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Rabbit and Rodent Ophthalmology
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Ocular disorders of rabbits and rodents are caused by genetic defects, infections, nutritional deficiencies, congenital malformations, as well as environmental and management problems. Their investigation and diagnosis rely on implementation of diagnostic approaches and instrumentation used commonly for other companion animal species. Special considerations of importance include the small size of the eye in some species, necessitating use of magnification for accurate assessment, and the vulnerability of some species to complications of parenteral and even topical antibiotic and anti-inflammatory therapy.

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Pet rabbits and rodents occasionally develop ocular disorders that resemble those which occur under laboratory conditions. Causes include genetic defects, infections, nutritional deficiencies, congenital malformations, as well as environmental and management problems. Owners notice ocular signs such as watery or mucoid discharge, ocular opacities, blepharospasm, or alterations in globe size. Intraocular disorders and those that affect the orbit are less apt to be noted.

Vision assessment in pet laboratory animals is limited in large part by their natural lifestyles and methods of management. Most are confined to familiar quarters and rarely venture into unknown areas. Nocturnal species such as rats, mice, and hamsters may avoid brightly lighted conditions where owners are more apt to observe them. Ocular examination of some species is made difficult by the small size of the eye, restraint difficulties, and the use of large conventional diagnostic instruments.1 Examination of the external eye and adnexa is greatly aided by the use of magnification (eg, a 20- or 30-diopter [D] condensing lens, a simple pocket magnifier, or head loupe). Several accessory ocular diagnostic tests commonly used in dogs and cats may also be used in small laboratory animals. Fluorescein dye examination of the cornea and nasolacrimal system is possible even in very small animals. Tonometry and Schirmer tear test evaluation are possible in rabbits. Normal intraocular pressure in rabbits is 25 mm Hg. Normal Schirmer tear test values for the rabbit are approximately 5 mm wetting per minute without topical anesthesia.2 Exfoliative conjunctival cytology collection and examination is both possible and useful in all laboratory species. Note: Topical anesthesia should be used only sparingly in these small animals because systemic toxicity is possible. Fundus examination of rodents and rabbits may be performed by effecting mydriasis with 1% tropicamide (Mydriacyl, Alcon Laboratories, Fort Worth, TX) and using indirect ophthalmoscopy with a 30- to 40-D indirect condensing lens. Pigmented irides of rodents may resist dilation because of binding of the mydriatic agent to uveal melanin. In such animals, mydriasis may be encouraged by instillation of one drop each of 1% atropine and 10% phenylephrine (AK-Dilate, Akorn, Abita Springs, LA) three times within a 15-minute period.

Although basic ocular and orbital anatomy of rabbits and rodents resembles that of a dog and cat, clinically relevant species variations must be appreciated in order to accurately assess ocular problems.

Rabbits
Anatomical features in rabbits include (1) a superior rectus muscle normally evident through the superior bulbar conjunctiva; (2) a single, large, inferior nasolacrimal punctum opening to a tortuous nasolacrimal duct of variable diameter along its course; (3) four glands within the orbit (lacrimal, intraorbital, Harderian, and gland of the nictitating membrane); (4) a merangiotic fundus in which retinal vessels are present only nasal and temporal to the optic disc (see R. Bell-
horn's article, this issue); (5) a heavily myelinated optic disc located superior to the visual axis with a large, deep, central depression; and (6) an extensive orbital venous plexus.1,3-8 Eyelid separation in rabbits occurs about 10 days postnatally. Important ocular problems in rabbits include epiphora, conjunctivitis/blepharoconjunctivitis, glaucoma, uveitis, and conjunctival pseudopterygium. Notable but less common problems include congenital eyelid abnormalities, cataract, corneal opacities, and ocular colobomas.

In rabbits with epiphora, a milky aqueous discharge is present which causes crusting of the facial hair near the medial canthus (Fig 1). The cause of the epiphora is either excessive lacrimation or inadequate tear drainage. Excessive lacrimation results from ocular irritation; a variety of external and intraocular disorders may be responsible. Acquired nasolacrimal duct obstruction may accompany or follow chronic rhinitis and/or dacryocystitis that may result from infection (Pasteurella) or possibly dental disease (tooth root inflammation).9 Skull radiography may be indicated. Relief of the obstruction may sometimes be accomplished by nasolacrimal duct irrigation with saline injected through the single inferior punctum with a conventional 22-gauge nasolacrimal cannula or an intravenous catheter. Simple nasolacrimal irrigation may not relieve acquired structural stenosis. Cannulation of the nasolacrimal duct with monofilament nylon (0 to 2-0) may relieve the obstruction; however, cannulation should be done with caution as it is often not possible in normal rabbits because of the natural bends and variable diameter of the duct.3,5 Presumed bacterial rhinitis and dacryocystitis may be responsive to systemic antibiotic therapy (enrofloxacin, 10 mg/kg twice a day intramuscularly (IM) or orally; procaine penicillin G, 60,000 IU/kg once a day IM for 10 days). Relapses occur frequently. Note: Prolonged therapy with β-lactam antibiotics may induce potentially fatal enterocolitis in rabbits.8

Pasteurella multocida is the most commonly incriminated cause of conjunctivitis in rabbits.1,5,8 This organism may be present in an otherwise normal conjunctival sac or may contaminate the ocular surface by extension from the nasal cavity through the nasolacrimal duct. P multocida also causes a wide variety of other manifestations that include orbital cellulitis, uveitis, pneumonia, otitis media and interna, vulvovaginitis, subcutaneous abscesses, pyometra, orchitis, and generalized septicemia. Major or minor conjunctivitis may accompany any of these signs or may occur in their absence. Conjunctivitis attributable to pasteurellosis in rabbits can be treated with topical chloramphenicol, ciprofloxacin, or gentamicin ophthalmic solution or ointment four times daily combined with systemic broad-spectrum antibiotic therapy (see above), if ineffective alone. The prognosis for cure of systemic pasteurellosis is poor. Signs of infection resolve with therapy and may recur following stress. Many animals appear to be chronic carriers. Other reported causes of conjunctivitis include Staphylococcus aureus,12 chlamydia,13 and Pseudomonas species.14

Blepharitis may be caused by infection with the spirochete Treponema cuniculi, the causative agent of rabbit syphilis.1,13 The vulva and prepuce are most commonly affected, but lesions may involve the eyelids, lips, nares, and anus. The disease is transmitted by young rabbits by an infected dam. Diagnosis is confirmed by identification of spirochetes and examination of skin scrapings. Infection may be successfully treated with three injections of 40,000 IU per kg of body weight of benzathine penicillin G/procaine penicillin G (Bicillin, Wyeth-Ayerst Laboratories, Philadelphia, PA) given at 7-day intervals. Eradication of treponematosis may require treatment of even asymptomatic animals in the colony. Squamous cell carcinoma mimicking blepharitis has been reported (Fig 2).5

Blepharoconjunctivitis may occur as a pri-
primary entity or may ensue following self-trauma stimulated by discomfort from other ocular disorders. Myxomatosis (a viral disease enzootic in wild rabbits in the Western United States, Europe, South America, and Australia) rarely occurs in domestic rabbits. This infection is transmitted by fleas and mosquitoes. Subcutaneous swellings involve orifices, eyelids, and the face. Diagnosis is presumptive based on clinical signs and typical gross and microscopic lesions. Virus isolation is required for definitive diagnosis. Specific therapy is unavailable and mortality is high. Mosquito and flea control is needed for prevention. Rabbit poxvirus may cause similar cutaneous lesions. Blepharoconjunctivitis associated with self-trauma merits diagnostic and therapeutic pursuit of other occult ocular disorders, eg, keratitis or uveitis. Other noninfectious causes of conjunctivitis, blepharitis, and ocular discharge in rabbits include environmental influences (dusty and filthy bedding), trauma, and eyelid malformations (entropion, distichiasis, and trichiasis). Entropion should be surgically corrected when evident. Distichia can be removed by simple epilation, electroepilation, or cryoepilation but only after all other causes of ocular irritation have been ruled out. Deformed cilia have been reported to cause trichiasis and secondary keratitis in French Rex Rabbits.

Primary glaucoma occurs in laboratory rabbits from New Zealand White stock and occasionally in apparently cross-bred pet rabbits. In New Zealand White Rabbits, this condition is inherited as an autosomal recessive trait with incomplete penetrance. Onset may be as early as the first month after birth, but it more commonly develops between 3- and 6-months of age. Unilateral or bilateral buphthalmos, generalized corneal edema, and blindness should prompt the clinician to measure intraocular pressure with a Schiotz tonometer or an applanation tonometer (Tonopen, Mentor, Norwell, MA) (Fig 3). Normal intraocular pressure is 25 mm Hg. Medical therapy is of limited benefit. The available topical miotic and β-blocking drugs may not reduce intraocular pressure soon or adequately enough to preserve vision. The recently introduced topical carbonic anhydrase inhibitor dorzolamide (Trusopt, Merck, West Point, PA) may be partially effective when administered two or three times a day. Surgical options include cyclocryotherapy and diode laser cycloablation for potentially sighted eyes and enucleation with silicone prostheses provided there is no orbital sinus involvement. Serious intraoperative hemorrhage from the orbital venous sinus is a possible complication of enucleation; more commonly, the bleeding is self-limited before serious consequences ensue.

Uveitis is associated with ocular trauma, ulcerative keratitis, systemic infection (especially pasteurellosis, staphylococcal disease), and spontaneous lens rupture (phacoclastic uveitis). The cause of the latter condition is unclear; although some affected rabbits harbor Encephalitozoon cuniculi organisms within their ruptured lenses. Control of uveitis in rabbits is similar to other species; topical anti-inflammatory and antimicrobial agents are indicated (eg, 1% prednisolone...
acetate, 1 g q three times daily [PredForte Allergen, Irvine, CA] or 0.3 ciprofloxacin [Ciloxan, Alcon Laboratories Inc, Fort Worth, TX]). The cause and extent of the intraocular inflammation determine the ultimate prognosis for resolution; severe infectious and phacoclastic uveitides often require enucleation.

Pseudopterygium is a problematic, but relatively uncommon, idiopathic condition of bilaterally symmetrical annular conjunctival overgrowth of the cornea. The conjunctiva is not adherent to the corneal epithelium but rather lies on its surface (Fig 4). Conjunctivitis is usually absent or minor at most. The objective of treatment is to prevent or reverse vision impairment from obstruction of the visual axis. Simple surgical excision of the advancing conjunctiva is deceptively easy; recurrence, however, is frequent.

Corneal opacities may be caused by ulcerative and nonulcerative keratitis caused by trauma and eyelid abnormalities; inherited corneal dystrophy (Dutch-Belted Rabbits); and dietary and inherited ocular lipidosis. Corneal ulceration is treated similarly to other species with topical antimicrobial and mydriatic/cycloplegic drugs (eg, neomycin-bacitracin-polymyxin B [Neosporin, Burroughs-Wellcome, Research Triangle Park, NC], 1 qqt four times daily; 1% atropine 1 qqt twice daily).

Clinically significant primary cataracts (ie, those not associated with other ocular diseases) are uncommon in pet rabbits. Secondary cataract formation commonly follows chronic uveitis. Diabetic rabbits may develop osmotic cataract similar to dogs. Cataract removal by phacoemulsion is possible. Rarely, heritable cataract, cyclopia, persistent hyperplastic primary vitreous, lens coloboma, and optic nerve coloboma have been reported.

Figure 4. Rabbit with pseudopterygium covering peripheral two-thirds of cornea. See Figure 29 on page 120.

**Rodents**

**Rats**

The relevant anatomic features of the rat eye include (1) extensive orbital venous plexus; (2) three lacrimal glands per eye: intraorbital, extraorbital, and harderian (associated with the third eyelid); (3) transient persistence of hyaloid artery in weanlings; (4) an extremely large spheric lens; and (5) holangiotic predominantly rod retina with radial arterioles and venules (see article by R. Bellhorn, this issue). In weanlings, eyelid separation occurs at postnatal days 12 to 16. Common ocular problems of rats include epiphora, conjunctivitis, keratoconjunctivitis, dacryoadenitis, and retinal degeneration. Epiphora may be caused by excessive lacrimation or nasolacrimal duct obstruction. Excessive lacrimation may be associated with bacterial or viral conjunctivitis or ocular irritation from soiled bedding. The harderian glands of mice, rats, and other rodents produce porphyrin pigments which stain the periocular hair reddish-brown if it is white or lightly pigmented when epiphora is present (chromodacryorrhea). Owners may mistake this red pigment for “bloody tears.” Soiled bedding generates ammonia vapors which act as a direct ocular irritant causing conjunctivitis and predisposing to secondary bacterial infection. Good husbandry with frequent cage cleaning and bedding replacement minimizes this problem.

Many of the common bacterial and viral agents that cause mild to moderate conjunctivitis in rats also cause subclinical to clinical upper and lower respiratory tract disease. These include Streptococcus pneumoniae, Pasteurella pneumotropica, Mycoplasma pulmonis, Pseudomonas aeruginosa, and Sendai virus. Bacterial culture and sensitivity testing might be indicated in individual instances especially if several animals are affected. Systemic antibiotic therapy may be indicated for animals with significantly debilitat-
ing signs. Recommended antibiotics include 6 to 10 mg/kg of oxytetracycline once or twice a day IM; 20,000 mg/kg of procaine penicillin G orally once daily; 15 to 20 mg/kg of tetracycline orally two to three times daily by mouth; or 2 to 4 mg/kg of tylosin once to twice daily IM.

Sialodacryoadenitis virus (SDAV) and rat coronaviruses cause inflammation of salivary and lacrimal glands and, frequently, ocular signs. Exophthalmos, epiphora, keratoconjunctivitis, uveitis, and multifocal retinal degeneration occur in various combinations. Sialodoacryoadenitis caused by sialodacryoadenitis coronavirus which is highly contagious and spreads rapidly by contact, aerosol, and fomite transmission. Rats of any age are susceptible to infection. When infection is enzootic within a colony, only young rats are affected. Infection lasts about 1 week, until the rats undergo seroconversion with no carrier state. No specific therapy is available. The best treatment is supportive. Permanent ocular clinical sequelae include corneal opacification, anterior and posterior synechiae, secondary cataract, secondary glaucoma, and multifocal retinal degeneration.

In laboratory animal colonies, dacryoadenitis often follows blood sample collection by cannulation of the orbital venous plexus with a capillary tube. This sequela may be avoided by blood collection from other sites.

Vitamin A deficiency causes chronic keratoconjunctivitis and xerosis. Systemic replacement therapy with vitamin A may resolve clinical signs. The use of commercially prepared diets should avoid this problem. Nasolacrimal duct obstruction and subsequent epiphora results from malocclusion of the incisor teeth. The gums and distal nasal cavity may become inflamed and secondary swelling may cause distal obstruction of the duct. Corrective dentistry usually improves the condition if accomplished before nasal cavity deformity occurs. Other reported causes of epiphora include stress, vitamin deficiency (riboflavin, pantothenic acid), water deprivation, and lack of grooming.

Blinding primary cataracts are relatively uncommon in rats; hereditary cataracts occur in some strains. Secondary cataract commonly develops as a sequela to anterior uveitis or generalized retinal degeneration. Grossly visible albeit transient cataracts can occur in rodents following prolonged eyelid separation (eg, during anesthesia) as a result of temperature and/or osmotic changes in the anterior chamber and lens. These usually resolve with recovery from anesthesia and/or reduced corneal exposure. Nutritional cataracts may develop in rats fed diets with continuous excessive sugar levels (galactose, sucrose, or xylose). The feeding of commercially prepared diets to pet rats should avoid this problem.

Primary and secondary retinal degenerations occur in rats. Primary retinal degenerations are inherited disorders. Secondary retinal degenerations follow excessive light exposure (duration and/or intensity, especially in albino strains) and SDAV infection. The SDAV retinopathy is usually multifocal, but rarely causes bilateral blindness. Phototoxic retinopathy, however, predictably causes blindness. It may be avoided by maintenance of approximately 12-hour light/dark cycles. Pet rats should have access to areas where they may retreat from light.

Secondary glaucoma occurs in rats following chronic uveitis, most commonly caused by SDAV. Congenital malformations in laboratory rats are many and varied. Microphthalmos alone or accompanied by cataract or retinal dysplasia may be a sporadic congenital defect or may be inherited. It must be distinguished from atrophy of the eye following severe uveitis.

Enucleation of the eye in rodents could result in serious hemorrhage from the orbital venous plexus, therefore, careful intraoperative hemostasis is recommended.

Mice

The clinically important ocular anatomical features of the mouse resemble those of the rat. Eyelid separation occurs at 13 to 14 days postnatally. Important ocular problems in mice include epiphora, conjunctivitis and keratoconjunctivitis, and retinal degeneration. The causes of epiphora in mice are similar to those for rats, including environmental contamination with ammonia, dental problems, dacryoadenitis, and infections. Pasteurella pneumotropica causes dacryoadenitis. Infectious agents incriminated in murine conjunctivitis include Pasteurella pneumotropica, Pseudomonas aeruginosa, Salmonella sp, Streptobacillus moniliformis, Streptococcus of the Lancefield serological classification group C, Corynebacterium dopschii, Mycoplasma pulmonis, and several viruses (mousepox/ectromelia, Sendai, and lymphocytic
choriomeningitis) (Fig 5). Bacterial culture and sensitivity testing and/or viral serology may be indicated in selected cases.

Cataracts occur as solitary genetic defects as well as combined with multiple ocular defects. Secondary cataract may result as a sequela of uveitis, retinal degeneration, and prolonged eyelid separation. Posterior lens capsule rupture in neonatal mice is inherited as a simple recessive trait in some strains.

Retinal degenerations in mice occur as genetic defects as well as associated with phototoxicity and retinal inflammation.

Many heritable ocular defects have been identified in laboratory strains of mice that may occasionally afflict pets include eyelid malformations, nanophthalmos, microphthalmos, cataract, retinal dysplasia, and optic nerve hypoplasia.

Guinea Pigs

Important anatomic features of the guinea pig eye include (1) a rudimentary nictitating membrane, which is merely a fold of conjunctiva near the medial canthus; (2) a large intraorbital lacrimal gland which occupies the lateral and anterior ventral aspects of the orbit; (3) an extensive zygomatic salivary gland which occupies the posterior, medial, and superior aspects of the orbit; and (4) a paurangiotic retina, in which a few inconspicuous capillary loops extend into the retina from near the optic disc (see article by R. Bellhorn, this issue). Guinea pigs are born precocious with open eyelids.

Clinical ocular problems of guinea pigs include blepharitis; corneal ulceration; conjunctivitis; "pea-eye," an inferior conjunctival mass; corneal and scleral mineralization; cataract; and panophthalmitis.

Blepharitis in young animals is sometimes caused by dermatophytes (Trichophyton, others). Treatment with topical antifungal drugs (miconazole, tolnaftate, or thiabendazole in paraffin) is usually curative.

Corneal ulceration, usually traumatic in origin, is diagnosed by fluorescein stain application and treated with topical broad-spectrum antibiotics (e.g., 0.3% ciprofloxacin 1 qqt three times a day).

Causes of conjunctivitis include infections and ascorbic acid deficiency (scurvy). Chlamydia psittaci is the best-documented infectious agent causing conjunctivitis in this species. Infection is primarily venereal, with the newborns infected at birth, causing "inclusion conjunctivitis" characterized by mild chemosis, follicular formation, and serous ocular discharge. In enzootically infected colonies, outbreaks may be cyclic. Lesions usually resolve within a month and treatment is considered unnecessary. Recovered animals may be resistant to reinfection. Diagnostic confirmation may be achieved by identification to typical intracytoplasmic chlamydial inclusions in epithelial cells obtained by conjunctival scraping and stained with Jimenez, Macchiavello's, or other cytologic stain. The zoonotic potential is uncertain. For severely affected animals, topical therapy with a broad-spectrum antibiotic (e.g., tetracycline [Achronmycin, Storz, St. Louis, MO]) for 7 to 10 days may be indicated.

Infectious conjunctivitis is potentially caused by numerous bacteria, including streptococci, Micrococcus, Staphylococcus aureus, Pasteurella multocida, Bordetella bronchiseptica, Proteus, and pneumococci. Conjunctival cytology indicative of bacterial conjunctivitis shows many neutrophils with or without intracellular bacteria. Topical broad-spectrum antibiotic therapy for 7 to 10 days is indicated.

Ascorbic acid deficiency causes conjunctivitis characterized by a flake-like ocular discharge. Improper diet, malocclusion, respiratory or renal disease may precipitate clinical signs.

Figure 5. Adult mouse with blepharokeratoconjunctivitis of indeterminate cause. See Figure 30 on page 120.
orexia causes the classic signs of scurvy such as ocular discharge, hunched posture, joint pain, rough pelage, and soft stool. Treatment consists of ocular cleansing as needed and correction of ascorbate deficiency by parenteral (10 mg/kg IM) and oral routes. The clinician must, of course, ascertain and correct the cause of the anorexia.

Corneal and scleral calcification occurs in guinea pigs unassociated with inflammation. Mineralization in other tissues may occur simultaneously. Complete physical examination is indicated. Specific ocular treatment is usually not required.

Occasionally, adult guinea pigs, especially pure-bred American shorthairs, develop a ventral subconjunctival nodule that protrudes from the inferior conjunctival sac in one or both eyes, described as pea-eye by some fanciers. Examination of biopsy specimens has shown them to be portions of the lacrimal or zygomatic glands. Some nodules cause ventral ectropion, lagophthalmos, and secondary axial corneal degeneration (Fig 6). Despite this, treatment is usually unnecessary. Surgical removal for cosmetic purposes is discouraged.

Cortical cataracts of suspected genetic origin have been commonly noted in guinea pigs of several breeds, including but not limited to Abyssinians and English shorthairs (Fig 7). Experimental 1-tryptophan deficiency produces cataracts, but the importance of nutritional deficiency in pet animals is uncertain.

Bacterial septicemia may be reflected in panophthalmitis. Streptococcus zooepidemicus causes cer-
resembles that of the pigmented rat. Facial dermatitis commonly afflicts gerbils. Signs include erythema, alopecia, and crusting of the external nares, forepaws, muzzle, and periocular areas. Causation is multifactorial; reduced grooming and cage bedding have been implicated. Ambient temperature affects gerbil grooming behavior but optimal temperatures for this have not been determined. Sand may be superior to other bedding materials in reducing incidence of facial dermatitis.

Other Rodents

Chinchillas have a rudimentary third eyelid, a vertical slit pupil, and an anangiastic retina. Asteroid hyalosis and cataract have been noted in aged animals. Traumatic corneal ulceration is relatively common. Treatment involves topical broad-spectrum antibiotic instillation; systemic antibiotic administration is indicated for serious ulceration.

Degus (Octodon degus) have anangiastic retinas. Neonatal-onset cataract possibly associated with diabetes mellitus has been reported.

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