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CHAPTER 37

Ophthalmologic Diseases in Small Pet Mammals

Alexandra van der Woerdt, DVM, MS,
Diplomate ACVO, Diplomate ECVO

RABBITS

Ophthalmic examination of rabbits can be performed easily.[69,70] The eyes are laterally located and have a round pupil. Evaluation of a menace response is difficult, but most rabbits will react to bright light by squinting. The dorsal rectus muscle can usually be seen as a large striated band of tissue under the conjunctiva. Some rabbits do not respond to topical application of mydriatic agents because of the natural presence of atropinase. In these rabbits, the addition of 10% phenylephrine may help to obtain mydriasis. Rabbits have a merangiotic fundus. The well-myelinated optic nerve is present above the visual axis and has a deep optic cup. Retinal blood vessels are present in a linear streak medial and lateral to the optic nerve. An extensive venous plexus is present in the orbit. Tear production in rabbits can be measured by using Schirmer tear test strips. Average tear production is 5 mm/min (standard deviation, ±2.4 mm/min)[1]; however, very low values can be measured in some normal rabbits. Normal intraocular pressure measured by applanation tonometry is between 10 and 20 mm Hg.

The nasolacrimal system of rabbits has a single nasolacrimal punctum. The punctum is located in the ventral eyelid 3 mm from the eyelid margin, near the medial canthus and ventral to the lacrimal caruncle (Fig. 37-1).[9,38] The lacrimal sac is immediately rostral to the punctum and caudal to the nasolacrimal duct aperture. The nasolacrimal duct extends from the orbit to the nasal fossa and runs within the part of the maxilla that forms the lateral wall of the maxillary sinus.[38] Approximately 5 to 6 mm within the maxilla, the duct curves sharply and decreases in diameter.[9] At the level of the palatine bone, the nasolacrimal duct leaves the bony nasolacrimal canal and makes a sharp turn at the nasolacrimal duct flexure, which is located just caudal to the caudal limit of the incisor tooth roots. The nasolacrimal duct narrows at this flexure in normal rabbits. The duct then follows the ventral margin of the nasoturbinates and exits on the ventromedial aspect of the alar fold just caudal to the mucocutaneous junction of the nares.

CONJUNCTIVITIS AND EPIPHORA

Conjunctivitis in rabbits is common. In normal rabbits with no ocular or respiratory disease, the most frequently isolated organisms from the conjunctival cul de sac include *Staphylococcus*, *Micrococcus*, and *Bacillus* species. Less common organisms include *Bordetella*, *Stomatococcus*, *Neisseria*, *Pasteurella*, *Corynebacterium*, *Streptococcus*, and *Moraxella* species.[14] However, *Pasteurella multocida* is a cause of conjunctivitis, epiphora, nasolacrimal duct obstruction, and dacryocystitis in rabbits.[27,38] A wide variety of other infectious agents also have been associated with conjunctivitis in rabbits, including *Staphylococcus aureus*, *Pseudomonas* species, *Haemophilus* species, *Treponema paraluiscuniculi*, mycoplasmas, chlamydiae, and myxoma virus.[27,45,60] In New Zealand white rabbits with conjunctivitis, upper respiratory disease, and pneumonia, bacterial isolates consisted of *Bordetella broncheptica*, *P. multocida*, *S. aureus*, and *Pseudomonas alcaligenes*.[50] Mucopurulent conjunctivitis and blepharitis with corneal ulceration have been associated with *S. aureus* infection in a rabbit.[45] Treatment with topical gentamicin ophthalmic ointment and systemic gentamicin was curative. Methicillin-resistant *S. aureus* was isolated in a rabbit with severe bilateral conjunctivitis. (K. Quesenberry, personal communication). Other causes of conjunctivitis in rabbits include foreign bodies, entropion, distichia, trichiasis, and high ammonia or dust content in the environment. Dental disease, including root elongation and dental abscesses, is also associated with conjunctivitis.

Unilateral or bilateral epiphora can be present in rabbits without conjunctivitis. The discharge often has a white, gritty appearance and may be intermittent and resistant to treatment with topical antibiotics. Root elongation of the maxillary

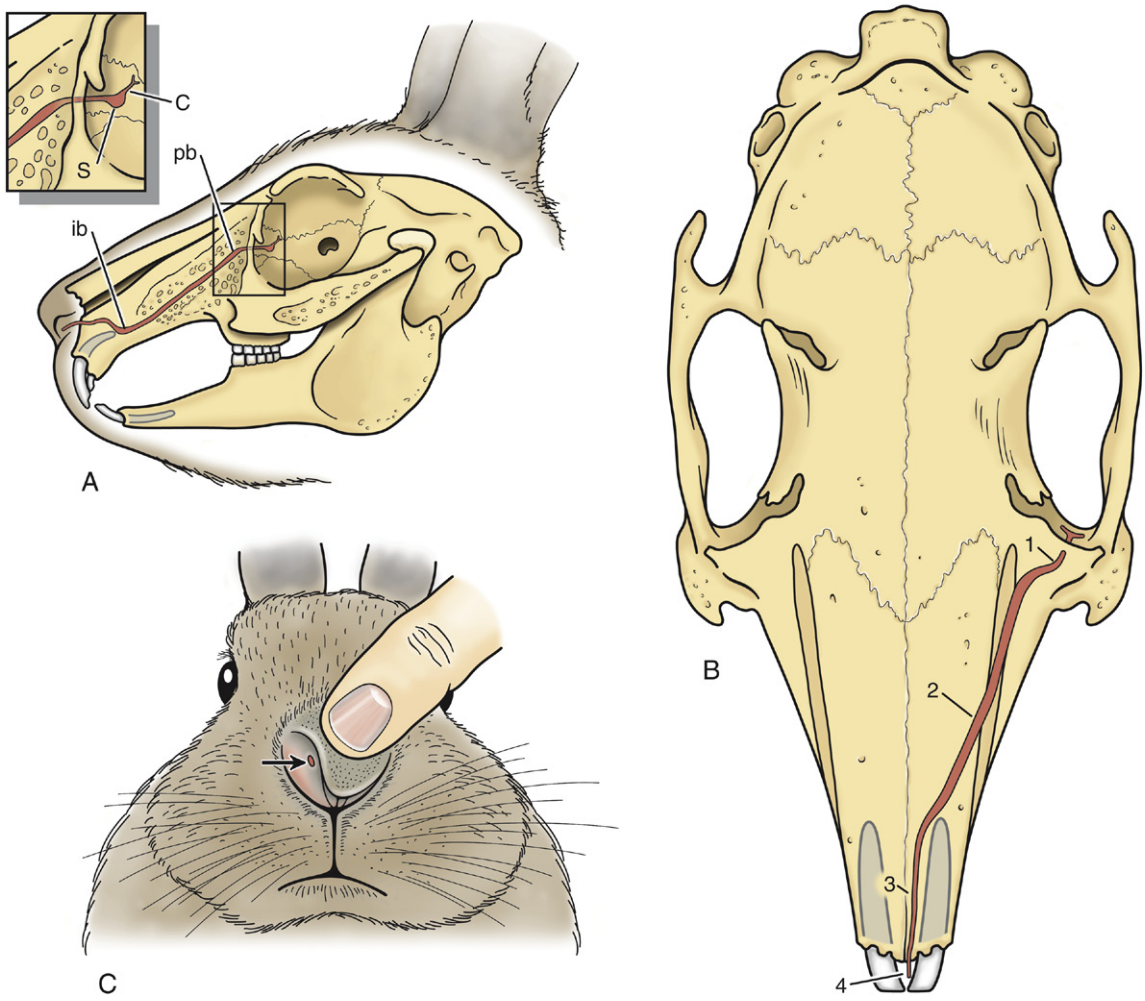

Fig. 37-1 Diagram of the rabbit nasolacrimal duct. **A,** Lateral view with inset. The two sharp bends, the proximal maxillary bend *(pb)*, and the bend at the incisor tooth *(ib)*, are indicated. The inset shows the canaliculus *(C)* and the lacrimal sac *(S)*. **B,** Dorsoventral view. *(1)* Proximal portion of the duct extending from the punctum through the proximal maxillary curve; *(2)* portion of the duct extending from the proximal maxillary curve to the base of the incisor tooth; *(3)* portion of the duct extending from the base of the incisor tooth to the end of the lacrimal canal; *(4)* distal portion of the duct extending from the end of the lacrimal canal to the nasal meatus. **C,** The nasal meatus of the nasolacrimal duct *(arrow)*. The opening is enlarged for diagrammatic purposes.

incisors is a common underlying cause.[27] The elongated roots can cause an obstruction of the nasolacrimal duct at its flexure just caudal to the roots of the incisors. Radiographs of the skull are needed to assess the incisors; excess curvature of the incisor roots is abnormal. In one report describing two affected rabbits and 13 normal rabbits, radiographs revealed a cystic dilation of the nasolacrimal duct immediately caudal to the duct flexure, and the incisors were more arched than in normal rabbits.[38] Irrigation of the nasolacrimal duct in affected rabbits yielded opaque, white, gritty fluid which, on cytologic examination, showed numerous macrophages, lipid-laden mesothelial cells, lipid droplets, and small numbers of bacteria and erythrocytes. Occlusion of the ducts was presumed to be attributed to fat droplets. Bacteriologic culture of fluids used to irrigate the nasolacrimal ducts of both normal and affected rabbits yielded similar bacterial isolates; therefore microorganisms may not be important in the pathogenesis of epiphora in rabbits. The most common bacterial isolates were *S. aureus,* coagulase-negative *Staphylococcus* species, *Moraxella* and *Neisseria* species, *Oligella urethralis,* and *Streptococcus viridans.* In a study of 28 cases of dacryocystitis in rabbits, 89% of cases were unilateral. The cause was determined to be dental malocclusion in 50%, no apparent cause in 35%, rhinitis in 7%, both rhinitis and dental malocclusion in 4%, and panophthalmitis in 4%. Most animals (98%) were treated with topical antibiotics, with a mean duration of 5.8 weeks.[21]

In rabbits with epiphora, the diagnostic value of bacterial culture of the irrigation fluid is questionable. However, bacterial culture is recommended if nasal discharge is present in conjunction with epiphora. Skull radiographs are useful to detect underlying dental disease. Dacryocystorhinography using contrast material injected into the nasolacrimal system can help localize the site of obstruction, differentiate between a complete and partial obstruction, and identify any dilation.

Treatment of epiphora in rabbits can be frustrating. Irrigation of the nasolacrimal duct is important to restore the patency

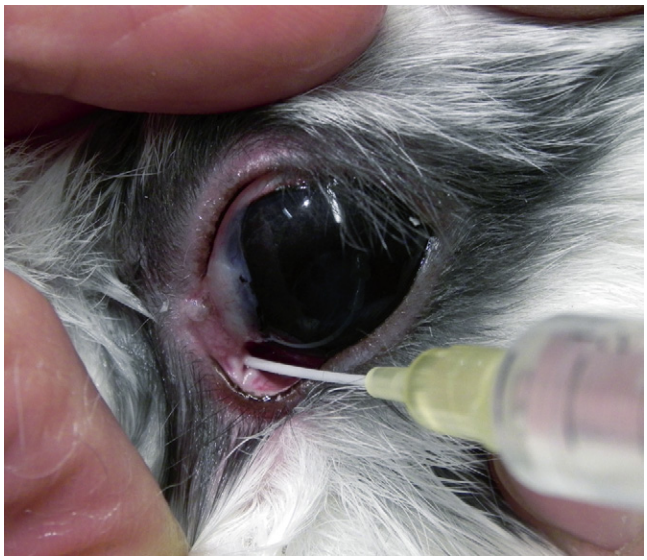

Fig. 37-2 Irrigation of the nasolacrimal duct in a rabbit with a 24-gauge Teflon intravenous catheter. Rabbits have a single nasolacrimal punctum in the ventral eyelid.

of the nasolacrimal system. After instilling a topical ophthalmic anesthetic, use a 23-gauge lacrimal cannula or a 22- to 24-gauge Teflon intravenous catheter to flush the duct (Fig. 37-2). Recurrence of the obstruction is common, and duct irrigation may need to be repeated every 2 to 3 days or weekly until a few consecutive clear irrigations are obtained. If topical antibiotic therapy is used, a broad-spectrum medication such as triple antibiotic solution is recommended. Topical nonsteroidal, anti-inflammatory ophthalmic medications, such as 0.03% flurbiprofen or 0.1% diclofenac, may help minimize irritation caused by the procedure. In rabbits with chronic or severe infections, concurrent topical ophthalmic and systemic antibiotic therapy may be needed. Suggested combinations include systemic enrofloxacin (Baytril, Bayer Corporation, Shawnee Mission, KS) or marbofloxacin (Zeniquin, Pfizer Animal Health, Exton, PA) and topical ciprofloxacin or gentamicin ophthalmic solution. In rabbits with evidence of underlying incisor root elongation, removing the incisors can be considered in severe cases.

CORNEA

Corneal dystrophy is the accumulation of cholesterol or lipid crystals in the cornea.[4] This may develop spontaneously, as has been reported in American Dutch belted rabbits,[47] or be due to high dietary cholesterol. It also occurs in breeds that are predisposed to hypercholesterolemia, such as the Watanabe rabbit with heritable hyperlipidemia.[22,35] In Watanabe rabbits, yellowish-white granules can develop along the corneal-scleral junction and in the iris. In rabbits without systemic lipid abnormalities, spontaneous corneal dystrophy is usually bilateral and symmetric and does not progress to visual impairment. In any rabbit with corneal dystrophy, carefully evaluate the fat content of the diet.

Progressive occlusion of the cornea with a conjunctiva-like membrane is occasionally seen in rabbits.[3,18,57,59] Membranous corneal occlusion, or pseudopterygium, is a pain-free condition that may affect one or both eyes (Fig. 37-3). Ophthalmic examination reveals a circular membrane that originates at the limbus (the junction of the cornea and sclera) and gradually

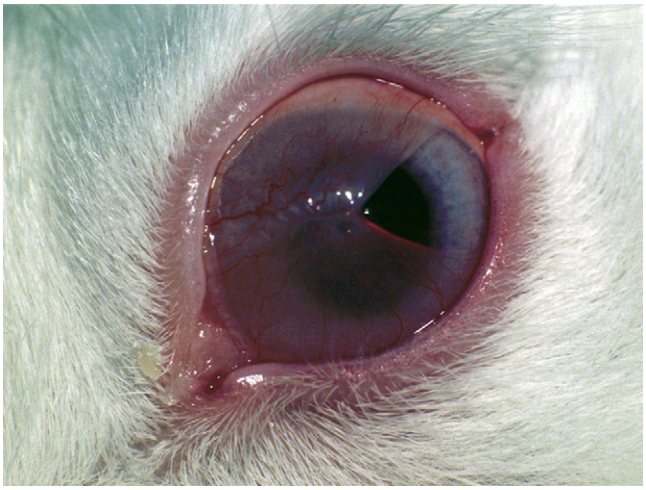

Fig. 37-3 Progressive occlusion of the cornea with conjunctiva-like tissue. This tissue is not adherent to the cornea. The disease is not painful. *(Courtesy David Wilkie, DVM, MS.)*

advances over the cornea. In severe cases, only a small central opening is present, allowing visibility of an otherwise normal globe. The membrane does not adhere to the cornea. The cause of this condition is unknown, although trauma has been suggested. Progressive membranous occlusion in rabbits has been compared with pterygium in humans. However, in humans the membrane is triangular and adherent to the cornea, whereas in rabbits it is nonadherent and circumferential from the limbus. Treatment with topical antibiotic or antibiotic-steroid medications has no effect. The membrane can be resected surgically and treated with topical antibiotics postoperatively; this usually results in quick recurrence of the membrane. However, if the membrane is resected a few millimeters beyond the limbus and the eye is then treated with a topical antibiotic-steroid combination, recurrence may be prevented. Good results have also been obtained with surgical resection and the use of topical cyclosporine with or without corticosteroids.[57] Another described surgical technique is to incise the membrane into four to six quadrants and suture each quadrant of the membrane to the inside of the eyelid.[3,59] With this technique, recurrence may be prevented for at least 1 year.

Superficial nonhealing corneal ulcers are occasionally seen in rabbits. Clinical signs are usually mild and include epiphora, conjunctival hyperemia, and blepharospasm. The ulcer is usually located in the paracentral cornea, is very superficial, and has redundant epithelial edges. The clinical appearance is that of an indolent ulcer, as seen in boxer dogs. Carefully examine the eyes of affected rabbits to eliminate potential causes such as abnormal hairs, lagophthalmos (inability to fully close the eyelid), facial nerve paralysis, or a foreign body. Treatment with a topical antibiotic solution or ointment usually fails to resolve the ulcer. Additional therapies such as corneal debridement, grid keratotomy, use of topical serum, application of corneal glue, tarsorrhaphy, or superficial keratectomy are usually necessary for the ulcer to heal.

UVEITIS AND DISEASES OF THE LENS

Encephalitozoon cuniculi may cause granulomatous encephalitis and renal lesions in rabbits. Many rabbits infected with *E. cuniculi* are asymptomatic, but neurologic signs can include

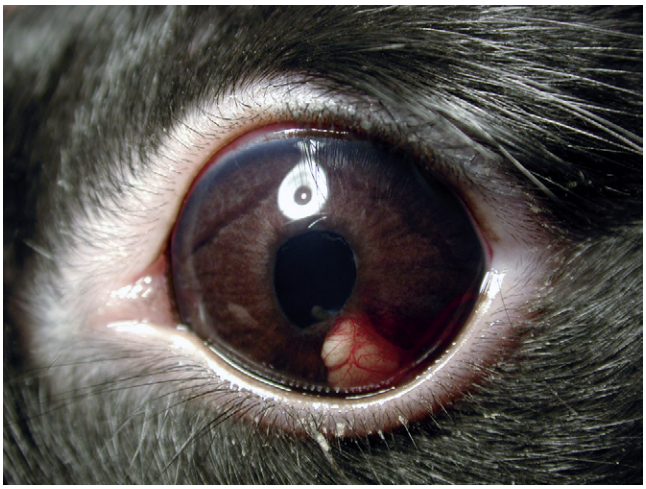

Fig. 37-4 Rabbit infected with *Encephalitozoon cuniculi*. A white lesion is present in the iris, protruding into the anterior chamber. Lens involvement with cataract formation is present underneath the iridial lesion.

convulsions, tremors, torticollis, paresis, and coma. *Encephalitozoon cuniculi* infection has also been associated with phacoclastic uveitis.[20,23,63] Most affected rabbits are young (less than 2 years of age), and dwarf rabbits appear predisposed to this disease. Clinically, a white mass is often seen protruding into the anterior chamber (Fig. 37-4). Careful examination of the anterior segment of the eye with slit-lamp biomicroscopy may reveal a break in the anterior lens capsule. The break is frequently hidden by inflammatory material and it may appear as if only the iris is involved in the inflammatory process. A focal cataract is often present in the area of the anterior lens capsule break. Signs of a severe pyogranulomatous anterior uveitis are usually present, such as conjunctival hyperemia, a swollen hyperemic iris, miosis, aqueous flare, and low intraocular pressure. The posterior segment of the eye is initially normal; however, if left untreated, severe uveitis and cataract formation can lead to blindness and possible phthisis bulbi or glaucoma. An abscess in the iris caused by *P. multocida* initially may resemble phacoclastic uveitis. Measurement of serum antibody titers for *E. cuniculi* and *P. multocida* may aid in the differential diagnosis. The treatment of choice is surgical removal of the lens by phacofragmentation. Because of the rabbit's ability to regenerate a lens after this procedure, insertion of an artificial lens after phacofragmentation is not recommended. Systemic treatment of *E. cuniculi* with albendazole (30 mg/kg PO q24h for 30 days, then 15 mg/kg PO q24h for an additional 30 days) has been reported.[63] Fenbendazole (20 mg/kg q24h for 28 days) has proved effective in both preventing experimental *E. cuniculi* infection in rabbits and treating naturally infected seropositive rabbits.[64] If the lens is not removed surgically, control of the uveitis with topical steroidal (such as 1% prednisolone acetate) and nonsteroidal anti-inflammatory medications as well as systemic fenbendazole or albendazole is necessary. Enucleation may be indicated if the uveitis cannot be controlled medically and a chronically painful eye is present.[23,74]

Spontaneous cataract formation is rare in rabbits. One report describes a low incidence of juvenile cataracts in laboratory New Zealand white rabbits.[49] Keratitis caused by Shope fibroma virus has been reported in one rabbit.[32] Cataracts developed 3 months after the keratitis, which may have been hereditary in origin and unrelated to the virus. Cataract and subsequent glaucoma can develop subsequent to ocular trauma, such as cat-scratch injury.

Intraocular sarcomas have been described in two adult rabbits; one had a nonvisual eye associated with chronic inflammation and the second had chronic uveitis, cataract formation, and glaucoma.[42] In both rabbits, intraocular spindle cell neoplasms closely associated with lens and lens capsular fragments were described. The histologic features of the tumors closely resembled posttraumatic ocular sarcomas in cats; chronic inflammation and trauma were considered as probable causes.

GLAUCOMA

Congenital glaucoma is inherited as an autosomal recessive trait in rabbits. In those with this condition, the intraocular pressure is high as early as 3 months of age.[10] With increasing age, progressive buphthalmos with a markedly enlarged cornea, structural abnormalities of the iridocorneal angle, atrophy of the ciliary processes, and excavation of the optic nerve develop. Topical glaucoma medications used in dogs, such as 0.5% timolol maleate and 2% dorzolamide, may also be used in rabbits. Because response to therapy is unpredictable in rabbits, carefully monitor the intraocular pressure during treatment. Enucleation, insertion of an intrascleral prosthesis, and laser cycloablation with a diode laser have also been used to manage glaucoma in pigmented pet rabbits. However, laser cycloablation cannot be used in albino rabbits. If left untreated in chronic cases, pressure-induced atrophy of the ciliary body may lead the intraocular pressure to return to normal.

ORBIT

Retrobulbar disease processes are occasionally seen in rabbits. Clinical signs include progressive exophthalmos, protrusion of the third eyelid, and inability to retropulse the globe. Exposure keratitis may be present if the ability of the eyelids to close properly has been affected (Fig. 37-5). Abscesses are the common cause of retrobulbar disease in rabbits; dental disease with tooth root abscessation is often a predisposing factor (see Chapter 32). Infection is caused by both aerobic and anaerobic bacterial species. A thorough dental examination and skull radiographs are indicated in any rabbit with a suspected retrobulbar mass (see Chapter 35). If available, a computed tomography (CT) scan is especially helpful in diagnosis *Taenia serialis coenurus* formation caused exophthalmos in a pet rabbit. Surgical removal was curative.[51] Retrobulbar neoplasia is uncommon in rabbits.

An abscess in the retrobulbar space of a rabbit can be very difficult to treat. Because of the thick nature of the abscessed material and the anatomy of the alveolar bulla, drainage of the abscess through the mouth, as performed in dogs and cats, may or may not be successful. If the abscess is caused by an abscessed tooth root, the tooth or teeth must be extracted to allow drainage and the rabbit treated with long-term systemic antibiotic therapy. In some cases aggressive surgical debridement may be necessary. This may include exenteration of the orbit and sacrifice of a sighted eye. Even with aggressive surgical and medical management, the prognosis for recovery is always guarded. Stomatoscopy-aided dental trimming, tooth removal, and debridement successfully treated a retrobulbar abscess in a rabbit.[39] Anecdotal reports suggest that some rabbits with retrobulbar abscesses respond to medical therapy with long-term (3-month) administration of benzathine/procaine penicillin G

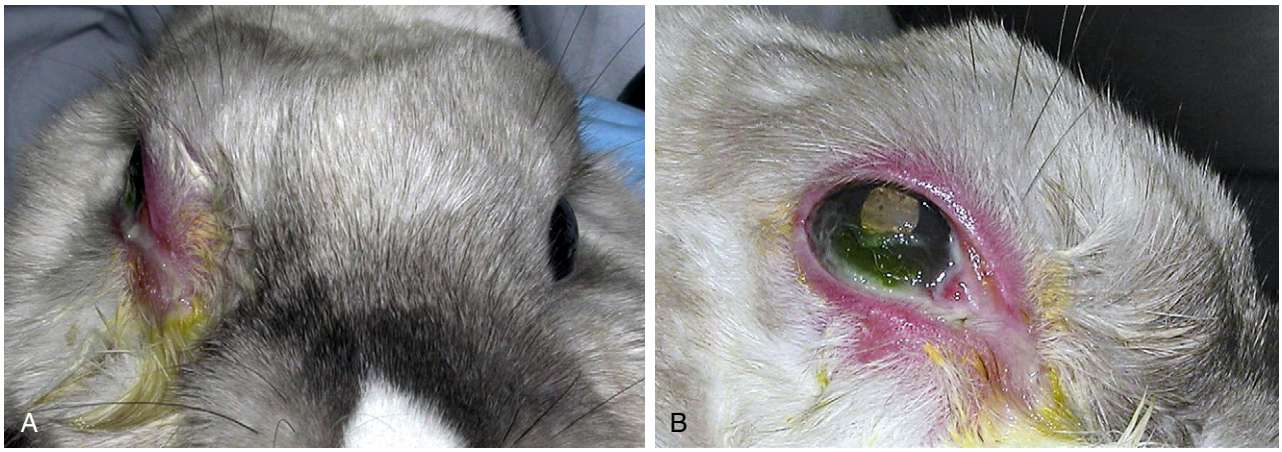

Fig. 37-5 Rabbit with retrobulbar disease. Note the exophthalmos, hyperemia of the eyelids, discharge, and exposure keratitis.

(for rabbits < 2.5 kg, 75,000 U per rabbit SC q48h; for rabbits > 2.5 kg, 150,000 U per rabbit SC q48h).[56]

Bilateral exophthalmos in rabbits is commonly associated with compromised vascular drainage of the head. Periodic exophthalmos is common in rabbits with thymomas (see Chapter 20).[34,53,68] In one report, a localized myasthenia gravis associated with the thymoma was suggested as a cause.[68] More probably, the presence of a large intrathoracic mass causes cranial vena cava syndrome, in which the mass compresses vessels in the cranial thorax.[53] This compression impedes blood flow in the right and left cranial vena cava as well as the external jugular veins and causes decreased vascular drainage from the head. Exophthalmos was also reported in a rabbit after the chronic placement of an external jugular catheter,[29] and bilateral exophthalmos can develop with thrombosis of both jugular veins. The external jugular veins are the largest vessels draining the head, whereas the internal jugular veins of rabbits are relatively small and primarily drain the brain, throat, and neck regions. Therefore occlusion of the external jugular veins will compromise vascular drainage of the head. In clinical cases, exophthalmos secondary to venous thrombosis often resolves spontaneously, presumably after the jugular veins become patent again. Exophthalmos in obese rabbits may be caused by excessive fat deposition in the orbit.

Rabbits have several glands in the orbit. The lacrimal gland is located dorsolaterally, and the accessory lacrimal gland, divided into three lobes, is located along the caudal and ventral orbital margin. The superficial gland of the third eyelid is a small gland located near the third eyelid's cartilage. The deep gland of the third eyelid, also known as the harderian gland, consists of two parts: dorsal (white) and ventral (pink) lobes. Prolapse of the deep gland of the third eyelid has been described in rabbits. Surgical correction with a pocket technique, as has been described in dogs, was successful in reducing the gland in one rabbit.[31]

FERRETS

Ferrets have prominent globes placed laterally in the skull; they have very limited binocular vision.[24] The ferret's pupil is a horizontal slit that quickly responds to light. Topical 1% tropicamide may have to be applied to evaluate the fundus. Like dogs and cats, ferrets have a holangiotic retinal vascular pattern. The projection of retinal ganglion cells from the temporal area of the retina in albino ferrets differs from that of pigmented ferrets.[48] In pigmented ferrets, 6,000 retinal ganglion cells project ipsilaterally to the brain, whereas in albino ferrets, only 1,500 retinal ganglion cells project ipsilaterally. The significance of this difference has not been established. Intraocular pressure can be measured with a tonopen (Tono-Pen XL, Reichart, Inc., Dewpew, NY). Normal values for intraocular pressure range from 14.5 ± 3.3[46] to 22.8 ± 5.5 mm Hg.[58] Tear production in normal ferret eyes is less than in dogs, with an average of 5.3 ± 1.3 mm/min measured by commercially available strips.[46]

Conjunctivitis in ferrets can be caused by viral or bacterial infection. Ocular signs of canine distemper virus, a fatal disease in ferrets, are mucopurulent oculonasal discharge, blepharitis, corneal ulcers, and keratoconjunctivitis sicca.[33] Conjunctival swelling and a proliferative lesion of the nictitans caused by infection with *Mycobacterium genavense* have been described in two ferrets. Other clinical signs in these ferrets included peripheral lymph node enlargement.[37]

Degeneration of corneal endothelial cells leading to progressive corneal edema and cloudiness of the cornea is seen in older mink (8-11 years of age).[26] Royal pastel females are predisposed. Unlike the disease in dogs, these mink do not develop corneal ulceration, pigmentation, or vascularization. There is no specific treatment for this condition, but symptomatic treatment with 5% sodium chloride solution or ointment two to four times a day may or may not improve corneal clarity.

A lymphoplasmacytic keratitis has been reported in a ferret with lymphoma.[55] An infiltrative lesion resembling corneal lesions reported in mink with Aleutian disease was present in the cornea.

Cataracts are common in ferrets.[67] Progressive cataract formation has been reported in two genetically unrelated populations of ferrets.[43] In 1-year-old ferrets, cataracts were observed in 47% of animals examined. Severity ranged from clinically insignificant, small cataracts in the posterior cortex of the lens to blinding, complete cataracts. By 18 months of age, cataracts were detected in virtually every animal, and in animals previously diagnosed, the cataracts had progressed. A genetically separate group had a combination of blinding cataract, microphthalmos, abnormal iris formation, and retinal detachment. In another ferret colony, microphthalmos, cataract, retinal dysplasia, and a persistent hyperplastic primary vitreous-type membrane were shown to be inherited as an autosomal

dominant defect.[17] Dietary factors may play a role in the development of cataracts in ferrets. A diet high in fat or deficient in vitamin E or protein may promote cataract formation.[43] In ferrets with cataracts, monitor the eyes regularly for the onset of secondary complications. Lens-induced uveitis can usually be controlled with topical 1% prednisolone acetate applied once or twice daily. Other complications caused by cataracts are lens subluxation or luxation and glaucoma. Ferrets that are blind because of cataracts usually adjust well in a home environment. However, cataract surgery can be performed successfully in ferrets. The lenses can be removed by phacofragmentation or by an extracapsular technique. Artificial lenses are not available in a size suitable for ferrets. Before cataract surgery, make sure the ferret becomes accustomed to frequent application of eye medications to facilitate easy treatment after surgery.

Retinal degeneration is seen in ferrets. Clinical signs are progressive loss of vision, which may not be noticed until the disease is advanced. Ophthalmic examination reveals mydriasis with a very poor pupillary light reflex. Cataracts may or may not be present. Retinal vascular attenuation and tapetal hyperreflectivity are seen in the fundus. There is no treatment for retinal degeneration.

Exophthalmos can occur in ferrets from several causes. Lymphosarcoma is a common disease in ferrets, and although orbital involvement has been reported in only two ferrets,[41] it is occasionally seen clinically. Exophthalmos is often the presenting complaint. Ophthalmic examination reveals unilateral or bilateral exophthalmos, decreased retropulsion of the globe, and protrusion of the third eyelid. Lagophthalmos may result in exposure keratitis with corneal ulceration and vascularization. In clinical cases, lymphosarcoma is usually present in other areas of the body. In the two ferrets described,[41] peripheral lymph nodes were affected in one ferret and involvement of the liver, spleen, intestines, kidneys, and adrenals was present in the other. Retrobulbar adenocarcinoma has been reported in one ferret,[40] and we have seen this clinically as a rare cause of unilateral exopthalmos (A. van der Woerdt, K. Quesenberry, personal communication, 2006).

In ferrets with exophthalmos, a CT scan of the head is the best method of diagnosing a retrobulbar mass and determining the extent of the lesion. Cytologic examination of a sample from the retrobulbar area obtained by ultrasound-guided fine-needle aspiration can confirm the diagnosis of neoplasia. However, this procedure can be difficult because of the limited size of the retrobulbar space. Instead, in cases of suspected orbital lymphosarcoma, diagnosis may be confirmed by obtaining samples for diagnostic tests elsewhere in the body, such as a fine-needle aspirate or wedge biopsy of an enlarged lymph node. In ferrets with lymphosarcoma, therapy is directed at treating the disease systemically with prednisone or chemotherapeutic agents. While treating the ferret for lymphosarcoma, temporary tarsorrhaphy may be necessary to protect the cornea if pronounced exophthalmos is present. If the corneal epithelium is intact, protect the eye of a ferret with exophthalmos with lubricating ophthalmic ointment applied two to four times daily. If an ulcer is present, treat with an antibiotic ophthalmic ointment, such as triple antibiotic or gentamicin ointment, applied three to four times daily. In cases of retrobulbar adenocarcinoma, treatment may include exenteration and radiation therapy.[40] Zygomatic salivary gland mucocele is another reported cause of exophthalmos in ferrets.[44] Fine-needle aspiration of a soft fluctuant swelling dorsotemporal to the eye yields a tenacious, blood-tinged fluid. Surgical excision is usually curative (see Chapters 3 and 11).

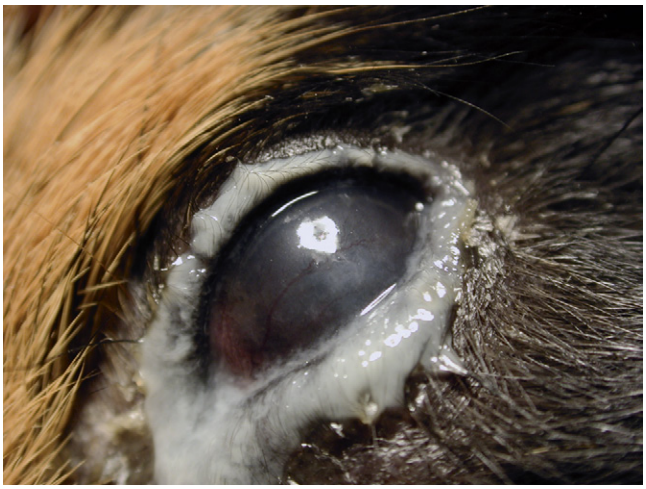

Fig. 37-6 Conjunctivitis and keratitis in a guinea pig. Note the abundant mucopurulent discharge, corneal vascularization, and fibrosis.

GUINEA PIGS

Guinea pigs have a paurangiotic retina that appears avascular on examination. Their eyelids are open from birth, and they have a rudimentary third eyelid. Guinea pigs produce a very small amount of tears, and measurement of tear production by using commercially available strips is not possible.[65] The phenol red thread tear test is a better option to assess tear production in guinea pigs.

Conjunctivitis is common in guinea pigs (Fig. 37-6). One common cause is *Chlamydophila caviae* (formerly *C. psittaci*),[33] which causes a self-limited disease manifesting as mild chemosis, ocular discharge, and follicle formation. Cytologic examination of a specimen from a conjunctival scraping may reveal intracytoplasmic inclusion bodies in epithelial cells. Treatment is generally considered unnecessary. Vitamin C deficiency in guinea pigs causes conjunctivitis with a flaky discharge. Treatment is directed at correcting the dietary deficiency.

A spontaneous outbreak of listerial keratoconjunctivitis has been reported in hairless guinea pigs.[13] Clinical signs ranged from serous lacrimation with hyperemic conjunctiva to purulent, ulcerative keratoconjunctivitis with corneal neovascularization. *Listeria monocytogenes* was cultured from the ocular discharge. Treatment was not attempted.

Blepharitis caused by dermatophyte infection may be seen in young guinea pigs.[5] Topical antifungal therapy is usually effective.

Lipid deposition within the conjunctiva of the eyelids, named "fatty eye" by guinea pig breeders is most commonly seen in overweight animals. There is no specific treatment for this condition.

Lymphosarcoma is rare in guinea pigs but has been reported to infiltrate the cornea.[62] Lymphosarcoma should also be considered as a differential diagnosis of conjunctival masses in guinea pigs.[2] Another differential diagnosis for conjunctival nodules in guinea pigs is a syndrome known as "pea eye." These nodules are protrusions of portions of the lacrimal or zygomatic glands and appear pale or pink. Treatment is not necessary because animals are usually not bothered by this condition.

A dermoid is a congenital lesion in which skin-like tissue is present in an abnormal location. Corneal and conjunctival

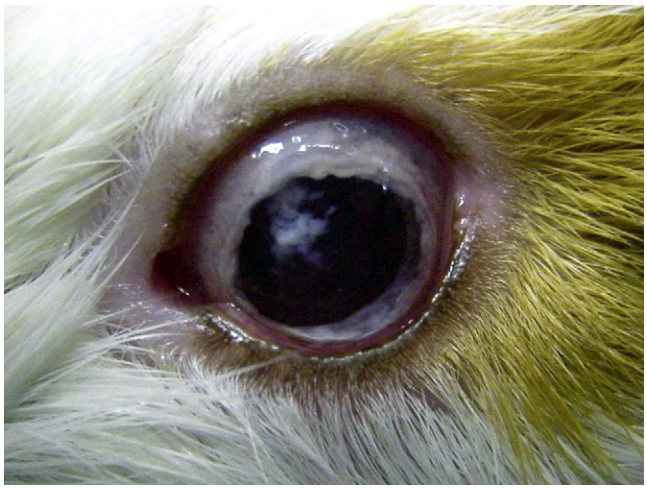

Fig. 37-7 Osseous metaplasia of the mesectodermal trabecular meshwork in a guinea pig. White opaque material is present in the iridocorneal angle. *(From Brown C, Donnelly T: What's your diagnosis? Heterotopic bone in the eyes of a guinea pig. Lab Anim 2002; 31:23-25.)*

dermoids have been reported in guinea pigs.[52,71] Irritation from abnormal hairs causes conjunctivitis, irritation, and possibly corneal ulceration. Surgical resection is curative.

Cataracts have been reported in guinea pigs. Mature cataract can be associated with diabetes mellitus.[73a] They can be removed surgically, but the procedure is difficult because of the small size of the globe, the large size of the lens, and the difficulty of intubating a guinea pig for general anesthesia.

Osseous metaplasia of the mesectodermal trabecular meshwork occurs in guinea pigs (Fig. 37-7).[8,25] Clinically, an arc of white, opaque material is visible in the anterior chamber, covering the iridocorneal angle. Vessels may be present overlying the osseous choristoma. Hematopoietic active bone marrow is present. This is usually an incidental finding and no specific treatment is necessary.

As in other rodents and rabbits, exophthalmos in guinea pigs may be related to dental disease. A tooth root abscess of a molar may result in maxillary sinusitis and orbital disease. Careful examination of the teeth is indicated in any guinea pig with exophthalmos.

CHINCHILLAS

Chinchillas have fine ciliae on the upper and lower eyelids.[35a] The third eyelid is rudimentary. The cornea is large and highly curved. The pupil is a slit or vertical oval when constricted. The lens is very large, occupying approximately 50% of the axial diameter of the globe. Chinchillas have prominent choroidal vasculature and an anangiotic retina. The retina has no fovea and no cones and a choroidal tapetum lucidum is absent. The optic nerve can be myelinated or nonmyelinated. Tear production is low, with an average reported value of 1.07 mm/min. The phenol red thread tear test can be used to measure tear production. An average of 14.0 mm/15 seconds has been reported.[48a] A menace response and dazzle reflex is absent in most chinchillas with normal eyes. Gram positive bacteria are predominant in the conjuntival flora of normal chinchillas. The most frequent bacteria found are *Streptococcus* species and *S. aureus*. Average intraocular pressure measured by applanation tonometry was 18.5 ± 5.75 mm Hg in one study, and 17.71 ± 4.17 in another study. Average intraocular pressure measured with rebound tonometry was 2.9 ± 1.8 mm Hg.[48a] Cataracts and asteroid hyalosis have been reported in older animals.[33]

The premolar, molar, and incisor teeth in chinchillas, as in guinea pigs, continue to grow and erupt throughout the animal's life. Insufficient wear can result in elongated roots of the premolar and molar teeth, resulting in progressive orbital disease including epiphora, decreased retropulsion of the globe, and proptosis (see Chapter 32).[16] Computed tomography is more sensitive than radiography in detecting early lesions.[15] The prognosis for advanced disease is poor. Exophthalmos secondary to an orbital *Taenia coenurus* has been reported. Surgical excision was curative.[28]

RATS, MICE, AND HAMSTERS

The retinas of rats, mice, and hamsters are holangiotic, with arteries and venules radiating from the optic nerve like spokes on a wheel.[73] Rats have three lacrimal glands: intraorbital, extraorbital, and harderian.

Inbred strains of rats and mice are commonly used in commercial laboratories to study naturally occurring ophthalmologic diseases. Diseases involving all parts of the eye have been described. Common abnormalities include retinal degeneration, as in the Royal College of Surgeons (RCS) rat strain[66]; microphthalmos; and cataracts. In addition to specific genetically determined ocular abnormalities in inbred strains, other spontaneous abnormalities occur. Ophthalmic examination of 6,000 Sprague-Dawley rats revealed a focal linear retinopathy in 3% and a fundic coloboma in 0.5%.[30] Spontaneous corneal degeneration has been described in Sprague-Dawley and Wistar rats,[7] and corneal dystrophy has been described in Fischer 344 rats.[36] Experimental infections also lead to ophthalmic abnormalities. Blepharitis with crust formation in the medial canthus and partial periocular alopecia were observed in mice experimentally infected with *Trypanosoma brucei*.[61]

Conjunctivitis in mice can be caused by numerous infectious agents, including *Pseudomonas aeruginosa, P. pneumotropica, Salmonella* species, *Streptobacillus moniliformis, Corynebacterium kutscheri,* Lancefield group C streptococci, *Mycoplasma pulmonis,* mousepox or ectromelia virus, Sendai virus, and lymphocytic choriomeningitis virus.[6,33] Bacteriologic culture and sensitivity testing may be indicated in individual rats and mice with persistent conjunctivitis. Epiphora in rats and mice can be caused by dental problems. Nasolacrimal duct obstruction can result from overgrowth or malocclusion of the incisors.

Chromodacryorrhea is red staining around the eyes seen in rats and mice. Inflammation of the harderian gland causes secretion of tears pigmented with porphyrin. Sialodacryoadenitis virus is a highly contagious coronavirus that replicates in the respiratory tract epithelium, causing rhinotracheitis, bronchitis, and alveolitis. The virus also causes sialoadenitis of the submandibular and parotid salivary glands and necrotizing dacryoadenitis of orbital and harderian lacrimal glands. Exophthalmos, epiphora, and keratoconjunctivitis may result. The infection usually resolves within 1 week in immunocompetent animals. In a study of athymic rats, infection persisted for more than 3 months, indicating that normal T-cell function is required for host defenses against the virus.[72] Infection with sialodacryoadenitis virus may also result in uveitis and multifocal retinal degeneration.[33] Complications from infection include corneal opacification, anterior and posterior synechiae, cataract, and glaucoma. Specific therapy is not available, and treatment is

supportive only. Other causes for red tears include infection with parainfluenza virus type 3 or Sendai virus as well as pain or stress. Ammonia vapor from soiled bedding can act as an ocular irritant, predisposing animals to secondary infection. Keeping the housing areas well ventilated is important in preventing infection with sialodacryoadenitis virus.

Of clinical significance is the effect of xylazine on the lens in rats and mice. A reversible cataract has been observed after systemic use of xylazine. Transcorneal water loss and altered aqueous humor composition caused by corneal exposure have been suggested as a pathogenesis of cataract formation.[12]

In hamsters, keratoconjunctivitis can result from ammonia vapor from soiled bedding. Dental problems including tooth root infection may result in facial or retrobulbar abscesses, with hemifacial swelling, proptosis, and exposure keratitis as common sequelae. Treatment with systemic antibiotics is often unrewarding, and such abscesses frequently lead to the animal's death.[33]

Insidious globe enlargement with loss of vision has been reported in four hamsters.[19] Ophthalmic examination of these hamsters revealed enlarged globes in both eyes, widely dilated pupils, lack of pupillary light reflex, a small optic nerve, and pale retinas. Histopathologic results suggested chronic open-angle glaucoma. Treatment of the suspected glaucoma was not attempted.

SUGAR GLIDERS

Sugar gliders have an avascular retina. Only a small residual tuft of fluorescein-impermeable vessels projects from the optic disk into the vitreous.[11]

Sugar gliders have prominent globes that are susceptible to trauma. Corneal ulcers may result from intraspecies fighting.[54] A retrobulbar abscess can result from a bite wound to the face or a molar root abscess. Corneal lipid infiltration may form in juvenile sugar gliders when the mother is fed a diet that is too high in fat. Although not reported, cataract formation is seen clinically.

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