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Survival Methods for the Equine Practitioner in Equine Ophthalmology

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1. Introduction

Equine practitioners can develop solid expertise in field ophthalmology with practice and effort. Getting known as someone who has a special interest in eyes is a great way to build a client base: eye problems are a serious concern to owners and are not typical “turf” for non-veterinary caregivers like massage therapists, holistic therapists, lay dentists, and farriers, although there are equine iridologists out there! Some problems will always have to be referred to specialists, but the average practitioner can examine, diagnose, and treat 85% of the eye problems that are seen in the field.

Items to carry for diagnosis and treatment of eye problems:

- direct ophthalmoscope
- transilluminator, and/or penlight
- 14-D magnifying lens
- digital camera (4.0-megapixel camera or higher)
- Hand-held slit-lamp (optional)
- applanation tonometer (optional)
- fluorescein and rose bengal dye strips (carry right in scope case)
- Schirmer tear test strips (carry right in scope case)
- tropicamide — topical agent to dilate pupils
- proparicaine (topical anesthetic); carbocaine (injectible local anesthetic)
- 30-ml bottles of 1 part Betadyne to 50 parts saline. Small bottles of sterile saline are available through www.floridainfusion.com; the 2% solution can be mixed in house. Larger pour bottle of same for cleaning periorbital region
- sterile cotton swabs bundled six swabs to a pack
- sterile individual polyester swabs
- sterile 4 × 4 gauze, non-sterile 4 × 4 gauze, sterile gloves
- scalpels blades, packed in sterile sleeves (any size as blunt end is used)
- 2-mm biopsy punches (for occasional “scooping” of foreign bodies)
- slide boxes with four microscope slides to a box (for cytology specimens)
IN-DEPTH: OPHTHALMOLOGY—SURVIVAL METHODS FOR PRACTITIONERS

- formalin in small jars (for preserving biopsy specimens)
- “write on” plastic bags—4 × 6 in (to dispense medications and clarify treatment schedules)
- thioglycollate culture broth tubes or agar plates, Porta-cult transport media
- serum separator tubes (to prepare autologous serum)
- fine suture material: 1-0 through at least 4-0, some use up to 6-0
- sedatives (xylazine, butorphanol tartrate, detomidine)
- subpalpebral lavage systems
- adhesive tape (to make butterfly holding wings for lavage systems)
- 20-gauge catheters and IV catheter caps (to use with lavage system)
- tongue depressors (to use with lavage systems)
- tuberculin and 3-ml syringes as well as larger sizes
- 25- and/or 23-gauge needles
- prescription pad
- halogen light on tripod or hook, and headlamp

Drugs to carry in your truck or buy locally for treatment of eye problems.

- antibiotic ointments: triple antibiotic, gentamicin, chloramphenicol—most others need to be scripted out at pharmacies
- corticosteroid ointments: triple antibiotic with 1% hydrocortisone, triple with 0.1% dexamethasone
- antifungal medications to buy from pharmacies: OTC Monistat, clotrimazole, silver sulfadiazine (BE CAREFUL—may be best for temporary use until compounded products can ship; off-label use; may be irritating)
- atropine ointment
- injectable antibiotics to make fortified solutions or inject SC: cephazolin 1-g bottles, gentamicin 100 mg/ml, amikacin 50 or 250 mg/ml
- 10-ml bottles of saline for mixing fortified solutions
- artificial tears
- cyclosporine ointment
- timolol maleate 0.5% drops
- be ready to prescribe many other drugs such as 5% NaCl, timolol maleate combined with dorzolamide (0.1% diclofenac, flurbiprofen, 0.3% tobramycin, oxacillin, ciprofloxacin, itraconazole, or idoxyuridine. Some specialized drugs may be kept at your clinic and dispensed/prepared on an as needed basis, like EDTA, topical in adefinial tears, fortified antibiotic preparations, etc. Serum will need to be centrifuged at your clinic. Some drugs (e.g., 1% liquid miconazole, idoxyuridine, itraconazole) are available only through compounding pharmacies. University pharmacies are often good sources for unusual ophthalmic preparations.
- non-steriodal anti-inflammatory drugs (NSAIDs): injectable and oral flunixin meglumine and phenylbutazone
- systemic corticosteroids: injectable and oral dexamethasone

Some tips regarding examination and diagnostic tests:

- Owners calling about horses with painful eyes or eye trauma should be told to have a dark examination area available and to have four bales of shavings or hay ready to use as head rest for standing surgery/diagnostics. A stool or other step stand may help if the horse is tall or the vet is short!
- Horses are often most relaxed if examined initially in stalls. Instruct the handler to stand on the side opposite to the side being examined. Always examine both eyes fully, even if there is an obvious problem in just one. Having the holder tilt the head one direction or another by applying pressure on the ear will help expose desired site of the eye.
- Chemical restraint is often needed but can be short lived. Be organized with all diagnostic and treatment supplies at hand before sedating—strive to work quickly! Butorphanol can cause troublesome head tremors.
- Use a small syringe to draw up and administer topical drugs (anesthetics and mydriatics). If the horse is sedated, distribution of topicals will be facilitated by rolling the head away from the examiner slightly so the target eye is more vertical. This position will aid in accurate “aiming” of medication that is sprayed or dripped on the surface of the globe.
- Get in the habit of performing a systematic evaluation of all regions of the eye, using information discussed below on the anatomy of each region. Use a penlight, direct ophtalmoscope, and slit lamp if available. Even if the problem is obvious, slow down and look at all regions: orbit, lids, conjunctiva, tear film, cornea, anterior chamber (AC), iris, lens, posterior segment, fundus, and optic nerve. Assess cranial nerve function. Assess visual axis using dazzle, direct and consensual pupillary light response (PLR), menace, and (when appropriate) blindfold obstacle maze navigation. Use dial settings to change lenses on the direct ophthalmoscope to focus on deeper layers of the globe. Use (or refer) for tonometry in suspected glaucoma cases.
- Vascularization of the cornea occurs in many disease processes. Superficial vessels are individual long branching vessels that lie within the epithelium or anterior stroma. Deeper, mid-stromal vessels are multiple—they are...
short and straight and may form a brush border on the limbus. Very deep vessels that rest on the Descemet’s membrane interface are individual long branching vessels.

- Buy at least one atlas or book on equine ophthalmology (several are listed in the references). Studying many fundic photos will hone skills in deciding what is “normal” and what is “abnormal.” Look at lots of “normal” eyes! At least 20% will have some finding of record!

- Develop a recording system to write down your observations. Start taking digital photographs of the eyes. With a little practice, and judicious cropping, it is easy to get great images using a camera with 4.0 megapixels or higher resolution. Archiving the images in the horse’s medical record and rephotographing at later intervals will help determine progress of your cases.

2. Ocular Examination

Reflex Testing

Making a quick, threatening motion toward the eye to cause a blink response and/or a movement of the head tests the menace response. This is a crude test of vision. Care is taken not to create air currents toward the eye when performing this test. Horses have a very sensitive menace response.

The horse should also quickly squint or “dazzle” when a bright light is abruptly shown close to the eye.

The palpebral reflex is tested by gently touching the eyelids and observing the blink response. Vision could be further assessed with maze testing with blinkers or a large towel alternatively covering each eye. The maze tests should be done under dim and light conditions. Barn aisles that have been cleared of hazards are good stages for maze testing; blindfolded horses that are visual in an ordered and systematic manner. It is important to approach each eye problem in the horse in an ordered and systematic manner. The majority of cases can be diagnosed by using standard ophthalmic clinical examination techniques. Infrared sedation, a nose or ear twitch, and supraorbital sensory and auriculopalpebral motor nerve blocks may be necessary to facilitate the examination.

The auriculopalpebral nerve (motor nerve to the orbicularis oculi muscle) can be palpated under the skin and blocked with 2–3 mL of lidocaine just lateral to the highest point of the zygomatic arch.

The frontal or supraorbital nerve (sensory to the medial two thirds of the upper lid) can be blocked at the supraorbital foramen. This foramen can be palpated medially at the superior orbital rim where the supraorbital process begins to widen. Line blocks can be used near the orbital rim to desensitize other regions.

Schirmer tear testing is a method to measure reflex tearing and should be used for chronic ulcers and eyes in which the cornea appears dry. The Schirmer tear test must be done before instillation of any medications into the eye. The test strip is folded at the notch and the notched end inserted over the temporal lower lid margin. The strip is removed after 1 min, and the length of the moist end is measured. Strips are frequently saturated in horses after 1 min, with values ranging from 14 to 34 mm wetting/min considered normal. Values <10 mm wetting/min are diagnostic for a tear deficiency state.

Corneal cultures using microbiologic culture swabs should be obtained before placing any topical medications in the eye. The swabs should be gently touched to the corneal ulcer and plated directly on a biplate or submitted for culture in transport media. Daeron swabs come packaged in sterile packs of two. These tightly woven synthetic swabs are superior to cotton swabs for culture because they do not leave fiber remnants that may confuse subsequent cytology analysis.

As an alternative method, one author (A.E.D.) uses a double-wrapped sterile scalpel blade for corneal cultures in ambulatory settings. The blade is applied to the target surface and dropped into a thiglycollate broth tube. The broth is carried in the truck and placed in an incubator at days end and observed overnight for turbidity. Samples with broth growth are plated out for microscopic analysis and in house sensitivity testing. A special sensitivity wheel stocked with discs that elute common antibiotics used against eye infections (cephazolin, chloramphenicol, gentamicin, amikacin, ciprofloxacin, oxacillin, and tobramycin among others) is kept at the clinic and used just for ocular sensitivity analysis. Useful results are often available in as little as 48 h when cultures are done in house; this approach is practical solution to the challenge that...
practitioners face with the long lead time needed to ship out culture samples and wait for results from reference laboratories.

Corneal scrapings to obtain cytology specimens to detect bacteria and deep fungal hyphal elements can be obtained at the edge and base of a corneal lesion with topical anesthesia and the handle end of a sterile scalpel blade. Superficial swabbing cannot be expected to yield the organisms in a high percentage of cases so removing the superficial debris can be helpful before collecting the sample. Cytology of eyelid and conjunctival masses can also be diagnostic. The foil wrapping that covers the blade can be folded around the blunt end of the blade and used as a handle for the scraping so that the sharp end remains cased. The sample is transferred to two or three glass slides by direct smear. Persistence will pay off—it takes patience to “chase” exfoliated corneal tissue around the corneal lesion and actually get it to adhere to the blade edge. Once the specimen is on the slide, it should air dry—fixative is not needed. The slides are best transported in plastic slotted boxes instead of cardboard slide carriers. It is not difficult to develop expertise in basic ocular cytology; with practice, slides can be read in-house so that treatment decisions can be made ASAP. The core concept of ocular cytology is to look for infectious elements (bacteria and/or filamentous fungi), “visiting cells” (neutrophils, eosinophils, lymphocytes and monocytes), and foreign material such as plant parts. All these elements are identifiable with Romanowskikm stain and gram stain, and patient examination using the oil immersion lens of the microscope. Slides will typically show a combination of large rafts of intact blocks of dense epithelial cells and smaller thinner sheets of individual cells. The smaller sheets, as well as areas that show individual “visiting” cells will provide the most useful information. Practitioners often forego cytology because of the expense and long turnaround time involved in using a reference laboratory. This challenge is eliminated if the cytology is performed by a technician or veterinarian right at the practice.

The cornea should be clear, smooth, and shiny. Placing fluorescein dye (use it nondiluted) in the eye to identify corneal ulcers should be routine in every eye examination of the horse. Small corneal ulcers will stain that might otherwise be undetected.

Seidel’s Test
Fluorescein can be used to detect perforated corneas or leaking corneal sutures.

Tear Film Break-Up Time
Normal tear film is continuous. Blinking maintains the tear film continuity. The tear film breaks up if blinking does not occur often enough. Dark dry spots will appear under cobalt blue–filtered light as part of normal evaporation and diffusion of tears. Fluorescein dye is placed on the cornea and not flushed off. The lid is manually blinked three times and held open to expose the tear film to evaporation. The time needed for a dry spot to appear on the corneal surface after blinking is referred to as the tear film break-up time (TFBUT). In a normal healthy eye, dry spots start occurring between blinks at ~10–12 s. A TFBUT of <10 s is abnormal and probably associated with instability of the mucin layer of the tear film.

Rose bengal dye should be used in selected cases after installation of fluorescein to identify the integrity of the tear film. Rose bengal dye strips are available at http://www.akorn.com.

To determine the patency of the nasolacrimal system, it is best to use irrigation from the nasal orifice with a nasolacrimal cannula or curved multipurpose syringe, although fluorescein dye penetration through the nasolacrimal system may also indicate patency.

The AC is best examined with a handheld or transilluminator mounted slit-lamp. The AC contains optically clear aqueous humor. Increased protein levels in the AC can be noted clinically as aqueous flare. White cells in the AC are called hypopyon, and red cells in the AC are called hyphema. Aqueous flare, hypopyon, and hyphema indicate uveitis.

The intraocular pressure (IOP) of horses is 16–30 mm Hg with applanation tonometer applanation tonometer. A mydriatic should be applied to the eye once the pupillary light response has been evaluated. The agent of choice is topical 1% tropicamide, which takes ~15–20 min to produce mydriasis in normal horses and has an action that persists for ~8–12 h. Atropine is used for therapeutic mydriasis because it can dilate the normal horse pupil for >2 wk.

The lens should be checked for position and any opacities or cataract. There are a number of lens opacities which may be regarded as normal variations: prominent lens sutures, the point of attachment of the hyaloid vessel, refractive concentric rings, fine “dustlike” opacities, and sparse “vacuoles” within the lens substance.

Cataracts are lens opacities and are associated with varying degrees of blindness. They can be congenital, secondary to previous uveitis, and be progressive or nonprogressive. In some horse breeds they may be hereditary.

Normal aging of the horse lens will result in cloudiness of the lens nucleus (nuclear sclerosis) beginning at 7–8 yr of age, but this is not a true cataract. The suture lines and the lens capsule may also become slightly opaque as a normal feature of aging.

The adult vitreous should be free of obvious opacities. Vitreal floaters can develop with age or be sequelae to equine recurrent uveitis (ERU). They are generally benign in nature.

The retina and optic nerve are examined with a direct or indirect ophthalmoscopes. The rotary lens setting of the direct ophthalmoscope should be set to 0 to examine the retina and optic nerve and to a “green” number 20 to focus on the lids and cornea.
Magnification of the fundic image with the direct ophthalmoscope is ×7.9 laterally and ×84 axially in horses. Magnification with the indirect ophthalmoscope and a 20-D lens is ×0.79 laterally and ×8.4 axially. The Panoptic™ ophthalmoscope has an intermediate level of magnification between the direct and indirect ophthalmoscopes. The fundus should be examined for any signs of ERU, such as peripapillary depigmentation. The nontapetal region ventral to the optic disc should be carefully examined with a direct ophthalmoscope, because this is the area where focal retinal scars are seen. Retinal detachments may be congenital, traumatic, or secondary to ERU and are serious faults because of their association with complete or partial vision loss.

B-scan ultrasound, computerized tomography (CT), and magnetic resonance (MR) imaging are important for evaluating intraocular and orbital lesions in the horse. CT and MR are usually performed at referral institutions. High-resolution ultrasound is a referral procedure, but B mode ultrasound using a 7.5- to 10-MHz tendon probe and conventional field units can often produce diagnostic imaging of large problems such as detached retina, lens luxation, or abnormal globe dimensions.

3. Common Eye Conditions Seen in the Field
Ocular Problems in the Foal
A newborn foal may exhibit lagophthalmos, low tear secretion, a round pupil, reduced corneal sensitivity, lack of a menace reflex for up to 2 wk, hyaloid artery remnants containing blood for several days after birth, prominent lens Y sutures, and a round optic disc with smooth margins.

Tapetal color is related to coat color and is usually blue-green but may be partially red, orange, or blue. Color dilute foals have a red fundic reflection from a lack of a tapetum and consequent exposure of choroidal vessels.

Dermoids (choristomas) are aggregates of skin tissue aberrantly located in the conjunctiva, cornea, or eyelid. Treatment would be a keratectomy for corneal dermoids and blepharoplasty for eyelid lesions.

Entropion is an inward rolling of the eyelid margin. This causes the eyelid hairs to rub on the cornea. It can be a primary problem in foals or secondary to dehydration or emaciation as in “downer foals.” It may be repaired to prevent corneal ulceration in the neonate by placing sutures at the lid margin in a vertical mattress pattern to evert the offending eyelid margin.

Lacrimal puncta agenesis or duct atresia may be unilateral or bilateral. Clinical signs are a chronic mucoid and eventually mucopurulent discharge (often copious) in a young horse. Presumptive diagnosis of duct agenesis may be made by noting a lack of a distal opening of the nasolacrimal duct or puncta at the mucocutaneous junction within the nares.

Persistent pupillary membranes (PPMs) seldom cause any visual impairment although focal lens or corneal opacities may be present. There is no therapy.

Congenital cataracts in foals are common congenital eye defects. Surgery is recommended.

Microphthalmos is a common ophthalmic congenital defect in the foal. A range of lesions may be present. The microphthalmic eye may be visual or associated with other eye problems that cause blindness.

Strabismus is deviation of the globe from its normal orientation and may be noted alone or with other congenital ocular deformities.

Congenital lens luxation is a severe eye problem that needs surgery for resolution.

Subconjunctival hemorrhage may be found in foals after dystocias.

Persistent superficial corneal erosions in the neonatal foal may be associated with decreased corneal sensation.

Iridocyclitis in the foal is generally secondary to septicemia and may be unilateral or bilateral. Fibrin, hyphema, and/or hypopyon may be present. Infectious and toxic etiologies are reported in foals. Severe unilateral, blinding, fibrinous uveitis secondary to plant toxins has been noted in primarily Thoroughbred foals and yearlings in the southern United States.

Congenital glaucoma and congenital retinal detachment are found periodically in foals and represent severe blinding eye problems.

Ocular Problems in Mature Horses Listed by Anatomical Region

Orbit

The orbit is composed of several bones forming a series of canals, fissures, and foramina that contain the globe, orbital fascia, the optic nerve and other nerves, blood vessels, muscle, fat, and glands. Prominence of the orbital rim renders it prone to injury, especially in situations where horses are kicked or trapped in narrow places like starting gates. Radiography may outline fractures of orbital bones, but image angling restrictions may make diagnostic views difficult. Ultrasonography sometimes can identify bone fragments. Severe sinus disease may invade the orbital space, and abscesses of the caudal upper molar roots may threaten orbital health. Common conditions include the following.

Exophthalmos, or anterior displacement of the globe, is associated with nictitans protrusion and lagophthalmos. It can result in corneal ulceration. Exophthalmos can be confused with buphthalmos, which is a marked increase in globe diameter associated with advanced glaucoma. Infectious, traumatic, inflammatory, or neoplastic disease processes involving the eyelids, the frontal, maxillary, and sphenopalatine sinuses, tooth roots, the guttural pouch, and nasal cavity may extend into the orbit to...
cause exophthalmos and/or strabismus. Retrobulbar hemorrhage and cellulitis associated with orbital trauma can cause exophthalmos.

Enophthalmos, or posterior displacement of the globe, is caused by dehydration, atrophy of orbital fat, orbital fractures, and ptosis.

Strabismus is deviation of the visual axis of one or both globes and can be found with neurologic deficits, visual difficulties, and abnormal head posture. In the neonatal foal, the horizontal axis of the pupil and globe is deviated slightly medially and ventrally with the eye reaching the normal adult position by 1 mo of age. Orbital asymmetry can develop secondarily to orbital rim fractures, orbital cellulitis and abscesses, orbital tumors, and orbital emphysema. Congenital strabismus (hyperopia) and dorso-medial strabismus are reported in Appaloosa foals and may be associated with equine congenital stationary night blindness. Esotropia (crossed eyes) is reported in mules. Strabismus may also result from space occupying lesions of the orbit or be caused by muscle avulsion from a traumatic proptosis.

Orbital fat prolapse can occur from trauma or idiopathic means.

**Orbital Trauma, Orbital Foreign Bodies, Contusions, and Periorbital Fractures**

Horses may injure the orbital region on the race track, in trailers or pastures, by rearing and hitting stall ceilings or starting gates, from gunshot, kicks from other horses, or when being disciplined. These should be taken seriously and treated aggressively, because infection secondary to trauma can lead to orbital cellulitis, which can be vision threatening or even fatal. Field diagnostics may be inconclusive for fracture or foreign body, so monitor closely and use common sense—if the peri-orbital region remains hot, painful, and swollen in the face of therapy, there may be a sequestra, abscess, or foreign body present even if you cannot image it. Monitor the globe and treat as necessary for secondary uveitis. Consider location of nasolacrimal duct obstruction. There is no treatment at present; if the condition may resolve spontaneously. Radiographs will confirm diagnosis.

Orbital fractures can be identified by palpation, facial deformity, and radiography. Blepharositis, epistaxis, orbital emphysema, corneal ulcers, uveitis, and limitations of global motility caused by entrapment by bone fragments may accompany orbital fractures. Orbital fractures can result in displacement of globe and have the potential for globe penetrating bone fragments. Minor orbital rim fractures may not need surgical correction unless fracture fragments are impinging on the globe or perfect cosmesis is required. Serious periorbital fractures should be surgically repaired quickly, because fibrous union of the fractured pieces begins within 1 wk after the injury to make elevation and realignment very difficult. Interosseous wiring with stainless steel suture, bone plating, and cancellous bone grafts may be necessary to immobilize and repair extensive orbital fractures.

**Foreign Bodies Can Lead to Orbital Abscesses**

Older horses tend to develop neoplasia, whereas foals and yearlings may be prone to acute orbital trauma and cellulitis. Cellulitis may be associated with fever, blepharositis, swelling of the super-orbital fossa, nictitans protrusion, chemosis, and corneal edema.

Head trauma can cause globe proptosis. Proptosis is forward displacement of the eye from the orbit. It is seen commonly with retrobulbar hemorrhage and edema after penetrating orbital trauma. In cases of traumatic globe proptosis, careful ophthalmic exam will dictate viability of the eye. Lack of an indirect pupillary reflex to the normal eye and miosis with severe hypotony and hyphema indicate severe trauma and poor visual prognosis. Temporary tarsorrhaphy is recommended for proptosis.

**Eyelids**

Thin and highly vascularized, the eyelids contain muscles, connective tissue, cilia, glands that produce components of the tear film, and a tarsal plate that gives the free edge of the lid support. They are lined with conjunctiva. The major issues that arise with eyelids are trauma and neoplasia. Occasionally lids can be malformed. Prompt, definitive therapy is needed when any condition threatens the anatomy or function of the lids. Eyelids can heal very well if the surgery is done with careful techniques.
Nictitans
This is the “third eyelid” consisting of a conjunctiva covered membrane, a T-shaped cartilage, and a sebaceous gland at the base. It functions to protect the cornea and distribute tear film.

Eyelid Lacerations
This is very common, especially when horses are irritated by insects and seek objects to rub their eyes on. The most common cause is entrapment of the lid in a bucket handle! Counsel owners to tape up bucket handles before injuries occur. Intralosomal anesthetics will make the horses positive for drug testing. Great care should be taken with the repair, including minimal debridement. Careful apposition of the torn pedicle, use of 4-0 suture, and closure of the tarsal plate using a figure of eight suture pattern that keeps suture tags from abrading the cornea. Standing surgery is helped by resting the chin of the sedated horse on bales and using a very short (5.5 in) Olsen Hagar needle holder and forceps. Be careful if you decide to use a two-layer closure—the inner layer must not abrade the cornea. In the field, single-layer closures with meticulous deep bites are often the safest repairs. Temporary tarsorrhapsies may stabilize the tarsal plate in severe tears. Check the globe carefully for concurrent injury or uveitis. Use NSAIDs and topical and/or systemic antibiotics for a few days.

Sarcoids
Sarcoids are common benign tumors that affect the adnexal region and threaten the eyes by expansion and compromise of lid function. Definitive diagnosis is by biopsy, but biopsy can stimulate tumor expansion. “On the farm” treatment includes topical application of a bloodroot-based paste—appropriate if the lesion is located away from the palpebral margin and is not compromising lid function. Shrinking the sarcoid lesion with antipсорiasis skin ointments and/or topical 5-fluorouracil (5-FU) for 2 wk may be beneficial before using Bacillus Calmette-Guérin (BCG). Surgical resection of necrotic tissue is controversial with some experts suggesting it will exacerbate the sarcoid.

Immunotherapy
Injecting attenuated Mycobacterium cell wall fraction (BCG) often is effective in promoting remission. Referral or “in clinic” treatments include cryotherapy, intralosomal chemotherapy with cisplatin, hyperthermia, CO₂ laser ablation, and intralosomal radiotherapy. The bovine papilloma virus (BPV) induces sarcoids, and intralosomal treatment with the BPV vaccine can be helpful. Transmission is thought to be linked to stable flies, but a genetic susceptibility probably also has a role.

Squamous Cell Carcinoma
Squamous cell carcinoma (SCC) is a common tumor that can affect the lids, nictitans, and globe. Appaloosas, draft breeds, and Paint horses are at increased risk. Solar radiation promotes neoplastic transformation, especially if the lids lack pigment. SCC is locally invasive and often accompanied by local necrosis and functional compromise of the affected tissue. Excision with wide margins may be curative, but submission of the mass for margin analysis is wise. The author has had good luck supplementing surgery with intralosomal cisplatin chemotherapy if the mass cannot be completely excised. Masses located in the nictitans may be treated by excision of the entire nictitans. This is a simple surgery that can be done in the field with good results—horses do not often suffer tear film problems afterward. Adjunctive therapy at referral hospitals includes excision with eyelid reconstruction, hyperthermia, cryosurgery (double freeze thaw cycle to −4 to −40°F), beta irradiation with strontium 90, brachytherapy using interstitial radiation with a variety of radioisotopes, and intralosomal chemotherapy. Other tumors that can affect the lids include melanoma, fibroma, fibrosarcoma, lymphoma, mast cell tumor, hemangioendothelioma, angiosarcoma, and osteoma. Fungal granulomas or mycetomas resemble neoplastic lesions—differentiate by histopathology.

Eyelid Edema
This often occurs unilaterally in summer months; it is thought to reflect an allergic reaction to insect bites. It may be quite dramatic but usually not painful. It responds promptly to topical and systemic corticosteroids. It is important to check globe carefully—uncomplicated cases will show normal pupil size and intact corneal epithelium.

Meibomianitis
These are cheesy abscesses of the meibomian glands of the tarsal plate. It may affect all four lid margins, and is idiopathic in origin. Treatment involves incision of the gland conjunctiva with a no. 15 scalpel blade and curettage of the debris with a small curette. Insipissated material may be gritty. Post-operative therapy includes topical and/or systemic antibiotics and corticosteroids. Recurrence is common.

Solar Blepharitis
This is common in Appaloosas and other horses with light pigmentation of the eyelid margins. It often accompanies chronic insidious uveitis. Palliative therapy includes the use of a UV light blocking fly mask and intermittent, judicious use of topical steroids. Topical cyclosporine A helps some horses.

Prolapse of the Nictitans
Bilateral prolapse can accompany systemic tetanus or an acute attack of Hyperkalemic periodic paralysis. Unilateral prolapse should prompt a thorough examination. After sedation and topical anesthesia, the nictitans should be pulled out and everted out to inspect for neoplasia, foreign body, or other
abnormality. Horner’s syndrome can cause unilateral prolapse with ptosis and sweating on the ipsilateral neck.

**Conjunctiva**

Conjunctiva is the mucous membrane that lines the lids (palpebral conjunctiva) and covers the sclera (bulbar conjunctiva). The membrane is filled with fine blood vessels and thus is a barometer of systemic or vascular abnormalities, including anemia or jaundice. Almost any ocular inflammation causes the “red eye” appearance of conjunctivitis. This term is not a diagnosis but a secondary symptom of ocular or systemic disease! Look for allergic, infectious, immune, neoplastic, parasitic disease, or foreign bodies as the primary cause of any observed conjunctivitis.

**Chemosis**

This is edema of the conjunctiva, usually associated with allergy. It is rapidly responsive to topical steroids.

**Dermoid**

Congenital abnormal growths that may affect the conjunctiva near the limbus. May have hair, glandular, or mineralized tissue elements. May be surgically removed under general anesthesia.

**Scleral Hemorrhage**

Reddish streaks under the conjunctiva around the limbus, commonly seen in neonatal foals as a result of birth trauma. Will resolve in about a week with no therapy.

**Neoplasias**

Include squamous cell carcinoma, melanoma, lymphoma, papilloma, and hemangioma/hemangiosarcoma. See notes under eyelids for therapies.

**Habronemiasis**

This is a parasite that can cause ocular granulomas involving the conjunctiva and limbus. Onchocerciasis can also cause conjunctivitis and keratitis and uveitis. Treatment involves administration of systemic ivermectin and topical steroids. Granulomas may require debridement under sedation and topical anesthesia.

**Nasolacrimal System**

The nasolacrimal system has both drainage and secretory functions. Drainage begins at the two puncta in the upper and lower medial canthus of each eye, continues through the nasolacrimal canal, into the nasolacrimal sac, and down the nasolacrimal duct, which runs through the lacrimal bone of the maxilla. Drainage exits at the nasal punctum. The tear film is a byproduct of secretions of the lacrimal and nictitans glands, the meibomian glands, and the conjunctival goblet cells. Much research is currently being done on the inflammatory mediators that reside in the precorneal tear film, because these have an important role in corneal ulcer progression and healing.

Dacryocystitis is inflammation of the lacrimal sac and nasolacrimal duct and is common. It can be secondary to obstruction of the drainage system that is congenital or acquired. It is seen frequently in summer months accompanying insect irritation or solar blepharitis. It is characterized by mucopurulent discharge at the medial canthus and mild conjunctivitis/hyperemia. Mild unobstructed cases are rapidly responsive to topical antibiotics or antibiotic/steroid combinations. Obstructed cases can sometimes be cleared by simple retrograde flushing (using a curved tip syringe, jugular catheter, or other small tubing attached to a syringe) of a saline solution that has been mixed with a small amount of steroid and/or antibiotic. Further diagnostics are warranted on cases where the duct is not patent. This occasionally can be secondary to dental disease. Contrast radiography can determine boundaries of duct system and location of obstruction, and referral for surgery (conjunctivorhinostomy) is optimal therapy.

Keratoconjunctivitis sicca (KCS) or “dry eye” in horses is a group of clinical signs related to a lack of tears. Corneal ulcers, corneal pigmentation, conjunctivitis, and blepharospasm may be seen. The Schirmer tear test values (normal is 14–34 mm wetting/min) are <10 mm wetting/min in horses with quantitative KCS. This condition is uncommon in horses. Qualitative KCS, where the Schirmers are normal to elevated and yet the cornea appears dry, is common in the horse.

KCS can be caused by nerve damage that affects parasympathetic system that innervates lacrimal gland. It is often accompanied by cranial nerve VII facial paralysis and an inability to close the lid. It is also seen with some cases of vestibular disease, eosinophilic keratitis, and with locoweed poisoning. Other cases are idiopathic. It may be very difficult to manage because of associated chronic corneal disease, exposure, and discomfort. Lubrication of eye using artificial tears in gel format is helpful. Topical cyclosporine A may alleviate signs; some idiopathic cases resolve spontaneously.

**Cornea**

**Corneal Anatomy**

The outer corneal layer is a relatively impermeable epithelium that is richly innervated and very thin (0.14 mm and six to eight cell layers thick), with an underlying basement membrane. Healing of defects in this layer is rapid and occurs by “sliding leapfrog” motion of adjacent cells without mitosis. The next layer is the stroma, which is the thickest layer. Stroma is composed of type I collagen fibrils that are arranged in a parallel lamellar lattice pattern. Disruption of the lattice causes opacity. Healing of stromal defects involves a balance of resorptive remodeling (facilitated by the proteinases that are released from bacteria, corneal cells, and infiltrating polymorphonuclear leukocytes [PMNs])
and restorative repair where fibroblasts lay down collagen to fill in the defect. Successful healing of defects is followed by several months of collagen remodeling that may return the tissue to a degree of its original transparency. In deep lesions, or lesions where healing is delayed, the collagen is laid down in a thick, random fashion, making an opaque scar. In severe cases, remodeling by proteinases is excessive, and keratomalacia (melting) or perforation results. The third layer of tissue is a thin (45 μm) basement membrane called Descemet’s membrane. The final layer of the cornea is a very thin monolayer of cells, the endothelium. The endothelium is no thicker than a single red blood cell (7 μm). This layer of cells has an Na-K–activated ATPase-dependent electrolyte pump that constantly works to keep the corneal stroma relatively dehydrated. Disruption of the normal pump activity results in edema of the endothelium and overlying stroma that can be permanent.1-5

Equine Corneal Ulceration

Equine corneal ulceration is very common in horses and is a sight-threatening disease needing early clinical diagnosis, laboratory confirmation, and appropriate medical and surgical therapy. Ulcers can range from simple, superficial breaks or abrasions in the corneal epithelium to full-thickness corneal perforations with iris prolapse. The prominent eye of the horse may predispose to traumatic corneal injury. Both bacterial and fungal keratitis in horses may present with a mild, early clinical course but need prompt therapy if serious ocular complications are to be avoided. Corneal ulcers in horses should be aggressively treated no matter how small or superficial they may be. Corneal infection and iridocyclitis are always major concerns for even the slightest corneal ulcerations. Iridocyclitis or uveitis is present in all types of corneal ulcers and must be treated to preserve vision. Globe rupture, phthisis bulbi, and blindness are possible sequelae to corneal ulceration in horses.

Proteinases in the Tear Film

Tear film proteinases normally provide a surveillance and repair function to detect and remove damaged cells or collagen caused by regular wear and tear of the cornea. These enzymes exist in a balance with inhibitory factors to prevent excessive degradation of normal tissue. Two major families of proteinases that may affect the cornea include the matrix metalloproteinases (MMPs) and the serine proteinases. MMPs predominate in the horse.1-5

Bacterial and fungal pathogens induce corneal epithelial cells, corneal stromal fibroblasts, and leukocytes (PMN) in the tear film to upregulate cytokines (interleukin [IL]-1, IL-6, and IL-8) that induce MMP production and elicit inflammatory and degradative processes. Proteinases that may contribute to corneal ulceration in the early stages of infection could be of bacterial or corneal cell origin. In the later stages as PMNs accumulate, PMN-derived proteinases predominate as the main factor in corneal tissue destruction. In pathologic processes such as ulcerative keratitis, excessive levels of these proteinases can lead to rapid degeneration of collagen and other components of the stroma, potentially inducing keratomalacia or corneal “melting.”

Corneal Sensitivity in Foals and Adult Horses

Corneal sensation is important for corneal healing. The cornea of the adult horse is very sensitive compared with other animals. Corneal touch threshold analysis revealed the corneas of sick or hospitalized foals were significantly less sensitive than those of adult horses or normal foals. The incidence of corneal disease is also much higher in sick neonates than in healthy foals of similar age. Ulcerative keratitis in the equine neonate often differs from adult horses in clinical signs and disease course. Foals may not show characteristic epiphora, blepharospasm, or conjunctivitis, and the ulcers may be missed without daily fluorescein staining. This decreased sensitivity may partially explain the lack of clinical signs often seen in sick neonates with corneal ulcers.

Corneal Healing in the Horse

The thickness of the equine cornea is 1.0–1.5 mm in the center and 0.8 mm at the periphery.1-5

The normal equine corneal epithelium is 8–10 cell layers thick but increases to 10–15 cell layers thick with hypertrophy of the basal epithelial cells after corneal injury. The epithelial basement membrane is not completely formed 6 wk after corneal injury in the horse, despite the epithelium completely covering the ulcer site. Healing of large-diameter, superficial, noninfected corneal ulcers is generally rapid and linear for 5–7 days and then slows. Healing of ulcers in the second eye may be slower than in the first and is related to increased tear proteinase activity. Healing time of a 7-mm-diameter, midstromal depth, noninfected corneal trephine wound was nearly 12 days in horses (0.6 mm/day).

Equine Corneal Microenvironment

The environment of the horse is such that the conjunctiva and cornea are constantly exposed to bacteria and fungi. The corneal epithelium of the horse is a formidable barrier to the colonization and invasion of potentially pathogenic bacteria or fungi normally present on the surface of the horse cornea and conjunctiva.1,5

A defect in the corneal epithelium allows bacteria or fungi to adhere to the cornea and to initiate infection. Staphylococcus, Streptococcus, Pseudomonas, Aspergillus, and Fusarium spp. are common causes of corneal ulceration in the horse. Infection should be considered likely in every corneal ulcer in the horse. Fungal involvement should
be suspected if there is a history of corneal injury with vegetative material, or if a corneal ulcer has received prolonged antibiotic and/or corticosteroid therapy with slight or no improvement.

Tear film neutrophils and some bacteria and fungi are associated with highly destructive proteinase and collagenase enzymes that can result in rapid corneal stromal thinning, descemetocele formation, and perforation. Excessive proteinase activity is termed “melting” and results in a liquefied, grayish-gelatinous appearance to the stroma near the margin of the ulcer.

Total corneal ulceration ultimately requires the degradation of collagen that forms the framework of the corneal stroma. Horse corneas show a pronounced fibrovascular healing response. The unique corneal healing properties of the horse in regards to excessive corneal vascularization and fibrosis seem to be strongly species specific.

Many early cases of equine ulcerative keratitis present, initially, as minor corneal epithelial ulcers or infiltrates, with slight pain, blepharospasm, epiphora, and photophobia. At first, anterior uveitis and corneal vascularization may not be clinically pronounced. Slight droopiness of the eyelashes of the upper eyelid may be an early, yet subtle, sign of corneal ulceration.

A vicious cycle may be initiated after the first injury to the cornea, with “second injury to the cornea” occurring because of the action of inflammatory cytokines. Ulcers, uveitis, blepharitis, conjunctivitis, glaucoma, and dacryocystitis must be considered in the differential for the horse with a painful eye. Corneal edema may surround the ulcer or involve the entire cornea. Signs of anterior uveitis are found with every corneal ulcer in the horse, and include miosis, fibrin, hyphema, or hypopyon. Persistent superficial ulcers may become indolent because of hyaline membrane formation on the ulcer bed.

Fluorescein dye retention is diagnostic of a full-thickness epithelial defect or corneal ulcer. Faint fluorescein retention may indicate a microerosion or partial epithelial cell layer defect because of infiltration of fluorescein dye between inflamed epithelial cell junctions. All corneal injuries should be fluorescein stained to detect corneal ulcers. Rose bengal retention indicates a defect in the mucin layer of the tear film.

Horses with painful eyes need to have their corneas stained with both fluorescein dye and rose bengal dye, because fungal ulcers in the earliest stage will be negative to the fluorescein but positive for the rose bengal.

Fungi may induce changes in the tear film mucin layer before attachment to the cornea. Early fungal lesions that retain rose bengal are multifocal in appearance and may be mistaken for viral keratitis.

Microbiologic culture and sensitivity for bacteria and fungi are recommended for horses with rapidly progressive, and deep corneal ulcers. Corneal cultures should be obtained first and followed by corneal scrapings for cytology. Mixed bacterial and fungal infections can be present.

Vigorous corneal scraping at the edge and base of a corneal ulcer is used to detect bacteria and fungal hyphae. Samples can be obtained with the handle end of a sterile scalpel blade and topical anesthesia. Superficial scraping with a cotton swab cannot be expected to yield organisms in a high percentage of cases.

A “crater-like” defect that retains fluorescein dye at its periphery and is clear in the center is a descemetocele and indicates the globe is at high risk of rupture. Descemet’s membrane does not retain fluorescein dye, whereas deep ulcers that continue to have stroma anterior to Descemet’s membrane will retain fluorescein. Deep penetration of the stroma to Descemet’s membrane with perforation of the cornea is a possible sequelae to all corneal ulcers in horses.

**Common Problems of the Cornea**

### Superficial Corneal Erosions

Erosions are defects that do not break into the stroma. If these do not get infected, they heal quickly without visible scars. Topical mydriasis and antibiotic therapy is indicated. In some older animals, the erosions become chronic, non-healing indolent ulcers because the epithelium does not generate a normal basement membrane for secure adherence. These cases may be helped by debridement or temporary tarsorrhaphy. If they do not heal in 2 wk, consider performing a superficial linear keratotomy. Using sedation and local anesthesia, carefully drag the point of a 22-gauge needle clamped very close to the jaws of a hemostat over the surface of the lesion to create a grid incision into the superficial stroma and thus provide a lattice for adherence of new epithelial cells. Refer to texts for exact description of technique. Grid keratotomies can seed pre-existing infection deeper into the stroma. Do not perform this procedure if infection with bacteria or fungus is suspected.

### Superficial Keratitis

This may present as punctate areas of stain uptake, as focal vascularization, as pigment deposition, as focal superficial opacities, or as bullous keratopathy where the epithelium takes on a faintly blistered appearance. Punctate keratitis may have a viral (herpes?) or an idiopathic etiology. May be painful or comfortable. Epithelium shows fluorescein stain uptake in a dot like pattern that is scattered over the surface. A trial of topical idoxyuridine may improve the condition. Topical NSAIDs, especially 0.1% diclofenac, may be very helpful. Other forms of keratitis may also respond well to topical NSAIDs or antivirals, whereas others respond to topical antibiotics. Caution is advised when considering the use of topical steroids in these cases—horses on steroid trial therapy should be monitored closely. Horses with unusual epithelial appearance or unex-
plained opacity should be checked for glaucoma by tonometry

Ulcerative Keratitis
Ulcers are defects that extend into the stroma (erosions are defects limited to the epithelium).

Healing of these defects is a balancing act: ideally, tear film proteinases remodel the stromal defect and native fibroblasts restore stromal integrity. Bacterial or fungal infections and various host factors may tip the balance toward excessive resorption, resulting in melting of stromal collagen or even perforation of the globe. Ulcers are very painful and are accompanied by secondary uveitis, so the syndrome is complicated by patient objection to topical therapy. Refer to the texts in the references for a full discussion of the subject. Adjunctive surgical therapy may involve debridement or keratotomy which can be done in the field. Complex cases may need keratotomy, conjunctival grafts, amniotic membrane grafts, or tarsorrhaphy and thus are referral cases. Very serious cases may need corneal transplantation by penetrating keratoplasty (PK) or penetrating lamellar keratoplasty (PLK).

Ulcers with bacterial infection can be diagnosed by cytology. Therapy choices are dictated by the type of bacteria seen on slides, and later may be adjusted according to clinical response and results of lesion culture/sensitivity. Initial therapy is intense, usually four to six times per day. Antibiotics are combined with mydriatics and topical antiproteinases. Systemic NSAIDs help control pain. Subconjunctival injection may be used to supplement topical therapy. Treatment of cooperative patients without obvious keratomalacia may be through ointments administered at home, and resolution may be straightforward. Treatment of factionious patients, or patients with very deep defects may be through liquids administered through a subpalpebral lavage (SPL) tube at home or at a referral hospital. Frequent monitoring will be necessary until it is clear that healing is occurring. The most common antibiotic drugs used on bacterial keratitis are chloramphenicol, cefazolin, tobramycin, gentamicin and systemically administered NSAIDs help control pain. Subconjunctival injection may be used to supplement topical therapy. Treatment of cooperative patients without obvious keratomalacia may be through ointments administered at home, and resolution may be straightforward. Treatment of factionious patients, or patients with very deep defects may be through liquids administered through a subpalpebral lavage (SPL) tube at home or at a referral hospital. Frequent monitoring will be necessary until it is clear that healing is occurring. The most common antibiotic drugs used on bacterial keratitis are chloramphenicol, cefazolin, tobramycin, gentamicin, ciprofloxacin, and amikacin (Table 1). Atropine application should be to effect. Topical antiproteinase therapy using serum application is routine and may include a combination of MMP inhibitors. Debridement should be judicious.

Melting or Very Aggressive Ulcers
The most serious, eye-threatening bacterial infections are those caused by beta hemolytic Streptococcus spp. and Pseudomonas aeruginosa—the prognosis is guarded if collagenolysis is extensive, and these infections will be expensive and time consuming to treat. Treatment must be immediate and aggressive—in referral hospitals, antibiotics and antiproteinases are administered every 1–2 h around the clock. Cephazolin is most effective against beta hemolytic Streptococcus spp. Amikacin, tobramycin, and gentamicin are most effective against Pseudomonas. Topical antiproteinase therapy usually involves a combination of agents (serum, acetylcysteine, EDTA, ilomastat) administered every 1–2 h initially. See Table 1 for more information on drug therapy and recommended strengths of fortified solutions. Fortified solutions of amikacin and gentamicin can easily be prepared from stock product used in general practice pharmacies for systemic or orthopedic therapy. Cephazolin is very inexpensive and readily available in 1-g bottles through veterinary distributors or local small animal hospitals. It may be reconstituted to a 50-mg/ml fortified solution.

Ulcers with Fungal Infection (Keratomycosis)
Fungi are normal inhabitants of the equine environment and conjunctival microflora, but can become pathogenic after corneal injury. Aspergillus, Fusarium, Cylindrocarpon, Curvularia, yeasts, and molds are known causes of fungal ulceration in horses.

Ulcerative keratomycosis is a serious, sight-threatening disease in the horse. Blindness can occur. The most often proposed pathogenesis of ulcerative fungal keratitis in horses begins with slight to severe corneal trauma resulting in an epithelial defect, colonization of the defect by fungi normally present on the cornea, and subsequent stromal invasion. Seeding of fungi from a foreign body of plant origin is also possible. Some fungi may have the ability to invade the corneal epithelium after disruption of the tear film. Stromal destruction results from the release of proteinases and other enzymes from the fungi, tear film leukocytes, and keratocytes. Fungi may produce antiangiogenic compounds that inhibit vascularization.

Fungi seem to have an affinity for Descemet's membrane, with hyphae frequently found deep in the equine cornea. Deeper corneal invasion can lead to sterile or infectious endophthalmitis. Saddlebreds seem to be prone to severe keratomycosis, whereas Standardbreds are resistant.

Diagnostic tests should include fluorescein and rose bengal staining, corneal cytology, corneal culture with attempted growth on both fungal and aerobic plates, and biopsy if surgery is performed. Prompt diagnosis and aggressive medical therapy with topically administered antifungals, antibiotics, atropine, and systemically administered NSAIDs will positively influence visual outcome and may negate the need for surgical treatment. Treatment must be directed against the fungi and against the iridocyclitis that occurs after fungal replication and fungal death. Therapy is quite prolonged and scarring of the cornea may be prominent. The fungi are overall more susceptible to antifungal drugs in this order: natamycin = miconazole > itraconazole > ketoconazole > fluconazole.

“Ulcer cocktails” are equal parts of equine serum, tobramycin, natamycin, and cefazolin that, when combined, are very effective against Staphylococcus,
<table>
<thead>
<tr>
<th>Key Drug</th>
<th>Drug Class</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cisplatin* (Platinol) 10 mg mixed in 1 ml H₂O and 2 ml pure medical sesame oil</td>
<td>Chemotherapy</td>
<td>3.3 mg/ml, inject at 1 mg per cm³, tumors up to 20 cm³</td>
<td>3 or more sessions at 2 week intervals</td>
<td>Intralesional*</td>
<td>Certain neoplasias, esp. squamous cell carcinoma. Can inject on same day as excisional surgery.</td>
</tr>
<tr>
<td>5-FU* (Efudix 5% cream) and 5-Fluorouracil (1%)</td>
<td>Chemotherapy</td>
<td>Cover the lesion with cream or inject 1 ml per cm³ of mass</td>
<td>q 12-24 hrs. for 7 days or 4 or more sessions at 2 week intervals</td>
<td>Topical*</td>
<td>Certain neoplasias, sarcoïdosis, certain neoplasias</td>
</tr>
<tr>
<td>blood root paste</td>
<td>Immunotherapy</td>
<td>Cover lesion with paste 1/8–1/4&quot; thick</td>
<td>Daily for 4–6 days. May repeat if no response</td>
<td>Topical paste</td>
<td>Sarcoïd, possibly other neoplasias</td>
</tr>
<tr>
<td>BCG</td>
<td>Immunotherapy</td>
<td>Inject 1 ml/cm³ of mass</td>
<td>Repeat q 2–4 wks for up to 6 treatments</td>
<td>Intralesional</td>
<td>Sarcoïd</td>
</tr>
<tr>
<td>Triple antibiotic with 1% hydrocortisone†</td>
<td>Corticosteroid with antibiotic</td>
<td>1/4 inch strip</td>
<td>q 6-24 hrs</td>
<td>Topical†</td>
<td>Chemosis, allergic or solar blepharitis, dacryocystitis</td>
</tr>
<tr>
<td>Triple antibiotic with 0.1%† dexamethasone</td>
<td>More potent corticosteroid with antibiotic</td>
<td>1/4 inch strip</td>
<td>q 6-24 hrs</td>
<td>Topical†</td>
<td>Chemosis, allergic or solar blepharitis</td>
</tr>
<tr>
<td>Triple antibiotic</td>
<td>Antibiotic</td>
<td>1/4 inch strip of ointment</td>
<td>q 6-12 hrs</td>
<td>Topical</td>
<td>Antibiotic for lid repair followup, minor purulent dacryocystitis</td>
</tr>
<tr>
<td>Optimmune cyclosporine A 0.2%</td>
<td>Immune modulator</td>
<td>1/4 inch strip of ointment</td>
<td>q 12-24 hrs</td>
<td>Topical</td>
<td>Solar blepharitis</td>
</tr>
<tr>
<td>Procaine penicillin G (PPG)‡</td>
<td>Antibiotic</td>
<td>3 ml per 100 lbs</td>
<td>q 12 h</td>
<td>Intramuscular only</td>
<td>Orbital infection-trauma in combo with gentocin</td>
</tr>
<tr>
<td>Gentamycin sulfate</td>
<td>Antibiotic</td>
<td>100 mg/ml: 3 ml per 100 lbs</td>
<td>q 24 h-assure 24 hrs between doses</td>
<td>Intramuscular, intravenous</td>
<td>Orbital trauma-infection-use in combo with PPG</td>
</tr>
<tr>
<td>Cefiotrofur sodium (Naxcel), 4 g per 80 ml bottle</td>
<td>Antibiotic</td>
<td>2 ml per 100 lbs of reconstituted drug</td>
<td>q 12-24 hrs</td>
<td>Intramuscular, Intravenous</td>
<td>Alternative broad spec antibiotic for orbital infection or severe trauma, $$$</td>
</tr>
<tr>
<td>Trimethoprim sulfa, double strength (960 mg)</td>
<td>Antibiotic</td>
<td>DS tabs: 1.5 tabs per 100 lbs</td>
<td>q 12 h</td>
<td>Oral</td>
<td>Antibiotic for lid laceration or corneal infection</td>
</tr>
<tr>
<td>Globe problems</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atropine HCL 1%</td>
<td>Mydriatic</td>
<td>1/4 inch strip or 0.1 ml (drops)</td>
<td>q 6–12 h Reduce freq. once dilated</td>
<td>Topical SPL</td>
<td>Dilate miotic pupil, Control pain of ciliary spasm</td>
</tr>
<tr>
<td>Triple antibiotic ointment</td>
<td>Antibiotic</td>
<td>1/4 inch strip</td>
<td>q 6–12 h</td>
<td>Topical</td>
<td>Ulcers, minor</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>Antibiotic</td>
<td>1/4 inch strip</td>
<td>q 2–8 h</td>
<td>Topical</td>
<td>Ulcers, gram positive and gram negative</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>Antibiotic</td>
<td>1/4 inch strip ointment or 0.1–0.2 ml (fortified is 33 mg/ml)</td>
<td>q 2–8 h, Subconjunctival injection</td>
<td>Topical SPL, can repeat</td>
<td>Ulcers, gram negative</td>
</tr>
<tr>
<td>Amikacin</td>
<td>Antibiotic</td>
<td>0.1 MI-0.2 of 10–15 mg/ml 25 mg</td>
<td>q 2–8 h, Can repeat</td>
<td>Topical SPL, Subconjunctival injection</td>
<td>Ulcers sensitive to drug, esp. gram negative</td>
</tr>
<tr>
<td>Tobramycin*</td>
<td>Antibiotic</td>
<td>1/4 inch strip or 0.1–0.2 ml of 15 mg/ml 20–40 mg</td>
<td>q 2–8 h, Subconjunctival injection</td>
<td>Topical SPL, Can repeat</td>
<td>Ulcers, gram positive and gram negative</td>
</tr>
<tr>
<td>Key Drug</td>
<td>Drug Class</td>
<td>Dose Range</td>
<td>Frequency</td>
<td>Route</td>
<td>Indications</td>
</tr>
<tr>
<td>--------------------------</td>
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</tr>
<tr>
<td>Ciprofloxacin</td>
<td>Antibiotic</td>
<td>1/4 inch strip or 0.1–0.2 ml human drops</td>
<td>q 2–8 h</td>
<td>Topical SPL</td>
<td>Ulcers, gram positive</td>
</tr>
<tr>
<td>Oxacillen</td>
<td>Antibiotic</td>
<td>0.1 ml of human drops</td>
<td>q 2–8 h</td>
<td>Topical SPL</td>
<td>Ulcers, sensitive to drug</td>
</tr>
<tr>
<td>Cephazolin (mix 1 g vial with artificial tears or saline)</td>
<td>Antibiotic</td>
<td>0.1 ml – 0.2 ml to mix 50 mg/ml fortified solution</td>
<td>q 2–8 h, Subconjunctival injection</td>
<td>Topical SPL, Can repeat</td>
<td>Ulcers, gram positive</td>
</tr>
<tr>
<td>2% povidone iodine solution</td>
<td>Anti-infective</td>
<td>0.1–0.2 ml</td>
<td>q 6 h</td>
<td>Topical</td>
<td>Suspected fungal ulcers</td>
</tr>
<tr>
<td>Silver sulfadiazine</td>
<td>Anti-infective burn cream</td>
<td>1/4 inch strip cream</td>
<td>q 12 h</td>
<td>Topical</td>
<td>Off label use, has antibacterial and antifungal activity</td>
</tr>
<tr>
<td>1% Itraconazole/30% DMSO in petroleum†</td>
<td>Anti-fungal</td>
<td>1/4 inch strip ointment</td>
<td>q 6 h</td>
<td>Topical, Subconj. inj.</td>
<td>Fungal ulcers. Must be compounded</td>
</tr>
<tr>
<td>Natamycin</td>
<td>Anti-fungal</td>
<td>0.1% 0.1–0.2 ml 5000 units</td>
<td>One time</td>
<td>Topical</td>
<td>Fungal ulcers</td>
</tr>
<tr>
<td>1% miconazole</td>
<td>Anti-yeast</td>
<td>1/4 inch strip cream</td>
<td>q 6 h</td>
<td>Topical</td>
<td>Fungal ulcers. Off label</td>
</tr>
<tr>
<td>Clotrimazole</td>
<td>Anti-yeast</td>
<td>1/4 inch strip cream</td>
<td>q 6 h</td>
<td>Topical</td>
<td>Ulcers with yeast infection. Off label</td>
</tr>
<tr>
<td>Itraconazole†</td>
<td>Anti-fungal</td>
<td>3 mg/kg</td>
<td>q 10 h</td>
<td>Oral</td>
<td>Fungal ulcers (compounded in KY)</td>
</tr>
<tr>
<td>5% NaCl (hypertonic saline)</td>
<td>Osmotic agent</td>
<td>1/4 inch strip ointment</td>
<td>q 10 h</td>
<td>Topical</td>
<td>Clear corneal edema</td>
</tr>
<tr>
<td>Flubiprofen</td>
<td>NSAID</td>
<td>0.1 ml</td>
<td>q 6–12 h</td>
<td>Topical</td>
<td>Keratitis, idiopathic</td>
</tr>
<tr>
<td>Diclofenac</td>
<td>NSAID</td>
<td>0.1 ml</td>
<td>q 6–12 h</td>
<td>Topical</td>
<td>Keratitis, painful keratopathy, $$ but often very effective</td>
</tr>
<tr>
<td>Adequan (dilute with art. tears)</td>
<td>Topical anti-inflammatory</td>
<td>Mix to 50 mg/ml</td>
<td>q 6–12 h</td>
<td>Topical</td>
<td>Keratitis non-healing ulcers</td>
</tr>
<tr>
<td>Serum, autologous or homologous</td>
<td>MMP inhibitor serine protease inhibitor treatment</td>
<td>Undiluted, 0.1–0.2 ml per 5–7 days</td>
<td>Topical</td>
<td>Melting ulcer Prevent collagenolysis Non-healing ulcer Melting corneal ulcer Melting ulcer Topical chelation of calcific keratopathy</td>
<td></td>
</tr>
<tr>
<td>Doxycycline</td>
<td>MMP inhibitor antibiotic</td>
<td>1 mg/kg</td>
<td>q 12 h</td>
<td>PO</td>
<td>Keratitis, painful keratopathy</td>
</tr>
<tr>
<td>Disodium EDTA (can be made by adding 1–5 ml water to EDTA blood tube)</td>
<td>MMP inhibitor chelating agent (Ca and Zn)</td>
<td>0.17% to 1.0% solution 0.1–0.2 ml</td>
<td>q 1–6 h</td>
<td>Topical</td>
<td>Melting ulcer Topical chelation of calcific keratopathy</td>
</tr>
<tr>
<td>N-acetylcysteine</td>
<td>MMP inhibitor chelating agent (Ca and Zn)</td>
<td>5–10%, 0.1–0.2 ml</td>
<td>q 1–6 h</td>
<td>Topical</td>
<td>Melting ulcer-prevent collagenolysis</td>
</tr>
<tr>
<td>Ilomastat</td>
<td>MMP inhibitor chelating agent 9Ca and Zn</td>
<td>0.1%, 0.1–0.2 ml</td>
<td>q 1–6 h</td>
<td>Topical</td>
<td>Melting ulcer—prevent collagenolysis</td>
</tr>
<tr>
<td>Tetanus anti-toxin</td>
<td>Immune globulin reduces collagenolysis injection</td>
<td>1 ml</td>
<td>One time</td>
<td>Subconjunctival</td>
<td>Extensive fibrin in Anterior chamber. Referral procedure, contraindicated if hemorrhage is present</td>
</tr>
<tr>
<td>Tissue plasminogen activator (TFA)</td>
<td>Fibrin dissolution</td>
<td>50–150 mg (kept frozen at referral centers)</td>
<td>One time</td>
<td>Inject in anterior chamber under general anesthesia</td>
<td>Punctate keratitis Some superficial keratitis</td>
</tr>
<tr>
<td>Idoxuridine</td>
<td>Antiviral</td>
<td>1/4* strip or 0.1–0.2 ml</td>
<td>q 4–6 h</td>
<td>Topical</td>
<td>Eosinophilic keratoconjunctivitis</td>
</tr>
<tr>
<td>Cromodyn sodium 1% NSAIDs</td>
<td>Mast cell stabilizer</td>
<td>See above</td>
<td>0.1 ml q 6–12 h</td>
<td>Topical drop</td>
<td>Various inflammatory conditions Various inflammatory conditions where the corneal epithelium is intact</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>See above</td>
<td>0.1 ml q 6–12 h</td>
<td>q† 6–12 h</td>
<td>Topical Systemic subconjunctival</td>
<td>Deep ulcer therapy Topical therapy for fractious horses</td>
</tr>
<tr>
<td>Lavage tube kits</td>
<td>Treatment device</td>
<td>“Florida tubes” are the longest and best</td>
<td>Treatment can be given as often as needed</td>
<td>Topical SPL</td>
<td>Deep ulcer therapy Topical therapy for fractious horses</td>
</tr>
<tr>
<td>Key Drug</td>
<td>Drug Class</td>
<td>Dose Range</td>
<td>Frequency</td>
<td>Route</td>
<td>Indications</td>
</tr>
<tr>
<td>--------------------------</td>
<td>--------------------</td>
<td>-----------------------------</td>
<td>-----------------</td>
<td>---------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Atropine HCL 1%</td>
<td>Mydriatic</td>
<td>1/4-inch strip or drops, 0.1 ml</td>
<td>q 6–48 h</td>
<td>Topical</td>
<td>Dilate pupil and decrease pain from ciliary spasm</td>
</tr>
<tr>
<td>Phenyl ephrine 10%</td>
<td>Mydriatic</td>
<td>Drops</td>
<td>q 6–8 h</td>
<td>Topical</td>
<td>Can be epitheliotoxic-use only if atropine fails to dilate pupil.</td>
</tr>
<tr>
<td>Prednisolone acetate 1%</td>
<td>Corticosteroid</td>
<td>Drops, 0.1 ml</td>
<td>q 1–6 h</td>
<td>Topical</td>
<td>Anti-inflammatory, good penetration</td>
</tr>
<tr>
<td>Dexamethasone HCL 0.5%–1%</td>
<td>Corticosteroid</td>
<td>Drops or ointment</td>
<td>q 4–6 h</td>
<td>Topical</td>
<td>Anti-inflammatory, good penetration</td>
</tr>
<tr>
<td>Flurbiprofen 0.03%</td>
<td>Topical NSAID</td>
<td>Drops, 0.1 ml</td>
<td>q 1–6 h</td>
<td>Topical</td>
<td>Non-steroidal anti-inflammatory</td>
</tr>
<tr>
<td>Diclofenac 0.1%</td>
<td>Topical NSAID</td>
<td>Drops, 0.1 ml</td>
<td>q 6–12 h</td>
<td>Topical</td>
<td>Non-steroidal anti-inflammatory, but potent</td>
</tr>
<tr>
<td>Cyclosporine A (0.2%)</td>
<td>Immunosuppressant</td>
<td>Ointment 1/4-inch strip</td>
<td>q 6–12 h</td>
<td>Topical</td>
<td>Suppresses inflammation, can be used on ulcerated eye, useful for insidious cases with solar blepharitis, KCS, NKU</td>
</tr>
<tr>
<td>Timolol maleate 0.5%</td>
<td>Beta blocker</td>
<td>Drops or gel</td>
<td>q 12 h</td>
<td>Topical</td>
<td>Glaucma secondary to uveitis</td>
</tr>
<tr>
<td>Dorzolamide 2% (Trusopt)</td>
<td>Carbonic anhydrase inhibitor to decrease aqueous</td>
<td>Drops, 0.1 ml</td>
<td>q 12 h</td>
<td>Topical</td>
<td>Glaucma secondary to uveitis</td>
</tr>
<tr>
<td>Timolol/Dorzolamide</td>
<td>Combo drug for glaucoma</td>
<td>Drops, 0.1 ml</td>
<td>q 12 h</td>
<td>Topical</td>
<td>Glaucma unresponsive to either drug alone.</td>
</tr>
<tr>
<td>Flunixin meglumine</td>
<td>NSAID</td>
<td>0.5 mg/kg PO, IV, or IM</td>
<td>q 12 h for 5 days, then</td>
<td>PO, IV, IM</td>
<td>Uveitis, pain, swelling</td>
</tr>
<tr>
<td>Phenylbutazone</td>
<td>NSAID</td>
<td>0.25 mg/kg PO</td>
<td>q 12 h as needed</td>
<td>PO or IV</td>
<td>Uveitis, pain, swelling</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>Corticosteroid</td>
<td>2.2–4.4 mg/kg</td>
<td>q 12 or 24 h</td>
<td>PO</td>
<td>Uveitis—be cautious of laminitis risk</td>
</tr>
<tr>
<td>Triamcinolone</td>
<td>Corticosteroid</td>
<td>5–10 mg per horse</td>
<td>q 24 h</td>
<td>IM</td>
<td>Uveitis</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>Antibiotic</td>
<td>2–6 mg</td>
<td>1x, can repeat in 7–21 days</td>
<td>Subconjunctival injection</td>
<td>Anti-leptospiral infection therapy</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>Antibiotic</td>
<td>10 mg/kg</td>
<td>q 12 h for 4 wk</td>
<td>Intravitreal injection</td>
<td>May decrease signs in leptospiral associated cases, but there are safety concerns.</td>
</tr>
<tr>
<td>Cyclosporine A</td>
<td>Immune suppression</td>
<td>2.5–5 mg per horse</td>
<td>q 24 h</td>
<td>IM</td>
<td>Anti-leptospiral infection therapy</td>
</tr>
<tr>
<td>Tacrolimus, sirolimus,</td>
<td>Immune suppression</td>
<td>4-mg injection</td>
<td>1x, can repeat in 7–21 days</td>
<td>Intravitreal injection</td>
<td>May decrease signs in leptospiral associated cases, but there are safety concerns.</td>
</tr>
<tr>
<td>Rapamycin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Uveitis—early, uncomplicated disease that is quiescent at surgery time.</td>
</tr>
<tr>
<td>Cylcoproprin A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Uveitis—Device currently unavailable</td>
</tr>
<tr>
<td>Implant (under development-</td>
<td>Immune suppression</td>
<td>Implant yields slow release of</td>
<td>Surgical implant under sclera</td>
<td>NC State, U Fla, and</td>
<td>Uveitis—some referral centers have access to these drugs.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>drug</td>
<td></td>
<td>Ohio State were doing these surgeries</td>
<td></td>
</tr>
<tr>
<td>Tacrolimus, sirolimus,</td>
<td></td>
<td>Research drugs under development</td>
<td>Awaiting results of clinical trials</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rapamycin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Under investigation for topical and intraocular use</td>
</tr>
</tbody>
</table>

* Not available through veterinary distributors, generally must write prescription for human pharmacy.
† Hard to find at all, generally must compound.
‡ Available over the counter at human pharmacies.
§ May only be available at referral institutes.
Streptococcus, and fungi. Natamycin, miconazole, itraconazole/DMSO, fluconazole, amphotericin B, 2% betadine solution, chlorhexidine gluconate, posaconazole, voriconazole, and silver sulfadiazine can be used topically against equine keratomycosis.

Although the use is off label, OTC 2% miconazole vaginal cream can be used topically in the eye, it can be irritating. It is best to order 1% miconazole solution through a compounding pharmacy. OTC 1% silver sulfadiazine cream can also be used topically on the cornea. Call university pharmacies to order other antifungal topical or systemic pharmaceuticals.

Uveitis may be worse the day after initiation of antifungal therapy because of fungal death.

Systemically administered itraconazole or fluconazole may be useful in recalcitrant cases.

Stromal Abscesses

Focal trauma to the cornea can inject microbes and debris into the corneal stroma through small epithelial ulcerative micropunctures. Some stromal abscesses may be secondary to systemic disease.

A corneal abscess may develop after epithelial cells adjacent to the epithelial micropuncture divide and migrate over the small traumatic ulcer to encapsulate infectious agents or foreign bodies in the stroma. Epithelial cells are more likely to cover a fungal than a bacterial infection. Re-epithelialization forms a barrier that protects the bacteria or fungi from topically administered antimicrobial medications. Re-epithelialization of stromal abscesses interferes with both routine diagnostics and treatment.

Corneal stromal abscesses can be a vision-threatening sequelae to apparently minor corneal ulceration in the horse. A painful, blinding chronic iridocyclitis may result.

Most stromal abscesses involving Descemet’s membrane are fungal infections. The fungi seem “attracted” to the type IV collagen of Descemet's membrane.

Both superficial and deep stromal abscesses do not heal until they become vascularized. The patterns of corneal vascularization are often unique, suggesting that vasoactive factors are being released from the abscess that influences the vascular response.

Medical therapy consists of aggressive use of topical and systemic antibiotics, topical atropine, and topical and systemic NSAIDs.

Superficial stromal abscesses may initially respond positively to medical therapy. If reduced inflammation of the cornea and uvea are not found after 2–3 days of medical treatment, surgical removal of the abscess should be considered.

Deep lamellar and PKs are used in abscesses near Descemet’s membrane and eyes with rupture of the abscess into the AC. PK eliminates sequestered microbial antigens and removes necrotic debris, cytokines, and toxins from degenerating leukocytes in the abscess.

PK for Deep Corneal Stromal Abscesses

Corneal transplantation is performed to restore vision, to control medically refractory corneal disease, and to re-establish the structural integrity of the eye. It is a referral procedure.

PK is considered high risk for rejection in infected, vascularized corneal tissue. Nearly all PKs in horses are in high-risk corneas.

Fresh corneal grafts are preferred in horse PKs, but frozen tissue can be used.

Vascularization of the grafts, indicating rejection, begins at 5–10 days post-operatively.

Few equine PK grafts remain clear after their vascularization. They form a therapeutic and tectonic function.

Corneal Foreign Bodies

Superficial foreign bodies can often be coaxed out with a sterile cotton swab, flushed out with lavage, or “scooped out” by careful undermining with a needle or small biopsy punch. The remaining ulcer should be swabbed and lavaged with 2% povidone iodine/saline solution and treated medically. Very deep or penetrating ulcers must be referred for surgery and supportive care.

Burdock Pappus Bristle Keratopathy

This is commonly seen in the northeast in the fall, when many pastures have tall burdock weeds. Burdock balls are commonly found in the tail and mane. Affected horses present with signs of corneal ulceration or erosion, particularly in the medial canthus under the nictitans. The tiny burdock bristles are not visible in field conditions, but there may be vessel patterns in the conjunctiva of the nictitans or on the cornea that “point” to their probable location. All suspect areas should be debrided.

Nictitans debridement is facilitated by everting the whole membrane with a small towel clamp or hemostat and gently scraping the conjunctiva with the serrated edge of a sterile hemostat until it bleeds. Resolution is prompt if the bristles have been completely removed. Treatment involves topical atropine and antibiotics and systemic NSAIDs.

Calcific Band Keratopathy

This is a troublesome, painful condition that complicates some cases of chronic uveitis and may be related to topical steroid application. Gritty plaques of Ca++ are deposited in the corneal epithelium and upper stroma. May require superficial keratectomy and/or chelation with topical EDTA. Recurrence is common.

Eosinophilic keratoconjunctivitis (EK) has an unknown etiology, but may be an immune-mediated disease.

All ages and breeds of horses can be affected, with many cases reported in the spring. Clinical signs include corneal granulation tissue, blepharospasm, chemosis, conjunctival hyperemia, mucoid discharge, and corneal ulcers covered by raised, white, necrotic plaques. EK resembles a corneal tumor in appearance.
KCS may develop in affected horses because of lacrimal gland inflammation. The lacrimal gland should be palpated to detect swelling.

Corneal cytology typically contains numerous eosinophils and a few mast cells to rule out similar appearing infectious and neoplastic causes.

Superficial lamellar keratectomy removes plaques and speeds corneal healing.

Topical corticosteroids (1% prednisolone acetate or 0.1% dexamethasone) four to six times a day in early stages (despite corneal ulcerations), antibiotics (e.g., bacitracin-neomycin-polymyxin or chloramphenicol), 1% atropine, and 0.03% phospholine iodide (q 12 h) in combination with systemic nonsteroidal antiinflammatory drugs are indicated. Topical cromolyn sodium (4.0%, q 8 h) or lodoxamide (0.1%, q 8 h), mast cell stabilizers, can also aid healing. Systemic corticosteroids may be necessary. Horses with EK should be dewormed twice with ivermectin 10 days apart. These lesions are typically slow to heal. Scarring of the cornea occurs.

**Herpes Keratitis**

Multiple, superficial, white, punctate or linear opacities of the cornea, with or without fluorescein dye retention, are found associated with equine herpes virus 2. The focal punctate corneal opacities may be found at the end of superficial corneal vessels, and may retain rose bengal stain. Varying amounts of ocular pain, conjunctivitis, and iridocyclitis are present. Multiple foals in a herd may be affected. Topically administered idoxuridine and triflurourhymidine (q 8 h) have been used with topical NSAIDs for treatment of equine herpes ulcers, but recurrence is common. Lysine administered 20–30 g/day, PO, may be helpful.

**Lens**

The lens is a biconvex transparent body that is suspended in the front third of the eye by the zonular fibers that attach to the ciliary body. The anterior capsule rests against the rear or the iris, and the posterior capsule abuts against the vitreous body. The outer edge of the lens is called the equator; this bounds the outer cortex region of the lens. The central axis of the lens pierces the nucleus or central region. Suture lines that reflect the embryological development of the lens are apparent on both the anterior and posterior poles in the nuclear region. Normal lenses may show faint concentric rings towards the equator or the nuclear sclerosis of advancing age. Small clear vacuoles may be visible as tiny bubbles in the lens substrate—these are of no clinical significance.

Cataract is, by definition, any opacity of the lens or lens capsule. Cataracts form when there is a decrease in soluble lens protein, a failure of the lens epithelial cell sodium pump that keeps the inner lens dehydrated, lens fiber swelling, or membrane rupture. Cataracts are described according to location: anterior polar, anterior subcapsular, perinuclear, nuclear, equatorial, or complete. They are also described by extent: dense, diffuse, or focal. Focal cataracts are often described by appearance: floriform, stellate, vermiform, crystalline, or elliptical.

Congenital cataracts have been reported in Morgans and a few other breeds. A complete dense congenital cataract in an otherwise normal eye is a candidate for surgical removal. Surgery should be done before 6 mo of age by phacoemulsification. Horses appear to see adequately when aphakic.

Acquired cataracts are the most common type of cataract. Most often they are a sequelae of repeat attacks of uveitis. Dense cataracts that are secondary to uveitis are often accompanied by extensive posterior synechia and sometimes phthisis bulbi. They also can form after severe blunt trauma or penetrating injury as release of lens protein to the systemic circulation causes an autoimmune reaction. Aging brings cataract changes to many horses, similar to humans and dogs. Surgery on acquired cataracts is rarely performed because the complication rate with accompanying intraocular inflammation is high.

**Lens Luxation/Subluxation**

Loss of lens position is a common sequelae of chronic uveitis, especially in Appaloosa horses. If the lens is subluxated, an “aphakic crescent” will be obvious on focal illumination. If the lens is completely luxated, an opaque molded body will be apparent on the ventral fourth of the globe in the posterior segment. Occasionally, the lens will be luxated anteriorly into the anterior chamber and be visible as a large white or yellow elliptoid object. Syneresis or liquefaction of the vitreous accompanies lens luxation. Treatment is complicated for lens luxation.

**Uveal Tract**

**Heterochromia Iridis**

Heterochromia iridis, or dual coloration of the iris (usually blue and brown), is common to the Appaloosa, palomino, chestnut, gray, spotted, and white horses, and is not considered a true pathologic condition.

**Aniridia/Iris Hypoplasia/Enlarged Corpora Nigra/Iris Colobomas**

Aniridia, or the complete absence of the iris, is reported in Quarter Horses and Belgians and is also seen with congenital cataracts in Thoroughbreds. Congenital thinning of the iris (hypoplasia) to a full-thickness hole in the iris (coloboma) may be noted in heterochromic eyes. Enlargement of the corpora nigra can obstruct the pupil to cause vision problems.

**Iris and Ciliary Body Cysts**

Uveal cysts are a hallmark of the hereditary anterior segment abnormalities of the Rocky Mountain Horse and the Connemara pony. Iris cysts may be found sporadically in other breeds. The cysts con-
tain a thick vitreous gel-like material. Cysts at the ventral pupil margin appear to cause more vision problems.

Equine Uveitis: the Leading Cause of Blindness in Horses
By definition, uveitis is inflammation of the uvea of the eye (iris, ciliary body, and choroid). The complex of diseases known as ERU refers to intraocular inflammation that recurs or persists causing various degrees of inflammation, scarring, degeneration, and dysfunction of multiple components of the eye. Several classification schemes are used to differentiate subsets of clinically observed disease.

Classification by Observed Inflammation Over Time
In classic uveitis, horses show repeated bouts of severe inflammation and pain in one or both eyes. In between bouts, the eye(s) appear comfortable. Horses who are experiencing their first bout of classic uveitis are called primary cases. Because not all uveitis is recurrent, a case is not termed “recurrent” until two or more classic episodes have occurred.

In insidious uveitis, the horse seems normal to the owner, and the animals do not exhibit overt ocular pain, although astute observers may notice subtle abnormalities such as “dull” appearance to the globe, intermittent tearing or conjunctivitis, small pupils, and asymmetric appearance to the eyes. This is most often seen in Appaloosas, draft horses, or European warmbloods. Examination of the globe with a direct ophthalmoscope or slit lamp shows deterioration of numerous ocular structures that is progressive over time. Pupils may be slightly miotic. Subtle signs include corneal haze, slight aqueous flare, muddy iris color, slight miosis, iris rim atrophy, corpora nigra atrophy, cataract, low grade vitreous haze, and retinal scarring.

Classification by Stage of Disease at Time of Examination
Acute cases are horses who are suffering from a flare up of classic uveitis. Signs of an acute bout include pain, lacrimation, photophobia, chemosis, conjunctival hyperemia, corneal edema, corneal vascularization, dot-like keratic precipitates on the corneal endothelium, aqueous flare, hyphema or hypopyon, miosis, vitritis, and ocular hypotension (pressure of 10–12 mm Hg).

Quiescent cases are horses in a “calm” time of their cycle of repeat inflammation. Slit-lamp examination will show subtle flare in the anterior chamber, indicating low-grade persistent inflammation. Ocular examination may reveal scarring or “footprints” of previous disease including chronic corneal edema, iris atrophy, iris color change, synchiae (adherence of the iris to the lens or cornea), pigment rests on the lens, cataract, densities or haze in the vitreous, or scarring around the optic disc. Optic disc scarring is seen as either a focal “bullet hole” pattern of numerous tiny scars, or a “butterfly” pattern where wing-shaped islands of depigmentation flank either side of the optic disc.

End-stage uveitis cases have eyes that have undergone severe degeneration and chronic scarring. They may show extensive corneal scarring, circumferential synchiae, dense cataract, lens luxation or subluxation, secondary glaucoma, retinal detachment, or phthisis bulbi. End-stage uveitis is associated with vision loss.

Diagnosis of uveitis is simplified by understanding that a horse may present anywhere along the spectrum from acute to end stage and as either a classic or insidious case. If three of more of the above signs are found in a horse, and the history is suggestive of either recurrent disease or breed associated insidious disease, a presumptive diagnosis of uveitis may be made. Note that some of the signs are related to acute inflammation, and some represent chronic ocular changes that are permanent scars.

Some experts prefer to refer to the syndrome as “equine persistent uveitis” because inflammation is detectable on a cellular basis in all of these conditions, including quiescent and insidious cases. Miosis is a hallmark of most acute forms of recurrent or persistent uveitis.

Aqueous flare may be the first sign to appear and the last clinical sign to disappear.

Classification by Primary Observable Region of Ocular Involvement
Anterior uveitis: The observable inflammation is restricted to the anterior segment (cornea, AC, iris).

Posterior uveitis: Inflammation is predominantly observable in the posterior segment (vitreous, retina, and optic nerve). Most often seen in warmbloods and Appaloosas.

Panuveitis: Cases where the entire eye is inflamed. Anterior or posterior disease may progress to panuveitis over time.

Care must be taken to assure that there is not another primary problem in the eye that is causing internal inflammation. The cornea must be examined closely for signs of corneal ulcer, stromal abscess, foreign body, neoplasia, and immunemediated or idiopathic keratitis. The globe should be examined for neoplasia. Ruling out concurrent ocular disease that incites ocular inflammation is critical, because corticosteroid therapy is contraindicated in many of these conditions.

Pathophysiology
Uveitis begins with compromise of the blood–ocular barrier. This barrier normally functions to keep the aqueous and vitreous clear as tight fenestrations between the cells of ocular capillary walls prevent cells and large molecules from passing through the blood vessels of the iris and the choroid. The blood–ocular barrier also serves to isolate intraocular structures from the normal traffic of immune surveillance, making the tissue of the inside of the eye an immune privileged site.
In acute uveitis, the uveal blood vessels in the iris, ciliary body and choroid thicken and become congested. Soon they become “leaky,” and cells and inflammatory mediators cross the compromised blood–ocular barrier and enter the inside of the eye. Most of the cells that cross the barrier initially are neutrophils. This is seen grossly as hypopyon, aqueous flare, and vitreous haze. Neutrophils that enter the eye are soon replaced by large numbers of lymphocytes, some of which infiltrate the connective tissue of the ciliary body and iris, forming large follicle-like clusters. Antibodies and inflammatory cytokines are detectable inside the eye and within ocular tissues. These substances probably react with host and (in some cases) infective factors to contribute to ongoing pathologic change. Numerous heavy exudates appear on intraocular tissues, most notably on the posterior surface of the iris and ciliary body, on the capsule of the lens, and in the layer between the retinal pigmented epithelium (RPE) and the photoreceptors of the retina. The exudates interfere with the function of the ocular tissue they adhere to. Cytokine activity mediates tissue destruction. With repeated or persistent inflammation, the various chronic changes occur. Vision loss results when dense cataract and synchiae obscure acuity, when the retina detaches or degenerates and no longer can transmit processed light signals to the brain, or when glaucoma causes ischemic damage and degeneration of the optic nerve.

Etiology

Decades of research have substantiated that recurrent uveitis is an immune-mediated disease. However, some bacterial, viral, and parasitic infections have been associated as triggering events for the syndrome. Factors include the following: bacterial infections (leptospirosis, Borrelia burgdorferi [Lyme disease], brucellosis, Streptococcus, Rhodococcus equi [foals], generalized septicemia); viruses (influenza, equine viral arteritis, parainfluenza); parasites (onchocerciasis, strongylus, toxoplasmosis); and host conditions (tooth root abscess, severe hoof abscess, septicemia, severe trauma).

Of all the possible infectious triggers, leptospirosis is the most significant worldwide. Leptospirosis-associated cases account for at least 60% of the cases of ERU seen in our practice, which is located in a temperate river valley in the northeast United States.

Leptospirosis and Uveitis

The most significant serovars associated with disease are *L. interrogans* serovar *Pomona* (seen often in the United States) and *L. interrogans* serovar *Grippotyphosa* (seen often in Germany and central Europe). Factors that increase the risk of leptospirosis in horses include the following: pasture access to cows, pigs, or deer; close proximity to streams or ponds frequented by the same; use of piped pond water for drinking water; heavy infestation of stable with rats; and rainy season with persistence of ground water.

Horses become infected when they drink water contaminated by the urine of a carrier animal (often a cow, deer, pig, or rat). The spirochete gains access to the horse’s bloodstream by mechanical penetration of mucous membranes. Bacteremia results in clinical illness, manifested by anemia, fever, and flu-like symptoms. Clinical disease is mild and self-limiting, and rarely diagnosed as a leptospiral infection when acute. Resolution of signs does not mean elimination of the bacteria from the body—the spiral organisms often colonize the kidneys of the horse and may persist for a few months, being shed in the urine. Leptospirosis has also been associated with numerous cases of abortion in mares. Recent research has documented that the organisms can persist in the eye and may be recovered from ocular media by culture in some cases.

Ocular signs of classic uveitis associated with leptospirosis rarely occur during acute leptospiral infection but rather begin months after infection. Initial signs subside with or without therapy but recur at unpredictable intervals. Subsequent episodes of inflammation may be more or less severe than the initial one. Inflammation associated with subsequent episodes eventually compounds and damage of intraocular tissues creates visual deficits. Blindness is a common final outcome.

The pathogenesis of the “lepto link” with equine recurrent uveitis has been the subject of much research and debate. The key findings are as follows: antibodies to pathogenic serovars can be found in the sera, aqueous, and vitreous of horses with leptospirosis; and studies have supported the hypothesis that intraocular synthesis of these antibodies is occurring; leptospiral organisms have occasionally been cultured from the ocular media of horses with uveitis; molecular homology has been shown between the equine cornea and leptospira; pineal inflammation has been shown to accompany lepto-associated uveitis in horses similar to experimental models of uveitis in laboratory animals; major histocompatibility complex (MHC) II reactivity has been shown on resident and infiltrating cells of horses with both natural and experimental leptospirosis-associated uveitis; and seroreactivity to equine retinal proteins has been found in horses with lepto-associated uveitis.

A unified theory has yet to appear to explain all the ocular events that accompany leptospiroplastic-associated uveitis.

- Is it a direct toxicity of an intraocular infection with the spirochete?
- Is it an autoimmune disease against “self” that is triggered by molecular mimicry between leptospira and host tissue?
- Are leptospira somehow modulating the immune response of the eye?
These questions continue to challenge researchers. Although systemic infection with pathogenic strains of leptospirosis is clearly a common trigger for vision-threatening recurrent uveitis, it is likely that the genetic makeup, specifically that of the MHC complex, of the individual horse that contracts the disease plays a role in determining both susceptibility to leptospirosis as an inciting trigger and severity of subsequent inflammatory episodes.

**Testing Horses for Exposure to Leptospirosis**
Serum from horses diagnosed with uveitis should be submitted for modified agglutination test (MAT) analysis against a panel of leptospiiral serovars. Many non-uveitic horses will show low titers to the bratislava, autumnalis, hardjo, or canicola serovars. These are judged to be insignificant findings in northeastern practice geography. Positive titers, especially titers >1:400, to *L. interrogans* serovar Pomona or *L. interrogans*, serovar Grippotyphosa, are judged to be significant and a likely indicator of leptospiiral associated etiology. Seroreactivity to *L. interrogans* serovar Icterohemorrhagica is often paired with reactivity to *L. interrogans* serovar Pomona.

Research has shown that horses with uveitis can occasionally be seronegative for antibodies to leptospira and still have leptospiral DNA or live organisms in the eye. Therefore, a negative titer does not fully rule out leptospirosis as an etiologic factor. A positive titer to serovars Pomona or Grippotyphosa is, however, strong cause for concern.

**Breed and Uveitis**
Recent work has also shown that certain breeds are at risk for uveitis, most notably Appaloosas, European warmbloods, and draft horses. The Appaloosa breed is 8.3 times more at risk than other breeds for uveitis. Appaloosas that have insidious disease often have overall roan or light coat colors rather than dark coats with a rump blanket. The skin around the lids of affected Appaloosas is often mottled or pink in pigmentation. Mane and tail hair may be sparse. It is theorized that these horses have genetic proclivity to uveitis because of aberrations in the major histocompatibility complex, specifically in their equine lymphocyte antigen subtype. Recent research from Germany has supported this concept in German warmbloods susceptible to disease.

**Unilateral Versus Bilateral Disease**
Little work has been done to document the incidence of ocular involvement in horses, but recurrent uveitis can be a unilateral or bilateral disease. In a study of 160 cases: 50% of the lepto-associated horses had unilateral disease and 50% had bilateral disease; >80% of the Appaloosas had bilateral disease; and 62% of the cases that were seronegative to lepto, and also non-Appaloosas were unilateral.

Uveitis may begin in one eye and later occur in the fellow eye. However, if a case is unilateral and no attacks are seen in the other eye for 2 yr after the initial attack, it is uncommon for uveitis to show up later in the contralateral eye.

**Therapy of Uveitis**
Mydriasis is essential therapy for all cases of acute uveitis. Initial application of atropine should be frequent until the pupil is fully dilated. Severe cases may show poor response to the action of mydriatics.

Topical corticosteroids and systemic NSAIDs are the core elements of anti-inflammatory field therapy for acute attacks. Therapy should be intense for ~2 wk and may be tapered over another 2 wk. Subconjunctival and/or systemic corticosteroids are indicated in severe cases.

Corticosteroid topical therapy may induce calcific band keratopathy, especially in horses with leptospiral associated uveitis. Treat with EDTA chelation/keratectomy. Horses with acute uveitis are very painful and may suffer secondary corneal ulcers from self trauma. Corticosteroids are contraindicated in these cases—use topical NSAIDs instead and treat for any infection/collagenolysis of the ulcer.

Anecdotal reports indicate that a 1-mo course of oral doxycycline may help cases that are associated with leptospiral infection.

Acupuncture therapy may help moderate the frequency or severity of episodes.

Therapy does little to alter the progression of disease in horses with insidious uveitis.

Some horses (especially Appaloosas) develop secondary glaucoma late in their disease. Glaucoma therapy is difficult, but timolol, dorzolamide or a combination of these drugs (Cosopt) may be tried. Judicious topical steroids may help as well.

Although horses in central Europe routinely undergo pars plana vitrectomy for ERU, results have been poor with this procedure in the United States, and the procedure is not often advised here. Dr. Brian Gilger of North Carolina State University has some success with a surgery where a suprachoroidal device that slowly elutes cyclosporine is implanted under the sclera. Currently, this device is not available for private cases, but work is in progress to obtain FDA approval.

**Prognosis of Uveitis**
Visual prognosis for horses suffering from multiple acute attacks of uveitis or insidious chronic disease is always guarded. Data on the incidence of blindness in uveitic horses are lacking, but it is clear that uveitis is the leading cause of blindness in horses worldwide. The authors have observed ocular inflammation serious enough to threaten vision in at least 1–2% of the practice population. Analysis of the visual outcome of 160 cases followed over 11 yr revealed the following trends: 56% of the case series (89/160) lost vision in one or both eyes; 20% of the cases (32/160) became completely blind; and 36% (57/160) lost vision in one eye.
Breaking the cases down further into those that were seropositive or seronegative to *L. interrogans* serovar *Pomona* and those that were Appaloosas or “non-Appaloosas,” the following trends were seen: horses that were seropositive to *L. pomona* and also Appaloosas had a very poor visual prognosis: 100% lost vision in at least one eye and 50% went completely blind (n = 14); horses that were Appaloosas and seronegative had substantial occurrence of blindness: 72% lost vision in at least one eye and 29% went completely blind (n = 28); horses that were seropositive to *L. pomona* and non-Appaloosas had a slightly lower rate of blindness: 50% lost vision in at least one eye and 17% went completely blind (n = 86); horses that were seronegative and non-Appaloosas had the best visual prognosis: 34% lost vision in at least one eye and just 6% went completely blind (n = 32).

Secondary complications and degeneration of ocular tissues are common sequelae of uveitis. Several interesting findings were noted in this series that are representative of sequelae seen in ERU horses in other geographic regions of the world.

**Cornea**
Focal scars, streaks, calcium deposits, and other corneal opacities were common. The seropositive horses experienced a high rate of calcific band keratopathy. Striae and dense corneal streaks were common in Appaloosas and were highly correlated with blindness.

**Iris**
Iris atrophy and color change were common, especially in Appaloosas and seropositive horses. Anterior synechiae were rare unless phthisis bulbi was present, but posterior synechia occurred in nearly one third of all cases and 40% of Appaloosas.

**Lens**
Diffuse cataract(s) developed in 41% of all cases, and nearly 75% of the Appaloosas. These were a common cause of blindness. Lens luxation was common in Appaloosas (29%).

**Posterior Segment**
Severe vitritis was observed in nearly one third of the cases. Peripapillary scarring was also present in about one third of the horses. Cataracts and synechiae often obstructed posterior segment evaluation, so inflammatory changes were probably underreported.

**Glaucoma and Phthisis Bulbi**
Appaloosas had the highest rate of glaucoma (21%). Phthisical eyes developed most often in Appaloosas and seropositive horses.

Owners often are concerned that horses with ERU will need enucleation. In the above series, only 4% (6/160) were enucleated for complications from corneal infection or glaucoma. Of more concern is the fact that 43 of the 160 horses (27%) were treated for corneal ulcers over the observation period. Risk of corneal ulcers in ERU horses should be stressed, because owners often choose to medicate horses with painful eyes themselves, and they may potentiate serious infections by applying corticosteroids and delaying proper diagnosis. Ten horses suffered from calcific band keratopathy. This is a troublesome complication that limits therapeutic options for ERU.

Future understanding of recurrent uveitis in all species will revolve around the study of several key questions. What genetic makeup predisposes individuals to develop disease? If the disease is autoimmune, which autoantigens participate in the initiation and perpetuation of inflammation? What immune mechanisms initiate the immune response and mediate tissue destruction?

Study continues to determine the genetic predisposition for the syndrome in certain horse breeds (i.e., Appaloosas and German warmbloods). If a genetic marker is associated with susceptibility, horses with this genotype could be identified and excluded from use for reproduction, thus decreasing the prevalence of the disease. New immunosuppressive therapies, such as tacrolimus (FK506) may offer hope in the medical management. Perfecting a device to deliver such a medication may also be feasible. Studies are also being done to determine the role of leptospirosis or other microorganisms in the initiation and pathogenesis of ERU. Effort continues to further quantify the immune events that characterize inflammation and mediate recurrence. Vaccine research is ongoing at several universities.

### 4. Equine Glaucoma

The glaucomas are a group of diseases resulting from alterations of aqueous humor dynamics that cause an intraocular pressure (IOP) increase above which is compatible with normal function of the retinal ganglion cells and the optic nerve. Horses with previous or concurrent uveitis, aged horses, and Appaloosas are at increased risk for the development of glaucoma. Iris and ciliary body neoplasms can cause secondary glaucoma. Congenital glaucoma is reported in foals and associated with developmental anomalies of the iridocorneal angle.1-3

The infrequency of diagnosis in the horse may be caused, in part, by the limited availability of tonometers in equine practice but also by the fact that large fluctuations in IOP, even in chronic cases, may make documentation of elevated IOP difficult.

Afferent pupillary light reflex deficits, corneal striae, decreased vision, lens luxations, mild iridocyclitis, and optic nerve atrophy/cupping may also be found in eyes of horses with glaucoma.

The systemically administered carbonic anhydrase inhibitors acetazolamide (1–3 mg/kg, PO, q 12 h), the topical carbonic anhydrase inhibitor dorzolamide (2%, q 8 h), and the β-blocker timolol maleate (0.5%, q 12 h) have been used to lower IOP in horses with varying degrees of success. The newer prostaglandin derivatives cause low-grade uveitis and may exacerbate the IOP in horses with glau-
coma. Topical atropine therapy was once thought to reduce the incidence of glaucoma in horses with uveitis, but it should be used cautiously in horses with glaucoma because it may cause IOP spikes. Anti-inflammatory therapy, consisting of topically and systemically administered corticosteroids, and/or topically and systemically administered non-steroidal anti-inflammatory agents also seem to be beneficial in the control of IOP.

Laser destruction of the ciliary body (cyclophotocoagulation) works the best at controlling IOP and preserving vision in horses.1-5

Contraindications/Possible Interactions

Conventional glaucoma treatment with miotics may provide varying amounts of IOP reduction in horses. Miotics and prostaglandins can potentiate the clinical signs of uveitis and should be used cautiously in horses with anterior uveitis. The horse eye seems to tolerate elevations in IOP for many months to years that would blind a dog; however, blindness is the end result. Buphthalmia can be associated with exposure keratitis. Topical atropine does not seem to have the benefit of lowering IOP in a majority of glaucomatous horse eyes as originally proposed.

5. Optic Nerve/Peripapillary Region

The optic nerve is formed by the bundling of ~750,000 axons of the ganglion cells of the retina. The portion visible on fundic exam is the optic disc that is located below and slightly temporal to the posterior pole of the globe. It is ovoid in shape, with the longer horizontal diameter measuring 5–7 mm. When viewed with a direct ophthalmoscope, the optic nerve and other fundic structures are under ×8 magnification. The vascularization of the optic disc is paurangiotic, with 40–60 small arteries and venules that can be seen radiating from the disc. Horses with light coat colors may have a sparsely pigmented non tapetum, revealing the underlying tigroid pattern of the larger choroidal vessels. The peripapillary region shows great variability in appearance. Equine practitioners should consult atlases and texts on equine ophthalmology to look at pictures of normal variations of peripapillary pigment, vascularization, disc color, and myelin distribution.

The most common pathologic findings observed on fundic examination of the peripapillary region include the following.

Chorioretinitis

Chorioretinitis is inflammation of the choroid and retina. Inactive lesions are more often reported than active lesions. The tapetal region is rarely affected. It is manifested in equine eyes as focal “bullet-hole” retinal lesions, diffuse chorioretinal degenerative lesions, horizontal band lesions of the nontapetal retina, and chorioretinal degeneration near the optic nerve head.1-5

Active chorioretinitis appears as focal white spots with indistinct edges and as large diffuse gelatinous gray regions of retinal edema. Inactive chorioretinitis appears as circular depigmented regions with hyperpigmented centers or large areas of depigmentation that appear in some cases as the wings of a butterfly flanking the nasal and temporal margins of the optic disc. Consult atlases of equine ophthalmology for photographs of chorioretinitis and pictures of benign conditions like coloboma or variations of pigments in the non-tapetal area that are commonly confused with pathologic changes.

Lesions can be caused by infectious agents (e.g., leptospirosis, equine herpes virus-1, Onchocerca cervicalis, Rhodococcus, Streptococcus equi, Lyme’s disease, brucellosis, toxoplasmosis, Halicephalobus gingivalis), immune-mediated uveitis of unknown origin, trauma, or vascular disease. Chorioretinitis is likely found with or without the signs of anterior uveitis found with ERU. Serologic testing may identify infectious causes of chorioretinitis. Systemic NSAID medication is administered for chorioretinitis. Flunixin meglumine, phenylbutazone, or aspirin are indicated. Topical medication does not reach the retina and is only indicated if signs of anterior uveitis are also present.

Senile Retinopathy

This is commonly seen in aged animals. It presents as bilateral multifocal islands of hyperreflective tissue bound by darkened irregular linear hyperpigmentation, most prominent in the peripapillary nontapetal region. Senile retinopathy resembles the spiculated microscopic appearance of bone tissue.

Optic Atrophy/Ischemic Retinopathy

Disc shows severe pallor and attenuation of vessels, and it may also show extensive retinal depigmentation and focal pigment hypertrophy. It follows infarction from head trauma, intracarotid injection, embolism, or optic neuritis.

Proliferative Optic Neuropathies

Typical finding is a focal, lobulated pink or white mass that extends from the margin of the disc into the vitreous. It is generally asymptomatic, benign, and non-progressive; lesion may be reactive glial cells. No treatment is needed. Should be monitored to check for enlargement because the differential includes optic nerve neoplasia. Astrocytomas, medulloepithelioma, and neuroepithelioma have been reported. Documentation of an enlarging mass off the optic disc with blindness is an indication for immediate exenteration.

Retinal Detachments

Retinal detachment is a separation of the nine layers of the sensory retina from the RPE. It is associated with slowly progressive or acute blindness in horses. It can be congenital in newborn foals or...
acquired later in life in adults. Retinal detachments can occur bilaterally or unilaterally and be partial or complete. Complete retinal detachments are seen clinically as free-floating, undulating, opaque veils in the vitreous overlying the optic disc. The tapetum is hyper-reflective.

Retinal detachments are a complication of ERU and are also associated with microphthalmos, head trauma, perforating globe wounds, cataract surgery, equine protozoal myeloencephalitis, and may be secondary to tumors or vitreal degenerative processes. Retinal detachments can also be idiopathic. If the media of the eye are so opaque (e.g., corneal edema, cataract) that the fundus cannot be visualized, b-scan ultrasound can be used to diagnose the classic “seagull sign” of retinal detachment. Laser surgery and pneumatic retinopexy to reattach the retina are well described for the dog but have not yet been reported for the horse.

Congenital Stationary Night Blindness

Congenital stationary night blindness (CSNB) is found mainly in the Appaloosa and was once thought to be inherited as a sex-linked recessive trait. Up to 25% of Appaloosas may be affected. Cases are also noted in Thoroughbreds, Paso Finos, and Standardbreds.

Clinical signs include visual impairment in dim light with generally normal vision in daylight and behavioral uneasiness and unpredictability occurring at night. CSNB does not generally progress, hence its name, but cases of progression to vision difficulties in the daytime are noted. Ophthalmoscopic examination is normal. Diagnosis is by clinical signs, breed, and electroretinogram (ERG) with decreased scotopic b-wave amplitude and a large negative, monotonic a-wave. CSNB seems to be caused by functional abnormality of neurotransmission in the middle retina. There is no therapy for this condition, but affected animals should not be bred.

Equine Motor Neuron Retinopathy

Retinal pigment epithelial cell accumulation of ceroid lipofuscin is found to be associated with the neurodegenerative condition, equine motor neuron disease (EMND). Generalized weakness, muscle fasciculations, weight loss, and muscle atrophy characterize EMND.

Ophthalmic lesions are found in 30% of affected horses. A mosaic pattern of dark to yellow brown pigmentation in the tapetum of affected horses is noticed, associated with a horizontal band of pigmentation at the tapetal–non-tapetal junction.

Consistent evidence of a plasma vitamin E deficiency (<1.799 µg/ml) in horses with EMND suggests that the RPE, retinal, and spinal lesions are a result of oxidative injury associated with a prolonged deficiency of nutritionally derived antioxidants.

Although visual deficits may not be consistently found, nyctalopia and ERGs with a 50% reduction in b-wave amplitude and normal appearing a-waves have been noted associated with EMND.

Supplementation with 6000 IU vitamin E per day in horses with EMND may stabilize the neurologic signs, but it is not known if this will affect the RPE and retinal changes.

6. Tips on Therapeutics for Equine Eye Problems

If ophthalmic ointments are prescribed, spend time with the owner showing the best way to apply them. Helpful tips include pointing out the orbital rim and telling the owners that they can rest the finger that is holding the lid open against, or slightly underneath, the rim dorsally. Having the owners touch the crease of the eyelid to lift it dorsally is another useful tip.

Dispense topical meds in a 4 × 6-in “Write on bag.” Write out the treatment schedule for that medication for the next several days by making circles on the bag label next to days of the week. This simple step increases compliance and keeps the medication clean.

Study ophthalmic textbooks for eyelid plastic surgery techniques. Practice some procedures on cadavers to get a feel for tissue handling. Become comfortable using fine 4-0 sutures for repairs. The author schedules suture removal of all skin sutures—whether resorbable or not—at 8–12 days, because the eyelid tissue retains some persistent inflammation if these sutures are left in.

The use of a fly mask or modified racing blinder hood helps protect the periorbital region after surgery. Be sure that there is not a “greenhouse” effect from a closed blinker—perforate the plastic if needed.

Decision making on handling tumors in the periorbital region is difficult because there are many options for treatment. If the tumor is large, close to the lid, or approaching the globe, referral may be the best option. Recurrence or ineffective treatment may have a disastrous result.

Nictitans removal is a simple surgical procedure that can be done at a clinic or in the field. I like to do it within stocks at our clinic.

Train owners to call for same day examination on any lids that show severe swelling.

Debridement of the cornea should be performed in superficial ulcers only. It can be as simple as swabbing the lesion with a sterile swab or as involved as actually cutting away some cornea with a no. 15 or no. 63 blade.

Subconjunctival injections are easiest if the horse’s head is supported by a table made of bales, and the handler, who is standing on the contralateral side of the horse, uses the ear of the horse to tilt the top of the head away from the clinician, thereby exposing the target sclera.

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Application of topical anesthetic using sterile cotton swabs will facilitate subconjunctival injections and work on the nictitans.

Lavage catheters (SPL tubes) are useful ways to deliver medication to the surface of the eye, especially in fractious horses. Order the longer “Florida catheters” from Mila. The footplate should be put in the fornix of the upper or lower lid. Tape the treatment end to one half of a tongue depressor to make injection of medication easier. The treatment end should be made of a 20-gauge catheter inserted in the tubing and plugged by a standard injection cap. Inject 0.1–0.2 ml of drug at a time and “chase” with 1.0 ml of air before injection of additional medications. Horses that rub should wear hoods so they do not dislodge the system. Adhesive tape “butterfly” tabs sewn to the skin work better for securing the tube than the guides that come with the kits. Weave the long delivery tube through braids in the mane to prevent inadvertent tearing of the tubing. Medication loss will be minimized and horse comfort maximized if injections of liquid and air are delivered slowly through the injection port. See detail in Table 1 for information on fortified solutions that can be administered through the lavage tube. Most horses with SPLs are on complex medication schedules that include a variety of anti-infective agents, a mydriatic, and one or more anti-collagenase therapies.

7. Key Prognostic Points

Prognosis of orbital and periorbital disease varies with the etiology, but prompt assessment, vigorous therapy, and judicious referral will optimize outcome. Client seminars or handouts discussing eye problems should stress that lumps, cuts, and asymmetry of the region around the eye can be very serious, and they should schedule examination of these conditions without delay.

Prognosis of eyelid lacerations or reconstruction of acquired scars is dependent on meticulous technique. Immediate repair of lacerations is desirable, but if the clinician is presented with an “old” injury, there may be plastic surgery strategies that will effectively reconstruct the eyelid. Prognosis of the repair is optimized if the clinician uses careful placement of small gauge sutures and takes care in reconstructing the tarsal plate. Some severe lacerations may be stabilized by temporary tarsorrhaphy. Placement of the head of the horse on a “table” made of bales will facilitate standing surgery.

Corneal ulcers can be simple traumas that resolve uneventfully or complex problems that are among the most complicated and expensive that horses experience. Often, prompt assessment and aggressive therapy wards off disaster. Practitioners should commit to seeing painful eyes on the date of occurrence.

Many drugs are used to treat corneal problems. Familiarity with the available medications will optimize success of treating the equine cornea. Many of these meds are human products that must be prescribed through pharmacies, compounded, or obtained at universities. Table 1 summarizes many of these products.

Cataracts are rarely operated on unless congenital and complete. Lens luxation and cataract are frequent complications of uveitis.

References and Footnotes