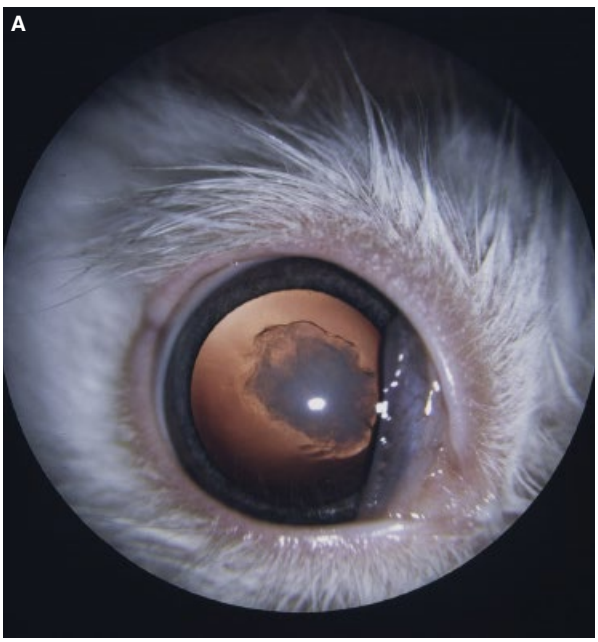


Figure 17.11 (A) Anterior uveitis in a rabbit secondary to *Encephalitozoan cuniculi*. An iridal granuloma is present in the lateral iris. The lens should be closely examined for evidence of cataract formation. (B) *E. cuniculi* abscess in the lens and iris in a dwarf rabbit. Note the neovascularization.





Figure 17.12 (A) Inherited congenital glaucoma in a New Zealand white rabbit. Intraocular pressure is elevated causing buphthalmia, mydriasis, and diffuse corneal edema. (B) Buphthalmia in a pigmented rabbit with glaucoma.



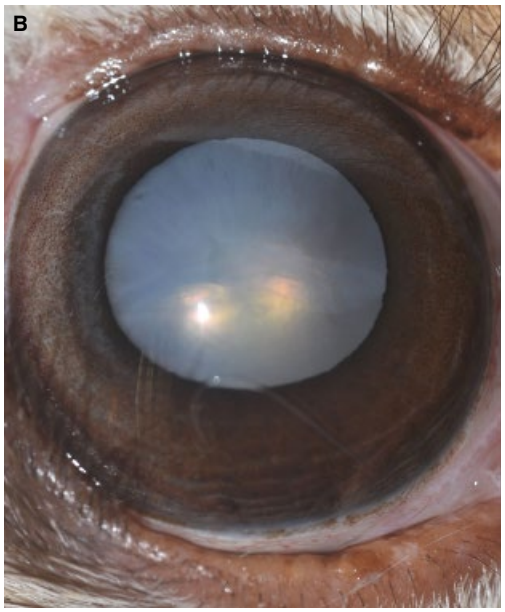


Figure 17.13 (A) Congenital cataract in a young rabbit. The lens opacity is limited to the nucleus. Progression of the cataract is unlikely. (B) Immature cataract in a dwarf rabbit.

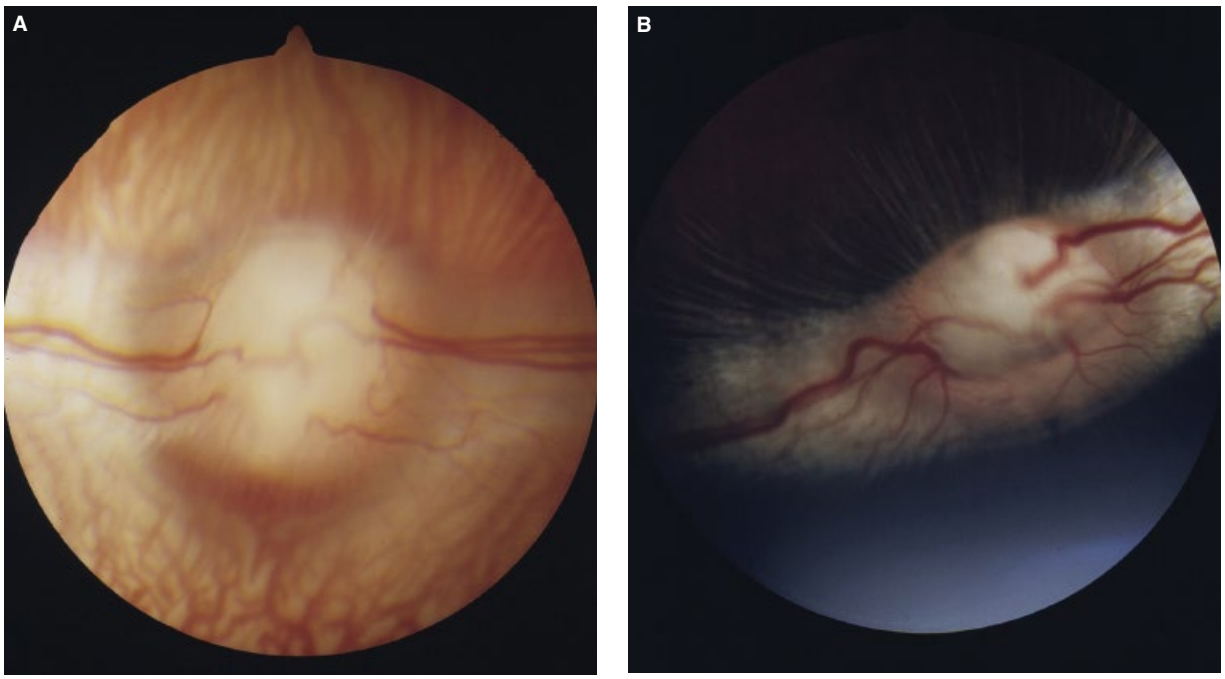


Figure 17.14 The normal rabbit ocular fundus varies somewhat depending on the animal's coat color. (A) Normal ocular fundus in an albino rabbit. Note the retinal vessels are primarily orientated medial and lateral of the optic disc, and on the myelinated optic nerve fibers. The absence of retinal and choroidal pigmentation, and tapetal fundus allow observation of both the retinal and choroidal blood vessels. There is also a very large physiologic cup within the optic disc. (B) Normal ocular fundus in a pigmented rabbit. The myelinated optic nerve fibers are prominent against the ocular fundus pigmentation. Note the large physiologic cup within the optic disc.

permitting direct visualization of the underlying choroidal vasculature. In pigmented rabbits, the entire ocular fundus is pigmented as the rabbit has no tapetal layer. The rabbit's retinal vasculature is merangiotic with prominent arteries and veins traversing the retina, and emerging from the optic disc's surface at the 9 and 3 o'clock positions. The optic nerve head often contains a large and deep physiologic cup (which can be confused with coloboma), and myelinated nerves fibers.

Ferrets

Microphthalmia is one of the most common congenital disorders in ferrets, and cataracts are often also present. Both albino and pigmented ferrets are affected (Figure 17.15). Inheritance of microphthalmia has not been demonstrated to date in ferrets.

Cataracts in ferrets have been reported in both normal sized as well as microphthalmic globes with retinal detachments. The cataracts range from fine multifocal punctate opacities to mature and hypermature types, and involve the lens nucleus and anterior and posterior cortices. Both nutrition and heredity causes have been suggested.

Ferrets can also present with ophthalmic manifestations of systemic disease. General illness, especially if related to infectious disease or neoplasia, can result in uveitis and conjunctivitis. Ferrets with lymphoma may have intraocular or orbital disease (Figure 17.16).

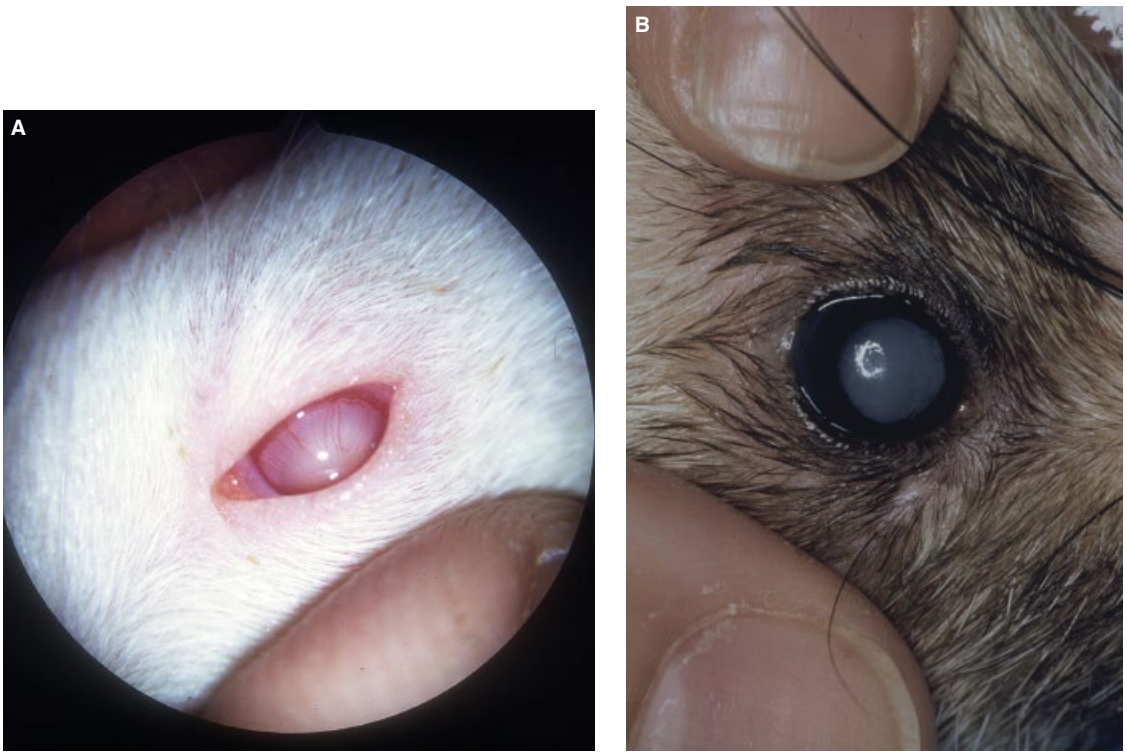


Figure 17.15 (A) In this white ferret, the cataract formation is limited to the large nuclear region. The vessels noted anterior to the lens are within the iris in this hypopigmented animal. (B) This ferret cataract affects the entire lens and is classified as mature. As cataract formation is common in microphthalmia in ferrets, globe size should be closely ascertained.

Figure 17.16 Bilateral exophthalmos and elevated nictitans in a ferret with lymphoma.



18

Systemic Diseases with Ophthalmic Manifestations

For convenience, this chapter is divided by major species (i.e., canine, feline, equine, and food and fiber animals). We selected the more common systemic diseases or those in which the ophthalmic manifestations assist in establishing the clinical diagnosis. Additional information about these diseases is available in the species-related chapters (canine, Chapters 4–13; feline, Chapter 14; equine, Chapter 15; and food and fiber animals, Chapter 16).

Ophthalmic Manifestations of Canine Systemic Diseases

Orbital Diseases

Merle Ocular Dysgenesis

There are a large number of popular breeds that have the merle gene and coat. Excessive white or partial albinism occurs in the canine breeds with the merling gene. In animals homozygous for the incomplete dominant merling gene (usually the result of breeding two merled parents), the hair coat is primarily white, iridal heterochromia occurs with multiple eye anomalies, and sometimes deafness occurs. The ocular anomalies have been studied extensively in the Australian Shepherd breed, and include microphthalmia, iridal heterochromia, cataract formation, large and variable equatorial staphylomas, retinal dysplasia, and retinal detachments (Figure 18.1; see also Figures 4.1, 10.2, and 10.5). The eye defects are often asymmetrical, and blindness is uncommon. However, cataract progression and/or retinal detachments can impair vision in later life. Dog fanciers should be advised that breeding of merled dogs will result in at least 25% of affected offspring; hence, it is recommended to breed merled dogs to a non-merle animal.

Dwarfism and Ocular Defects

Dwarfism-associated ocular defects occur in the Samoyed and Labrador Retriever breeds. In the Labrador Retriever, the eye defects are inherited as an incomplete dominant trait while the skeletal defects are transmitted as an autosomal recessive trait (Figure 18.2). In affected animals the ocular anomalies range from focal or geographic retinal dysplasia usually dorsal of the optic disc (thought to be heterozygous) to multiple ocular anomalies (microphthalmia, cataract formation, hyaloid artery remnants, retinal dysplasia, and complete retinal detachments, thought to be homozygous).

The eye defects in dwarfism in the Samoyed breed are thought to be inherited in an autosomal recessive mode and are similar, although not as severe, to those in affected Labrador Retrievers. Breeding of affected animals as well as adult animals known to have had affected offspring is not recommended.

Congenital Hydrocephalus

Congenital hydrocephalus can cause enlargement of the developing calvarium and displacement of the orbits (Figure 18.3). The result is displacement of both globes ventrolaterally, resulting in a change in the animal's facial features. Papilledema is occasionally present.

Infectious Diseases

The canine distemper virus (RNA paramyxovirus) affects the ophthalmic structures in several ways, resulting in mucopurulent conjunctivitis, acute keratoconjunctivitis sicca, iridocyclitis, chorioretinitis, optic neuritis, and central (occipital cortex) blindness (Figure 18.4). Conjunctivitis, keratoconjunctivitis sicca, and iridocyclitis occur with the other systemic (gastrointestinal and respiratory disease) signs of distemper while the retinal, optic nerve, and central nervous system (CNS) abnormalities occur several days after the initial viremia when the virus invades the neural tissues.

Chorioretinitis is characterized by multifocal round to oval lesions scattered throughout the tapetal and nontapetal fundi. In one study, 41% of the dogs with the neurologic form of disease had ocular fundus lesions, while

Color Atlas of Veterinary Ophthalmology, Second Edition. Kirk N. Gelatt and Caryn E. Plummer.
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Figure 18.1 In the homozygous merle Australian Shepherd, microphthalmia and multiple ocular anomalies occur. Note the bilateral heterochromia irides and asymmetrical microphthalmia in this puppy.

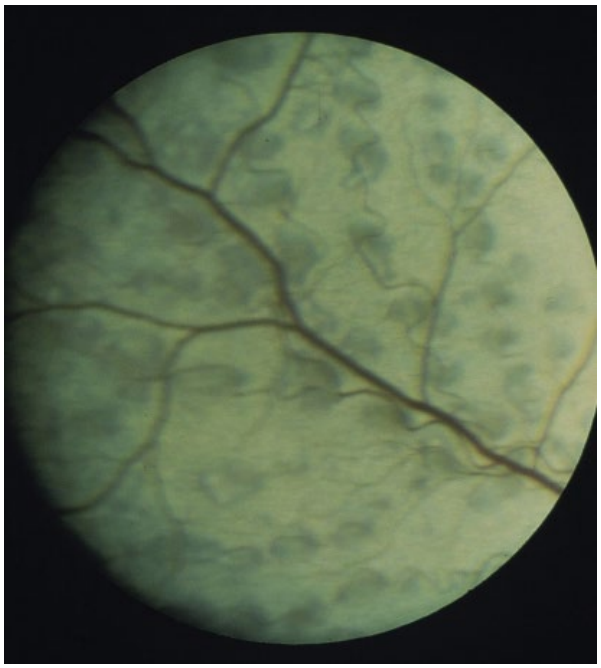


Figure 18.2 Oculoskeletal dysplasia is inherited in the Labrador Retriever. In this mildly affected puppy, note the numerous areas of retinal dysplasia in the tapetal fundus.

another report on dogs with chronic leukoencephalopathy syndromes noted 83% of the dogs had chorioretinal lesions. Hence, dogs with neurological diseases require a complete fundus examination.



Figure 18.3 Hydrocephalus in the dog can cause enlargement of the calvarium and in this Chihuahua has displaced the eyes laterally.

Optic neuritis can cause temporary to complete blindness when both nerves are involved. Mydriasis and blindness are the presenting signs. The inflamed papillae appear raised or elevated, and peripapillary hemorrhages and retinal vascular congestion can occur. Often, the inflammation extends a few disc diameters into the peripapillary retina (neuroretinitis). Optic nerve degeneration follows weeks later, and appears as ophthalmoscopically as depressed, smaller than normal, darkened optic discs with retinal vessel attenuation. (For other distemper ophthalmic manifestations see Figures 3.20, 3.21, 13.6A,B,D, and 13.19.)

Infectious Canine Hepatitis

Infectious canine hepatitis (ICH) caused by canine adenovirus-1 (CAV-1) has been associated with ophthalmic disease since its initial report in 1947 (Figure 18.5). Anterior uveitis and profound corneal edema occur with the natural disease (20% of cases) or following vaccination with CAV-1. Vaccination with canine adenovirus-2 (CAV-2) conveys protection against ICH while eliminating the adverse ocular reactions that occur with CAV-1.

Ophthalmic disease is thought to be due to a delayed Arthus reaction, and occurs 7–21 days post-CAV-1 contact. The local immune response is directed at virus in the corneal endothelium and anterior uvea. The anterior uveitis is bilateral in 12–28% of patients, and usually resolves without sequelae. However, persistent corneal edema, secondary glaucoma, and phthisis bulbi occasionally result. Treatment is symptomatic with topical corticosteroids and mydriatics (see Figure 10.10).

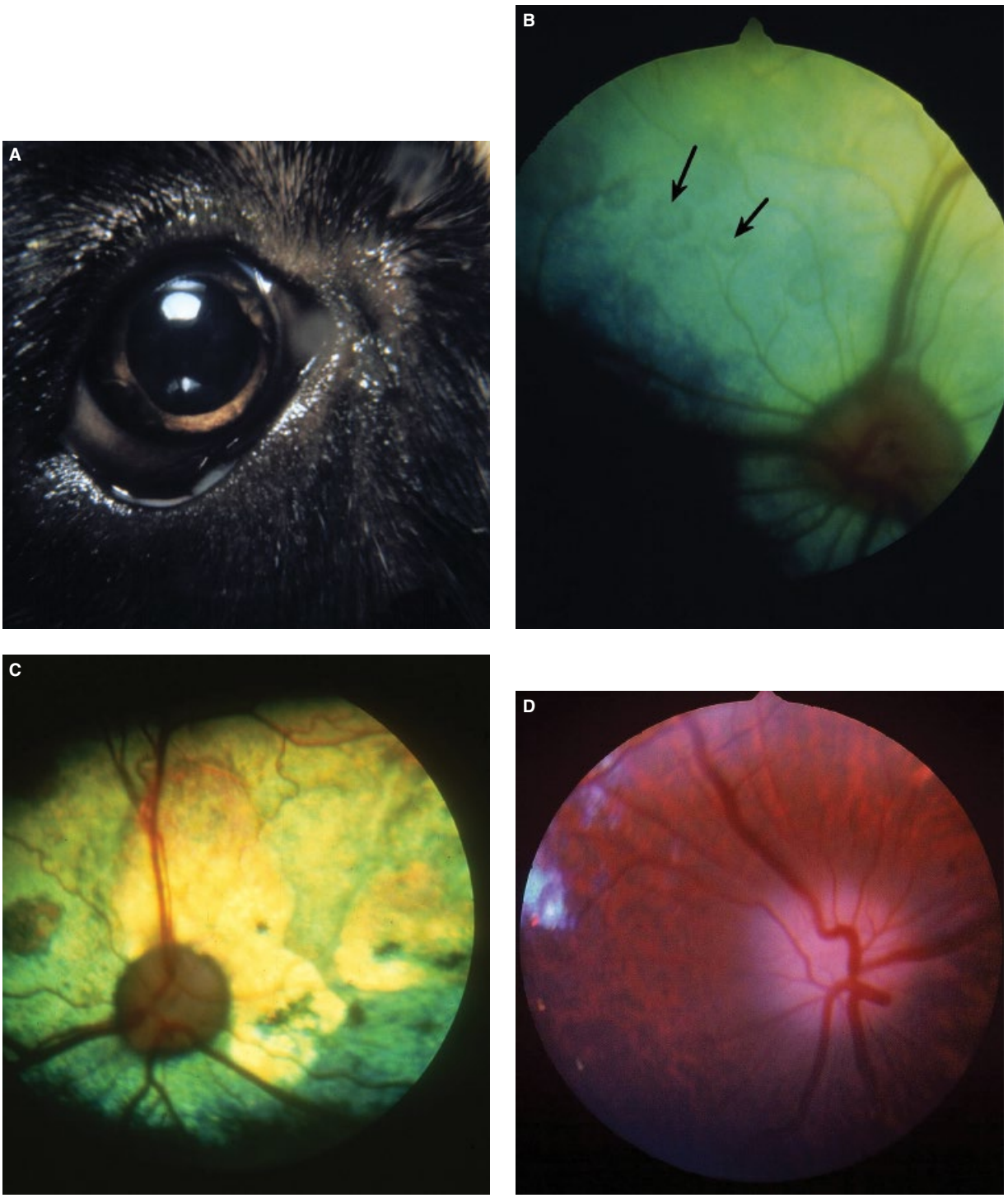


Figure 18.4 (A) Canine distemper frequently causes mucopurulent conjunctivitis. In some of these eyes, reduced tear production will also develop. (B) In this dog, canine distemper has caused an acute chorioretinitis. Note the round to oval translucent inflamed areas (arrows) within the tapetal fundus. (C) A chorioretinal scar in a dog that has recovered from infection with canine distemper virus. Sometimes, these scars are referred to as "gold medallion" lesions. However, they are not pathognomonic for distemper. (D) Optic neuritis associated with canine distemper. Note the hyperemic optic disc and its fuzzy, indistinct margins. The retinal vessels appear to be raised up on to the protruding disc.

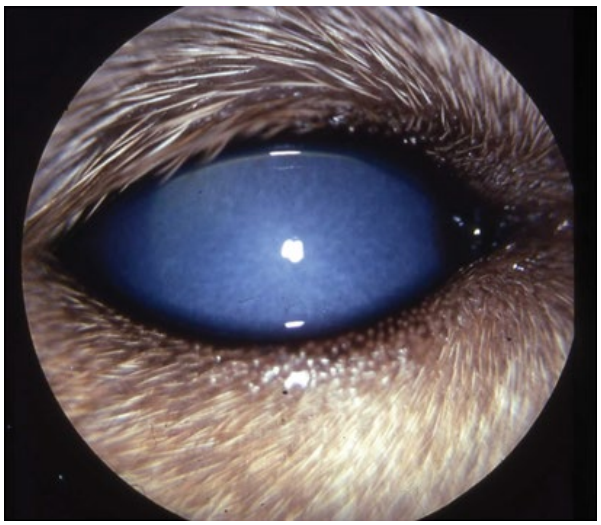


Figure 18.5 The ocular signs of infectious canine hepatitis include iridocyclitis with profound corneal edema.



Figure 18.6 Focal papilloma affecting the medial canthus of a St. Bernard puppy.

Canine Viral Papilloma

The canine papilloma virus can result in focal papillomas affecting the mucus membranes of the mouth, eyelids, conjunctiva and nictitating membrane, and cornea (Figure 18.6). Younger dogs are most frequently affected. Most oral papillomas regress spontaneously, but the ophthalmic tumors may persist. The tumors appear as solitary raised cauliflower-like masses that are sometimes slightly pigmented. Recommended therapy is wide excision of the papilloma and cryotherapy of its base.

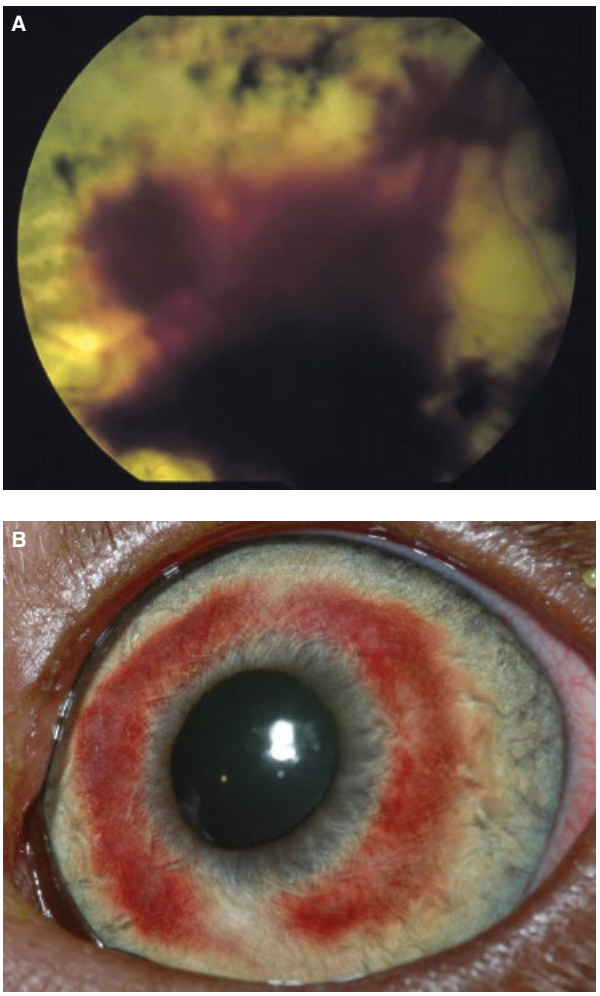


Figure 18.7 (A) Rocky Mountain spotted fever can cause hemorrhages in many of the body's tissues and the retina is no exception. Note in this dog, recent and resorbing preretinal hemorrhages are present. (B) In this dog affected by Rocky Mountain spotted fever there are small hemorrhages throughout the iris stroma.

Rickettsial Infections

Rickettsial infections such as ehrlichiosis and infectious cyclic thrombocytopenia (*Ehrlichia canis*, *Ehrlichia platys*, and *Ehrlichia equii*) and Rocky Mountain spotted fever (*Rickettsia rickettsii*) produce ocular signs (Figure 18.7; see also Figures 3.15E and 10.8). The ocular lesions produced by *E. canis* and *R. rickettsii* infections are very similar, and include conjunctivitis, chemosis, anterior uveitis, and retinal vasculitis. Petechial hemorrhages occur in the conjunctiva, iris, and retina. The systemic antibiotic of choice is oral doxycycline or tetracycline. Ophthalmic therapy is supportive and usually includes corticosteroids and mydriatics. When they occur, ophthalmic

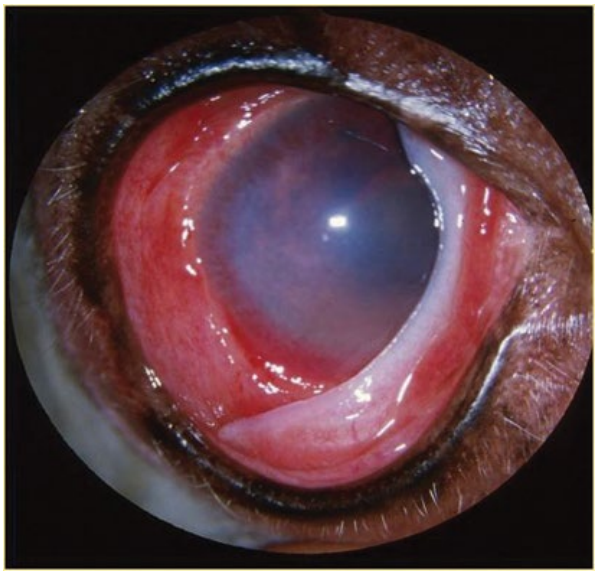


Figure 18.8 Canine brucellosis (*Brucella canis*) in a Miniature Dachshund dog has produced anterior uveitis that responded poorly to broad-spectrum antibiotics. Note the episcleral injection, corneal vascularization and edema, swollen iris, and miosis. *B. canis* was cultured from the aqueous humor.

complications are usually related to intraocular hemorrhage and inflammation and include secondary glaucoma, cataracts, and retinal detachments.

Canine Brucellosis

Brucella canis is an infrequent cause of anterior uveitis and endophthalmitis in the dog (Figure 18.8). Systemic signs include abortion, infertility, and vaginal discharges in bitches, and sterility and testicular and epididymal disease in males. The ophthalmic disease is usually a mild but persistent uveal inflammation. This disease is considered zoonotic, and neutering of an affected animal should be encouraged.

Mycoses

Mycotic infections or dermatophytosis can affect the canine eyelids, and are frequently associated with *Microsporum canis*, *Microsporum gypseum*, or *Trichophyton mentagrophytes* (Figure 18.9; see also Figure 5.16). Mycoses usually present as dry crusty alopecia affecting the eyelid and face. Diagnosis is made by Wood's lamp inspection, culture, and/or microscopic examinations of scrapings.

Fungal corneal ulcers are uncommon in the dog (in contrast to the horse and human), and are often associated with corneal foreign bodies, local or systemic immunosuppression, or chronic antibiotic therapy (see Figure 8.7). Mycotic corneal ulcers have a variety of clinical appearances ranging from nonhealing superficial ulcers to progressive antibiotic-resistant corneal ulcers with malacia or surface plaques that appear pigmented. Diagnosis is usually made with cytology and fungal culture. Treatment consists of topical antifungals and antibiotics, and in some cases surgical debulking and stabilization is necessary. With severe anterior uveitis, topical mydriatics and systemic antifungal agents should be considered.

Blastomycosis

Blastomycosis is endemic in the river valleys and also occurs in Canada, Europe, Mexico, Latin America, and Africa. Canine blastomycosis (*Blastomyces dermatitidis*) is a common regional systemic mycotic infection in the USA that often affects the ophthalmic tissues. At least 40% of affected dogs will show ophthalmic changes (Figure 18.10). Ophthalmic lesions include anterior uveitis (miosis, conjunctival hypermia, episcleral injection, aqueous humor flare, low intraocular pressure, and iridal swelling), posterior uveitis, panophthalmitis, and retinal detachments (see Figure 10.11A). Posterior segment granulomas are common. Most cases are bilateral, but can be asymmetrically affected. Spread to the eyes is via the hematogenous route, hence the disease primarily affects the choroid initially. Although the signs of a "red eye" or anterior uveitis may attract the attention of the pet owner and result in presentation for diagnosis and therapy, the most serious inflammation involves the posterior uvea, which can result in vitreal inflammation, exudative retinal detachments, and blindness. Cataract formation, secondary glaucoma, and phthisis bulbi are frequent sequelae. Treatment consists of long-term systemic antifungals and local anti-inflammatory therapy and can be expensive. Eyes with exudative retinal detachments do not usually regain vision. Enucleation may become necessary for patient comfort and can make control of the systemic condition easier.

Coccidioidomycosis

Coccidioidomycosis (*Coccidiodes immitis*), or Rift Valley fever, is a regional mycotic infection in the dog that occurs primarily in the southwestern USA, Mexico, and Central America. The arthrospores usually enter via the respiratory tract, and spread to infect the bones, skin, eyes, visceral organs, testicles, CNS, and the heart. In one study, 42% of patients with eye lesions had no systemic clinical signs, and most (80%) patients had unilateral infections (see Figure 10.11C). Clinical abnormalities include keratitis (49%), anterior uveitis (43%), and glaucoma (31%). Posterior segment involvement occurs in



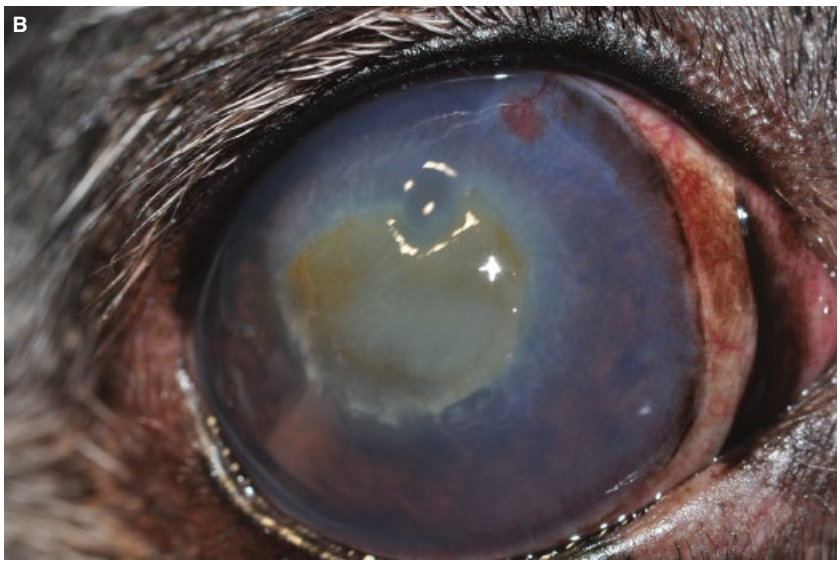


Figure 18.9 (A) Mycotic lid infections or dermatophytosis are common in puppies and appear as dry and crusty alopecia of the eyelids. (B) Mycotic corneal ulcerations are rare in the dog, and often associated with foreign bodies or systemic immunosuppression. In this dog, tissue plaque of necrotic tissue, inflammatory cells, and fungal elements are present in the axial cornea and there is peripheral corneal vascularization, corneal edema, and miosis, which signals anterior uveitis.

about 50% of patients, and can be masked by the anterior uveitis and/or opaque media (Figure 18.11). Treatment consists of long-term systemic antifungals and local anti-inflammatory medications. Eyes with exudative retinal detachments do not usually become visual.

Histoplasmosis

Histoplasmosis (*Histoplasma capsulatum*) is an infrequent ophthalmic infection in small animals which occurs in endemic areas in the Ohio, Missouri, and Mississippi river valleys. Systemic signs usually affect the respiratory or gastrointestinal systems. The ophthalmic manifestation is typically pyogranulomatous choroiditis that extends into the retina and can result in exudative retinal detachments (Figure 18.12). Diagnosis is by cytology, biopsy, or agar gel immunodiffusion test.

Cryptococcosis

Cryptococcosis (*Cryptococcus neoformans*) occurs worldwide, and affects a variety of animal species. Both CNS and ophthalmic clinical signs result, although not necessarily together. The ophthalmic signs are usually acute onset of mydriasis and blindness, related to granulomatous or pyogranulomatous optic neuritis and chorioretinitis (Figure 18.13). The optic neuritis probably results from the direct extension of the organism from

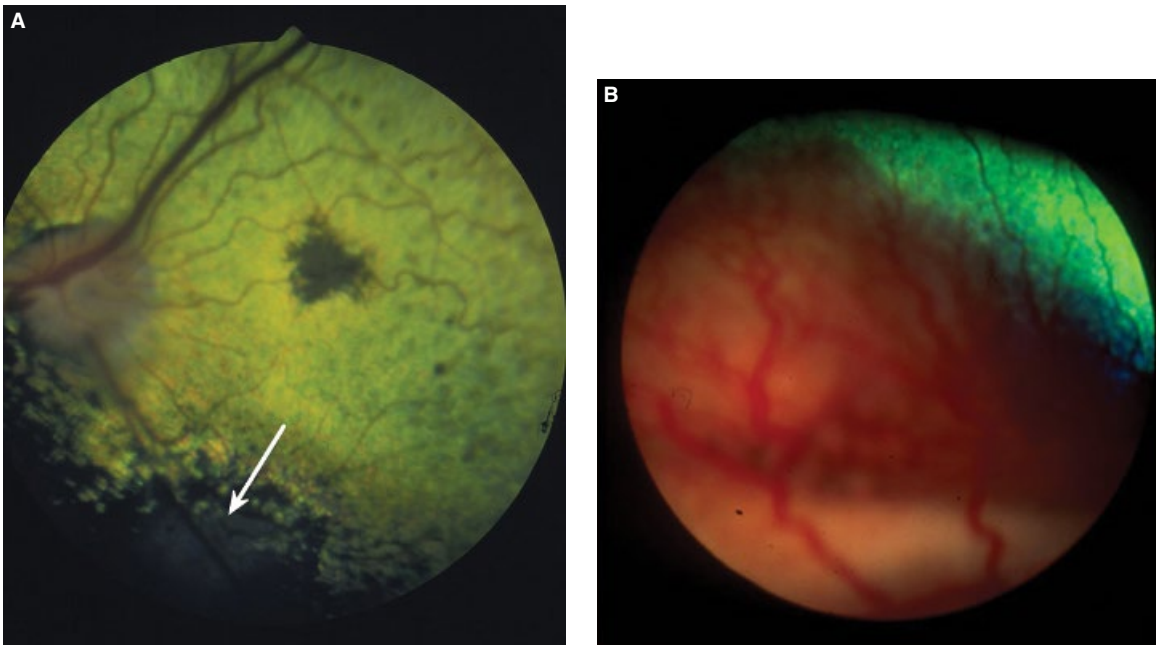
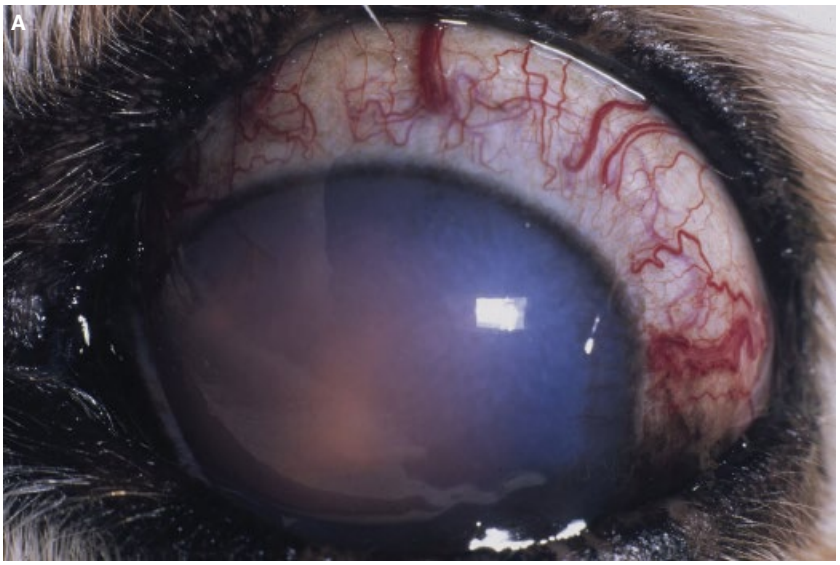
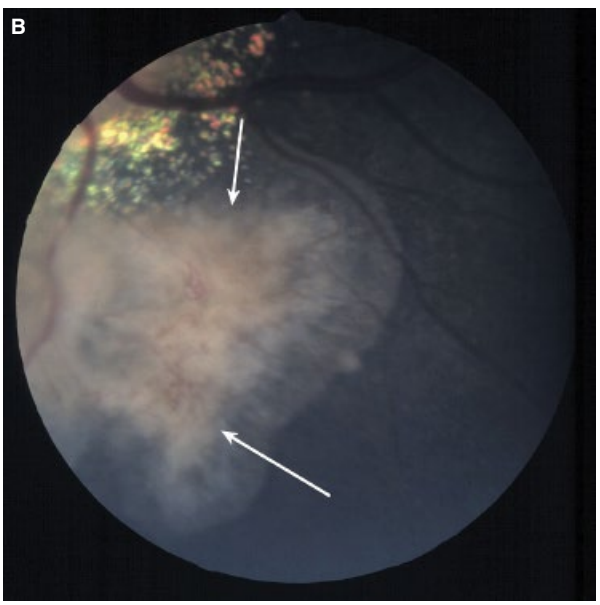


Figure 18.10 (A) Early blastomycosis in a dog causing chorioretinitis. Note the numerous pigmented foci in the tapetal fundus, and a single white granuloma (arrow) in the nontapetal fundus. (B) A blastomycosis granuloma in the peripheral fundus of a dog. Note the profound subretinal exudate underlying the retinal vessels.

Figure 18.11 (A) Coccidioidomycosis in a dog causing anterior uveitis and secondary glaucoma. Note the inflammatory debris in the anterior chamber, corneal edema, episcleral congestion, enlarged globe (buphthalmos), and deep corneal vascularization in the peripheral cornea. (B) Coccidioidomycosis also affects the posterior segment. In this dog note the diffuse chorioretinitis and focal granulomas (arrows) in the tapetal fundus. Courtesy of Ron L. Sigler.





the brain and cerebrospinal fluid (CSF). Diagnosis can be made with CSF tap or vitreocentesis demonstrating the thick-walled spheroidal yeast organisms. If attempted, long-term antifungal therapy is necessary.

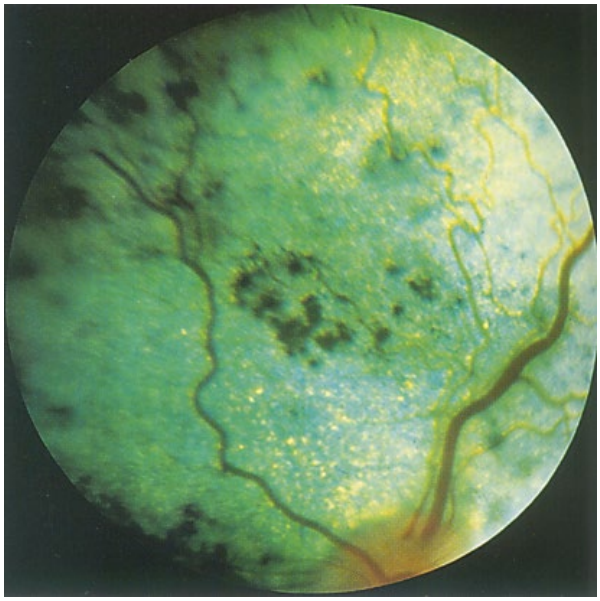


Figure 18.12 Histoplasmosis in a dog causing chorioretinitis. Note the pigmented areas within the fundus. Courtesy of Charles Martin.

Aspergillosis

Aspergillosis also affects the dog, and many variants are involved (*Aspergillus terreus*, *Aspergillus fumigatus*, *Aspergillus flavus*, *Aspergillus niger*, and *Aspergillus nidulans*). For reasons not yet defined, systemic aspergillosis seems to predominantly affect the German Shepherd breed (Figure 18.14). Systemic clinical signs often include diskospondylitis (vertebral pain, paresis, and paralysis). Ophthalmic signs include chorioretinitis, anterior uveitis, retinal detachments, and endophthalmitis (see Figure 10.11B). The prognosis is very poor.

Toxoplasmosis

Toxoplasmosis (*Toxoplasma gondii*), a protozoan, causes disease worldwide in many species of animals, including humans. The disease occurs clinically more frequently in cats than in dogs; however, affected dogs have been reported. Anterior uveitis, retinitis, choroiditis, extraocular myositis, scleritis, and optic neuritis have been associated with toxoplasmosis infections (Figure 18.15). Toxoplasmosis can occur concurrently with canine distemper.

Leishmaniasis

Leishmaniasis is usually thought of as a canine disease endemic to the Mediterranean region. However, several reports in the USA have recently documented the disease in resident dogs without travel histories. Systemic disease

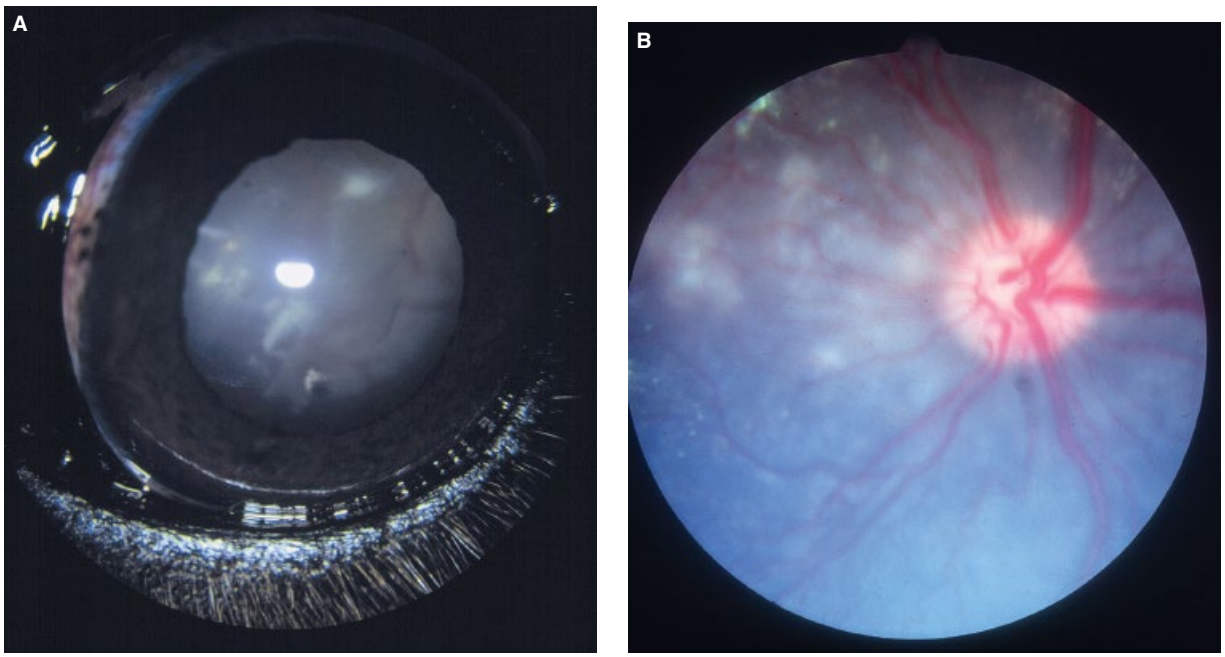


Figure 18.13 (A) Cryptococcosis in the dog tends to affect primarily the posterior segment causing chorioretinitis, optic neuritis, and exudative retinal detachment. In this dog, note the inflammatory retinal detachment and the dark foci of chorioretinitis.
(B) Cryptococcosis in dog with multiple granulomas within the retina. The highest granuloma concentration is adjacent to the disc.



Figure 18.14 Ocular aspergillosis in a German Shepherd female primarily affecting the posterior segment, causing retinal detachment and diffuse inflammation of the vitreous (causing the vitreous to appear yellow). *Aspergillus* organisms were aspirated from the vitreous.

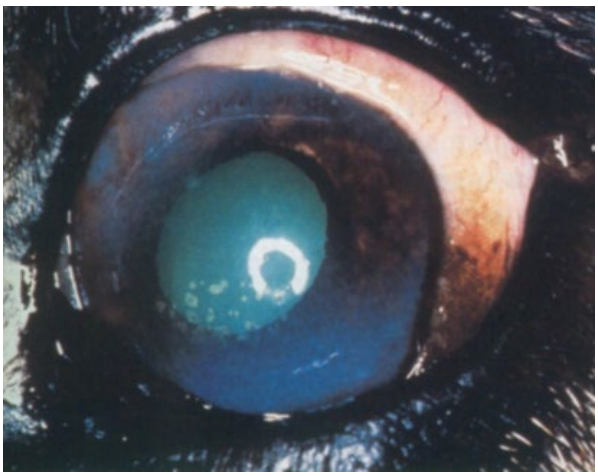


Figure 18.15 Toxoplasmosis in infrequent in the dog, and can cause anterior and posterior segment inflammations. In this dog, episcleritis, anterior uveitis, keratic precipitates, and darkened iris are present.

signs include emaciation, chronic renal failure, and chronic dermatitis (Figure 18.16). *Leishmania donovani infantum* (Old World countries) and *Leishmania donovani chagasi* (New World countries) produce several different ophthalmic abnormalities including blepharitis, granulomatous blepharitis, conjunctivitis, scleritis, anterior uveitis, and, infrequently, chorioretinitis.

Protothecosis

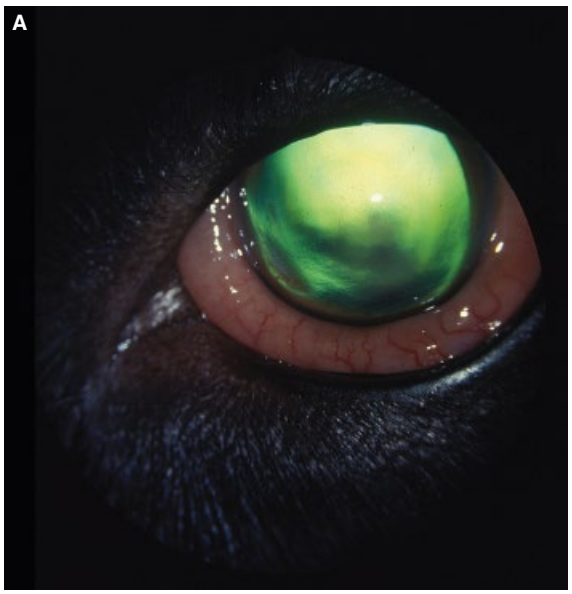
Protothecosis (*Prototheca wickerhamii* and *Prototheca zopfii*) infrequently affect dogs. Clinical signs involve the gastrointestinal and respiratory tracts, although heart, eyes, and brain can also be involved. Ophthalmic signs usually include acute mydriasis, blindness, usually due to chorioretinitis and/or retinal detachments and anterior uveitis, and/or endophthalmitis (Figure 18.17). Vitreous centesis, biopsy of infected tissues, rectal cytology, and urinary sediment are common avenues for diagnosis. Therapy includes antifungal agents, but is difficult and often the dog's poor condition merits euthanasia.

Parasitic Diseases

Dirofilaria immitis is the most frequent intraocular parasite in dogs, although aberrant migration of *Toxocara canis*, *Angiostrongylus vasorum*, *Ancylostoma* sp., and *Onchocercia* sp. have been also reported. Dirofilaria presents as an acute anterior uveitis, severe corneal edema, and the presence of a moving fourth stage (usually) larva within the anterior chamber or, less frequently, in the vitreous (Figure 18.18; see also Figure 10.9). The larva may be mobile and, when illuminated, may move away from the light from the anterior chamber through the pupil into the posterior chamber and vitreous. Therapy is removal of the intact larva from the anterior chamber, and medical treatment of the anterior uveitis. Some residual corneal edema may persist.

Ophthalmomyiasis

Ophthalmomyiasis is the migration within the anterior and posterior segments of the eye by fly larva (*Diptera* sp.). Migration of these larvae can cause direct damage to the ocular structures or secondary iridocyclitis, vitritis, and chorioretinitis (Figure 18.19). Systemic clinical signs are not usually present. Entry appears to be the ophthalmic adnexal skin or conjunctiva. Larval migration through the ocular fundus causes irregular curvilinear tracts through the tapetal and nontapetal fundi. Occasional retinal hemorrhages result when the





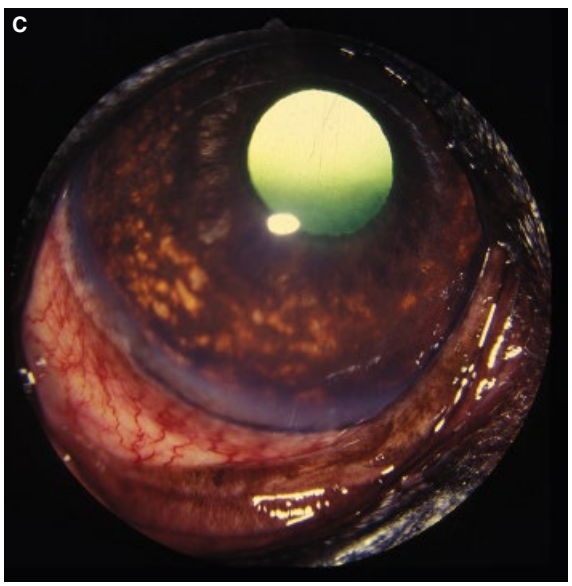


Figure 18.16 Leishmaniasis can affect all parts of the eye. (A) The conjunctiva is diffusely inflamed with leishmaniasis. The bulbar conjunctiva is elevated and hyperemia from the granulomatous inflammation. (B) Typical of systemic disease, both corneas are inflamed with leishmaniasis, and the dog is blind. Note the granulomatous inflammation with corneal edema and heavy neovascularization. (C) An intense anterior uveitis with peripheral corneal edema and neovascularization, loss of iridal pigmentation, and moderate iridal swelling. Courtesy of Alain Regnier.

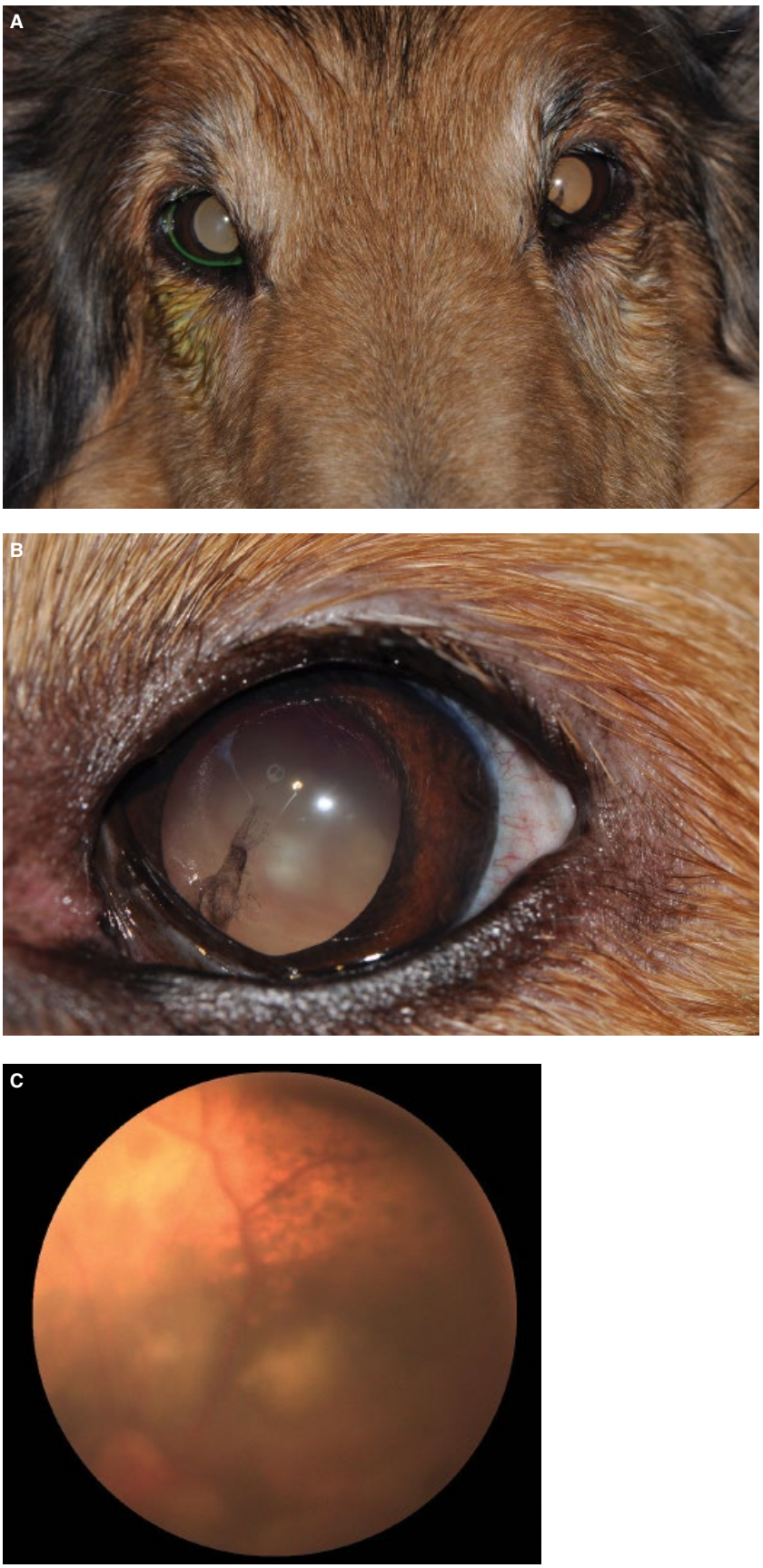


Figure 18.17 (A) Protothecosis in this dog has produced an acute anterior uveitis, severe posterior segment inflammation (white haze to vitreous), and retinal detachments. (B) Same dog as in part A. Note the retinal detachment just posterior to the lens. The pigment on the anterior lens capsule is caused by a ruptured uveal cyst, possibly an incidental finding. (C) Inflammation of the anterior and vitreal chambers obscures the fundic view of this eye. Note, however, the patches of subretinal infiltrate in this dog with protothecosis.

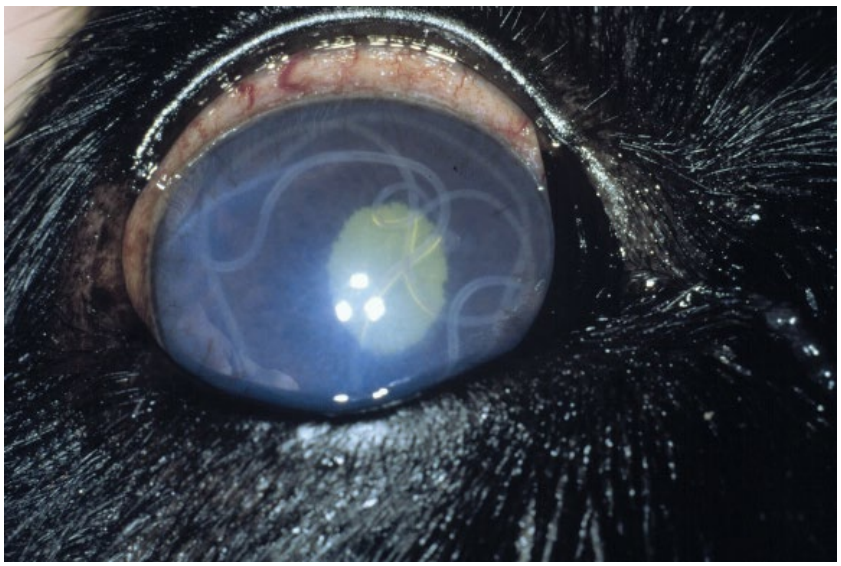


Figure 18.18 Canine heartworm (*Dirofilaria immitis*) is the most frequent intraocular parasite in the dog. In this mixed-breed dog, the dirofiliaria larva appears as a long mobile parasite in the anterior chamber. A secondary iridocyclitis is present. Note the diffuse corneal edema, conjunctival hyperemia and episcleral injection, and iridal swelling. Courtesy of Andras Komaromy.

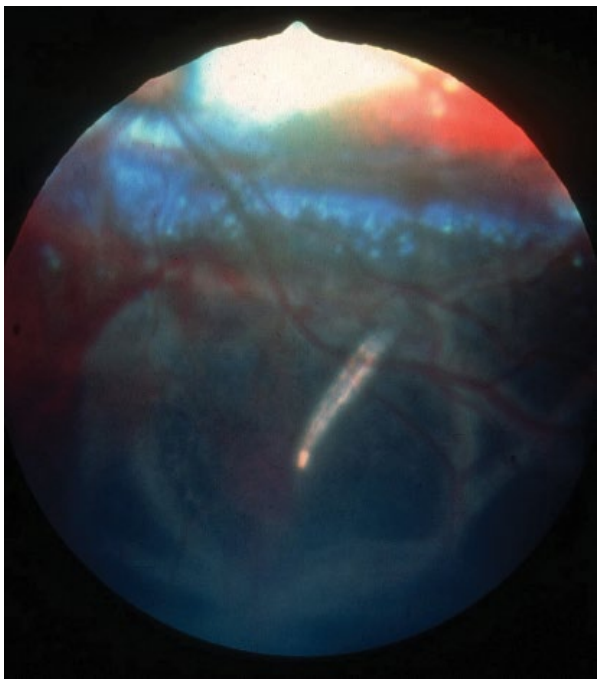


Figure 18.19 In ophthalmomyiasis interna, the fly larva migrates throughout the posterior segment. In this dog, the irregular tracts caused by the larva occur within the nontapetal fundus; the end of the larva is protruding in the vitreous.

wandering larva contacts a retinal blood vessel. The parasite may or may not be visualized. Manual removal of the larva is indicated, although this can require an invasive surgical procedure. Systemic corticosteroids can lessen the retinal damage.

Demodex

Demodex (*Demodex canis*) can produces dermatitis in young dogs, typically affecting the eyelids, lips, and fore-



Figure 18.20 In this young dog, Demodex dermatitis appears as dry scaly lesions affecting upper and lower eyelids.

legs (Figure 18.20; see also Figure 5.16). The circumscribed skin lesions are usually dry, scaly, and hairless. Diagnosis is by skin scrapings and observing the mites microscopically. Treatment for progressing lesions consists of antiparasitic preparations.

Metabolic Diseases

Diabetes mellitus affects both the dog and cat, but most clinical reports have focused on the dog. Ophthalmic signs of diabetes mellitus in the dog include acute onset of cataracts (usually resulting in blindness), ocular surface disease (dry eye, decreased tear film break-up times,

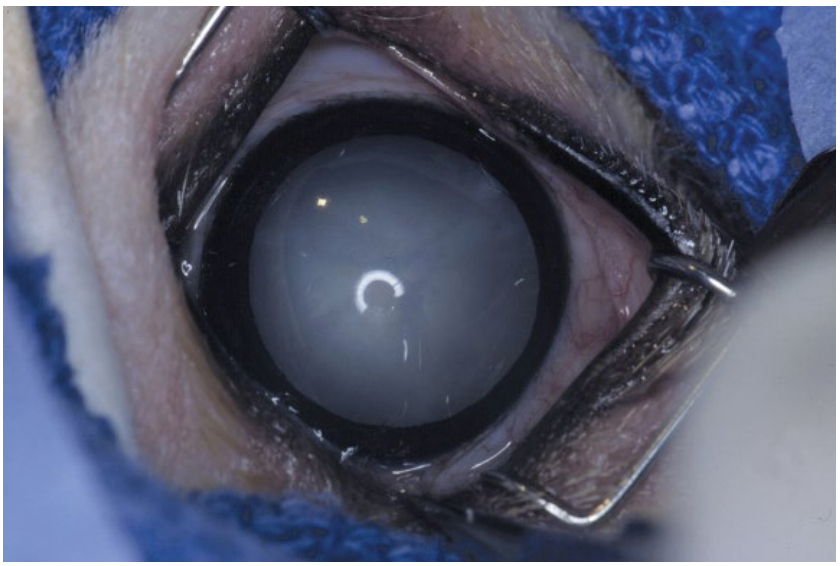


Figure 18.21 Diabetes mellitus in a dog has resulted in rapidly developing cataracts, which have caused blindness.

conjunctival epithelial dysplasia, and reductions in goblet cell density), impaired corneal wound healing (decreased corneal sensation, peripheral neuropathy), and, infrequently, retinal hemorrhages (Figure 18.21; see also Figure 11.10A,B). Retinal lesions are often masked by the presence of cataract and may not be appreciated until after surgical removal of the cataracts.

Altered lipid metabolism in affected patients can contribute to lipemia retinalis or lipid-laden aqueous humor (associated with anterior uveitis). The proliferative phase of diabetes mellitus (severe diabetic retinopathy and neovascular glaucoma), which is so destructive in humans, has not been reported in animals. Approximately 50–70% of diabetic dogs develop cataracts within 6 months of the diagnosis of the diabetes. Surgical removal of canine diabetic cataracts yields similar results to those in nondiabetic dogs. Often, cataract formation is quite rapid in dogs, resulting in a swollen lens with occasional anterior lens tears and extrusion of lens material (for the early signs of the diabetic cataract see Figure 11.10A,B).

Systemic Hypertension

Systemic hypertension occurs infrequently in domestic animals, but has become common in older domestic cats. It often has a preceding or causative underlying etiology. Essential hypertension in dogs is very rare. A dog diagnosed with systemic hypertension should be evaluated for renal, cardiac, and endocrine disease as well as other metabolic abnormalities and neoplasia. Determination of the cause of the hypertension affects therapy and prognosis. The ophthalmoscopic abnormalities appreciated with systemic hypertension include retinal, pre-retinal and sub-retinal hemorrhages, as well as retinal detachments and occasional chorioretinal infarcts (segmental degeneration) (Figure 18.22; see also Figure 13.8). Anterior uveitis may be observed. Ophthalmoscopy of the ocular fundus abnormalities permits convenient noninvasive monitoring of the response to drug therapy and result of normal blood pressure levels.

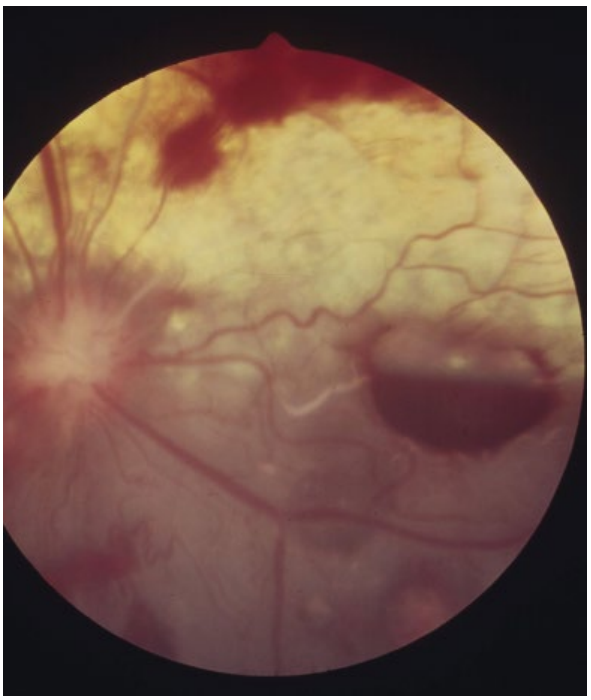


Figure 18.22 Fundus view of a dog with systemic hypertension. Note the intraocular hemorrhages, retinal hemorrhages, and variable retinal detachments. Located near the optic disc is a preretinal ("keelboat") hemorrhage.

Hyperlipidemia

Hyperlipidemia indicates elevated serum levels of cholesterol, triglycerides, or both. As serum proteins can be involved, the term hyperlipoproteinemia is used.

Causes of hyperlipidemia include pancreatitis, hypothyroidism, diabetes mellitus, hyperadrenocorticism, renal and hepatic diseases. Increased lipid blood levels can be readily observed when viewing the retinal vasculature, which can appear as pink engorged blood vessels (Figure 18.23; see also Figure 10.6E). When the blood–aqueous barrier has been compromised, as in anterior uveitis, serum lipids can also enter the anterior chamber and be confused with aqueous flare or hypopyon.

Chronic hyperlipoproteinemia has also been associated with corneal lipidosis. Hence, in the clinical evaluation of corneal lipidosis serum triglycerides, cholesterol, serum proteins, and thyroid function are recommended diagnostics. Therapy and prognosis are based on the underlying disease(s) (see Figure 8.18).

Other Circulatory Disorders

A number of circulatory disorders that affect blood flow and composition can produce vascular-related abnormalities affecting the anterior uvea (iridal hemorrhages and hyphema) and posterior segment (retinal and vitreal hemorrhages, and retinal detachments). These vascular conditions include but are not limited to polycythemia, severe anemia, thrombocytopenia, icterus, hyperviscosity syndrome (elevations of large molecules – IgM or polymerized IgA) and neoplasia (such as lymphoma).

Polycythemia usually produces highly tortuous conjunctival and retinal blood vessels that appear darker than normal. Small retinal hemorrhages can also result from severe anemia (hematocrit <5% or 7%). Thrombocytopenia (from many causes) can produce conjunctival hemorrhage, hyphema, retinal hemorrhages, and even retinal detachments. In hyperviscosity syndrome (most commonly encountered with multiple myeloma and lymphoma), secondary glaucoma, secondary cataract formation, papilledema, and retinal detachments can result. Diagnosis of the underlying disease directly influences therapy and prognosis (Figure 18.24).

Excessive Intravenous Therapy

Acute intensive intravenous fluid therapy in the dog, as for renal failure, can produce acute bilateral, limited to complete retinal detachments (Figure 18.25). The clinical signs with complete retinal detachments are acute mydriasis and blindness. Blood pressure is within normal levels and retinal hemorrhage is not usually present. Resolution occurs once the intensity of fluid therapy is reduced, or the renal problem is corrected.

Inherited or Breed-related Diseases

Uveodermal syndrome affects the Arctic breeds of dogs primarily, but has also been described in a wide variety of breeds including the Irish Setter, Golden Retriever, Shetland Sheepdog, St. Bernard, Old English Sheepdog, Rat Terrier, and Australian Shepherd. Affected dogs are young to middle-aged and present with progressive loss of pigmentation of the eyelid margins, lips, foot pads, scrotum, and nose (Figure 18.26). Ophthalmic lesions include blepharitis and loss of lid margin pigment, iridocyclitis and loss of iridal pigmentation, chorioretinitis and loss of nontapetal pigmentation and optic neuritis (Figure 18.27; see also Figures 5.19, 10.7, and 10.14). Secondary glaucoma, cataract formation, retinal detachment, and optic nerve degeneration are common. The unrelenting and chronic immune-mediated disorder targets melanin and melanocytes; therefore, any heavily pigmented tissues in the body can be affected. The diagnosis is confirmed with histopathology of the skin. Chronic systemic anti-inflammatory and immunomodulating therapy, in combination with topical corticosteroids and mydriatics for the anterior signs, is required for this disease (see Figures 4.10B and 10.21).

Neoplastic Diseases

Lymphoma is the most common secondary intraocular tumor in the dog and the most common secondary (metastatic or generalized) neoplasm of the eye. In one study in which 37% of dogs with lymphoma had ophthalmic disease (most were in advanced stages of the disease), the ocular signs included anterior uveitis (49% of affected dogs), posterior uveitis (9%), panuveitis (14%), retinal detachments (23%), and adnexal lesions (6%) (Figure 18.28). Orbital masses producing exophthalmia, as well as subconjunctival masses in the nictitating membrane and eyelids, are possible. Secondary retinal hemorrhages, retinal detachments, glaucoma, and cataract formation are possible sequelae. Systemic chemotherapy should be accompanied by topical anti-inflammatory therapy (see Figure 4.8B).

Ophthalmic Manifestations of Feline Systemic Diseases

The cat has fewer inherited eye diseases than the dog, but more viral diseases that can involve the eye and have serious systemic effects: feline herpes virus (FHV-1), feline infectious peritonitis (FIP), and feline immunodeficiency virus (FIV).

Infectious Diseases

Feline herpesvirus

Feline herpesvirus (FHV-1) affects cats worldwide, with most cats becoming infected at an early age and subsequently developing latency, so that they are carriers of

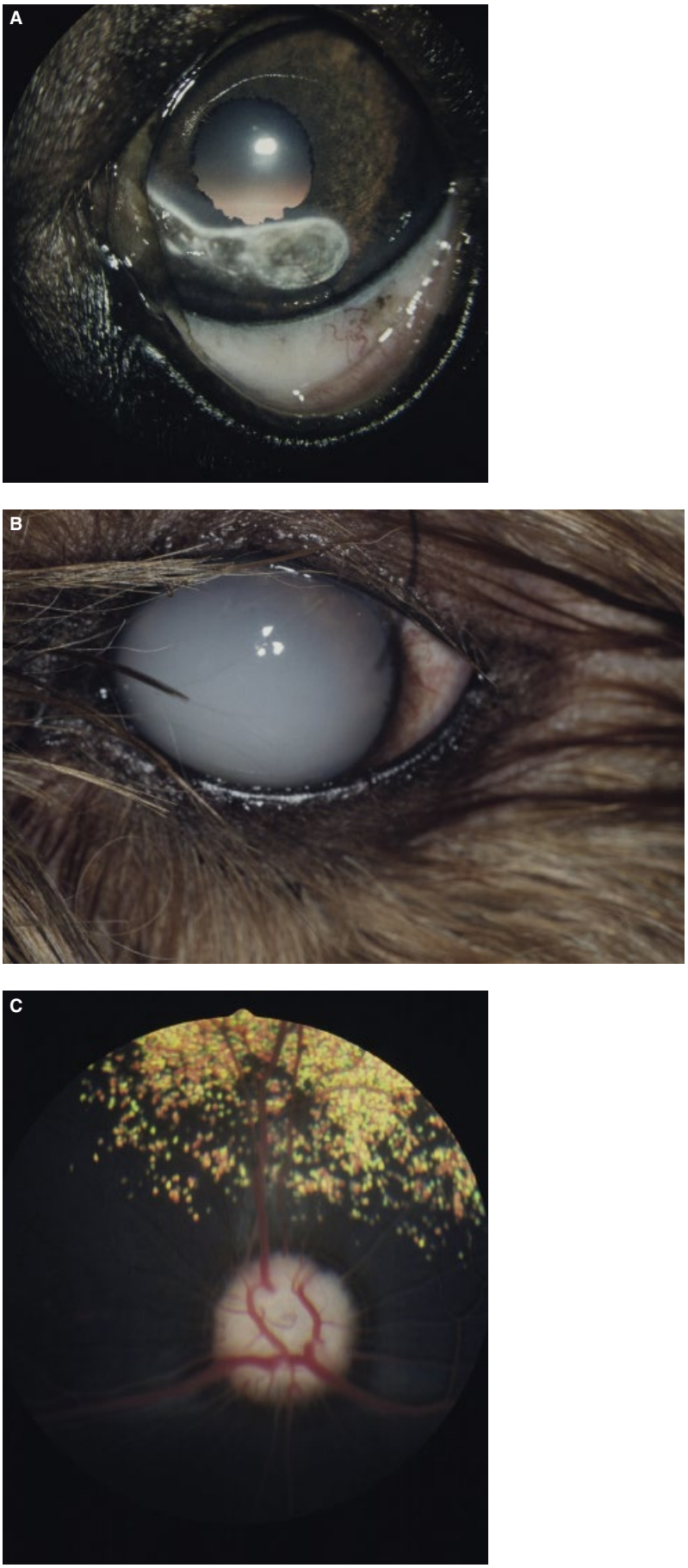


Figure 18.23 (A) Focal peripheral corneal lipidosis in a German Shepherd dog secondary to hyperlipidemia. (B) Lipids within the anterior chamber in a dog. Note that it is difficult to visualize the iris and the pupil, which places the opacity anterior to these structures. (C) Lipemia retinalis in this dog manifests as a pink color to the retinal vasculature.

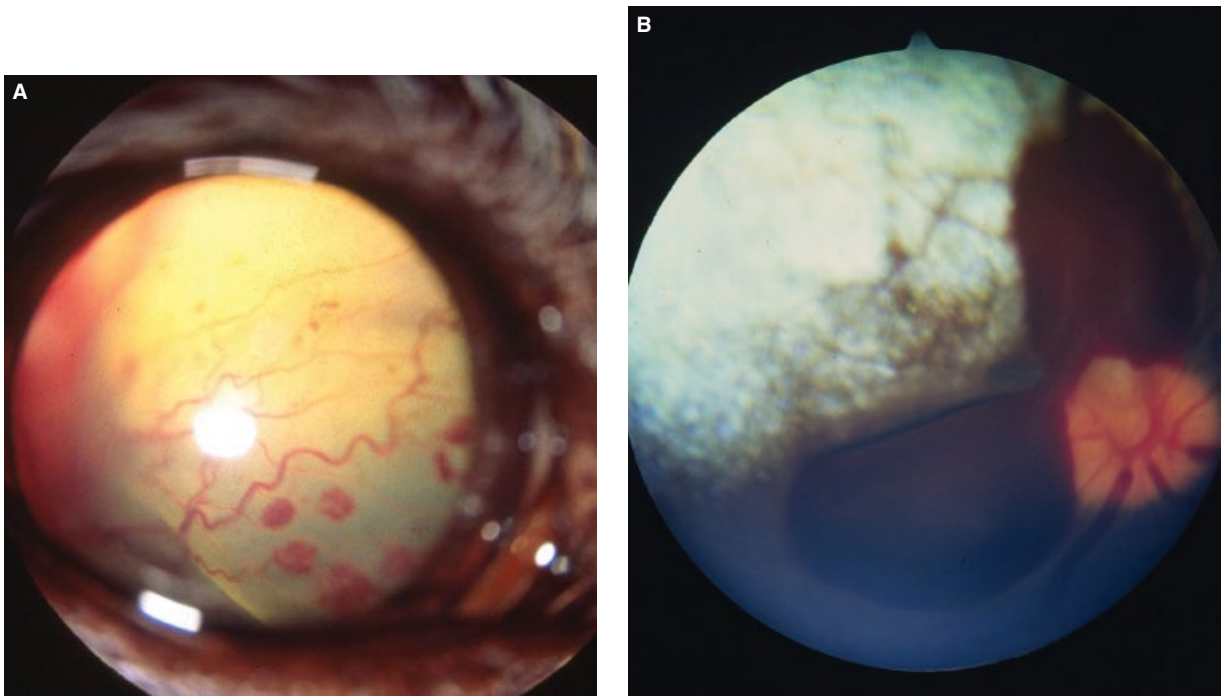


Figure 18.24 (A) Hyperviscosity in a dog associated with a cardiac anomaly. Note the tortuous, dilated, and dark red veins in this detached retina and multifocal retinal hemorrhages. (B) Pre-retinal hemorrhage in a Boston Terrier associated with hyperviscosity syndrome and lymphoma.

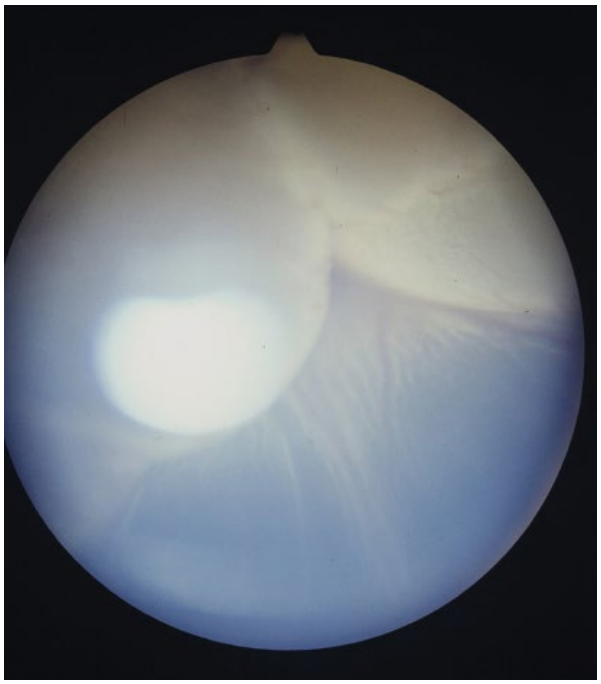


Figure 18.25 This dog was being treated aggressively with intravenous fluids for renal failure. Note the complete retinal detachment in the absence of inflammation and hemorrhage.

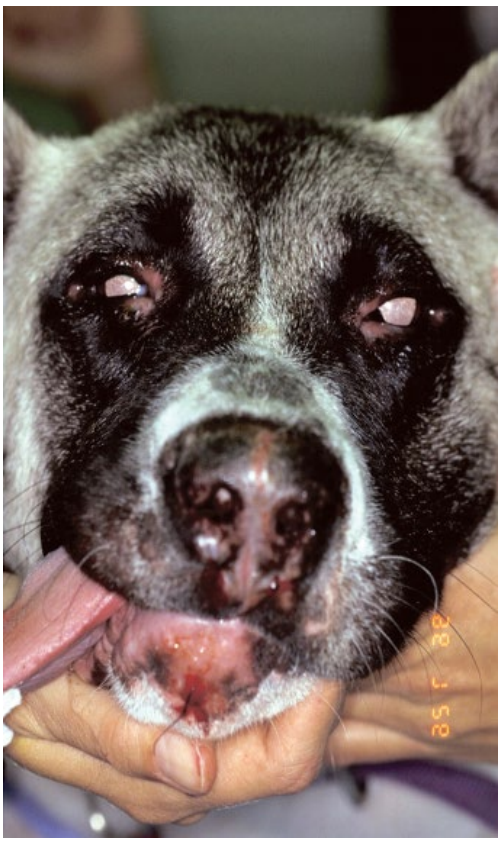


Figure 18.26 Uveodermal syndrome (UDS or Vogt–Koyanagi–Harada-like syndrome) in an Akita dog. There is depigmentation of the nose, lips, and eyelids, in addition to iridocyclitis and chorioretinitis.

Figure 18.27 A dog with UDS of several months' duration. Note the iridal depigmentation, iridocyclitis, and secondary keratitis.



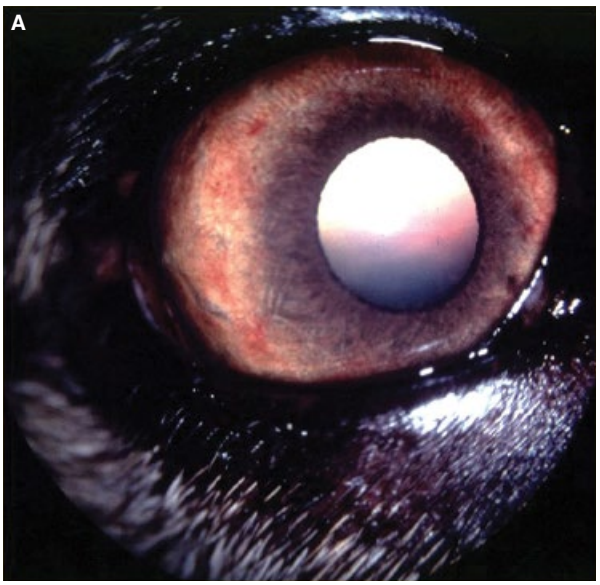






Figure 18.28 (A) Early anterior uveal lymphoma. The tufts of new blood vessels on the iridal surface surround small iridal lymphoma nodules. (B) Lymphoma in a dog with anterior uveitis and secondary glaucoma. Note also the enlargement of the facial lymph nodes beneath the orbits. (C) Lymphoma in another dog with iridocyclitis, hyphema, and posterior segment hemorrhage. Note the marked hyperemia of the bulbar conjunctiva and episcleral injection. Posterior synechiae are developing.



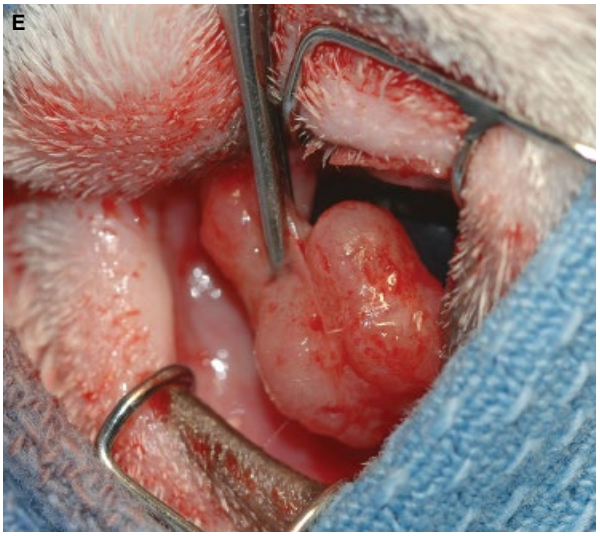


Figure 18.28 (*Continued*) (D) Resolving hyphema (clot attached to anterior lens capsule) and multifocal posterior synechiae in an eye following treatment for anterior uveitis associated with generalized lymphoma. (E) Adnexal lymphoma. The normal architecture of the nictitans has been effaced by neoplastic infiltrates.

the virus for life. FHV-1 has been associated with several ophthalmic diseases including acute, chronic, and recurrent conjunctivitis, dendritic and geographic corneal ulcers, stromal keratitis, keratoconjunctivitis sicca, corneal sequestrum, neonatal ophthalmia, and symblepharon (Figure 18.29). In adult cats, most often the ophthalmic manifestions of FHV-1 are recurrences.

Herpes-associated conjunctivitis appears with hyperemia, blepharospasm, chemosis, and mucopurulent exudates (see Figures 14.10, 14.11, 14.14, and 14.18). The corneal ulceration is quite characteristic with dendritic or geographic corneal ulcers that can also affect the conjunctiva (see Figures 14.16 and 14.17). Conjunctivitis and corneal ulceration are signs that develop in the cytolytic form of an active FHV-1 infection (active viral replication and epithelial cell lysis). When stromal keratitis occurs, it is usually an immune-mediated manifestation of disease, wherein signs of damage to ocular tissues is the result of self-damage by the host. It tends to be chronic and appears with corneal edema, stromal cellular infiltration, and deep corneal vascularization.

Therapy for the acute conjunctivitis is usually limited to supportive care with topical antibiotics and lubricants. For the recurrent and severe conjunctivitides and corneal ulcerations, topical and or systemic antiviral medications may be indicated. Stromal keratitis usually requires topical anti-inflammatory therapy, often combined with antiviral agents.

Chlamydophila

Chlamydophila infections in cats are usually mild and consist of conjunctival hyperemia, chemosis, serous ocular discharge, and blepharospasm (Figure 18.30). Often one eye is affected initially, but the opposite eye becomes involved a few days later. Asymptomatic carriers can spread the disease in catteries.

Feline Infectious Peritonitis

FIP is caused by a coronavirus, and is a serious and fatal systemic disease in the cat. Ophthalmic abnormalities occur most often with the non-effusive ("dry") form, and include bilateral granulomatous anterior and posterior uveitis, mutton-fat keratic precipitates, chorioretinitis, retinal hemorrhages, retinal detachments, and optic neuritis (Figure 18.31; see also Figure 14.26).

The definitive diagnosis of FIP is difficult without confirmation with histopathology; however, it can be suspected or presumed based on clinical signs, especially when they occur in young cats with elevated serum protein levels. Serum coronavirus titers are not reliable as FIP and feline enteric corona virus cross-react. Therapy for the anterior uveitis includes corticosteroids and mydriatics. Often, however, FIP uveitis will progress in spite of aggressive therapy.

Feline Immunodeficiency Virus

FIV can have ophthalmic manifestations, causing chronic anterior and posterior uveitis (Figure 18.32). Pars planitis occurs occasionally with FIV and is characterized by cellular infiltrates within the anterior vitreous. As a result of the chronic uveal inflammation, secondary cataract formation and glaucoma often develop (see Figure 14.28). FIV is the most frequent cause of chronic iridocyclitis and secondary glaucoma in the cat. Treatment of the uveitis requires topical corticosteroids and mydriatics.



Figure 18.29 (A) Recurrent feline herpesvirus (FHV-1) conjunctivitis and corneal ulcer in a young cat. Note the marked conjunctival inflammation (hyperemia and chemosis). (B) Dendritic corneal ulcers in a cat with FHV-1. (C) Kitten with symblepharon as the result of FHV-1. The palpebral conjunctiva has adhered to itself, the conjunctiva of the nictitans, and to the cornea.



Figure 18.29 (*Continued*) (D) Stromal keratitis in a cat secondary to FHV-1.



Figure 18.30 Chlamydophila (*Chlamydophila psittaci*) conjunctivitis in a young cat. Note the exudate at the medial canthus.

Toxoplasmosis

Toxoplasmosis occurs not infrequently in cats and is caused by *Toxoplasma gondii*, an obligate intracellular protozoal organism (Figure 18.33). Cats are the definitive host to *T. gondii*, and become infected by ingesting infected meat or water. Both anterior uveitis and chorioretinitis with exudative retinal detachments occur. Clinical signs relate to either anterior uveitis (blepharospasm, conjunctival hyperemia, aqueous flare, hypopyon, miosis, and iridal swelling) or chorioretinitis (inflammatory retinal detachments, impaired pupillary reflexes, and blindness) (see Figure 14.29).

Feline Leukemia Virus

FeLV frequently affects the feline eye and presents with inflammation of the anterior and posterior uvea, retina and optic nerve, and/or as overt masses in the orbit, eyelids, subconjunctival tissue, nictitating membrane, anterior chamber, and the posterior segment (Figure 18.34). The "D" or reverse "D" pupil in cats has been associated with positive FeLV titers (see Figure 19.7). Aqueous flare, hypopyon, dark iridal nodules within the iridal stroma, secondary glaucoma, and secondary cataracts with posterior synechiae are also common (see Figures 14.27 and 14.35).

Because of the variability of clinical presentation, titers for FeLV (using the ELISA method) are part of the routine clinical work-up for cats with anterior and posterior uveal diseases. Diagnosis is confirmed with FeLV titers, and biopsies of bone marrow, lymph node, or other affected tissues. Therapy for the ophthalmic manifestations is supportive with topical and systemic corticosteroids and mydriatics.

Cryptococcosis

Cryptococcosis (*Cryptococcus neoformans*) affects both dogs and cats, but is more frequently diagnosed in the cat (Figure 18.35). The organism enters the eye by direct meningeal extension from the brain and optic nerve, and/or hematogenously. CNS involvement and neurologic signs often precede ocular involvement. Ophthalmic signs usually include mydriasis and vision impairment or blindness. Anterior segment involvement is usually mild or occurs late in ophthalmic disease. The most frequent ophthalmoscopic findings are granulomatous optic neuritis and chorioretinitis with focal retinal detachments (see Figure 14.48).

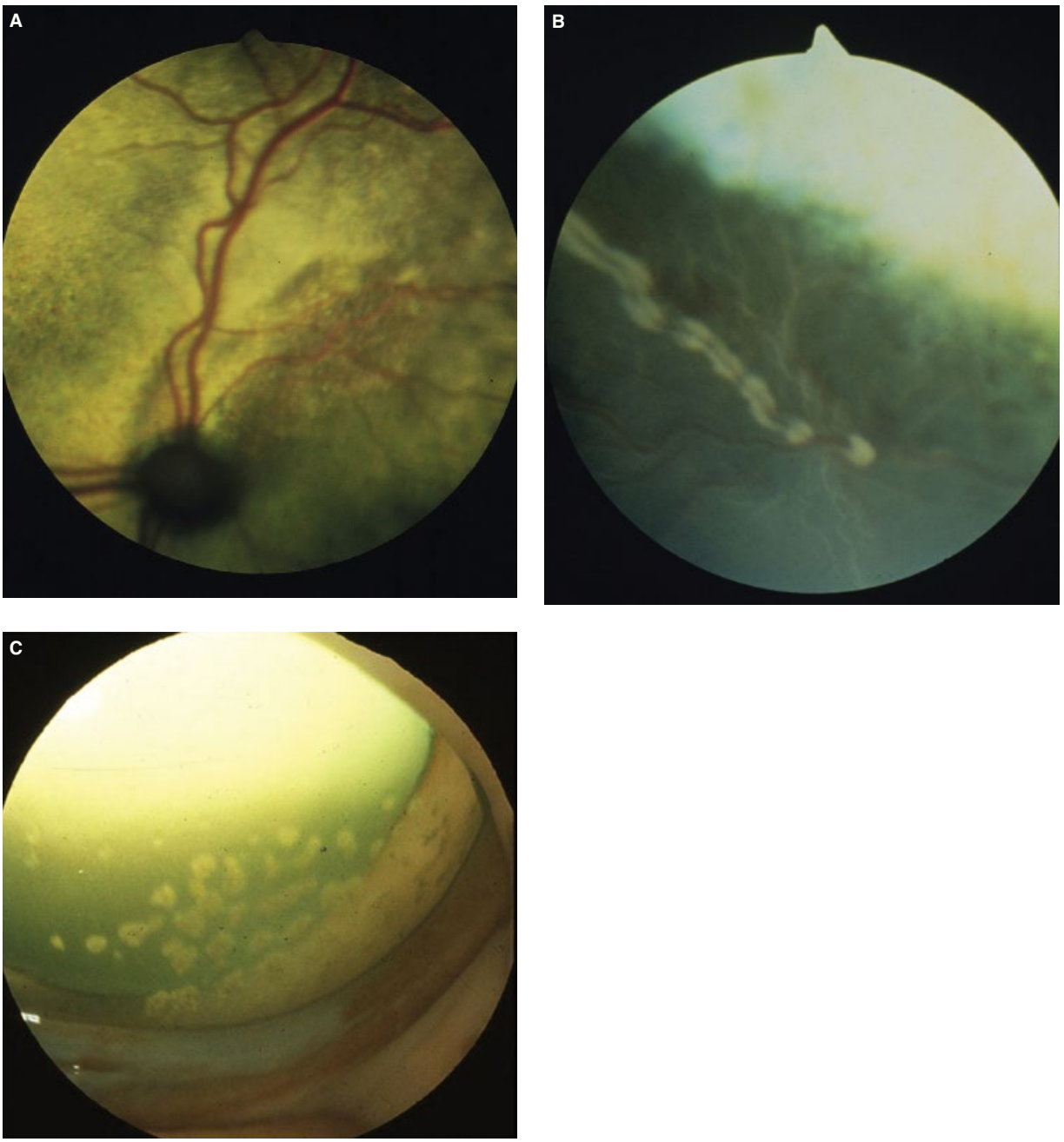


Figure 18.31 (A) Chorioretinitis in a young cat caused by feline infectious peritonitis (FIP). Note the diffuse involvement of the fundus. (B) Perivascular cupping of retinal blood vessels and chorioretinitis in a cat with FIP. (C) Anterior uveitis in a cat with FIP. Note the large, coalescing keratic precipitates on the corneal endothelium.

Infectious Feline Enteritis

Infectious feline enteritis in the developing feline fetus causes retinal dysplasia and cerebellar hypoplasia. The retinal dysplasia is usually concentrated in the tapetal fundus immediately above the optic disc, and appears as focal round or oval hyperreflective and pigmented areas (Figure 18.36). The retinal disease does not usually produce clinical visual impairment (see Figure 14.44).

Systemic Hypertension

Systemic hypertension is a common condition in older cats (<10 years of age). In as high as 80% of affected cats, signs of mydriasis and blindness initiate the first presentation to the veterinarian. The ophthalmic signs include hyphema, intravitreal hemorrhage, retinal hemorrhages (sub-retinal, intraretinal, and pre-retinal), and retinal



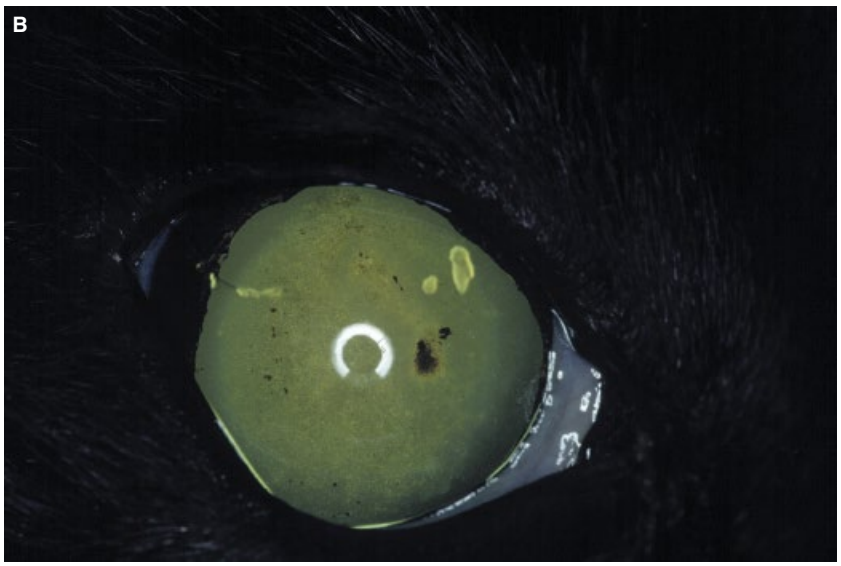


Figure 18.32 (A) Anterior uveitis in a cat caused by feline immunodeficiency virus (FIV). Note the conjunctival hyperemia, iridal swelling, iridal neovascularization, and posterior synechiae. (B) Anterior uveitis in a cat with FIV. Note the inflammatory cells on the anterior and posterior lens capsules, and a focal hemorrhage on the anterior lens capsule.



Figure 18.33 Anterior uveitis in a cat secondary to toxoplasmosis. Note the iridal swelling and inflammatory cells on the posterior cornea.

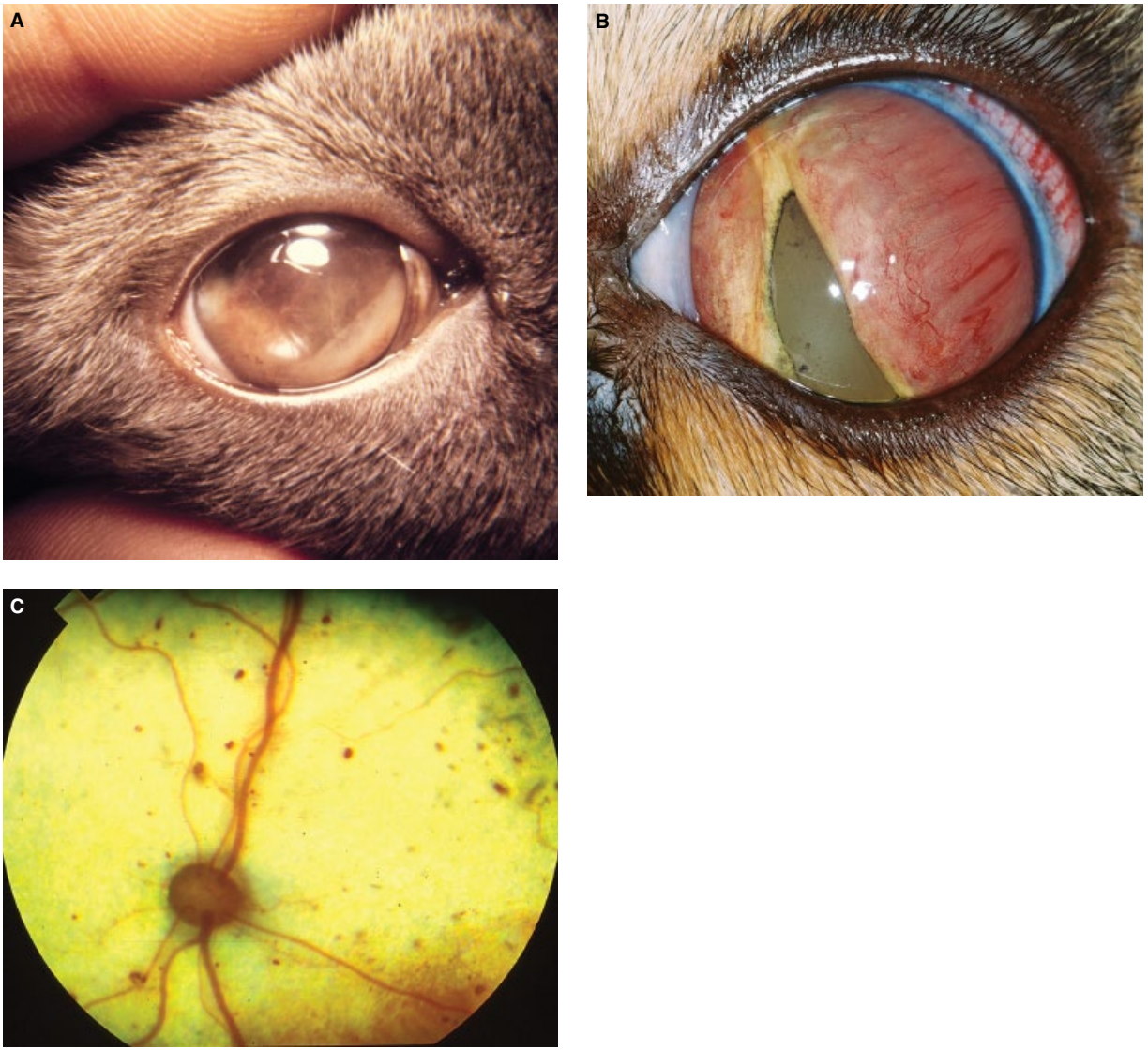


Figure 18.34 (A) Anterior uveitis and hypopyon in cat caused by feline leukemia virus (FeLV). (B) Neoplastic mass (lymphoma) involving the iris in a cat with FeLV. (C) Multifocal intraretinal hemorrhages in a cat with anemia secondary to feline leukemia.

Figure 18.35 Cryptococcosis chorioretinitis in cat. Note the retinal granulomas beneath a retinal vein in the tapetal fundus and the subretinal hemorrhage.

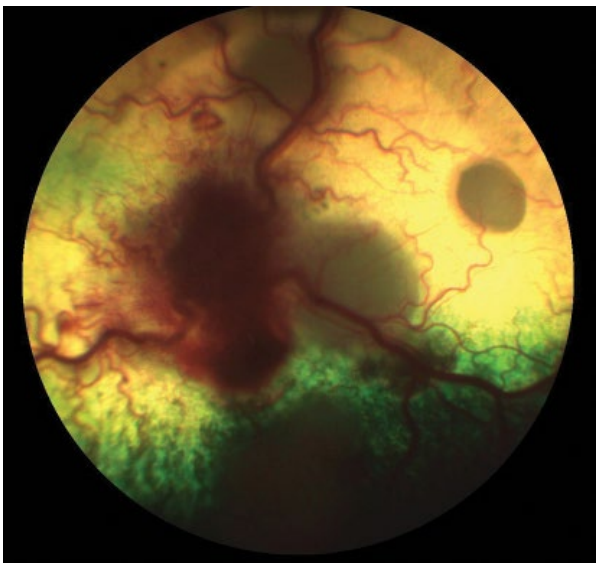




Figure 18.36 Feline panleukopenia in kittens can cause retinal dysplasia, often involving the tapetal fundus. Note the focal hyperreflective area with some focal pigmentation.

detachments (Figure 18.37). The onset of the retinal detachments causes the loss of vision. Other systemic conditions, such as renal disease, cardiac disease, and endocrinopathies, often precede and cause the development of hypertension. Hence, cats with hyphema and retinal hemorrhages and detachments require a complete medical and cardiovascular workup. Systemic antihypertensive therapy should be initiated as soon as possible in addition to therapy for the underlying etiology because timely resolution of the retinal detachment is critical to preserve as much retinal tissue and function as possible. Resolution of the systemic hypertension will allow the retina to reattach and remain or regain function in many cases if the detachments are not chronic and retinal damage is not severe (see Figure 16.9).

Ophthalmic Manifestations of Equine Systemic Diseases

There are many ocular manifestations of systemic disease in the equine species (horse, mule, and donkey) that can assist in establishing a systemic disease diagnosis. Some of these systemic diseases are presented here and others are included in Chapter 15.

Coat Color and Ophthalmic Diseases

Coat color is associated with iris color and heterochromia iridis (see Figure 15.6). There is also a condition known as lethal white foal syndrome that occurs in white, typically blue-eyed, foals with both parents overo-colored. The foals are born with two copies (homozygous) of a mutated endothelin-β receptor gene. They also have aganglionic colon resulting in gastrointestional dysfunction (which causes death), and often deafness. Lethal white foal syndrome is inherited as an autosomal recessive trait; there is a genetic test available (http:www.horsetesting.com/LWO.htm: Veterinary Genetic Laboratory, University of California, Davis, CA).

Coat Color and Congenital Stationary Night Blindness

Coat color and congenital stationary night blindness (CSNB) occur mainly in the Appaloosa breed. The leopard spotted coat is an important phenotype in Appaloosa horses with CSNB. Genes determining the characteristic coat color of Appaloosa horses have been identified.

Ocular Anomalies in Rocky Mountain Horses

Congenital ocular defects have been reported in the Rocky Mountain Horse breed, and are, in part, related to coat color. About 45% of Rocky Mountain Horses with chocolate-colored coats with white manes and tails have multiple ocular anomalies including megalocornea, congenital miosis, ciliary cysts, and retinal dysplasia (Figure 18.38). About 12%, 6%, and 4% of the affected horses with chocolate-colored coat with flaxen mane and tail, chestnut-colored coat, or some other coat color, respectively, have multiple ocular anomalies.

Habronemiasis

Habronemiasis (Summer Sores; Swamp Cancer) results from the aberrant migration of the larvae of *Habronema muscae*, *Habronema majus*, and *Draschia megastoma*. These adult parasites are found within the stomach of horses. Flies, including *Musca domestica* (house fly) and *Stomoxys calcitrans* (stable fly), are the intermediate hosts, and deposit the larvae in wounds or near the eye; larval migration through these tissues results in a granulomatous inflammatory response. These lesions affect the conjunctiva, nictitans, and/or periocular area, and these papules present with an irregular often yellow ("sulfur granules") appearance (Figure 18.39).

Diagnosis of cutaneous habronemiasis is based on the consistent clinical signs, an histologic granulomatous inflammatory infiltrate with eosinophils and mast cells, and collagenolysis. Larvae may not be present. Therapy consists of systemic ivermectin and topical topical corticosteroids to reduce the inflammatory response to the dying larvae.



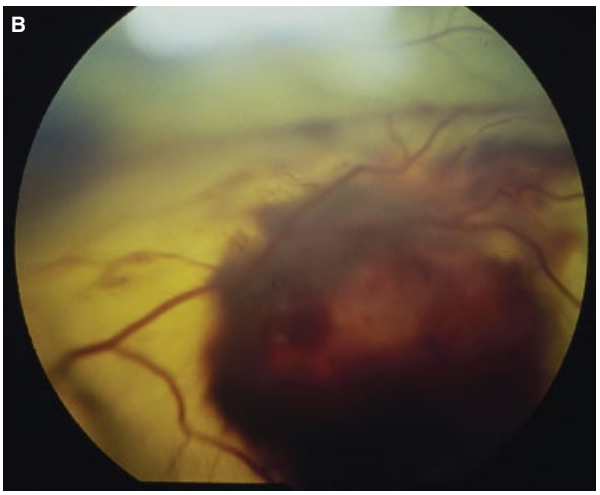


Figure 18.37 (A) Hyphema in a cat with systemic hypertension. The hemorrhage has nearly filled the anterior chamber and has obscured the pupil. (B) Focal retinal hemorrhage and detachment in a cat with systemic hypertension. The adjacent dorsal tapetal retina remains attached.



Figure 18.38 (A) Rocky Mountain Horse with multiple ocular defects with a chocolate-colored coat and white mane.

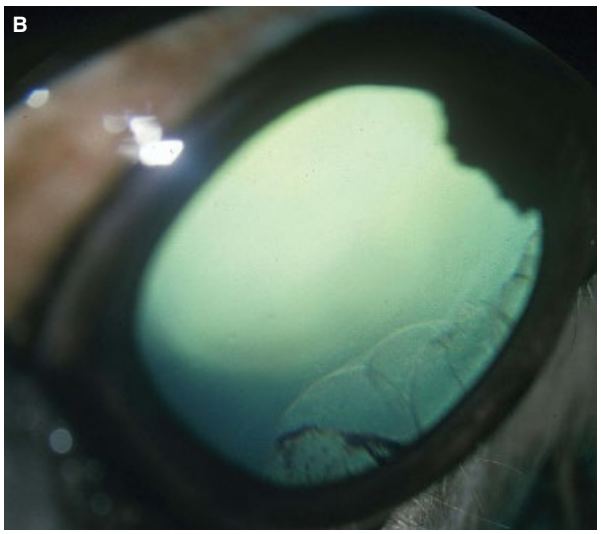


Figure 18.38 (*Continued*) (B) These ocular anomalies include megalocornea (most noticeable), congenital miosis (do not dilate fully with topical 1% tropicamide), ciliary cysts (shown here), and retinal dysplasia (the latter two defects may require ophthalmoscopy). Affected horses are usually visual.



Figure 18.39 Habronemiasis in the horse. Note the raised inflammatory mass at the base of the nictitans.

West Nile Virus

West Nile virus (WNV) is a single-stranded virus of the family Flaviviridae that is maintained and spread by a bird–mosquito relationship. Birds are the reservoir while the mosquitos transmit the virus to a number of species including birds, horses, cats, dogs, bats, alligators, and humans.

The disease typically affects young adult unvaccinated horses, although any aged horse can be infected. Clinical signs include ataxia, paresis, generalized muscle fasciculations, pyrexia, hyperesthesia, general malaise, lip droop, circling, and recumbency. Ophthalmic clinical signs include blindness, facial nerve paralysis, keratitis, and protrusion of the nictitans (Figure 18.40).

Diagnosis of West Nile Fever is based on elevated IgM concentrations in the serum and CSF, amplifying WNV DNA using polymerase chain reaction (PCR) or isolating the virus. Therapy consists of supportive care and systemic anti-inflammatory drugs to reduce cerebral edema. Prognosis is guarded, especially for unvaccinated horses.

Lymphoma and Lymphosarcoma

Lymphosarcoma is the most frequent neoplasm to affect the orbit and eye. Lymphosarcomas occur with eyelid swelling and masses of the nictitans, conjunctiva, and orbit, and uveitis (Figure 18.41; see also Figure 15.13).

Ophthalmic Manifestations of Bovine Systemic Diseases

Although the cow is an infrequent ophthalmic patient, several eye diseases have significant herd and financial effects. Fortunately, several of these diseases can be



Figure 18.40 (A) Horse with West Nile fever and profound forelimb weakness. These horses are usually very sick. Courtesy of Rob Mackay. (B) Horse with facial nerve paralysis and West Nile fever.

successfully treated. Additional information on these diseases can be found in Chapter 16.

Microphthalmia: Inherited, Avitaminosis A, or Infectious

Microphthalmia in calves occurs spontaneously, and can be inherited with other ocular defects in the Hereford and Shorthorn breeds (Figure 18.42; see also Figure 16.1). It occurs more often in calves from older dams. In the Shorthorn breed, microphthalmia, cataracts, retinal dysplasia, and retinal detachments appear inherited as an autosomal recessive trait. Microphthalmia and a prominent nictitans are accompanied by a palpebral fissure that is reduced in length. Cataracts are often present. There is no therapy.

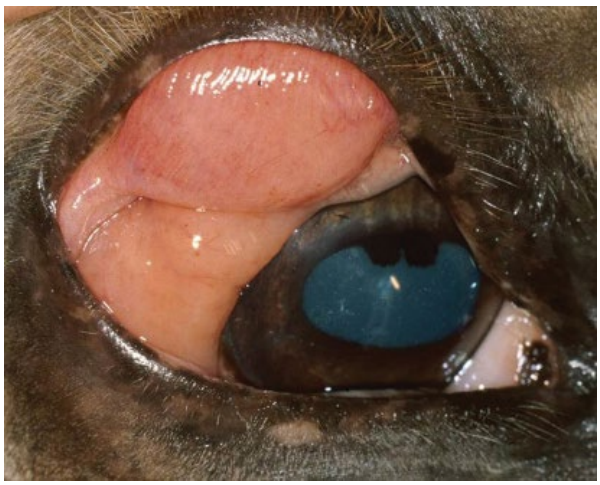


Figure 18.41 Conjunctival lymphoma in a horse.

Bovine Viral Diarrhea

Bovine viral diarrhea (BVD) virus causes prenatal ophthalmic abnormalities in calves when the dams are exposed to the virus between 75 and 150 days post-conception. Concurrent CNS abnormalities include microencephaly, cerebellar hypoplasia, hydranencephaly, and hydrocephalus. The ophthalmic abnormalities include microphthalmia, cataracts, retinal dysplasia and degeneration, optic nerve gliosis, and optic neuritis (Figure 18.43; see also Figure 16.13). Hence, calves with multiple ocular anomalies should be carefully examined for evidence of BVD.

Infectious Diseases

Infectious Bovine Rhinotracheitis

The infectious bovine rhinotracheitis (IBR) virus causes ophthalmic disease including conjunctivitis, corneal



Figure 18.42 Unilateral microphthalmos in a calf. The etiology was not determined.



Figure 18.43 Intrauterine exposure of the developing calf to bovine viral diarrhea can induce a number of ophthalmic anomalies. Note in this calf microphthalmia, persistent pupillary membranes, and cataract.



Figure 18.44 Anterior uveitis and corneal edema in a heifer secondary to systemic infectious bovine rhinotracheitis (IBR). Note the conjunctival hyperemia, and deep corneal vascularization.

edema, and anterior uveitis, which can be confused with infectious bovine keratoconjunctivitis (IBK). The conjunctivitis is characterized by raised red to white plaques of lymphocytic follicles, chemosis, and exudates that rapidly become mucopurulent. The keratitis is nonulcerative and characterized by edema that may be severe and corneal vascularization (Figure 18.44). The edema can become so dense that blindness results. Anterior uveitis is usually mild.

Bovine Thromboembolic Meningocephalitis

Bovine thromboembolic meningocephalitis is caused by *Haemophilus sommus*, and occurs primarily in feedlot cattle. The clinical signs include depression, neurologic signs, mydriasis, "red" eye, and vision impairment. Anterior uveitis, chorioretinitis with hemorrhages and white exudates, exudative retinal detachments, and endophthalmitis have been documented with this disease . Other severe septic infections, affecting the udder, respiratory tract, and uterus, can cause bacteremia that also cause anterior and posterior uveitis, chorioretinitis, and exudative retinal detachments (Figure 18.45).

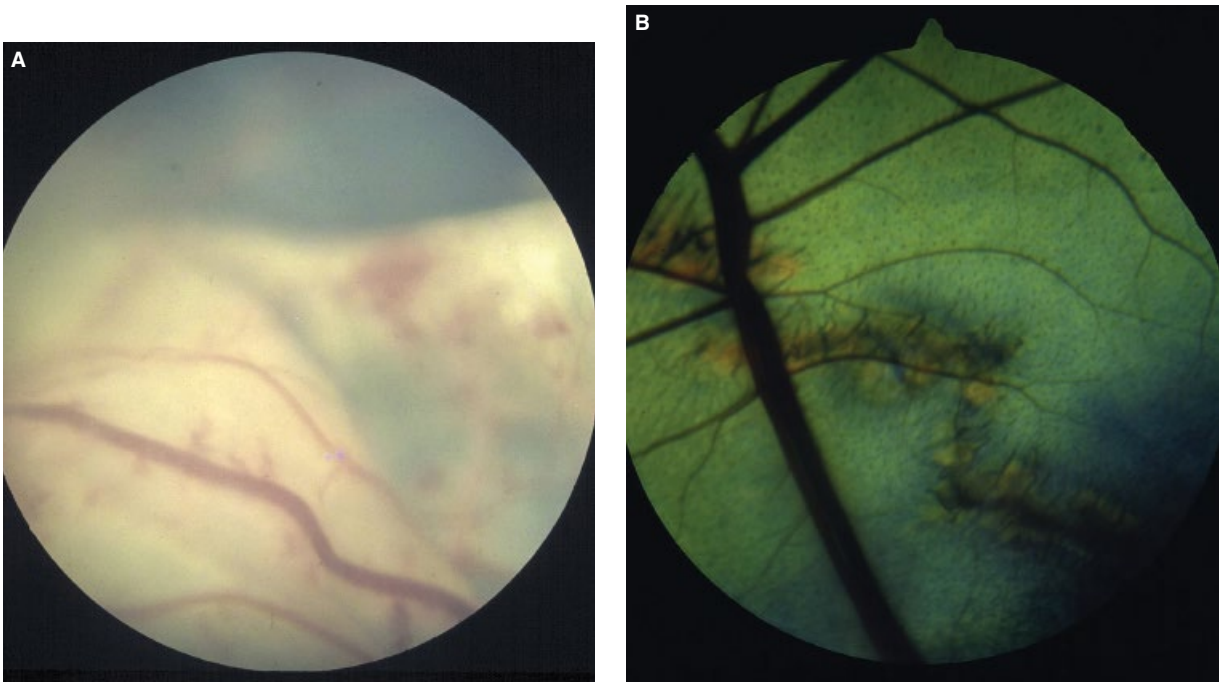


Figure 18.45 (A) Severe chorioretinitis, retinal hemorrhage, and exudative retinal detachment secondary to bovine thromboembolic meningoencephalitis in a feedlot steer. Courtesy of Glenn Severin. (B) Previous septic choroiditis secondary to bacteremia in a cow. Note the destruction of the tapetum fibrosum and limited pigmentation in these previous inflamed areas.

19

Neuro-ophthalmic Syndromes

Neuro-ophthalmic syndromes occur as the result of an insult to the segments of the nervous system that innervate or influence the functions of the eye and adnexa. Neuro-ophthalmology is a complex field, and although there are several syndromes recognized in veterinary medicine, their diagnosis and understanding can be difficult and incomplete. Patients with these syndromes present to either the ophthalmologist or the neurologist; often in large and referral veterinary practices these two disciplines will examine the patient, and combine their talents and experiences for the benefit of the patient. Optic neuritis that can be grouped with these syndromes is discussed in Chapter 13. Prognosis and treatment of neuro-ophthalmic conditions depends upon the location and etiology of the lesion.

Horner's Syndrome

Horner's syndrome is best understood and most frequently diagnosed in the dog. However, Horner's syndrome occurs in all of the most common domestic species and can indicate significant neurological disease.

Horner's Syndrome in the Dog

Horner's syndrome in the dog results from an interference of the sympathetic innervation to the eye and periocular structures. It results in miosis, ptosis (drooping of the upper eyelid), enophthalmos (eye appears recessed), and protrusion of the nictitating membrane (Figure 19.1). With these clinical signs, anisocoria (difference in pupil size between the two eyes) and a narrowed palpebral fissure also occur.

The clinical signs seen in Horner's syndrome occur when damage has been incurred anywhere along the three neuron pathways that dictate sympathetic control of the eye. Provocative pharmacologic testing (topical cocaine, 10% phenylephrine, and 1% hydroxyamphetamine) can be used to localize the lesion and differentiate between first and third order lesions.

The most frequent causes for Horner's syndrome in the dog are trauma, brachial plexus root avulsion, otitis media/interna, thoracic and intracranial tumors. Prognosis is determined by the etiology. Idiopathic Horner's syndrome occurs fairly commonly in the Golden Retriever. Signs and causes of Horner's syndrome are similar in the cat.

Horner's Syndrome in Large Animals

The clinical signs of Horner's syndrome in horses include miosis, ptosis, enophthalmia, and protrusion of the nictitating membrane (Figure 19.2). In addition, cutaneous facial and cervical hyperthermia and sweating occurs on the affected side. The causes of Horner's syndrome in horses include mycotic guttural pouch infections, trauma of the basisphenoid area, polyneuritis equi syndrome (cauda equina neuritis), equine protozoa myeloencephalitis, esophageal rupture, and after intravenous injections of xylazine, vitamin C/selenium, and phenylbutazone. Prognosis is determined by the contributing cause and response to therapy. Horner's syndrome in bovids manifests with ipsilateral anhydrosis (absence of sweating) of the nasal planum, rather than upregulated facial sweating as in the horse.

Facial Nerve Paralysis and Neuroparalytic Keratitis

Facial nerve paralysis is one of the more frequent neuro-ophthalmic disorders in the dog. Presentation is often based on nonophthalmic facial changes. The clinical signs of facial nerve paresis and/or paralysis include ear movement, drooping of upper and lower lips and commissures of the mouth, drooling, food collecting in the buccal area, and lack of nostril movements (Figure 19.3). The eyelids (especially the upper lid) fail to blink and protect the eye and often result in neuroparalytic keratitis (see also Figure 8.10). Touching the eyelids fails to induce the blink reflex. Because the lids do not function to protect the globe and distribute tear fluid, the globe is at risk for exposure keratitis and ulceration. Keratoconjunctivitis

Color Atlas of Veterinary Ophthalmology, Second Edition. Kirk N. Gelatt and Caryn E. Plummer.
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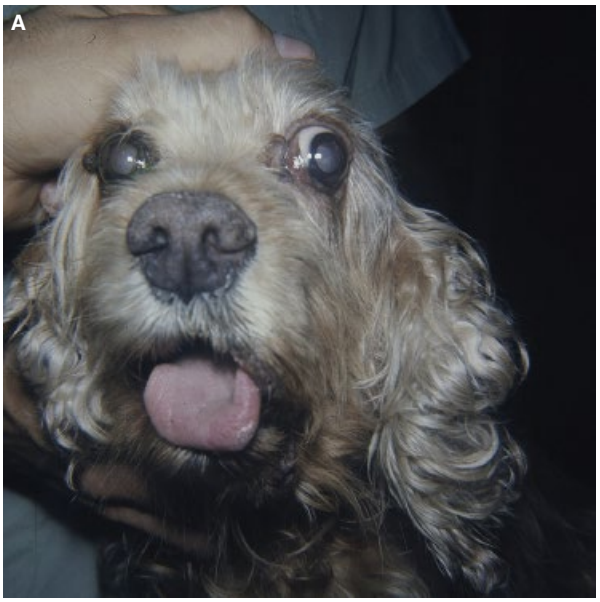




Figure 19.1 (A) Horner's syndrome in the dog is characterized by miosis, protrusion of the nictitating membrane, ptosis or drooping of the upper eyelid, and relative enophthalmia. (B) Idiopathic Horner's syndrome in a Golden Retriever. (C) Horner's syndrome in a cat.



Figure 19.2 Horner's syndrome in the foal is exhibited as miosis, protrusion of the nictitating membrane, ptosis or partial drooping of the upper eyelid, relative enophthalmia, and regional sweating of the area behind the orbit (note the wet hair). Guttural pouch infections and trauma are common causes of Horner's syndrome in the horse.



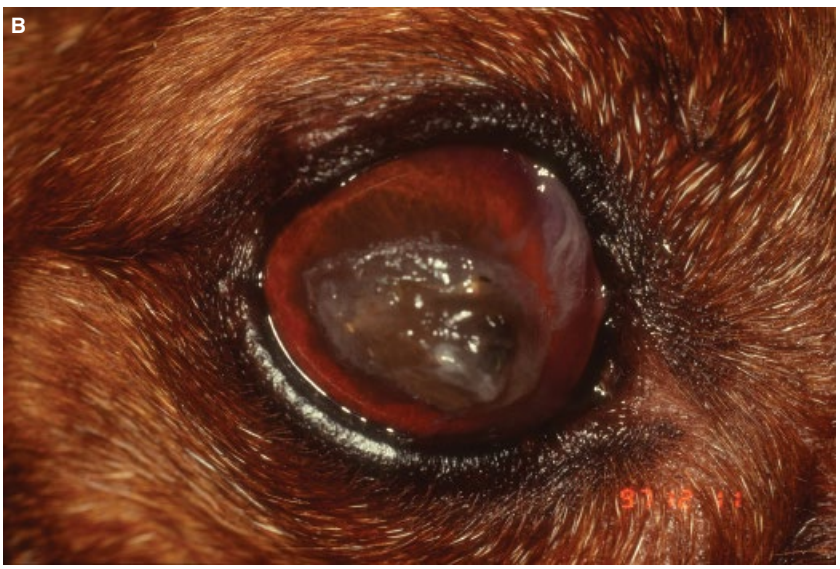


Figure 19.3 (A) Facial nerve paralysis in the dog is evidenced as an impaired or absent blink reflex, variable ptosis, and a drooping lower eyelid. Additional facial nerve abnormalities, such as drooping of the upper lip, may also be present. Manipulation of this dog's upper eyelid resulted in no attempt to close the palpebral fissure. (B) Neuroparalytic keratitis results from dysfunction of the facial nerve, impairment of the blink reflex, and reduced protection of the eyelid to the cornea and conjunctiva. The result in this dog is drooping of the lower eyelid and conjunctivitis, with the possibility of future keratitis or corneal ulceration.

sicca (KCS) can also be present, so tear production should be measured with Schirmer's tear test when the diagnosis of facial paralysis is made.

Facial nerve lesions are central (tumor, inflammation) or peripheral (trauma, otitis media or interna), with the latter having a better prognosis. Causes of facial nerve dysfunction in dogs and cats are surgical and nonsurgical trauma, neoplasia, and otitis media or interna. The condition can be idiopathic in some cases. In cattle, facial nerve paralysis has been associated with dehorning and *Listeria* sp. infections.

Short-term medical therapy includes topical lubricants and oral or topical pilocarpine to stimulate tear production when indicated. Long-term strategies to protect the cornea usually include temporary or permanent partial tarsorrhaphy. If facial nerve function does not improve and the globe is compromised, enucleation may be indicated.

Hemifacial Spasms in the Dog

Hemifacial spasms result in a narrowed palpebral fissure and an asymmetrical face secondary to the facial muscles' contractions (Figure 19.4). Horner's syndrome may also be present, and when combined with hemifacial spasms usually occurs secondary to otitis media. Treatment is directed at the underlying condition.

Neurotropic Keratitis and Fifth Nerve Paralysis

In neurotropic keratitis, the trigeminal (or its ophthalmic branch) nerve functions are impaired or absent, and the cornea and conjunctiva lose their sensation. As a result, the external ocular surfaces are unable to detect any insult and the eyelids fail to blink and adequately protect the outer eye; the result is the development of keratoconjunctivitis, and even corneal ulceration (see also Figure 8.11). Gentle touching of the cornea with a wisk of cotton or with the Cochet–Bonnet esthesiometer (quantitates corneal sensitivity in millimeters) fails to elicit a reflex response (Figure 19.5). The normal corneal reflex includes globe retraction, nictitans protraction, and a vigorous blink. If other branches of the trigeminal nerve are affected, muscle atrophy of the head will be noted concurrently.



Figure 19.4 In this dog, hemifacial spasms occurred, as evidenced by a narrowed palpebral fissure and distorted face.

Neurogenic Keratoconjunctivitis Sicca and Xeromycteria in the Dog (Ophthalmic Branch of the Trigeminal Nerve; Fifth Cranial Nerve)

In neurogenic KCS, the parasympathetic innervation to the lacrimal (and presumably the superficial gland of the nictitating membrane) is impaired resulting in KCS (Figure 19.6). Because of the pathways of these nerves, other clinical neural diseases, such as facial nerve paralysis, and Horner's syndrome may also be present. Causes include extraperiorbital sheath myositis, orbital abscesses and foreign bodies, caudal maxillary dental abscesses, iatrogenic injury from dental manipulations and orbital drainage procedures.

Treatment includes oral or topical pilocarpine which stimulates the hypersensitive pre- and post-ganglionic nerve endings to increase tear production. Topical cyclosporine is not usually successful for these patients as immune-mediated dacryoadenitis is not present. However, it may help with ocular surface inflammation.

Pupil Changes in the Cat

Each half (medial and lateral) of the iris in the domestic cat has distinct innervation, which contributes to the vertical slit orientation of the pupil. The lateral iridal sphincter is innervated by the malar nerve and the medial sphincter muscle is innervated by the nasal nerve. Both of these nerves are branches from the ciliary ganglion located within the orbit. Dysfunction of either nerve results in a "D" or reverse "D" shaped pupil.

Feline Hemidilated Pupil Syndrome

When one of the parasympathetic nerves innervating the feline pupil is impaired, that side of the pupil will dilate while the other side remains responsive (Figure 19.7).



Figure 19.5 (A) Testing for corneal sensitivity using a wisp of cotton. A vigorous blink, nictitans protrusion, and retraction of the globe into the orbit is the normal corneal reflex. (B) Neurotropic keratitis in a dog. Often, corneal ulceration is present, usually a vascular keratitis develops. (C) Temporalis and masseter muscle wasting in a dog with a trigeminal neuropathy.





Figure 19.6 (A) Neurogenic keratoconjunctivitis sicca (KCS) in the dog is caused by the interruption of the parasympathetic innervation to the tear glands, and these dogs also exhibit a dry nose. This relationship predicts a favorable response to pilocarpine therapy (causing a direct drug stimulation of the tear glands). Note the corneal pigmentation that has developed secondary to decreased tear production. (B) An example of a dog with neurogenic KCS with a dry nose. This case is more acute and has fewer signs of chronicity.



Figure 19.7 Feline hemidilated pupil occurs when either the lateral (malar) or the medial (nasal) parasympathetic innervation to the cat's iridal sphincter muscles is impaired. In this cat, the malar nerve is involved creating a reverse "D" pupil.



Figure 19.8 In Haw's syndrome in young cats variable bilateral protrusion of the nictitating membrane occurs. These protrusions, if severe, can impair vision. The condition is generally self-limiting.

This results in a hemidilated pupil that takes on a D-shaped or reverse D-shaped pupil. Most affected cats test positive for feline leukemia virus (FeLV). This condition should be distinguished from pupillary abnormalities associated with posterior synechiae, feline spastic pupil syndrome, and feline dysautonomia.

Haw's Syndrome in Cats

Haw's syndrome occurs in young cats as a bilateral protrusion of the nictitating membranes (Figure 19.8). Other ophthalmic and systemic abnormalities are usually absent, although there may be a history of a self-limiting diarrhea. The protrusions of the nictitans usually extend to cover part of the cornea, and in bright illumination (and the resultant miosis) visual impairment can be present.

The cause is unknown, although temporary inflammation of the superior cervical sympathetic ganglion has been proposed. The condition can persist for several weeks, eventually resolving with the gradual return of the nictitans into its normal position.

Treatment is not usually necessary unless visual impairment occurs. Either 1–2% epinephrine or 10% phenylephrine to stimulate contraction of the nictitans smooth muscles and retraction of the nictitans toward the medial canthus, or a mydriatic to dilate the pupil can be used.

Strabismus

Strabismus, or deviation of the globe from its normal axis, is usually characterized as either esotropia (convergence) or exotropia (divergence) in animals. The most common cause of acquired strabismus is trauma, especially in brachycephalic animals that have suffered a proptosis and damage to the medial rectus muscle. Orbital disease (neoplasia, abscesses or cellulitis, mucoceles) can also cause strabismus. Neuro-ophthalmic strabismus in animals is uncommon, but occurs most frequently in cats, where it is associated with inherited major neural pathways abnormalities.

Feline Strabismus

Strabismus in cats occurs most frequently in the Siamese and Himalayan breeds (Figure 19.9). The strabismus occurs as esotropia (the eyes are converged or crossed), and is often accompanied by nystagmus. The esotropia and nystagmus occur as a result of an abnormal route for the major retinal pathways with nearly all of these projections crossing to the pretectum and superior colliculus. As a result, binocular vision and stereopsis are denied, and eye is unable to fixate. The esotropia may be a compensatory effort to position the functional temporal visual fields to a more frontal position as well as enhance stereopsis. Siamese cats also possess lower percentages of Y to X-type retinal ganglion cells. Surgical attempts to correct estropia in cats have not been successful, in part because the extraocular muscles in the cat are very small.

Fibrosing Strabismus

First described in the Shar Pei breed, this condition is unilateral or bilateral, and typically occurs in young dogs (Figure 19.10). Other breeds reportedly affected include Irish Wolfhound, Akita, Golden Retriever, and Dalmatian. Extraocular muscles can be involved singly or in combination, but the medial rectus muscle appears



Figure 19.9 Feline strabismus or esotropia (convergence strabismus) most often occurs in the Siamese, Himalayan, and seal-point coated cats.



Figure 19.10 Fibrosing strabismus in the dog results in progressive esotropia, enophthalmia, and vision impairment to blindness. In this dog the strabismus is so severe that the entire cornea is hidden by the lower conjunctiva, and vision is absent. Courtesy of Ingrid Allgoewer.

most frequently affected, resulting in esotropia, enophthalmia, and visual impairment.

The initial lesion is inflammatory(myositis) followed by fibrosis of the extraocular muscle(s) as the disease develops chronicity. Therapy attempts to suppress the myositis, usually with systemic corticosteroids or immunomodulating medications, and is most effective when begun early in the course of the disease. Surgical resection of the fibrosed muscles can release the strabismus if medical therapy is ineffective.

Lateral Strabismus

Lateral strabismus (exotropia) occurs in primarily the brachycephalic breeds, such as the Boston Terrier, Pekingese, and English Bull Dog (Figure 19.11). The condition is unilateral or bilateral, and congenital. It seems nonprogressive and not associated with abnormal vision. Tonic eye reflexes (optokinetic eye movements) are usually normal. Possible paresis or an abnormally caudal insertion of the medial rectus muscle are possibilities. No treatment is recommended. Unilateral post-traumatic strabismus is very common in brachycephalic animals.

Convergence Strabismus (Esotropia) in the Horse

Strabismus is uncommon in horses, but may be more frequent in mules (Figure 19.12). Strabismus can be temporary in neonatal foals (ventromedial strabismus) and disappears at about weaning time. Strabismus in horses appears as excess "sclera show" and slanting of the pupil. The affected horse may demonstrate stumbling

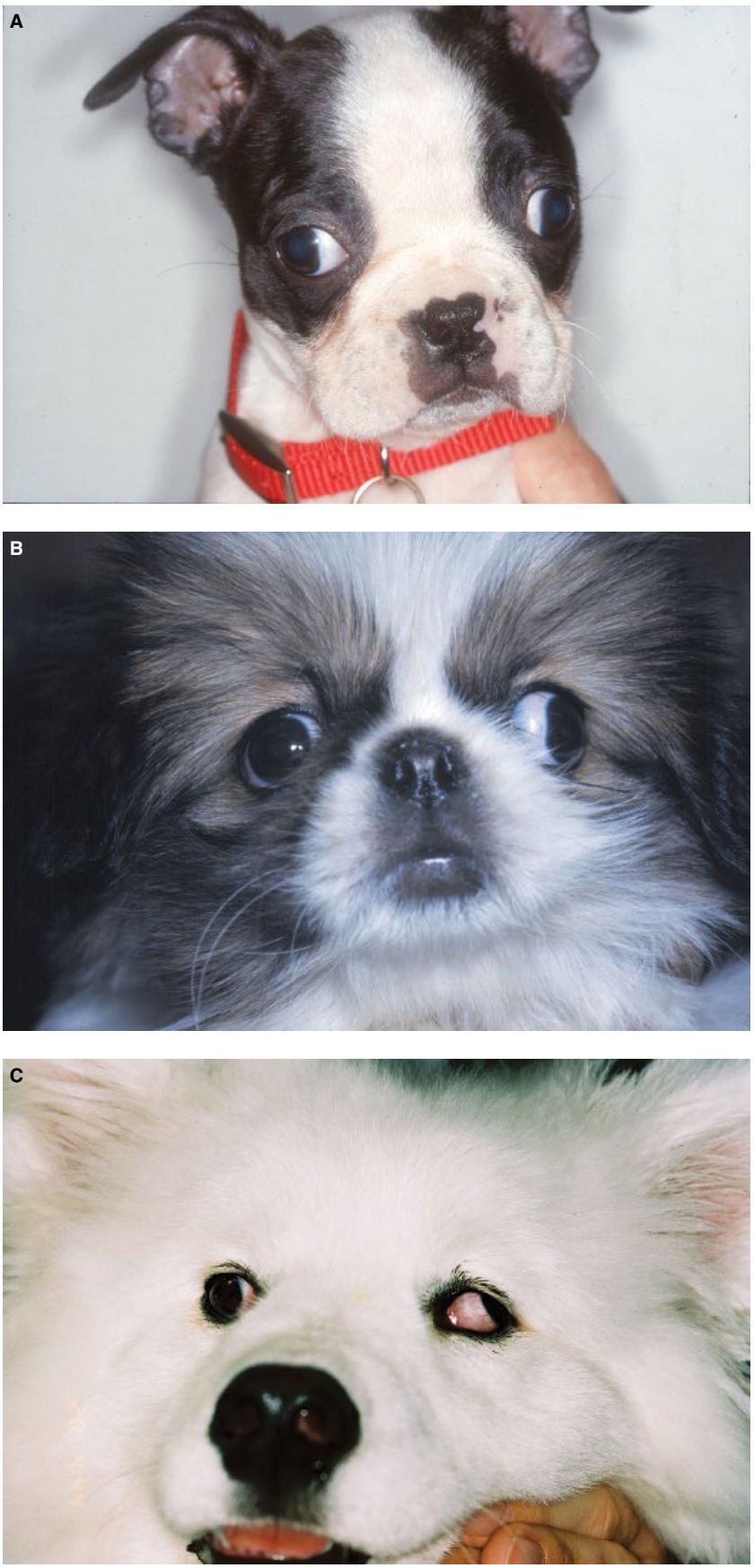


Figure 19.11 (A) Lateral strabismus (exotropia) in this Boston Terrier puppy has not been investigated, but in the brachycephalic breeds unilateral or bilateral exotropia (divergent strabismus) occurs. Clinical vision seems unaffected, and the condition is not progressive. (B) Unilateral exotropia in a Shih Tzu. (C) Unilateral strabismus and microphthalmia in a young Samoyed.



Figure 19.12 Convergence strabismus or esotropia that occurred acutely in an old horse. Note the large amount of exposed lateral sclera.

and increased nervousness. Strabismus in the Appaloosa breed has also been associated with inherited night blindness. This breed needs to be examined in a dark environment vision obstacle course as the horse in a lighted obstacle course will behave normally.

Strabismus in adult horses results from trauma or space-occupying masses of the orbit. Prognosis and treatment depends on the underlying disorder. The length of the extraocular muscles in the horse can be shortened or lengthened, using procedures described in humans.

Strabismus in Other Species

Deviations of the visual axis in cattle are uncommon, and most of those reported appear inherited in certain breeds. Bilateral esotropia and exophthalmia are inherited in the Jersey, Holstein, Brown Swiss, and Shorthorn breeds (Figure 19.13). Vision can be adversely affected, with affected animals demonstrating difficulties in unfamiliar environments. Breeding of affected animals is not recommended.



Figure 19.13 Bovine strabismus, as demonstrated in this Holstein cow, is usually esotropia and often associated with exophthalmia. In most instances the condition is inherited.

Ophthalmoplegia in the Dog

Ophthalmoplegia or a fixed globe (inability to move in any direction) results from lesions in the oculomotor nerve (third cranial nerve) and/or its nucleus (Figure 19.14). The oculomotor nerve innervates the majority of the extraocular muscles including the dorsal, medial, and ventral rectus muscles, the ventral oblique muscle, and levator palpebral muscle (assists in raising the upper eyelid). The oculomotor nerve also contains parasympathetic fibers that innervate the iridal sphincter muscles. The lateral rectus muscle (which is sometimes injured) is innervated by the abducens nerve. Ophthalmoplegia is divided into three clinical groups:

1) *External ophthalmoplegia:* no vision defects and dilated nonresponsive pupil;
2) *Internal ophthalmoplegia:* ventrolateral strabismus and/or ptosis of the upper eyelid;
3) *Complete ophthalmoplegia:* combination of external and internal ophthalmoplegia.



Figure 19.14 Internal ophthalmoplegia or cavernous sinus syndrome in a dog. Note the unequal pupils.

Internal ophthalmoplegia is the most frequent type and is often called cavernous sinus syndrome. Topical pilocarpine is used to demonstrate unusual sensitivity and results in miosis. Magnetic resonance imaging is used to image the cranial soft tissues. Treatment is directed to the cause, including targeted radiotherapy and/or chemotherapy for neoplastic masses.

Appendix A

Glossary: Frequently Used Veterinary Ophthalmology Terms

Common Ophthalmic Roots

blepharo – lid
cor – pupil
cyclo – ciliary body
dacryo – tears
hyal – vitreous
hyp – anterior chamber
irido – iris
kerato – cornea
ophthalmo – globe or eye
papilla – optic disc
phako/phaco – lens

Common Words

ablation Removal of globe or part of the eye (as destruction of the ciliary body).
ablepharon Partial or complete congenital absence of the eyelids.
accommodation Changes in the shape (and power) of the lens for seeing near objects. Limited in the domestic species.
amaurosis Total loss of vision.
amblyopia Reduced or partial loss of sight without detectable lesions in the eye and optic nerve.
aniridia Iris is absent clinically; some remnants can usually be demonstrated histologically.
anisocoria Unequal pupils.
ankyloblepharon Adhesion of the eyelids to each other. Physiologic in kittens and puppies for the first 10–14 days.
anophthalmia Congenital absence of the globes. Usually a severe hypoplasia of the globe (i.e., microphthalmia).
anterior uvea Iris and ciliary body.
aphakia Absence of the lens.
aphakic crescent Area of the pupil not covered by a luxated or displaced lens.
aqueous flare Aqueous humor with increased levels of proteins. Best demonstrated by a focal light beam projected across the anterior chamber or by a commercial laser flaremeter.
asteroid hyalosis Calcium lipid bodies and/or opacities suspended in the vitreous.
Bergmeister's papilla Remnants of posterior hyaloid artery appearing as small white projection from the center of the optic disc's surface.
Bernard–Horner syndrome (commonly called Horner's syndrome) Ptosis, miosis, enophthalmos, and protrusion of the nictitating membrane (also regional sweating in the horse).
biomicroscopy Microscopic examination of the various ocular structures.
blepharitis Inflammation of the eyelids. Can be diffuse or focal.
blepharospasm Contractions of the orbicularis oculi muscles.
blepharostenosis (blepharophimosis) Inability to open the palpebral fissure to a normal extent; smaller than normal palpebral fissure.
buphthalmos (hydrophthalmia or megaloglobus) Enlargement of the globe.
canaliculus Connects each lacrimal punctum to the nasolacrimal sac.
canthotomy Incision of either the lateral or medial canthus.
caruncle Small mass often covered with hair at the medial canthus in front of nictitating membrane.
cataract Opacity of the lens and its capsules.
chalazion Chronic and often granulomatous inflammation of the tarsal or meibomian glands.
chemosis Edema of the conjunctiva.
cherry eye Prolapse of the nictitans tear gland.
chorioretinitis Inflammation starting in the choroid and extending to the retina.

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choroid Posterior uvea; consists of pigmented cells, blood vessels, and in some animal species a special layer in the dorsal fundus, the tapetum lucidum (tapetum cellulosum in carnivores; tapetum fibrosum in herbivores).
choroiditis Inflammation of the choroid.
ciliary body Part of the anterior uvea; primary source of aqueous humor.
ciliary flush Hyperemia of the bulbar conjunctiva usually associated with intraocular inflammations.
cilium/cilia Another name for eyelash(es).
coloboma A defect of the eye. Commonly divided into typical (6 o'clock position) and atypical (all other positions).
conjunctiva Mucous membrane connecting eyelid margin to limbus of the globe. Divided into palpebral, fornix (or cul-de-sac), and bulbar.
conjunctivobuccostomy Surgically created fistula for the passage of tears from the ventral conjunctival fornix to the mouth.
conjunctivorhinostomy Surgically created fistula between the medial conjunctival fornix and the nasal cavity as an alternate drainage route for tears.
consensual pupillary reflex Constriction of the pupil when the opposite eye (retina) is stimulated. Sometimes called the indirect pupillary response.
corectasia Dilation of pupil.
corectopia Off-center pupil.
coreoplasty Construction of a new pupil.
corneal dystrophy Bilateral inherited corneal disease characterized by the deposition of materials, usually lipid substances. Distinguish from the unilateral corneal degeneration secondary to other corneal diseases.
corpora nigrum/nigra More recent term is granula iridica. Pigmented irregular mass on the dorsal and occasionally the ventral pupillary margin of the iris in herbivores.
cryoextraction Removal of the lens with ultra-cold devices.
cul-de-sac Junction of the palpebral and bulbar conjunctiva; also termed fornix.
cyclitis Inflammation of the ciliary body.
cyclocryothermy Application of ultra-cold probe on the sclera to destroy the ciliary body epithelium and reduce aqueous humor formation.
cyclodialysis Surgical fistula from anterior chamber beneath the iris and ciliary body to exit thorough the sclera.
cycloplegia Usually drug-induced paralysis of the ciliary body and the resultant loss of accommodation.
dacryoadenitis Inflammation of the lacrimal gland.
dacryocystitis Inflammation of the nasolacrimal sac.
dacryocystorhinography Radiopaque study of the nasolacrimal apparatus.
dermoid Congenital mass involving the eyelids, conjunctiva, nictitating membrane, and/or cornea consisting of normal skin and its components. Sometimes called choristoma.
descemetocele A deep corneal ulcer characterized by exposure and possible protrusion of Descemet's membrane. Does not stain with topical fluorescein.
dialysis Retinal tear at the ora ciliaris retinae with separation of the neurosensory retina from the retinal pigment epithelium.
diathermy Application of heat to various parts of the eye (especially the ciliary body).
diopter Refracting power of a lens whose focus in 1 m (D = 1/meter).
disc Refers to the optic nerve head.
discission Incision of a structure, usually the anterior capsule of the lens.
distichiasis Presence of abnormal eyelashes (cilia).
dyscoria Irregular pupil.
ectasia Protrusion of the cornea or sclera.
ectopic cilium Aberrant cilia emerging from the dorsal palpebral conjunctiva.
ectopic lentis Luxation or displacement of the lens.
ectropion Outward folding of the eyelid and its margin.
electroretinography Recording of retinal electrical potentials generated by a rapid change in illumination.
emmetropia Normal eye in refraction. Image focused on the retina with the eye at rest.
endophthalmitis Inflammation of the globe involving the inner structures.
enophthalmos Recession of the globe in the orbit.
entropion Infolding of the eyelid and its margin.
enucleation Surgical removal of the globe.
epilation Manual removal by forceps of cilia (eyelashes).
epiphora Overflow of tears onto the medial canthus or eyelid margin. Can signal overproduction and/or inadequate drainage.
equine recurrent uveitis Recurrent (or chronic) iridocyclochoroiditis of horses.
esotropia Convergent strabismus.
evisceration Removal of structures of the eye leaving only the sclera or cornea and sclera.
exenteration Removal of all contents from the orbit.
exophthalmos Protrusion of the globe; eyelids can usually still function and cover the cornea.
exotropia Divergent strabismus.
external hordeolum (stye) Inflammation and frequent abscessation of the glands of Zeis and Moll.

flare Increase in protein levels in the aqueous humor with a positive Tyndall phenomenon.
fluorescein Resorcinolphthalein: water-soluble compound that yields a bright green fluorescence. Used for detection of corneal ulcers, chorioretinal circulation time, integrity of the blood–aqueous barrier, and patency of the nasolacrimal apparatus.
glands of Moll Apocrine (sweat) glands near the eyelid margin. When inflamed – stye (or external hordeolum).
glands of Zeiss Sebaceous glands of the eyelid margins. When inflamed – stye (or external hordeolum).
glaucoma Abnormal increase in intraocular pressure and optic neuropathy.
gonioscopy Examination of the anterior chamber angle (iridocorneal angle and sclerociliary cleft) using a special contact lens.
granula iridica Pigmented mass of the edge of the upper and lower pupil in herbivores.
haw Lay term for the nictitating membrane.
hemeralopia Day-blindness.
heterochromia iridis Two or more colors in an iris, or between two irides in an individual.
hordeolum (internal) Also called chalazion. Inflammation of the meibomian gland; an abscess or granuloma. External hordeolum – inflammation of the glands of Moll and Zeiss (also called stye).
Horner's syndrome Sympathetic denervation of the eye and orbit. Clinical signs include miosis, nictitating membrane protrusion, enophthalmos, ptosis, and in some animal species regional vascular hyperemia and sweating.
hyaloid Vitreous.
hydrophthalmos Congenital globe enlargement. A dated term – buphthalmos – is preferred.
hyperopia Farsightedness. Objects seen distinctly when at a distance. Image is not in focus at the level of the retina (actually in focus behind the retina).
hyphema Hemorrhage in the anterior chamber.
hypopyon Purulent exudate (pus) in the anterior chamber.
hypotony Abnormally low intraocular pressure of usually less than 5 mmHg.
intraocular lens Artificial lens placed after cataract surgery to replace preoperative lens optics.
iridectomy Excision of the iris. Divided into basal, peripheral, and sphincterectomy.
iridencleisis A surgical procedure used to treat glaucoma consisting of a pillar of iris (functions as a wick) through a scleral incision.
iridocorneal angle The angle created by the iris and cornea. In nonprimate mammals, the angle is composed of the basal iris, ciliary body (ciliary cleft), and the sclera. Also termed anterior chamber angle and/or filtration angle.
iridocyclitis Inflammation of iris and ciliary body. Also called anterior uveitis.
iridodonesis Tremulousness of the iris, usually associated with lens instability, intraocular lens, or a small lens. Observed with lens luxation, aphakia, hypermature cataracts, and cataract resorption.
iridoplegia Pupillary dilation and paralysis of the iridal sphincter muscles.
iridotomy Incision of the iris.
iris bombé Focal or generalized bulging of the iris, indicating impairment of pupillary passage of aqueous humor. Associated with focal or annular posterior synechiae.
iritis Inflammation of the iris.
keratectomy Excision (usually superficial) of the cornea.
keratitis Inflammation of the cornea.
keratocentesis Puncture of the cornea and removal of aqueous humor.
keratoconjunctivis sicca Inflammation of the cornea and conjunctiva caused by a deficiency of the aqueous component of the tears (precorneal film).
keratoconjunctivitis Inflammation of the cornea and conjunctiva.
keratoconus (anterior) Conical anterior protrusion of the center of the cornea.
keratoglobus Enlarged cornea, usually with buphthalmos.
keratoplasty Corneal grafts. Divided into lamellar (superficial) and complete (full-thickness).
keratotomy Incision of the cornea.
Krause's glands Accessory lacrimal glands of the upper and lower conjunctiva fornix.
lacrimation Used clinically to imply excessive rates of tear formation.
lagophthalmos Inability to close the palpebral fissure; usually the inability of the upper eyelid to close.
lenticular Lens.
lenticulodenesis Instability of the lens associated with zonulary loss.
leukoma A dense corneal scar.
limbus The transitional zone between the cornea and sclera. Sometimes called the "blue zone."
luxation Dislocation of a structure in ophthalmology. The term is used for the globe (proptosis) and dislocation of the lens.
manometry Measurement directly of intraocular pressure by needle inserted into the anterior chamber or vitreous body.
megalocornea Enlarged cornea, usually with buphthalmos.

microcornea Small or hypoplastic cornea.
microphakia Abnormally small lens.
microphthalmia Abnormally small eye.
miosis Contraction of the pupil.
miotic Drug that causes constriction of the pupil. Two types are available: direct (parasympathomimetic) and indirect (anticholinesterase).
Mittendorf's dot Hyaloid vascular remnant at lens posterior pole.
morgagnian cataract Type of hypermature cataract with liquified cortex and solid nucleus.
motility Movement of the globe.
mydriatic Drug that produces dilation of the pupil. Two types are available: parasympatholytic and sympathomimetic.
myopia Nearsightedness. Objects seen distinctly when close. Image is not in focus at the level of the retina (actually in focus in front of the retina).
nevus Focal pigmented area in the iris, choroid, and so on.
nictitating membrane Also called third eyelid and membrana nictitans.
nyctalopia Night-blindness.
nystagmus Oscillation of the globe.
oculi unitas (OU) Both eyes.
oculus dexter (OD) The right eye.
oculus sinister (OS) The left eye.
ophthalmia neonatorum Conjunctivitis in the neonate with physiologic ankyloblepharon (in kittens and pups).
ophthalmoplegia Paralysis of the extraocular muscles. Divided into internal and external types.
ophthalmoscopy Examination of the ocular fundus by an ophthalmoscope (direct and indirect methods).
pannus Invasion of the cornea with subepithelial neovascularization and pigmentation.
panophthalmitis Inflammation of the globe involving all layers of the globe.
papilla Another term for the optic nerve head (or disc).
papilledema Edema of the optic disc or papilla.
papillitis Inflammation of the optic disc or papilla.
periodic ophthalmia Older term for equine recurrent uveitis.
peripheral anterior synechia Inflammatory attachments of the basal iris and the peripheral posterior cornea.
persistent pupillary membrane Congenital remnants of the prenatal pupillary vascular membrane that extend from the collarette region of the iris to the cornea, lens, or other areas of the iris.
phacodonesis Instability of the lens, usually secondary to the loss of zonulary attachments.
phacoemulsification Type of cataract surgery in which the lens matter is emulsified *in situ* using high frequency ultrasound waves.
photophobia Increased sensitivity to light.
photopic Under light or bright illumination conditions, as with photopic vision or electroretinography.
phthisis bulbus Atrophy of the globe with low intraocular pressure, usually associated with trauma and inflammation.
polycoria Two or more pupils in an eye. False (no sphincter muscle) and true types (with sphincter muscle). Differentiate from iris atrophy.
proptosis Forward displacement of the globe out of the orbit.
provocative tests Corticosteroid, water, mydriatic, and darkroom. Procedures to demonstrate tendency toward glaucoma in an eye.
pterygium Invasion of the cornea by the bulbar conjunctiva. Not documented in animals.
ptosis Drooping of the upper eyelid.
pupil Opening in the central iris.
rose Bengal Topical ophthalmic stain used to outline dead and degenerating corneal and conjunctival cells.
rubeosis iridis Neovascularization of the iris, with frequent involvement of the anterior chamber angle, pupil, anterior surface of the lens, and ciliary body processes.
scleritis Inflammation of the sclera.
sclerotomy Incision of the sclera.
scotopic Dark conditions, as with vision or electroretinography.
sequestrum Necrotic stromal tissue; usually refers to condition in the cat affecting the cornea.
sicca Dryness.
spherophakia Round or spherical-shaped lens.
squint Strabismus.
staphyloma Protrusion of the cornea and/or sclera lined with uveal tissue. Can be congenital, traumatic, or surgical.
strabismus (heterotropia) Visual axes of the eyes are not parallel. Common types: (i) strabismus convergens (internal squint), and (ii) strabismus divergens (external squint).
striate keratopathy Irregular linear lines in the cornea usually associated with glaucoma (breaks) and phthisis bulbi (folds), and changes in Descemet's membrane.
stye Inflammations of the glands of Moll and Zeiss.
subluxation Associated with partial loss of the lens zonule; partial lens displacement.
symblepharon Adhesion of the eyelid or both eyelids to the conjunctiva and/or cornea.
synchysis scintillans Liquified vitreous with cholesterol crystals.
synechia Adhesions of the iris to cornea (anterior synechia), iris to lens (posterior synechia), and in the iridocorneal angle (peripheral anterior synechia).
syneresis Liquefaction of the vitreous.

tapetum lucidum A special reflective layer within the dorsal choroid providing additional reflection of light permitting double retinal stimulation of the retina. Tapetum cellulosum in carnivores;tapetum fibrosum in herbivores.

tarsorrhaphy Temporary (by sutures) or permanent apposition of part (partial) or all (complete) eyelids.

tear film breakup The development of dry spots (measured in seconds) in the tear film, as observed by biomicroscopy with the preocular film stained with fluorescein.

Tenon's capsule Fascia bulbus.

tonography Continuous measurement of intraocular pressure to estimate pressure-sensitive aqueous humor outflow (in µL/mmHg/minute).

tonometry Measurement of intraocular pressure. Divided into indentation, applanation, and rebound.

transillumination Passage of light through tissues. Can facilitate solid and hollow tissue masses.

trichiasis Contact of hair with the eye; as in entropion or nasal fold trichiasis.

uvea Iris, ciliary body, and choroid. Anterior uvea: iris and ciliary body. Posterior uvea: choroid.

uveitis Inflammation of the uvea tract. Anterior uveitis – inflammation of the iritis and ciliary body; posterior uveitis – inflammation of the choroid. Panuveitis – inflammation of the iris, ciliary body, and choroid.

vibrissa Large tactile hair about the eyelids and face of large animals.

Wolfring gland Accessory lacrimal gland of the dorsal palpebral conjunctiva.

xerophthalmia Same as keratoconjunctivitis sicca or xerosis. Dryness of the cornea and conjunctiva.

zonules Suspensory ligaments connecting the lens periphery (equator) to the ciliary body.

zonulolysis Enzymatic (alpha chymotrypsin) or surgical transection of the lens zonules.

Appendix B

Eye Diseases in the Brachycephalic Breeds

Breeds Predisposed

Boston Terrier, Japanese Chin, Lhasa Apso, Pekingese, Pug, Shih Tzu, and others.

Anatomic Characteristics

Very shallow orbit; very short muzzle; macropalpebral fissure; long upper eyelid; very prominent globe (exophthalmia); nasal folds; caruncle hairs.

Physiologic Characteristics

Central corneal exposure; thin central precorneal film; lower sensitivity of central cornea (less corneal nerve endings in central cornea); blink rate suspicious; incomplete palpebral closure during sleep; often central rose Bengal retention by central cornea. In older age, lower tear production.

Predisposed Eye Diseases and Therapy

Macropalpebral Fissure

With a wider palpebral fissure, sudden forces applied to the head and orbit can result in the forward movement of the globe through the palpebral fissure. The eyelids quickly constrict, but the globe is beyond the fissure, and this action "traps" the globe in this forward position. Retrobulbar hemorrhage, edema, traction on the optic nerve, and possible tearing of the retrobulbar muscles can occur. As the medial rectus muscle is the shortest, it is at risk for transection (usually just behind the insertion of the muscle to the globe). Within minutes, the proptosed globe begins to dry. Corneal drying results in acute corneal necrosis (malacia). (For additional details including therapy see Chapter 4.)

Nasal Folds

Trichiasis from upper nasal fold hairs touch the nictitans, conjunctiva, and the cornea. Low grade irritation incites lacrimation; corneal irritation (superficial corneal vascularization, superficial keratitis), and eventually corneal pigmentation. If trichiasis is severe, corneal ulceration can develop. (For additional details including therapy see Chapter 5.)

Medial Lower Entropion

The inversion of the medial lower eyelid can result in closure of the lower lacrimal punctum and cause epiphora. Chronic spillage of the tears can result in blepharitis and pigmentation of the medial canthal region. (For additional details see Chapter 5.)

Caruncle Hairs

Not all brachycephalic dogs have hairs growing from the caruncle at the base of the nictitans and in the medial canthus. If these coarse hairs extend onto the medial canthus and beyond the palpebral fissure, they serve as a bridge for the tears to continuously moisten the medial canthal region resulting in chronic dermatitis. (For additional details including therapy see Chapter 5.)

Prominent Globe – Exophthalmos and Exposure

The collective anatomic abnormalities can have a significant adverse effect on the cornea and especially the central cornea. Onset of corneal ulceration is acute or chronic. Once the corneal epithelia has been traumatized and lost, the corneal stroma is entered by harmful bacteria or other infectious agents (often normal inhabitants of the conjunctival sac), and results in destruction of the stroma by these agents and proteases. The central cornea is at special jeopardy because it is already compromised, and the healing process requires additional time to migrate to its center. (For additional details including therapy see Chapter 8.)

Other Contributing Factures

In older dogs, vitreous syneresis, retinal giant tears, and retinal detachments occur, especially in the Shih Tzu and Boston Terrier breeds. How these intraocular diseases relate to the "brachycephalic ophthalmic syndrome" has not been documented.

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Appendix C

Inherited Cataracts in the Dog

Part 1: Inherited Canine Cataracts

Breed	Mode of inheritance	Age of onset
Afghan Hound	Autosomal recessive	Congenital to 2 years
American Cocker	Autosomal recessive/polygenic	Congenital and juvenile (1.5–7 years)
Beagle	Incomplete dominant	Congenital to 4 months
Boston Terrier	Autosomal recessive	Congenital to 4 months
Chesapeake Bay Retriever	Incomplete dominant	6 months to 7 years
Cavalier King Charles Spaniel	Unknown	Congenital
German Shepherd	Incomplete dominant Autosomal recessive	Congenital to 2 years
Golden Retriever	Autosomal recessive	Congenital
Irish Setter	Unknown	4.5 months to 2 years
Labrador Retriever	Dominant	Juvenile
Labrador Retriever	Autosomal recessive	Congenital
Miniature Poodle	Autosomal recessive	Juvenile
Miniature Schnauzer	Autosomal recessive	Congenital
Old English Sheepdog	Autosomal recessive	Congenital
Siberian Husky	Recessive (suspected)	4–18 months
Staffordshire Bull Terrier	Autosomal recessive	6 months +
Standard Poodle	Autosomal recessive	Congenital to 2 years
Toy Poodle	Autosomal recessive	Juvenile
Welsh Corgi	Recessive (suspected)	Congenital to 2 years
Welsh Springer	Autosomal recessive	Congenital to 8 weeks
West Highland White Terrier	Autosomal recessive	Congenital to 6 years

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Part 2: Characteristics of Inherited Canine Cataracts

Breed	Characteristics	Progression	Associated ocular diseases
Afghan Hound	Equatorial cortical vacuoles	Rapid	None
American Cocker Spaniel	Congenital – nuclear and cortical Juvenile – posterior axial	Moderate to slow	None
Beagle	Posterior axial to diffuse	Moderate	None
Boston Terrier	Nuclear and posterior sutures	Slow	None
Chesapeake Bay Retriever	Nuclear and cortical	Variable	None
Cavalier King Charles	Nuclear and posterior cortex	Unknown	Posterior lenticonus
German Shepherd	Cortical Nuclear	Slow	None
Golden Retriever	Homozygote – cortical and nuclear diffuse Heterozygote – posterior axial subcapsular triangular	Progressive Non-Progressive	None None
Irish Setter	Cortical	Rapid	None
Labrador Retriever	Cortical Posterior axial subcapsular triangular Non-progressive	Moderate to rapid	Retinal dysplasia (?)
Miniature Poodle	Cortical	Moderate to rapid	PRA, None
Miniature Schnauzer	Posterior subcapsular, cortex and nuclear	Rapid	Microphthalmos
Old English Sheepdog	Cortical and nuclear	Moderate to rapid	Multiple
Siberian Husky	Posterior subcapsular and equatorial	Slow	None
Staffordshire Bull Terrier	Nuclear Posterior sutures and cortex	Slow	None
Standard Poodle	Equatorial cortex	Moderate	None
Toy Poodle	Cortical	Moderate	PRA
Welsh Corgi	Posterior subcapsular or equatorial	Slow	None

PRA, progressive retinal atrophy.

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