

Pinnal Alopecia	522
Ear Margin Seborrhea	523
Sebaceous Adenitis	523
Auricular Hematomas	524
Equine Aural Plaques	524
Necrotic Ear Syndrome in Swine	524
Miscellaneous Diseases	525

## OTITIS EXTERNA 527

## OTITIS MEDIA AND INTERNA 531

## TUMORS OF THE EAR CANAL 534

Ceruminous Gland Tumors	535
Nasopharyngeal Polyps	535

# OPHTHALMOLOGY

## PHYSICAL EXAMINATION OF THE EYE

The initial examination of the eye should assess symmetry, conformation, and gross lesions; the eye should be viewed from 2–3 ft (~1 m) away, in good light, and with minimal restraint of the head. The anterior ocular segment and pupillary light reflexes are examined in detail with a strong light and under magnification in a darkened room. Baseline tests like the Schirmer tear test, fluorescein staining, and tonometry (intraocular pressure measurement) may be followed by ancillary tests such as taking corneal and conjunctival cytology and cultures, everting the eyelids for examination, and flushing the nasolacrimal system to evaluate the external parts of the eye, including the anterior segment. Diseases of the vitreous and ocular fundus are evaluated by direct and indirect ophthalmoscopy (usually performed after inducing mydriasis) and vision testing (menace reflex, obstacle course, dazzle reflex, etc).

Schirmer tear tests and cultures should be performed before topical anesthetic is instilled. Fluorescein staining and eversion of the eyelids do not require topical anesthesia, but tonometry, examination of the bulbar surface of the nictitating membrane, conjunctival and corneal cytology, gonioscopy, and lavage of the

nasolacrimal system usually do. To avoid false-positives, samples for corneal and conjunctival cytology that will be analyzed by fluorescent antibody procedures should be collected before topical fluorescein staining.

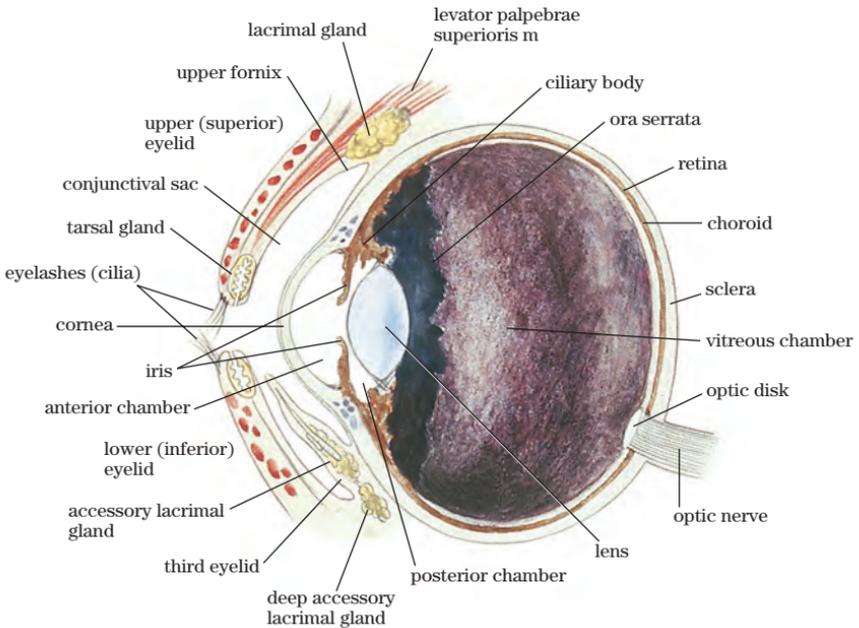
Special examinations such as slit-lamp biomicroscopy, ultrasonography, fluorescein angiography, and electroretinography may require sedation or local, regional, or general anesthesia, depending on the species.

## EYELIDS

The eyelids consist of four parts: 1) the outer very thin and mobile skin; 2) the strong and encircling orbicularis oculi muscle anchored at the medial canthus; 3) the thin and poorly developed fibrous tarsus, which contains the sebaceous Meibomian glands and attaches the lid to the bony orbital rim; and 4) the thin and flexible palpebral conjunctiva, which continues to the conjunctival fornix or conjunctival cul-de-sac. Eyelid disorders may be associated with facial and orbital abnormalities, specific breeds, and adjunct skin diseases, as well as with many systemic diseases.

### Conformational Abnormalities

**Entropion** is an inversion of all or part of the lid margins that may involve one or both



The eye and eyelids, median section. *Illustration by Dr. Gheorghe Constantinescu.*

eyelids and the canthi. It is the most frequent inherited eyelid defect in many canine and ovine breeds and may also follow cicatrix formation and severe blepharospasm due to ocular or periocular pain. Inversion of the cilia (or eyelashes) or facial hairs causes further discomfort, conjunctival and corneal irritation, and if protracted, corneal scarring, pigmentation, and possibly ulceration. Early spastic entropion may be reversed if the inciting cause is quickly removed or if pain is alleviated by everting the lid hairs away from the eye with mattress sutures in the lid, by subcutaneous injections (eg, of procaine penicillin) into the lid adjacent to the entropion, or by palpebral nerve blocks. Temporary stay sutures or surgical staples left in place for 2–3 wk may be used to treat entropion in very young puppies. Established entropion usually requires surgical correction.

**Ectropion** is a slack, everted lid margin, usually with a large palpebral fissure and elongated eyelids. It is a common bilateral conformational abnormality in a number of dog breeds, including the Bloodhound, Bull Mastiff, Great Dane, Newfoundland, St. Bernard, and several Spaniel breeds. Contracting scars in the lid or facial nerve paralysis may produce unilateral ectropion in any species. Conjunctival exposure to

environmental irritants and secondary bacterial infection can result in chronic or recurrent conjunctivitis. Topical antibiotic-corticosteroid preparations may temporarily control intermittent infections, but surgical lid-shortening procedures are often indicated. Mild cases can be controlled by repeated, periodic lavage with mild decongestant solutions.

**Lagophthalmos** is an inability to fully close the lids and protect the cornea from drying and trauma. It may result from extremely shallow orbits (in brachycephalic breeds), exophthalmia due to a space-occupying orbital lesion, or facial nerve



Bilateral entropion, Shar Pei puppy. *Courtesy of K. Gelatt.*

paralysis. Corneal scarring, pigmentation, and ulceration usually result. Unless the cause can be corrected, the therapy is frequent topical lubrication and surgical shortening or closure of the lateral canthi either temporarily or permanently. Excessive nasal skin folds and facial hair may aggravate the damage caused by lagophthalmos.

**Abnormalities of the cilia** include extra (distichia) or misdirected eyelashes on the lid margin. Epiphora, corneal vascularization, and corneal ulceration and scarring may result. In many instances, anomalous cilia are very fine, the same color as the surrounding eyelid hair, and result in neither clinical signs nor damage. However, ectopic cilia protruding through the dorsal palpebral conjunctiva can cause profound pain. If the corneal or conjunctival damage is caused by the extra cilia, excision, cautery, or cryotherapy of the cilia follicles is indicated. Anomalies of the cilia are common and probably inherited in some dog breeds but are rare in other animal species. Distichiasis is not treated unless corneal and/or conjunctival disease results. Successful removal of distichia requires destruction of the follicular base of the eyelids while not injuring the eyelid margin. The most popular method is cryotherapy applied at the base of the cilia beneath the palpebral conjunctiva at the eyelid margin. Depigmentation of the eyelid margin may result after cryotherapy but usually re-pigments in the subsequent months. Inadequate cryotherapy can result in distichia recurrence.

## Inflammation

**Blepharitis** (inflammation of the eyelids) can result from extension of a generalized dermatitis, conjunctivitis, local glandular infections, or irritants such as plant oils or solar exposure. The lids can be the original site of involvement for agents that lead to a generalized dermatitis. Dermatophytes (all species), *Demodex canis* (dogs), *D cati* or *D gatoi* (cats), and bacteria such as staphylococci often are involved. The mucocutaneous junction of the skin and conjunctiva can be the site of lesions of immune-mediated diseases such as pemphigus. Skin scrapings, cultures, and biopsies may be required for an accurate diagnosis. Localized glandular infections may be acute or chronic (stye [glands of Zeis and Moll] and chalazion [Meibomian glands]).

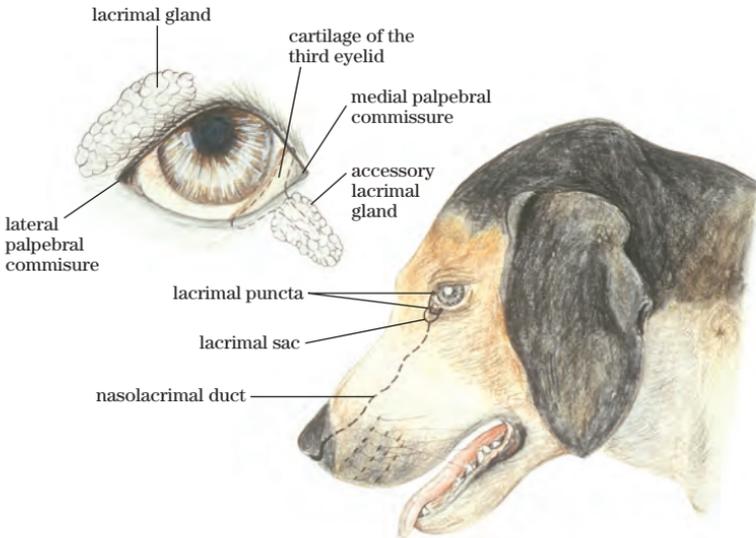
In generalized blepharitis, systemic therapy often is indicated in addition to topical treatment. Supportive therapy of hot packing and frequent cleansing is indicated in acute cases. Nonophthalmic preparations can be used to treat the eyelids, but caution in application is indicated to avoid corneal and conjunctival contact and possible irritation.

## NASOLACRIMAL AND LACRIMAL APPARATUS

The tear production and drainage system is vital for health of the outer eye. Tear glands within the orbit (lacrimal and in some species Harder gland) as well as the superficial tear gland of the nictitating membrane (third eyelid) produce the collective preocular or precorneal tear film. This film consists of three layers: outer lipid (from the Meibomian glands), middle aqueous layer (from lacrimal and third eyelid glands), and deep layer (mucus) from the goblet cells within the conjunctiva.

The tear drainage system consists of two lacrimal puncta (except in the rabbit and pig, which have only one punctum), two canaliculi, the lacrimal sac (within the bony lacrimal fossa), and the long and often tortuous lacrimal duct (to empty tears within the forward nasal cavity).

Hypertrophy, inflammation, and prolapse of the gland of the nictitating membrane (**cherry eye**) is common in young dogs and certain breeds (eg, American Cocker Spaniel, Beagle, Lhasa Apso, Pekingese, English Bulldog). In the acute stage, the red glandular mass swells and protrudes over the leading margin of the nictitans, and there is a mucopurulent discharge. Although the swelling may recede for short periods, the gland eventually often remains prolapsed. Because it is a major tear gland, it should be preserved if possible; the gland should be replaced and anchored with sutures to the orbital rim, periorbital fascia, or nictitans cartilage, or covered with adjacent mucosa (envelope or pocket techniques). Partial excision should be avoided. Complete excision may predispose to keratoconjunctivitis sicca (*see* p 492) in 30%–40% of dogs in later life. Surgical or medical resolution of cherry eye still predisposes ~20% of these dogs to future keratoconjunctivitis sicca. Therefore, these dogs should be monitored for several years after undergoing surgery.



Lacrimal apparatus, dog. Illustration by Dr. Gheorghe Constantinescu.

**Dacryocystitis** (inflammation of the lacrimal sac) usually is caused by obstruction of the nasolacrimal sac and proximal nasolacrimal duct by inflammatory debris, foreign bodies, or masses pressing on the duct. It results in epiphora, secondary conjunctivitis refractory to treatment, and occasionally a draining fistula in the medial lower eyelid. Irrigation of the nasolacrimal duct reveals an obstruction of the duct, reflux of mucopurulent discharge from the lacrimal puncta, or both. Radiographs of the skull after injection of contrast material into the duct (dacryocystorhinography) may be necessary to establish the site, cause, and prognosis of chronic

obstructions. Therapy consists of maintaining patency of the duct and instilling topical antibiotic solutions. Tubing (polyethylene or silicone) or 2-0 monofilament nylon suture temporarily catheterized in the duct may be necessary to maintain patency during healing. When the nasolacrimal apparatus has been irreversibly damaged, a new drainage pathway can be constructed surgically (conjunctivorhinostomy or conjunctivorolostomy) to empty tears into the nasal cavity, sinus, or mouth.

**Imperforate lacrimal puncta** are an infrequent cause of epiphora in young dogs. In foals, atresia of the nasal (distal) end of the nasolacrimal duct is a common



Bilateral inflammation and prolapse of the nictitating membrane (cherry eye), young Boston Terrier. Courtesy of K. Gelatt.



Exposure of inflamed, prolapsed nictitating membrane using thumb forceps, Boston Terrier. Courtesy of K. Gelatt.

cause of early epiphora and chronic conjunctivitis. In calves, multiple openings of the nasolacrimal duct may empty tears onto the lower eyelid and medial canthus, causing chronic dermatitis. Therapy in dogs and foals consists of surgically opening the blocked orifice and maintaining patency by catheterization for several weeks during healing.

**Keratoconjunctivitis sicca (KCS)** is due to an aqueous tear deficiency and usually results in persistent, mucopurulent conjunctivitis and corneal ulceration and scarring. KCS occurs in dogs, cats, and horses. In dogs, it is often associated with an autoimmune dacryoadenitis of both the lacrimal and nictitans glands and is the most frequent cause of secondary conjunctivitis. Distemper, systemic sulfonamide and NSAID therapy, heredity, and trauma are less frequent causes of KCS in dogs. KCS occurs infrequently in cats and has been associated with chronic feline herpesvirus 1 infections. In horses, KCS may follow head trauma.

Topical therapy consists of artificial tear solutions, ointments, and, if there is no corneal ulceration, antibiotic-corticosteroid combinations. Lacrimogenics such as topical cyclosporin A (0.2%–2%, bid), tacrolimus (0.02%, bid), or pimecrolimus (1%) may increase tear production; cyclosporine increases tear formation in ~80% of dogs with Schirmer tear test values  $\geq 2$  mm wetting/min. Ophthalmic pilocarpine mixed in food may be useful for neurogenic KCS (dogs 20–30 lb [10–15 kg] should be started on 2–4 drops of 2% pilocarpine, bid). Mucolytic agents (eg, 10% acetylcysteine) lyse excess mucus and restore the spreading ability of other topical agents. In chronic KCS refractory to medical therapy, parotid duct transplantation is indicated. In general, canine KCS requires longterm (often for life) topical lacrimogenic therapy.

## CONJUNCTIVA

The conjunctiva consists of the palpebral conjunctiva (lining the posterior eyelids), the fornix or conjunctival cul-de-sac where the palpebral and bulbar conjunctiva connect, and the bulbar conjunctiva (covering the anterior globe or episclera) and nictitating membrane. The conjunctiva has important roles in tear dynamics, immunologic protection, ocular movement, and corneal healing. Because it is loosely attached to the episclera, the bulbar conjunctiva is a useful tissue to graft to weakened, ulcerated corneas.

**Subconjunctival hemorrhage** may arise from trauma or blood dyscrasias, von Willebrand factor deficiency, and certain infectious diseases. It does not require therapy, but close inspection is warranted to determine whether more important intraocular alterations have occurred. If definite evidence or history of trauma is not present, then systemic examination is indicated to determine the cause of the spontaneous hemorrhage.

**Chemosis, or conjunctival edema,** is seen to some degree in all cases of conjunctivitis, but the most dramatic examples are seen with trauma, hypoproteinemia, allergic reactions, and insect bites. The latter are treated with topical corticosteroids and usually resolve rapidly. Specific therapy for the etiologic agent is indicated.

**Conjunctivitis** is common in all domestic species. Primary infectious conjunctivitis caused by different bacteria, viruses, mycoplasma, fungi, and parasites affect several species. The etiologic agents vary from infectious to environmental irritants. The signs are hyperemia, chemosis, ocular discharge, follicular hyperplasia, and mild ocular discomfort. The appearance of the conjunctiva usually is not sufficiently distinctive to suggest the etiologic agent, and specific diagnosis depends on history, physical examination, conjunctival scrapings and culture, Schirmer tear test, and occasionally biopsy. Unilateral conjunctivitis may result from a foreign body, dacryocystitis, or keratoconjunctivitis sicca (see above). In cats, feline herpesvirus 1 (FHV-1), *Mycoplasma*, or *Chlamydia psittaci* may produce conjunctivitis that begins in one eye and becomes bilateral after ~1 wk. Specific diagnosis is made most rapidly by demonstrating the inclusions or the agent in conjunctival scrapings. Bilateral conjunctivitis is common in viral infections in all species. Herpesviruses produce conjunctivitis in cats, cattle, horses, and pigs. Purulent discharge indicates a bacterial component, but this may be opportunistic because of debilitation of the mucous membrane. Environmental irritants and allergens are common causes of conjunctivitis in all species. If a mucopurulent exudate is present, topical antibiotic therapy is indicated but may not be curative if other predisposing factors are involved. Mechanical factors such as foreign bodies, environmental irritants, parasites, and eyelid conformational defects should be removed or corrected. Selected antibiotics are indicated for

chlamydial and mycoplasmal infections; topical antiviral preparations (eg, 1% idoxuridine, 3% adenine arabinoside, or 1% trifluorothymidine (often instilled tid-qid and administered for 7–14 days) are indicated for herpesvirus infections when both the cornea and conjunctiva are involved. Oral supplementation in cats with 250–500 mg of L-lysine daily (often placed in the treats) may reduce the severity and frequency of recurrence of FHV-1 conjunctivitis and keratitis.

## CORNEA

The size of the nearly round to oval cornea (vertical/horizontal) varies by animal species: dog (8.5 × 9.5 mm), cat (8.4 × 8.9 mm), horse (16.6 × 17.9 mm), and cow (15.2 × 16.4 mm). The animal cornea consists of the superficial epithelium and basement membrane, large and relatively acellular stroma, deeper Descemet membrane, and deep single layer endothelium. The cornea maintains a strong and durable barrier between the eye and environment, as well as a transparent medium to permit passage of light and images into the posterior segment. Corneal diseases are common in most animal species and fortunately can be treated successfully by medical, surgical, or a combination of these methods. The accessibility of the cornea permits several detailed and noninvasive diagnostic techniques.

**Superficial keratitis** is common in all species and is characterized by corneal vascularization and opacification, which may be due to edema, cellular infiltrates, pigmentation, or fibroplasia. If ulceration is present, pain—manifest by epiphora and blepharospasm—is an outstanding sign. Unilateral keratitis frequently is traumatic in origin. Mechanical factors, such as lid conformational defects and foreign bodies, should always be eliminated as possible causes, because improvement will not occur until they are resolved. Ulcerative keratitis may be complicated by secondary invasion by bacteria and, in horses, by saprophytic fungi. Bilateral superficial keratitis may be immune-mediated or associated with a lack of tears, eyelid conformational defects, or infectious agents.

**Pannus**, or Ueberreiter disease, is a specific, bilateral, progressive, proliferative, chronic, superficial keratitis that begins laterally and/or medially at the limbus and eventually extends from all quadrants to cover the cornea. Inflammatory cells (lymphocytes and plasma cells) infiltrate the cornea from the limbus, accompanied



Chronic superficial keratitis, early, German Shepherd. The cornea is progressively infiltrated with superficial blood vessels, followed by inflammation (lymphocytes and plasma cells), and lastly pigmentation. Courtesy of K. Gelatt.

by superficial blood vessels. This immune-mediated keratitis is common in German Shepherds, Belgian Tervurens, Border Collies, Greyhounds, Siberian Huskies, and Australian Shepherds. Specific therapy consists of topical antibiotics, antiviral or antimycotic agents when appropriate, removal of any mechanical irritants, tear replacement when deficient, and corticosteroids or cyclosporin A (or both) when immune-mediated. The latter may need to be continued indefinitely and the frequency varied depending on the response. Chronic superficial keratitis when immune-mediated is a lifelong disease, requiring lifelong topical anti-inflammatory therapy. The disease appears more aggressive in young dogs and in dogs that live outside in higher altitudes. Generally, topical 1% prednisolone, 0.1% dexamethasone, or 0.2%–1% cyclosporine instilled in both eyes bid-qid is sufficient to control the disease and prolong vision. The intensity of the inflammatory response in both eyes is quite variable and may change by age, season, amount of time the dog spends outside, and other factors. To minimize costs and adverse effects, but control the disease, topical therapy is adjusted to the individual animal (topical therapy ranges from one drop in the affected eye every other day to as frequent as one drop in the affected eyes qid).

**Interstitial keratitis** is a deep involvement of the corneal stroma that represents one of the clinical signs associated with all chronic and many acute cases of anterior uveitis. The corneal vascularization is less branching, finer, and deeper than in superficial keratitis; if the endothelium has been disrupted, corneal edema is often marked. Systemic diseases, such as infectious canine hepatitis, bovine malignant catarrhal fever, systemic mycoses in many species, and neonatal