Equine Ophthalmology

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This seminar is a review of advances in medication; medication delivery techniques employed in equine ophthalmology; and the diagnosis and treatment of selected ophthalmic diseases.   Author's address: 1701 S. Whitcomb St., Fort Collins, CO 80526-1932.   © 1998 AAEP.

1. Therapeutics Review

This review is intended to describe briefly some of the medications, techniques, and devices currently employed in equine ophthalmology.   One should not assume that the products and techniques mentioned are the only ones acceptable for treating any given disease, but they will provide a foundation for the sound management of ocular disease.   Some of the drugs and techniques are new, whereas others are not.

Since the successful medical management of ophthalmic disease is directly related to the method of medication used and the selection of appropriate drugs, a brief review of medication techniques is necessary before drugs can be discussed.   The methods of medicating the eye are topical, subconjunctival, intraocular, and systemic administration.   The disease presented will determine the medication technique or combination of techniques that will be most effective.   The efficacy of topical medication is determined by the chemical characteristics of the drug, the health of the cornea and conjunctiva, and the frequency of application.   In the equine, it is further limited by the diluting effect of high tear production and the difficulty in administering drops or ointments to a painful eye.   The use of subpalpebral delivery systems is the most common method of providing intensive topical medication.   They re-quire only chemical restraint and local anesthesia for placement.

2. Topical Medication

Drops are often difficult to administer to horses, especially those with a painful eye.   A small syringe with the hub of a 25-gauge needle that has been broken off can be used to direct a fine stream of medication across the eye.   This is especially useful during an examination when a topical anesthetic or fluorescein solution is instilled.

Subpalpebral delivery systems are easy to place (Fig. 1) but have disadvantages, which include slippage of the tubing and infection or irritation of the eyelid.

Double-passage tubing can be easily placed but has a higher potential of corneal contact, which may result in a corneal ulcer (Fig. 2).

Single-passage tubing (Fig. 3) can be made by using silicone tubing with a small oval of silicone sheeting glued to the end to prevent the tubing from pulling through the eyelid.   A commercial silicone tubing for transpalpebral medication is available from Mila International[a] and is sold with trocar, adapter tip, and suture clips.   After placement, the tubing can be connected to a bottle for gravity flow or a micrometered delivery system for continuous therapy.

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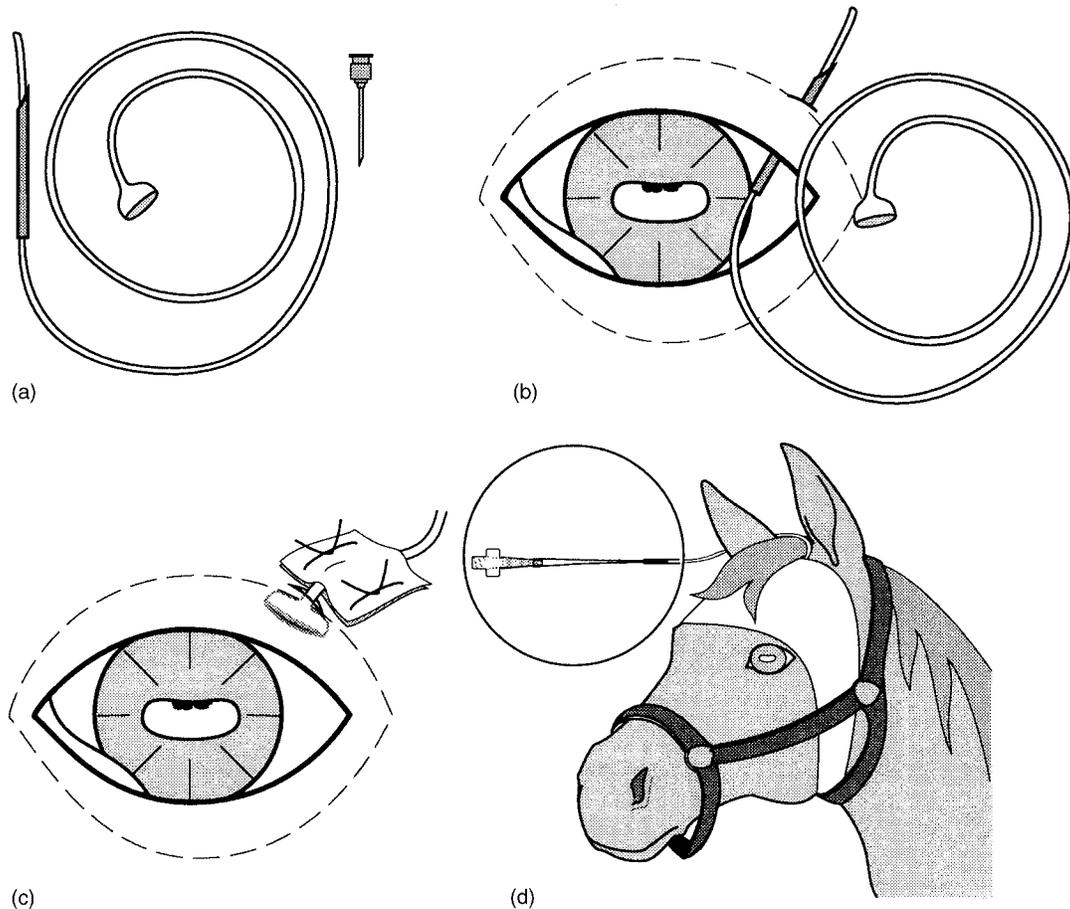

Fig. 1.   Placement of single-passage tubing: (a) fluted tubing, a 12-gauge needle with hub removed, and a needle to cannulate tubing; (b) needle tubing is passed through the upper fornix; (c) medication tube is seated in the superior fornix and anchored to the skin with butterfly tape; (d) medication tube is covered with tape.

Nasolacrimal systems are more difficult to position but cause less irritation, allowing them to remain in place for weeks.   A commercial nasolacrimal medication tube, made by Arnold's Veterinary Products Limited, England, is available from Jorgensen Laboratories.[b]   The tubing is positioned approximately 15 mm up the nasolacrimal duct.   An 8 French nasal feeding tube can be positioned in a similar manner (Fig. 4).   The backflow of medication along this type of nasolacrimal tube requires a larger volume of medication to treat the ocular surface adequately.

A nasolacrimal medication tube extending to the palpebral punctum can be positioned by using a 5 French, 36-in. (~91 cm) feeding tube or Stillman urinary catheter (Fig. 5).   With this type of tubing, all of the medication is delivered directly to the eye.

3.   Subconjunctival Injection

A subconjunctival injection may have a short-term or prolonged effect, depending on the medication administered, and will it result in increased drug levels in the tear film as well as direct ocular penetration at the injection site.   Subconjunctival injections provide a means of drug delivery to the equine eye when frequent topical applications are not possible or when supplemental therapy is necessary.   The injections are safe to perform and easily administered (Table 1).   The injection should be given under the bulbar conjunctiva to be effective. The injection of drugs into the eyelid or under the tarsal conjunctiva is not effective, because the rapid uptake of such drugs is into the eyelid circulation and away from the globe.   Prior to giving a subconjunctival injection, the veterinarian should be sure of the diagnosis and the desired effect of the drug being administered.   It would be disastrous to inject a corticosteroid in an eye affected with a mycotic keratitis.

4.   Intraocular Injection

An anterior chamber (intracameral) injection (Fig. 6) is usually restricted to (1) the use of 1:10,000 epinephrine in a physiological saline solution to control anterior uveal hemorrhage or dilate the pupil during intraocular surgery; or (2) proteases such as plasminogen activator (tPA) to dissolve fibrin clots.

An intravitreal injection (Fig. 7) is usually restricted to cases of suspected bacterial or mycotic endophthalmitis.   For most drugs, the appropriate
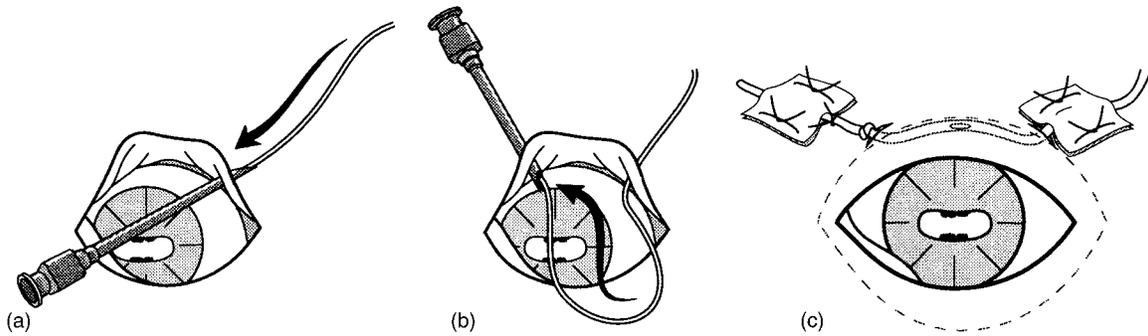
Fig. 2.   Double-passage tubing: (a) needle is passed through conjunctiva and the upper eyelid at the temporal fornix.   Tubing has been placed in the needle before it is withdrawn.   (b) Needle is passed through the eyelid, entering the nasal fornix, and tubing is placed before the needle is withdrawn.   (c) Small opening is made in the tubing before it is secured with butterfly tapes and suturing.
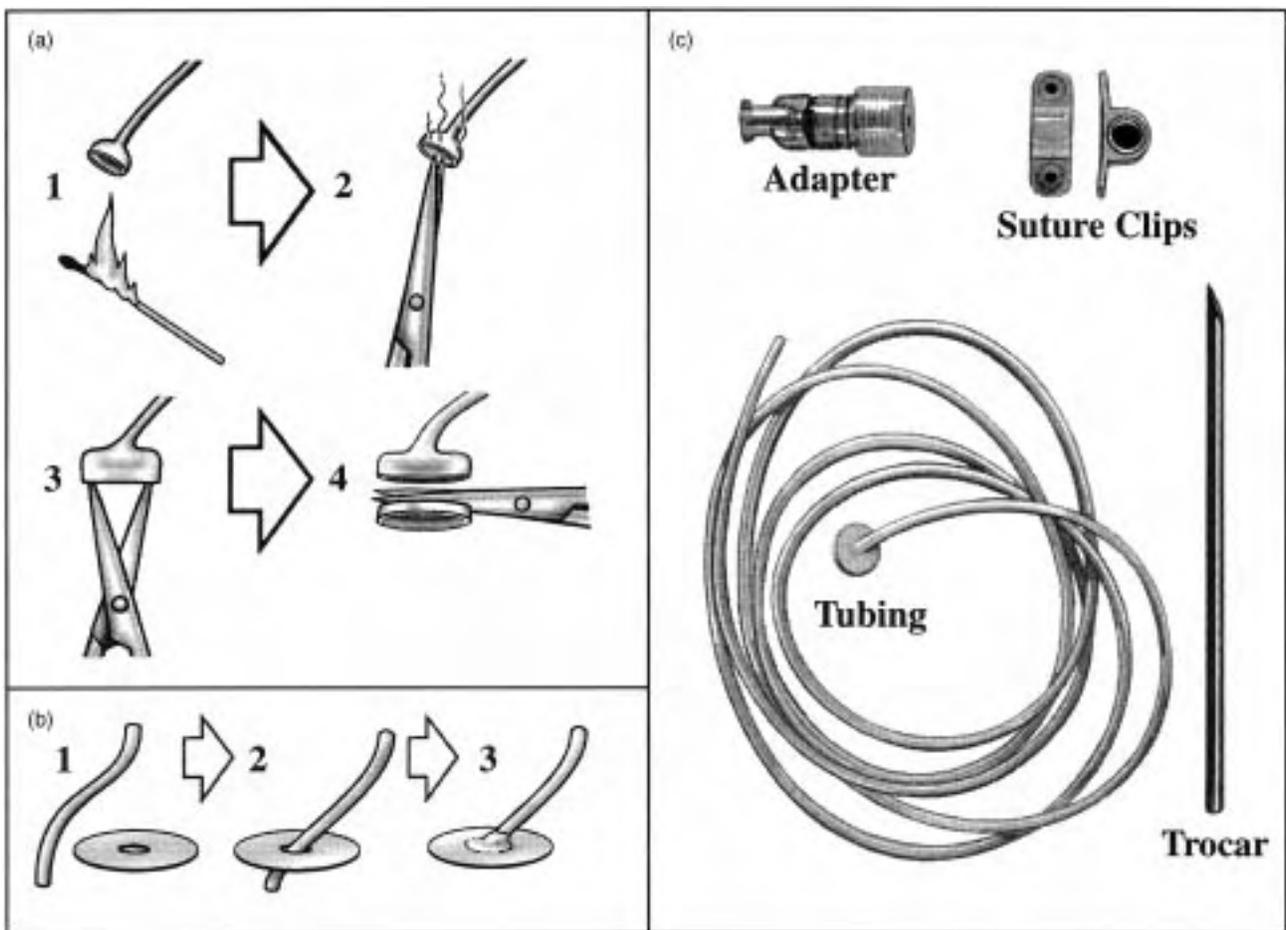

Fig. 3.   Single-passage subpalpebral delivery systems: (a) Fluted medication tube using polyethylene is shown.   1.   Tubing is heated over a flame.   2.   Scissors are inserted into the bell tip of the hot tubing.   3.   Scissors are opened to form a fluted tip.   4.   Edge is trimmed if needed.   (b) Silicone tubing is glued to silicone sheeting with silicone adhesive.   Allow several days for silicone adhesive to set up.   This has the fewest complications.   1.   Silicone tubing and a strip of silicone sheeting with a small hole are shown.   2.   Tubing is slipped through the hole in sheeting.   3.   Appearance of tubing after adhesive is applied and excess tubing is trimmed.   (c) Silicone tubing with trocar, skin suturing clips, and infusion adapter.   Continuous infusion units are available but not shown.

equine dosage has not been established; therefore, I use one to two times the maximum human dosage recommended by the manufacturer.   The intravitreal administration of an oculotoxic mixture of gentamicin and dexamethasone should be considered as a nonsurgical alternative to treat a blind eye with persistent pain secondary to equine recurrent uveitis or chronic glaucoma.   Unfortunately, a small percentage of blind equine recurrent uveitis and glaucoma patients have pain that cannot be con-
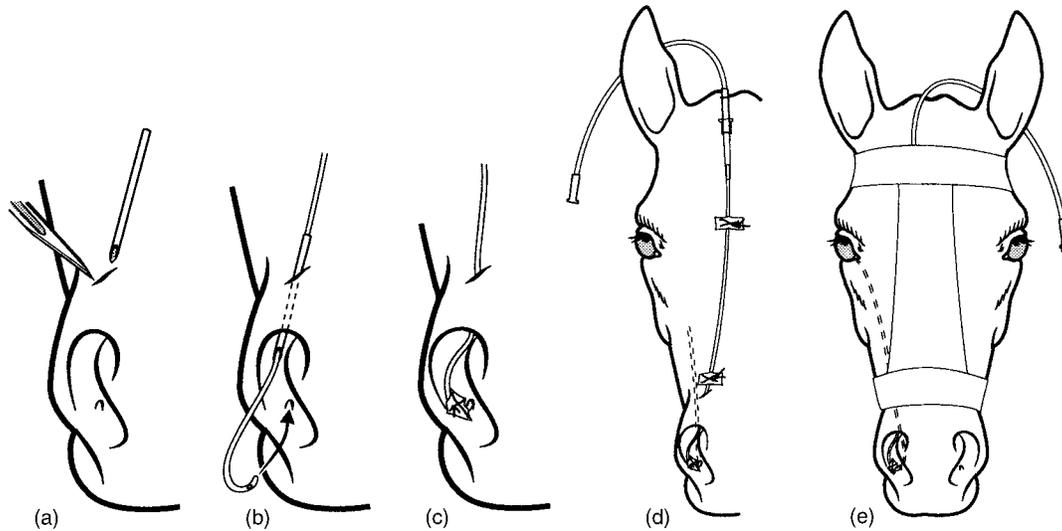
Fig. 4.   Placement of an 8 French nasal feeding tube: (a) Incision is made over false nostril with scalpel blade.   (b) 10-gauge hubless needle is passed through the incision, allowing an 8 French feeding tube to be drawn through the needle.   (c) Needle is withdrawn; the feeding tube is passed up the nasolacrimal duct.   Butterfly tape is placed on the tube and secured at the nasal punctum with nonabsorbable figure-8 suture.   (d) Tubing is secured to the skin with additional butterfly tape.   (e) Tubing is covered with Elasticon tape.

trolled medically.   In the past, surgical removal or an intraocular prosthesis was the only choice to make the animal comfortable.   The intravitreal injection of a mixture of gentamicin 25–50 mg and dexamethasone 1–2 mg will frequently arrest the process, resulting in a cosmetic and pain-free eye. The owner should be advised that the dosage is estimated on the basis of the size of the eye and the severity of the lesion.   The owner should be advised that (1) an injection may require repeating if signs persist; or (2) the eye may develop phthisis bulbi if the response to the injection is greater than expected. The patient is usually much more comfortable 2–3 days after injection, with the maximal response being in ~20 days.   Eyes are usually more sensitive to a second injection; therefore, reducing the dosage is recommended.   If the horse continues to have problems, the eye can always be removed later.

5.   Antifungals

Mycotic infections involving the cornea represent frustrating and devastating processes if not diagnosed and treated promptly.   One should always consider that a nonresponsive chronic ulcer has the potential of having a mycotic component.   All horses with mycotic keratitis should have concurrent topical broad-spectrum antibiotic therapy to prevent bacterial infection of the devitalized corneal tissues.

1.   Natamycin was the first ocular antimycotic drug approved for topical use.   It is available in a 5% ophthalmic suspension.   The spectrum includes *Candida, Aspergillus, Cephalosporium, Fusarium,* and *Penicillium.*

2.   Miconazole is a synthetic imidazole antifungal solution that is available in 20-ml ampules as a 1% solution for intravenous infusion.   Miconazole has a broad spectrum of activity against yeast and filamentous organisms, although some resistant forms of *Fusarium* may be encountered.   It is not approved for topical use on the eye but is safe and effective as a topical medication.   Five to 10 mg may be injected subconjunctivally daily for 5 days to supplement topical therapy.   It was my first choice of treatment for mycotic keratitis, but is not readily available now.   Miconazole can be used concurrently with natamycin.

3.   Clotrimazole is readily available over the counter as a 1% lotion and ointment, e.g., Lotrimin, for the treatment of dermatomycosis in man.   These preparations are effective for treating ocular mycosis, but unfortunately the bases are irritating to the eye.   Therefore, it can only be recommended until other antifungal medications can be located.

4.   Thiabendazole (TBZ) is effective but not commercially available for ocular use.   The use of 14.2% and 43% thiabendazole worming pastes applied directly to the eye has been reported to be successful but is irritating.   Diluting these preparations to 4% in artificial tears is recommended.

5.   Fluconazole in a 0.2% intravenous solution can be used topically, and 2.5–5 mg/kg can be divided every 12 h systemically.

6.   Ketoconazole as a 2% dermatologic cream can be used topically, but it may be irritating.   It can also be used orally at 2 mg/kg.

7.   Itraconazole, in the form of topical ophthalmic preparations, is not available.   One percent drops can be prepared by using artificial tears.   A 1% itraconazole–3% dimethyl sulfoxide ointment has been reported to be effective.[1]

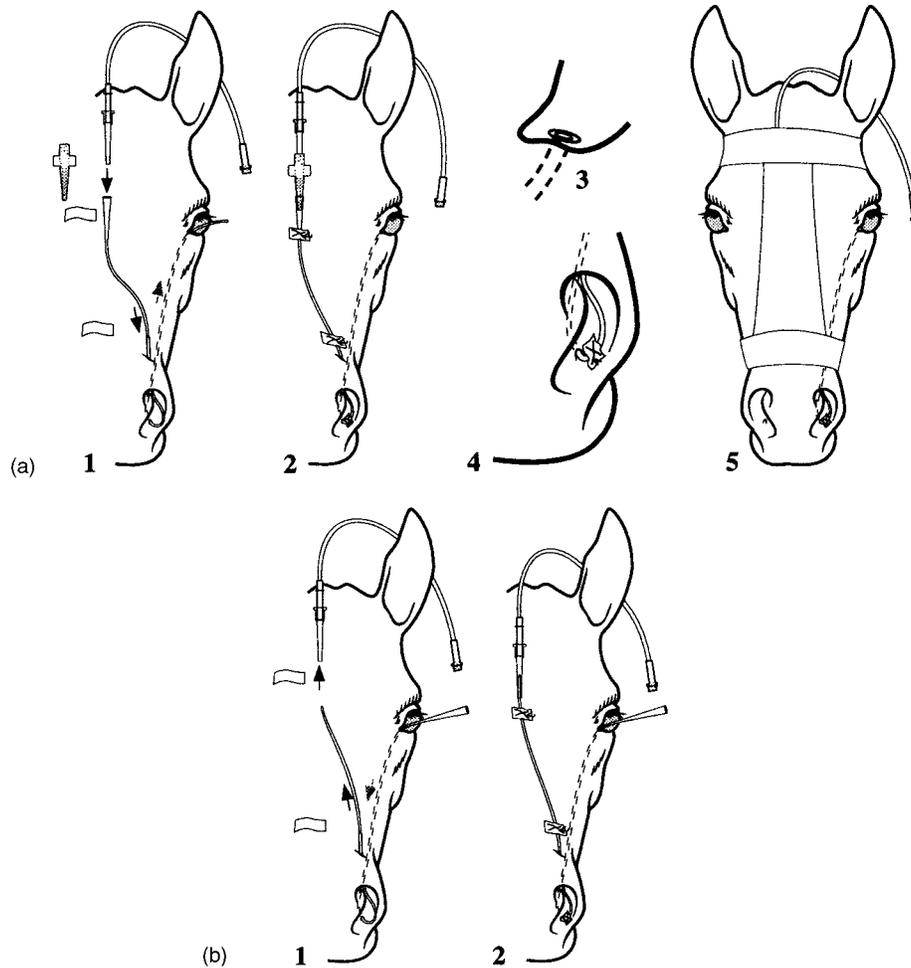8.   Silver sulfadiazine as a 1% ointment can also be used.

Fig. 5.   Placement of a 5 French nasal feeding tube: (a) Retrograde passage from nasal to palpebral punctum is shown.   1.   With the technique described for placing an 8 French feeding tube, a 5 French nasal feeding tube is passed retrograde from the nasal punctum until it emerges at the palpebral punctum.   A Christmas tree adapter and IV extension tube may be needed for additional length.   2.   Final positioning and securement of the tubing with butterfly tapes are shown.   3.   Tubing end is removed flush with the palpebral punctum.   4.   Position of tubing and butterfly tape after they are sutured in the nostril is shown.   5.   Tubing covered with Elasticon tape is ready for use.   (b) Passage of tubing through upper palpebral punctum if retrograde passage is unsuccessful is shown. 1.   Tubing is passed and the blunt end of the feeding tube is cut off and ready to be forced into the catheter adapter for connection to an IV extension.   2.   Final placement of tubing before it is cut flush with the palpebral punctum is shown.   Tubing will be covered with Elasticon tape as shown in (a.5).

6.   Anti-Inflammatory Drugs

Anti-inflammatory agents include corticosteroids and antiprostaglandins.   Prednisolone acetate 1%, prednisolone phosphate 1%, and dexamethasone 0.1% are the most effective topical corticosteroids.   Topical antiprostaglandins can be used when ulcerative keratitis would make corticosteroids contraindicated. Flurbiprofen sodium 0.03%, profenal 1%, and diclofenac 0.1% are used in human ophthalmology to control miosis during cataract surgery.   Diclofenac has been approved for treating intraocular inflammation.   An indomethacin 1–2% suspension can be made from indomethacin capsules or powder for solution.

Delayed-absorption corticosteroids are frequently used for subconjunctival injections (Table 1).   Triamcinolone 40 mg/ml is excellent.   Methyl predniso-

lone is longer acting but has the potential of causing a persistent mineralized plaque at the injection site. This plaque is irritating and may require surgical removal.

The long-term systemic administration of high levels of corticosteroids may be dangerous because of the potential to cause secondary laminitis.   Because of this danger, the antiprostaglandin agents flunixin meglumine, phenylbutazone, or aspirin are recommended in combination with topical and subconjunctival corticosteroids.   Flunixin meglumine 1 mg/kg q 24 h is very effective in controlling ocular pain from any cause and is my initial choice of systemic anti-inflammatory agent for acute ocular inflammation.   Ketoprofen 10% IV solution, 1 mg/lb q 24 h is also effective.   Phenylbutazone 1–2 g, q 12 or 24 h can also be used, but in my experience it is not as

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Table 1.   Recommended Dosages for Subconjunctival Injections

Drug	Dosage
Antibiotics	
Ampicillin	250 mg
Cephaloridine	100 mg
Chloramphenicol succinate	200 mg
Erythromycin	50–100 mg
Gentamicin	50 mg
Kanamycin	20 mg
Lincomycin	150 mg
Penicillin G	500,000 IU
Tobramycin	5 mg
Antifungals	
Amphotericin B	125 µg (3 times/week for 3 weeks)
Miconazole	5–10 mg q 24 h for 5 days
Nystatin	5000 IU
Autonomic Drugs	
Atropine (large animal 2 mg/ml)	1–2 mg[a]
Phenylephrine (10 mg/ml)	5–10 mg[a]
50:50 mixture of above (1 mg of atropine + 5 mg of phenylephrine/ml)	0.5–1 ml[a]
Corticosteroids	
Short-term effect	
Prednisolone acetate suspension	5–10 mg
Dexamethasone injectable	2 mg
Depot effect	
Betamethasone	3 mg
Triamcinolone	20–40 mg
Methylprednisolone acetate	20–40 mg
Anticollagenase	
Equine tetanus antitoxin	1 ml

[a]I usually use the lower end of the dosage range.

effective in acute ocular inflammation as flunixin meglumine. Aspirin is the preferred systemic drug for chronic disorders. The initial treatment is 25 mg/kg q 12 h for 5–30 days, followed by 30 mg/kg q 24 h indefinitely. I have not observed any untoward reaction to the aspirin administered at these dosage levels, and I feel the recurrence rate is higher if the dosage rate is reduced before 2 weeks. In some animals, treatment can be reduced to every other day.

7. Glaucoma

Primary equine glaucoma is uncommon. Treatment with 2–4% pilocarpine q 6 h has been successful, but this frequency of medication is difficult to maintain for extended periods. A polymerizing gel containing 4% pilocarpine (Pilopine HS Gel[c]) is effective with twice daily treatment. Humorsol 0.5% is an indirect-acting parasympathomimetic that can be used twice daily.

Other new drugs are also available. Dipivefrin 0.1% is a pro-drug of epinephrine that requires biotransformation to epinephrine before therapeutic activity occurs. Dorzolamide HC1 2% is a topical carbonic anhydrase inhibitor that is applied three times daily. If it is given with other topical medications, allow 10 min between medications. β-adrenergic blocking agents currently are the most frequently prescribed medications for human glaucoma. This has made it difficult to locate some of the older medications. Examples of β-blockers are betaxolol 0.5%, carteolol 1%, levobunolol 0.5%, metipranolol 0.3%, and timolol 0.5%. Latanoprost 0.005% ophthalmic solution can be given as one drop daily or twice daily. This is a new prostaglandin $F_{2\alpha}$ analog that reduces intraocular pressure by increasing uveoscleral outflow. This may be helpful in horses because they have a greater uveoscleral outflow. If topical medication is not possible, cyclodestructive procedures such as cryotherapy or transscleral laser therapy may be beneficial.

Secondary (obstructive) glaucoma is generally chronic and a complication to chronic uveitis [see Fig. 11 (b.4) below]. It is usually nonresponsive to medical treatment. If the eye is still visual, cryotherapy or transscleral laser therapy should be considered. If the eye is blind, enucleation, an intraocular prosthesis, or the intravitreal injection of gentamicin and dexamethasone should be considered.

8. Keratoconjunctivitis Sicca

1. The goals in treating keratoconjunctivitis sicca (KCS) are to supplement tear formation, stimulate lacrimal activity, control infection, cleanse the eye, and control corneal changes. Tears can be supplemented with artificial tears, ointments with or without antibiotics, and viscoelastics. Artificial tears have been improved with the use of artificial mucin, which increases contact time. Viscoelastic agents such as sodium hyaluronate and hydroxypropyl methylcellulose 2% are used for intraocular surgery and are also effective as a corneal protectant. Unfortunately, they are expensive. A less expensive alternative is sodium hyaluronate preparations for equine joint disease (e.g., Equron,[d] Hyalovet,[e] and Synacid[f]).

2. Keratoconjunctivitis sicca is not common in the horse and when seen is usually a transient disease that can be managed temporarily with tear substitution two to six times daily until tears return. I have seen it as a primary inherited disease, but it is usually seen with facial paralysis or guttural pouch infection. A topical cyclosporine ophthalmic ointment 0.2% is the drug of choice if long-term treatment is needed. Small-animal practitioners sometimes prepare a 1% solution in corn oil. Some animals that do not respond to this drug may respond to a 0.5% ointment made by liquefying USP petrolatum in a hot water bath and then adding a 10% cyclosporine solution to make a 0.5% concentration. This is stirred and then put into a 6-ml syringe before it solidifies.

3. A solution can be prepared that includes antibiotics to control infection, acetylcysteine to dissolve mucus, and pilocarpine to stimulate tear production.
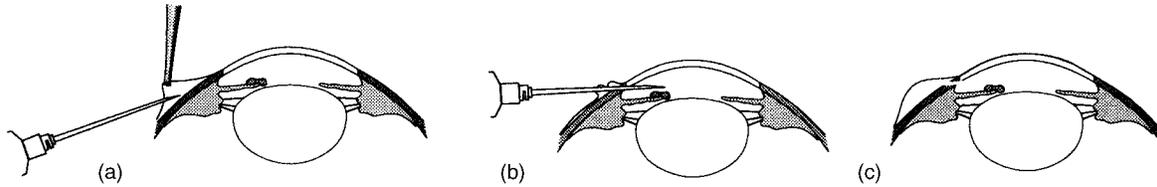
Fig. 6.   Anterior chamber injection: (a) Pick up conjunctiva several mm away from the dorsal or dorsolateral limbus.   Introduce a 25- or 27-gauge needle at this spot and tunnel beneath the epithelium as the needle is advanced toward the limbus.   (b) Traverse the limbus, using a drilling motion with the needle.   Withdraw aqueous or inject the desired amount of solution.   (c) When the needle is withdrawn, a subconjunctival aqueous bleb will develop.   This disappears in several hours.

9.   Orbital Prosthesis

Enucleation of the equine eye results in a deep cavity that, even though covered with skin, is objectionable in appearance.   Cosmetic results can be obtained when the orbital cavity is filled with a silicone rubber ball implant.[g]   These implants are available in colors (I prefer black) and sizes 28, 30, 33, 36, 38, 40, 43, 45 and 47 mm in diameter at a cost of approximately $18.00 each.   Following surgery, the skin contracts over the implant and results in a smooth contour across the orbit.

10.   Technique

The globe and third eyelid are removed by using a transpalpebral approach (Fig. 8).[2]   Care is taken to ensure the removal of the orbital lacrimal gland and the gland of the third eyelid, while removing as little as possible of the extrinsic eye muscles and retrobulbar fat.

After the eye has been removed, the largest implant that will easily fit into the orbit is selected. One side of the implant is trimmed flat until it lies level with the orbital rim when facing forward in the orbit.   The implant is secured into position by sutures placed in the periorbital and subconjunctival tissues.   This suture pattern will resemble the lacing across a shoe.   An optional subcuticular suture may be placed before closing the skin.   Before skin sutures are complete, 0.5–1.0 million units of potassium penicillin are injected around the implant. A compression bandage applied over the orbit for 24–48 h is recommended.   Systemic antibiotics are administered for 5 days.

Trauma to the orbit at a later date may displace the prosthesis or create a wound, with subsequent loss of the implant.

11.   Intraocular Implant

Chronic uveitis or glaucoma often results in blindness.   If a blind eye is continually inflamed and painful, requiring medication, it may be feasible to replace the contents of the globe with a silicone implant (Fig. 9).   If the eye is not significantly buphthalmic, a T incision in the sclera may be needed to insert the implant (Fig. 10).

The intraocular implant is not a guarantee of freedom from ocular problems.   Corneal necrosis may develop or the eye may become infected.   In either case, enucleation would be required.   Blind eyes are more likely to be injured; therefore, the client should be instructed that any change in the appearance of the eye or the presence of an ocular discharge or pain warrants an immediate examination.

12.   Equine Recurrent Uveitis: Etiology, Signs, and Management

Equine recurrent uveitis (ERU) is an immune-mediated disease affecting the anterior ocular segment (iris, ciliary body, cornea, and lens), the posterior ocular segment (choroid, retina, optic nerve, and vitreous), or both.   It is the most common cause for blindness in horses and mules.   Synonyms for the disease include iridocyclitis, moon blindness, and periodic ophthalmia.

The exact etiology of ERU is seldom determined because the initiating uveitis may pass unnoticed before the owner is aware ERU exists.   Uveitis is
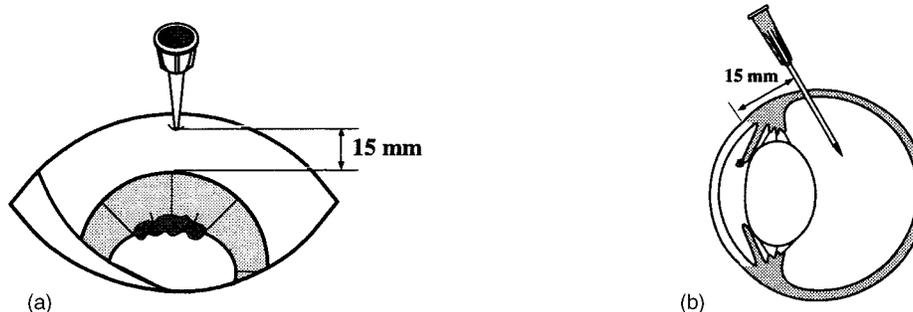

Fig. 7.   Vitreous injection: (a) anterior and (b) sagittal views.   A 23-gauge needle is inserted through the conjunctiva and sclera ~15 mm posterior to the dorsal limbus or 10 mm at the lateral limbus with the needle directed toward the optic nerve to avoid the lens.

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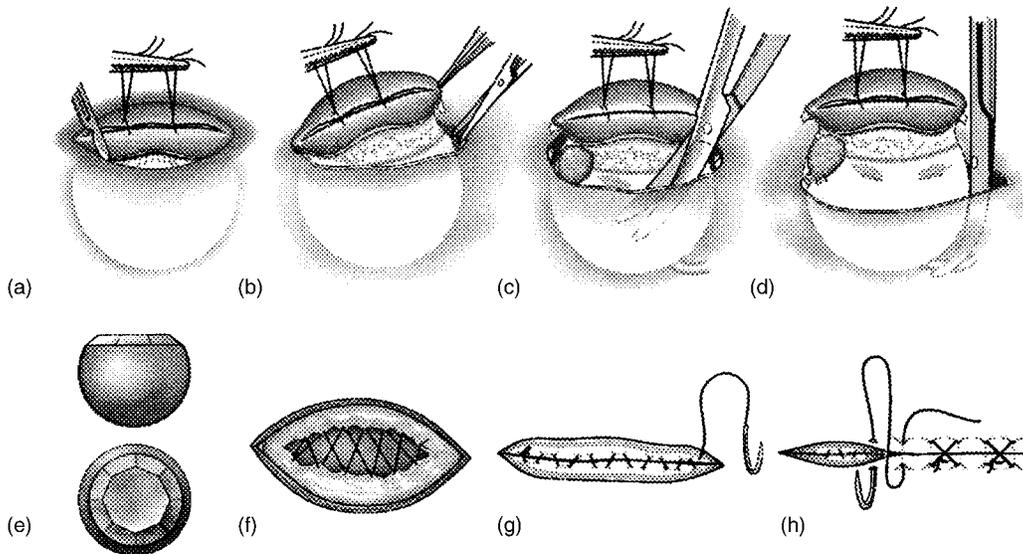
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Fig. 8.   Transpalpebral enucleation: (a) Eyelids are sutured together and skin is incised ~5–7 mm from the palpebral border. (b) Nasal and temporal attachments of eyelids are severed.   Blunt dissection with scissors is continued to the conjunctival attachment of the limbus.   (c) Extrinsic muscle insertions are separated from the sclera with scissors.   Care should be taken to remove the temporal lacrimal gland and the gland of the third eyelid.   (d) Carefully elevate the globe enough so that the optic nerve and retractor oculi muscle can be severed with enucleation or curved Mayo scissors.   (e) Silicone rubber orbital implant is trimmed for placement. Side view is above; front view is below.   (f) Periorbital fascia and tissues are closed over the implant with absorbable sutures. Penicillin G is injected behind the implant (250,000 IU).   (g) Subcutaneous tissue is closed and penicillin G is is injected in front of the prosthesis.   (h) Skin is closed with a cruciate suture pattern.

the most common intraocular lesion in horses and has many causes, which include trauma, intraocular surgery, corneal ulcers, interstitial keratitis, local or systemic infection, extension of central nervous system diseases, immune disorders, and severe toxemia. Infectious diseases that have been associated with uveitis are leptospirosis, brucellosis, *Staphylococcus equi, Rhodococcus equi,* toxoplasmosis, systemic fungi, herpesvirus, and equine viral arteritis.   *Onchocerca cervicalis* has been long proposed as a cause, but the high distribution of this parasite and its incidence in many animals with normal eyes make a causal relationship difficult to establish. It has been demonstrated that when the parasite dies, inflammation can occur.   Keratoconjunctivitis or keratouveitis have been observed when large numbers of microfilaria migrate into the conjunctiva, superficial stroma of the cornea, and intraocular tissues. Migrating *Setaria* and *Dirofilaria immitis* may gain access to the anterior chamber, where they are quite active and startling to see.

The initial uveitis may heal uneventfully or lead to a persistent immune-mediated response resulting in ERU.   This immune response may be a type I, immediate hypersensitivity; type III, arthus immune complex hypersensitivity; type IV, cell-mediated hypersensitivity involving antigen and sensitized T lymphocytes; or ocular tissue autoimmunity that may result from ocular trauma or be spontaneous. Phacolytic uveitis is associated with the escaping of lens protein from an injured or cataractous lens.

The most frequent observations alerting an owner to have his or her horse examined include ocular discharge, blepharospasm, change in appearance of the eye (this may be a cloudy cornea or a red eye), and, less frequently, signs of disturbed vision.

The examination should begin outdoors in bright light.   The examiner takes a position in front of the patient, where he or she can determine if the lesions are unilateral or bilateral.   The examiner evaluates the eyes and surrounding structures for the presence of ocular discharge, notes the position of the eye in the orbit as well as the size of the eye, and determines if there is any periorbital swelling.   As the horse is led indoors to the examination area, it should be observed for any responses to the environment that would suggest a visual deficit.   The examining area should be well lighted and be capable of being darkened so the anterior segment can be re-examined with a transilluminator and ophthalmoscope.   A portable surgical lamp is an excellent adjunct to a lighted room examination.   Chemical restraint (xylazine, detomidine, or butorphanol) local nerve blocks, or both may be needed if the horse is uncooperative.   If the lesion is unilateral, always examine the normal eye first.

Clinical signs of anterior segment involvement vary depending on the acuteness of the disease, the tissues involved, and whether the lesion is unilateral or bilateral.   Mild involvement may demonstrate only epiphora, conjunctival catarrh, and slight photophobia.   In acute uveitis [Fig. 11(a)] the initial signs are photophobia, blepharospasm, and enophthalmos with marked protrusion of the third eyelid.   A ca-
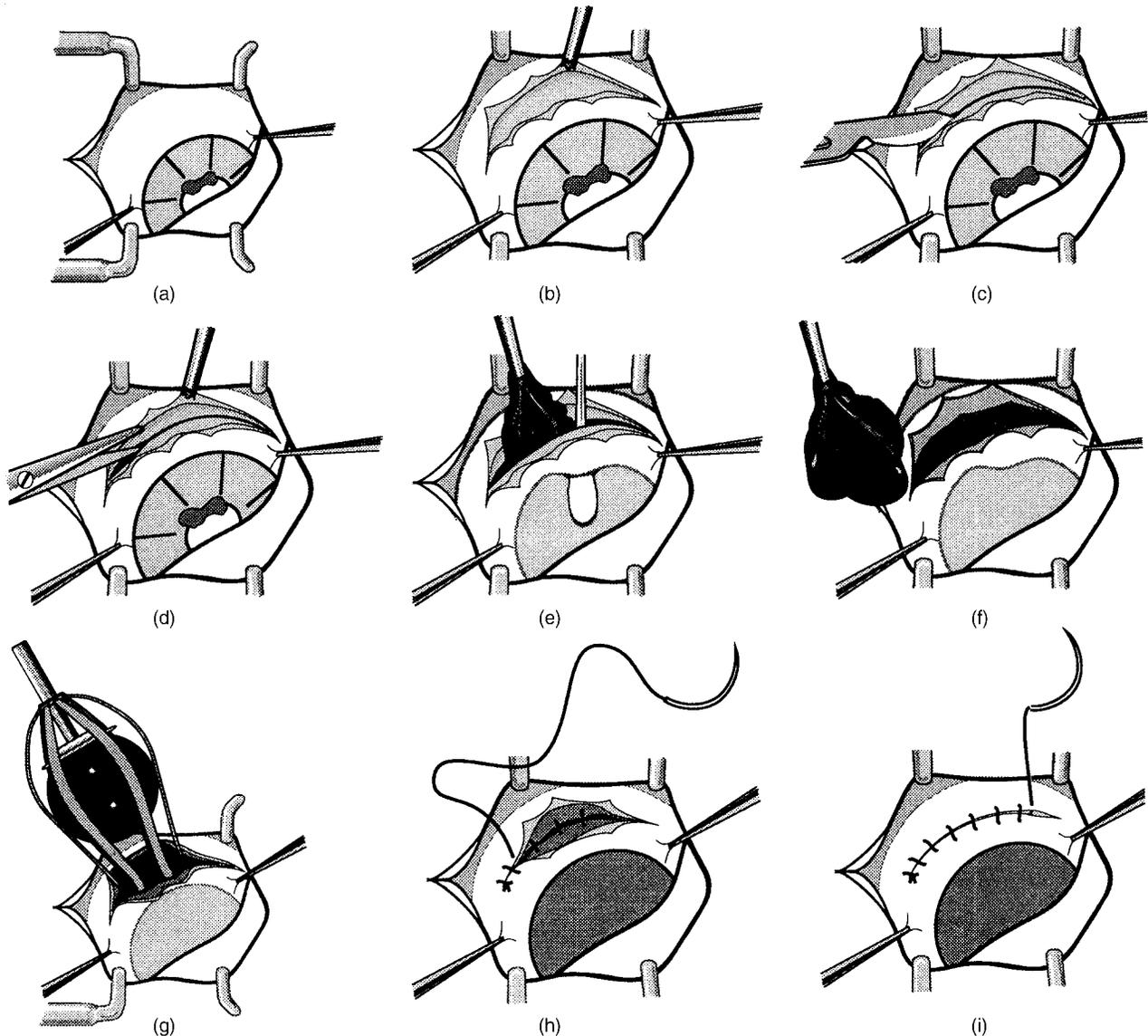
Fig. 9. Intraocular prosthesis for a blind glaucomatous eye: (a) Eye is prepared for surgery. Lateral canthotomy and eyelid speculum provide exposure. Eye is stabilized with fixation or mosquito forceps at 9 and 3 o'clock. (b) Conjunctival incision is completed and sclera is exposed. (c) Scleral incision initiated with scalpel results in collapse of the eye. (d) Incision can be completed from the 10 to 2 o'clock positions with Mayo scissors. (e) Ciliary body is separated from sclera with a Green spatula. (f) Appearance of the eye after evisceration. Ocular contents are visible to the left of the globe. (g) Carter sphere introducer with silicone rubber sphere ready to transfer to the globe are shown. (h) Sclera is sutured with a continuous pattern after 3 appositional sutures are placed. Note the eye is filled with blood. (i) Continuous suture pattern in conjunctiva is shown.

tarrhal conjunctivitis will be present; this results in epiphora that may become seropurulent after 24 h. Episcleral injection will be seen. The iris will be swollen, dull in color, and have a miotic pupil (Fig. 11). An examination of the aqueous with a slit beam of light will reveal aqueous flare. The horse will be head shy and may have signs of diminished vision.

Tonometry will reveal a very soft eye. If a tonometer is not available, digital tonometry can be performed by directly touching the cornea or by palpating the globe through the eyelids. I prefer to touch the cornea directly. After the eye is topically anesthe-

tized, the examiner can estimate intraocular pressure by touching the cornea with his or her finger or a muscle hook. When the cornea of a hypotonic eye is touched with the examiner's finger, the cornea will indent before the examiner is aware that pressure has been put on the cornea. If the intraocular pressure is normal, the examiner is aware that slight finger pressure must be applied before the cornea begins to flatten. In glaucoma, obvious pressure is needed to compress the corneal surface. If the lesion is unilateral and both eyes are examined by this technique, significant differences of intraocular pressure can be detected. When a muscle hook is

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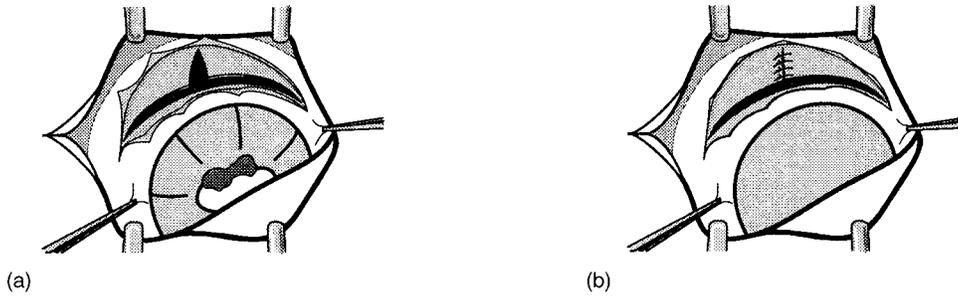

Fig. 10.   T scleral incision for an equine intraocular implant: (a) incision is started at the 12 o'clock position and extended toward the equator of the globe; (b) the incision is closed with interrupted sutures before the parallel incision is closed.

touched to a hypotonic eye, an obvious indentation in the cornea occurs before corneal resistance can be felt.   In the normal eye, resistance will be felt before the cornea indents; when glaucoma is present, obvious pressure will be needed to indent the cornea. This technique does not have as much of the inherent error that is associated with palpating the globe through the eyelid.

After the anterior segment has been examined, the pupil should be dilated and the posterior segment should be examined by using the ophthalmoscopy techniques described in my previous presentations. The posterior segment changes will be described later in this article.

During the next 1–3 days the eye may change rapidly.   These changes are progressive eyelid edema and chemosis.   The ocular discharge becomes more purulent.   Corneal edema develops, which may interfere with an examination of the deeper structures. A perilimbal corneal flush develops as a result of congestion of limbal corneal vascular loops.   Keratitic precipitates appear on the inferior one half of the cornea.   The nonpigment epithelium of the ciliary body becomes toxic and the blood–aqueous bar-

rier breaks down, resulting in clotted fibrin or blood in the anterior chamber.   The iris becomes extremely edematous and posterior synechia begin to form.   If the pupil is dilated at this time, fibrin, sloughed uveal pigment, and portions of the corpora nigra may be seen adhering to the lens.   Intraocular pressure continues to be low.

Corneal vascularization begins 3–6 days after onset and if inflammation persists, may proceed centrally at a rate of approximately 1 mm/day. Severe corneal endothelial damage may result in corneal edema, and in some patients edema may be so severe that the cornea may show a ripple effect when the patient blinks.   This may progress to a bullae formation that may rupture, resulting in corneal ulceration.

If systemic disease is suspected as a cause for ERU, then a complete blood cell count, urinalysis, and blood chemistry are indicated.   If infectious diseases such as leptospirosis, brucellosis, toxoplasmosis, or systemic fungi are suspected, then serological tests should be considered.   In some cases, an autoimmune profile may be indicated.

If anterior segment disease, whether mild or se-
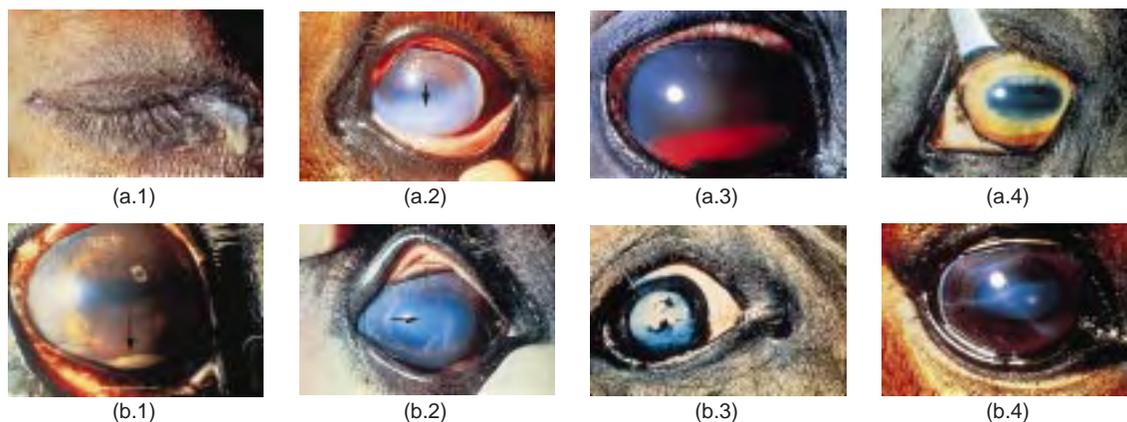
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Fig. 11.   Anterior uveitis: (a) Acute.   1.   Horse has photophobia and lacrimation.   2.   Lacrimation, conjunctival injection, corneal edema, keratic precipitate (arrow), miotic pupil, and swollen iris are shown.   3.   Hyphema—note the hemorrhage is stratified with dark red blood near the lower limbus and more recent hemorrhage above.   The pupil has been dilated and the lens appears cloudy. 4.   Corneal vascularization near the limbus indicates several days' duration, moderate corneal edema, and swollen iris with miotic pupil.   (b) Chronic.   1.   ERU relapse, showing miotic pupil, hypopyon (arrow), dark iris with superficial vascularization, and episcleral vascular injection.   2.   Quiescent uveitis with corneal vascularization (arrow), mild corneal edema, and dark iris. 3.   Quiescent ERU with secondary cataract.   Note uveal pigment on the lens that was exposed by the dilated pupil.   4.   Obstructive glaucoma.   The white lines in the cornea (Haab's striae) are due to rupture of Descemet's membrane.   The iris is very dark.

vere, were to resolve with treatment at this time, the eye could heal without further deterioration. But unfortunately in many cases the mechanism for a persisting immune-mediating disease is established and ERU results. Clinical signs may subside and go through quiescent periods followed by mild recurrent episodes to acute exacerbations. One of the most consistent signs of chronicity is darkening of the iris in the diseased eye. It is not unusual to have a horse presented with a history of no previous eye disease that upon examination is found to have a unilateral dark iris, which indicates that this is ERU and not an initial uveitis episode.

The sequelae [Fig. 11(b)] that may result from anterior segment ERU are corneal scarring, corneal endothelial degeneration with or without bullous keratitis, anterior and posterior synechia, complicated cataracts, lens luxation, and vitreous opacities. Severely damaged eyes may develop phthisis bulbi from extensive ciliary body degeneration. Obstructive glaucoma occurs when the filtration angle becomes obstructed. The signs include buphthalmos, corneal striae (interconnecting parallel lines caused by the rupture of Descemet's membrane), and a deep anterior chamber.

Symptomatic treatment of anterior ERU with anti-inflammatory therapy and mydriatics should be started immediately. Specific treatment is indicated when an etiology has been determined. Anti-inflammatory drugs are the most important part of ERU therapy. They inhibit the immune-mediated response and consequently reduce the uveal congestion so that the uveal blood–aqueous barrier can return to normal.

My routine protocol for the initial treatment of acute uveitis consists of the intravenous administration of flunixin meglumine 1 mg/kg, a subconjunctival injection of triamcinolone 20 mg or betamethasone 3 mg, and a separate subconjunctival injection of 1 mg of large-animal atropine. If there is evidence of synechia or profound miosis, 5 mg of injectable phenylephrine can be included in the atropine injection. I do not routinely use systemic prednisolone or dexamethasone because I feel the synergistic effect of flunixin and subconjunctival and topical steroids is as effective as systemic and topical steroids. This drug combination eliminates the laminitis risk seen with steroid therapy, especially with dexamethasone.

Topical treatment should be started with antibiotic–steroid ophthalmic ointments containing prednisolone (e.g., chloramphenicol and prednisolone) or dexamethasone (e.g., Maxitrol[h]) four times daily if possible. Mydriasis can usually be maintained with 1% atropine ophthalmic ointment four times daily. I have never found it necessary to use concentrations higher than 1% and therefore have never observed atropine-related colic. If topical medications cannot be properly administered, subconjunctival injections may be repeated as needed or a medication delivery system can be placed.

Systemic antibacterial agents are indicated when systemic infections are suspected or as a prophylactic measure in acute cases. The animal should be confined or the eye should be protected with a hood. Warm compresses are beneficial in the presence of periocular swelling. When systemic disease is diagnosed, specific treatment should be initiated and the symptomatic treatment of uveitis should be continued as long as needed.

Maintenance therapy consists of continuing the flunixin 5–7 days. If the eye is improving, aspirin 25 mg/kg PO q 12 h can be substituted. If the eye is not doing as well as expected, phenylbutazone or systemic corticosteroids can be given.

As the patient improves, atropine is reduced and can eventually be given as infrequently as every 4–5 days before being discontinued. Atropine will remain in the iris of horses longer than in the iris of other species.

Animals with severe corneal edema will benefit from treatment four times daily with 5% sodium chloride ophthalmic ointment. This edema is usually transient and will be self-limiting when the corneal endothelium returns to normal. Persistent corneal edema suggests either a continuing endothelial immunological response or permanent corneal endothelial degeneration. Topical or subconjunctival corticosteroids will be beneficial if an active immunological response exists. If corneal endothelial degeneration has occurred, corticosteroids will not help and an unfavorable prognosis must be given.

Glaucoma is usually chronic when diagnosed. If the eye is still visual, medical or surgical management is recommended. If the eye is blind, an intravitreal injection with 1–2 ml of a 50:50 mixture of 5% gentamicin and 0.2% dexamethasone will control glaucoma. The owner should be advised that the response to this injection is variable. Some eyes will be extremely sensitive, becoming hypotonic and developing phthisis bulbi. In either event the eye eventually becomes comfortable and may remain cosmetic. If glaucoma still persists 2 weeks after the first injection, a second injection should be given. Blind ERU eyes that are refractory to medical treatment and continue to be painful can be treated with 1 ml of mixture in the manner recommended for glaucoma.

Posterior ERU is rarely presented during the active stage unless blindness results. A blind eye may go undetected by the owner unless the animal involved is a performance horse. Some animals will be presented with a history of sudden blindness, but an examination will reveal that one eye has been blind for a long time and now the other eye has recently become blind. In the active stage of posterior ERU, optic neuritis or chorioretinitis or both are seen. In the chronic stage (Fig. 12), optic nerve atrophy, retinal scarring and degeneration, or retinal detachment may be present. Mild lesions will

COLOR

(a)

(b)

(c)

(d)
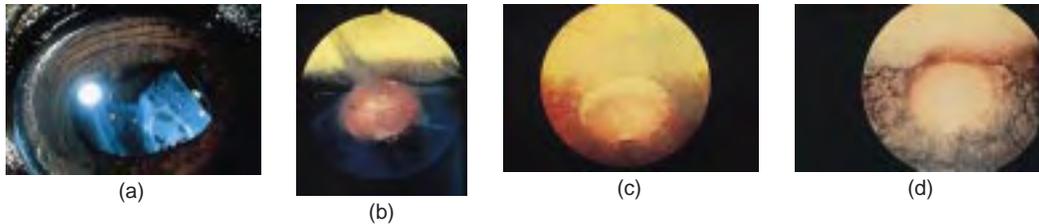
Fig. 12.   Chronic posterior ERU: (a) Lens luxation reveals the detached retina; (b) optic nerve atrophy and retinal folds are shown; (c) optic nerve atrophy and retinal degeneration dorsal to the optic nerve are shown; (d) chorioretinal degeneration in the nontapetal area surrounding the optic disk is shown.

be frequently seen as an incidental finding on a health or insurance examination.

Animals with a visual deficiency may be presented with a history of personality change, shying, poor performance, or becoming awkward.   The blind side may be difficult to examine if blindness is recent and associated with rapid onset.

The examination should be performed in the same manner as that previously recommended, i.e., examining the patient outdoors and then indoors in a darkened room.   Pupillary reflexes should be checked before the pupils are dilated with 1% tropicamide.   The modified otoscope is an adequate ophthalmoscope for a screening examination.   When abnormalities are noted, a thorough examination should be completed with a direct ophthalmoscope and, if available, an indirect ophthalmoscope, which will give a better visualization of the peripheral retina.

Optic neuritis is referred to as papillitis.   The optic nerve appears red as a result of congested blood vessels.   Small hemorrhages may be seen.   The physiological cup disappears as papillitis progresses.   Circumpapillary edema will make the blood vessels near the optic disk appear suspended above the choroid.   Exudate and small blood clots may be seen in the adjacent vitreous.

Optic nerve atrophy will be seen more frequently than optic neuritis.   The optic disk appears chalky white and flattened.   Blood vessels will be attenuated in early atrophy and absent in advanced atrophy.   In rare instances one half of the nerve will be atrophied and the remainder will be normal.

Active circumpapillary chorioretinitis will have congested blood vessels; the retina may appear hazy.   White infiltrates and small hemorrhages are usually seen.   If large areas are involved, subretinal fluid may build up, leading to detachment.

As inflammation regresses and the lesion becomes quiescent, retinal degeneration and chorioretinal depigmentation will develop.   Later some irregular areas of repigmentation occur and the lesion may take on a butterfly appearance.   Tapetal lesions tend to be multiple, more linear in appearance, and have areas of hyperreflectivity and pigment reorganization.   Rarely is the retinal degeneration extensive enough to cause blindness.

Retinal detachment is usually complete, but sectional detachments may occur.   Acute optic neuritis and chorioretinitis should be treated with systemic prednisolone or dexamethasone and prophylactic antibiotics until laboratory tests are available.   This treatment can be risky if infectious diseases are not ruled out first, but the treatment must be aggressive if vision is to be saved.   If subretinal edema is present, the patient should receive furosemide 0.5 mg/kg IV q 24 h to assist in the removal of fluid and to promote reattachment of the retina.   Treatment is not recommended for patients with optic nerve atrophy, retinal scars, or chronic retinal detachment.

13.   Eyelid and Corneal Lacerations

Lacerations of the eyelids and cornea are common and, except for minor injuries, require surgical intervention for proper healing.   The abundant blood supply of the eyelid ensures rapid healing of eyelid lacerations, whereas severe corneal injuries heal slowly, requiring neovascularization before healing is complete.   Common causes for eyelid and corneal lacerations are owner-inflicted trauma, and the animal's striking its head during loading or unloading, catching the eye or eyelids on a sharp object, or fighting with other horses.   Owner-inflicted lacerations result when the horse is struck with a sharp object.   A blow from a blunt object routinely results in contusion to the lids and periorbital soft tissues.   If the globe is involved, a corneoscleral rupture may result.

A.   Eyelid Lacerations

Eyelid lacerations would be expected to bleed profusely for a short period of time and then gradually stop unless the patient continues to injure the area.   An eyelid laceration with minimal tissue loss that has received proper wound closure and after care will heal without deformity.   Immediate treatment is indicated for wounds with a duration of <4 h.   A delayed repair is indicated if the patient is in shock, has other serious injuries, or if the laceration has been present for >12 h.   Proper cleaning and bandaging of the wound will reduce swelling and control infection, thereby allowing better surgical repair and increasing the possibility of first-intention healing.   If the wound is 4–12 h old, the surgeon should use his or her own judgment as to immediate or delayed repair.   If treatment is to be delayed, the wound should be gently cleaned with

0.2% saline dilution povidone-iodine solution (1/50 strength 10% Betadine solution[i]).

The eyelids should then be covered with a cotton square that has had a liberal application of nitrofurazone 0.2% ointment and should be bandaged with stretch adhesive tape. The laceration can be repaired the next day in the same manner as that for a fresh wound.

I prefer general anesthesia for optimal wound apposition on all eyelid lacerations, but minor lacerations can be repaired by using chemical restraint and infiltration of a local anesthetic. After the area has been clipped or shaved, the wound, adjacent skin, and the eye are cleansed with a dilute povidone-iodine solution for 2 min and covered for 2 min more with a sponge saturated with this solution. Standard surgical instruments and sutures are adequate to repair eyelid lacerations, but conjunctival suturing is simplified with an ophthalmic needle holder, conjunctival fixation forceps, strabismus scissors, and ophthalmic suture.

After the wound is draped, it is minimally debrided. If there is severe swelling and the conjunctiva has retracted, it will be beneficial to place an apposition suture at the palpebral edge. If this is not the case, suturing the conjunctiva first is preferred. The conjunctival sutures are continuous and placed so that the knots are buried in the wound and therefore will not rub the eye. The appositional skin suture is placed along the sharp edge of the eyelid, with the suture going through the meibomian glands. The suture ends should be cut short so they will not irritate the cornea. An alternative would be to leave the ends long and place them near the wound beneath later sutures. The next suture is placed in the skin ~3 mm from the edge of the eyelid. This suture should be deep (nearly to the conjunctiva) and incorporates the meibomial–tarsal plate. This is the tension suture that holds the wound together. As many sutures as needed are placed to finish closing the wound. An eyelid sutured in this fashion will heal with a smooth palpebral edge (Fig. 13). If there is swelling or eyelid tissue loss that could result in postsurgical lagophthalmos, it is recommended that the eyelids be sutured together with split-thickness horizontal mattress sutures. Bandaging the eye will reduce postoperative swelling and protect the eyelid during anesthesia recovery. Postoperative drugs should include flunixin meglumine to control pain and inflammation and systemic antibacterials. The bandage can be removed in 2–3 days. If possible, avoid all forms of ophthalmic medications and advise the owner not to manipulate the eyelids. If a corneal injury exists that requires treatment, a nasolacrimal medication tube would be indicated. A warm compress may be beneficial if swelling persists. The horizontal mattress sutures can be removed as soon as the eyelids appear normal. The skin sutures in the wound should be left in a minimum of 2 weeks and longer if they do not cause any local reaction (Fig. 14).

B. Corneal Lacerations

Corneal lacerations may be superficial, deep, or perforating (Fig. 15), with the method of management dependent on the severity of the injury, the lapsed time since the injury occurred, the amount of corneal edema, and the presence or absence of infection. A superficial injury will usually heal in 24–48 h without treatment unless the source of the laceration inoculates pathogenic organisms in the stroma. Because of this danger, a topical antibacterial ophthalmic preparation should be applied until the cornea is fluorescein negative. A deep laceration with minimal corneal damage will often heal with conservative treatment consisting of topical medications recommended for ulcer therapy.

If treated conservatively, this patient should be examined daily. If healing does not proceed as anticipated, the laceration should then be treated in the same fashion as a wound extending to Descemet's membrane.

Lacerations extending to Descemet's membrane require surgical attention. Corneal suturing or an adhesive is the treatment of choice. The next best choice would be a conjunctival flap. If these are not available, indirect corneal support with a third eyelid flap or a tarsorrhaphy with or without a soft contact is indicated. Indirect corneal support has a high risk of perforation and a guarded prognosis. A laceration with severe corneal damage should be treated medically like an ulcer and the cornea should be supported or protected. Lacerations that produce corneal flaps should be sutured if presented before corneal edema develops. After corneal edema is present, the best procedure is to debride the flap and treat the corneal lesion as a deep ulcer.

Small puncture wounds without iris incarcerated in the wound will have local corneal edema, a pinpoint pupil, plasmoid aqueous that seals the wound, hypotony, and severe pain. Fluorescein staining may be needed to demonstrate the wound. These wounds will not require surgical treatment but the potential of intraocular infection will necessitate systemic treatment for infectious anterior uveitis. The eye should be checked every few days. If the wound starts to ulcerate or develop a corneal abscess or stromal necrosis, appropriate treatment should be started.

Perforating lacerations with iris prolapse require surgery for best results. The examination should be stopped immediately and the patient should be prepared for surgery. Further manipulation at this time may cause additional damage to the eye. The patient should be premedicated with systemic antibacterials and flunixin meglumine. The blood–aqueous barrier has been altered, and any antibacterial drug capable of maintaining a therapeutic blood level will also be present in the aqueous in therapeutic levels until the normal blood–aqueous barrier returns. Do not put topical ointments on an eye with a perforating injury, because of the danger that the petroleum base will cause uveitis. The
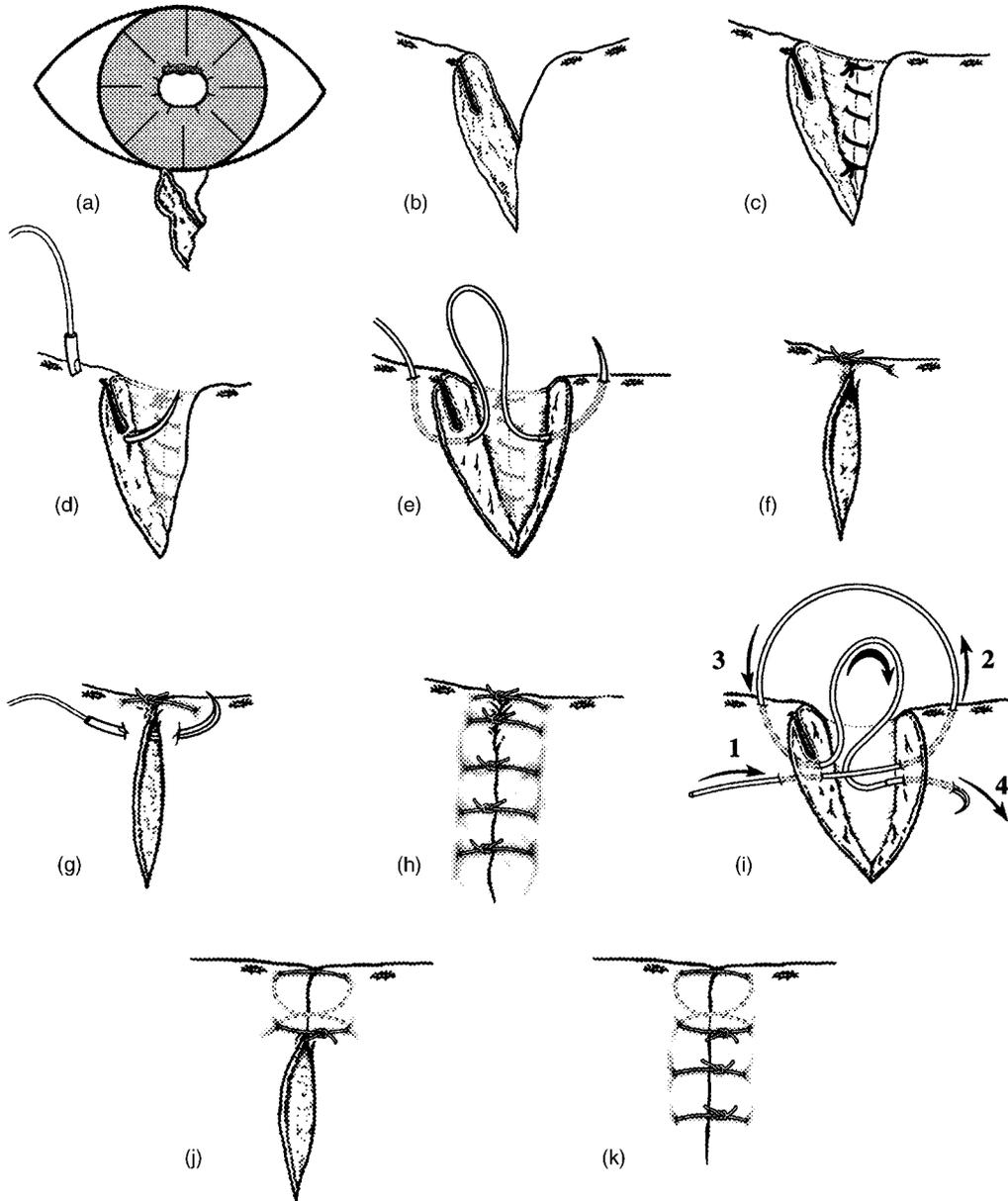
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Fig. 13. Eyelid wound with little tissue loss: (a) wound is shown before debridement; (b) wound is cleaned and debrided; (c) conjunctiva is sutured with continuous absorbable suture; (d) appositional suture is started along the edge of the lid; (e) opposite side of appositional suture is placed; (f) suture is tied and knotted on the edge of the eyelid; (g) tension suture is placed; (h) final appearance of wound closure is shown. (i) Figure 8 suturing pattern can be used as an alternate to the appositional and tension sutures. Final step of suture placement is shown. (j) final appearance of figure 8 suture and (k) final appearance of the wound closure.

Fig. 14. Eyelid laceration after 2 days, showing the appearance of the laceration (a) at entry, (b) the next day after an overnight Furacin bandage, (c) after repair, and (d) 2 weeks later after suture removal.
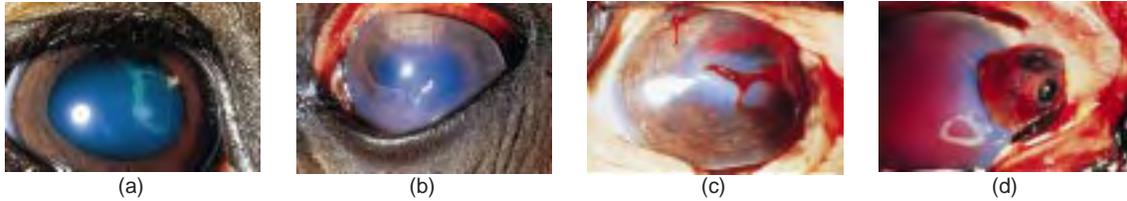
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Fig. 15. Corneal lacerations: (a) Superficial, stained with fluorescein; (b) deep; (c) perforating, with iris prolapse; (d) perforating limbal, with prolapsed anterior uvea, caused by blunt trauma.

ophthalmic instruments and sutures recommended include an ophthalmic needle holder, corneal suturing forceps, Arruga capsule forceps, corneal scissors, cyclodialysis, irrigation needle with a side port, 6-0 or 7-0 absorbable ophthalmic suture, and 6-0 silk with corneal needles.

Inhalation anesthesia is preferred. The same surgical preparation described for eyelid lacerations is used. A lateral canthotomy may be necessary to improve the exposure of the laceration. The eyelids can be separated with a speculum or sutured open. Silk stabilization sutures (6-0) are placed in the sclera near the limbus to assist in positioning of the eye. If the eye has nystagmus or has rotated downward, interfering with positioning of the eye, a four-point infiltration of 1.5 ml of lidocaine 2% into the rectus muscles will be helpful.

Perforating corneal wounds can be treated by corneal suturing, adhesives, conjunctival flaps, and corneal transplants, depending on the characteristics of the injury and the skills of the surgeon.

Regardless of the method of laceration closure, if the wound is less than 1 h in duration and the iris is not severely prolapsed or damaged, the iris can be replaced into the anterior chamber. Otherwise the iris is amputated so that the corneal edges can be repositioned without entrapping iris. In either case the iris is separated from the cornea with an iris spatula or cyclodialysis needle by rotating it 360° around the wound. Iris amputation will result in a larger postsurgical pupil. The hemorrhage following iris amputation can be controlled with a 1:10,000 epinephrine solution. This will also help maintain mydriasis during surgery.

Corneal suturing is the most accepted method of repair (Fig. 16). If the wound has straight edges and is not edematous, a simple interrupted or continuous suture pattern with 7-0 absorbable suture is recommended. Unfortunately the equine cornea rapidly develops edema, which will make it necessary to combine horizontal mattress sutures to appose the corneal edges properly. An air bubble placed in the anterior chamber makes suturing easier and reduces postsurgical synechia. Before the final sutures are placed, an attempt should be made to remove as much fibrin and clotted blood as possible. If hemorrhage occurs when the iris is manipulated or the anterior chamber is cleared, stop further manipulations. Blood will eventually absorb from the anterior chamber, but this can result in additional synechia formation.

Adhesives are indicated in small lacerations with smooth wound edges and minimal iris prolapse. Conjunctival flaps and corneal transplants should be considered when the edges of the wound cannot be apposed because of the loss of corneal tissue. After the wound has been closed, the surgeon may choose a nictitating membrane flap or eyelid suturing to give additional support to the wound.

Postsurgical ocular medication may be applied directly to the eye or with a medication tube. Topical medications should include atropine to maintain mydriasis and cycloplegia, and antibiotics to control infection. Systemic antibacterials and flunixin should be given for 5 days followed by oral aspirin as long as deemed necessary. Eyelid sutures can be removed at 2 weeks. Topical medications are generally continued until the corneal sutures come out between 3 and 4 weeks.

The prognosis is dependent on the severity of the ocular damage at the time of injury. An uncomplicated laceration has a favorable prognosis. A guarded to unfavorable prognosis is made if the injury extends into the sclera, there is severe anterior uveal damage, lens rupture or laceration, vitreal hemorrhage, or retinal detachment. Severe anterior uvea damage will generally manifest as phthisis bulbi that develops 2–3 weeks after injury.

14. Management of Corneal Ulcers

The essential steps of management of corneal ulcers are to determine the cause, establish an appropriate treatment, and if needed, protect or support the cornea. The causes are mechanical, infectious, metabolic, and neurogenic. Mechanical causes include abrasions from ropes or whips, self-inflicted trauma from rubbing the eye or striking an object, trauma from fighting with other animals, and corneal or adnexal foreign bodies. In foals, examine for entropion and congenital abnormalities such as dermoids or eyelid agenesis. In adult horses, eyelid tumors and acquired eyelid deformities should be considered. Exposure keratitis leading to ulceration can occur with exophthalmos.

Infectious agents may be opportunistic, following injury, drug therapy, or primary corneal infections. Common bacterial isolates are *Pseudomonas* spp., *Streptococcus* spp., *Staphylococcus* spp., and *Bacillus.* Keratomycosis is frequently preceded by corneal injury from plant material or the treatment of corneal ulceration with a topical antibiotic–corticoste-

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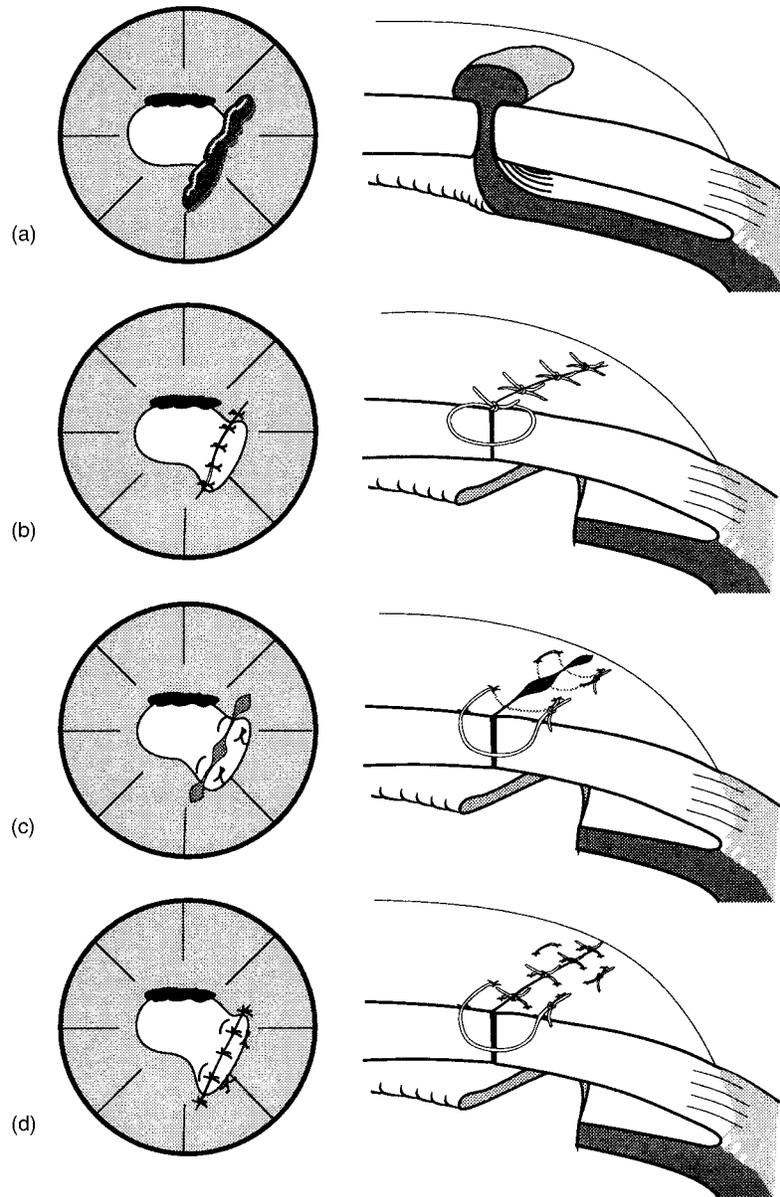

Fig. 16. Corneal laceration with prolapsed iris: (a) Frontal and cross-sectional views of perforating corneal laceration. (b) The prolapsed iris has been amputated, corneal iris adhesions reduced with an iris spatula, and the wound sutured with simple interrupted sutures. (c) Eyes with severe corneal edema should be sutured with horizontal sutures. This will seal the endothelial surface. (d) Simple interrupted sutures are placed between the horizontal mattress sutures to close the epithelial edge.

roid combination therapy. The mycotic agents frequently seen are *Aspergillus* spp., *Fusarium* spp., *Penicillium* spp., and the Phycomycetes group.

An altered corneal metabolism from inadequate or abnormal tear production will result in epithelial degeneration and ulceration. Corneal epithelial basement membrane disorders result in indolent corneal erosions. Corneal endothelial disease will lead to corneal edema predisposing to subepithelial bullae that can rupture, resulting in ulceration.

Neurogenic disorders causing corneal anesthesia, blepharoparesis, or absolute keratoconjunctivitis sicca will lead to corneal ulceration.

Corneal ulcers are characterized by pain, ocular discharge, loss of corneal transparency, and change in corneal contour (Fig. 17). The abundant nerve endings in the corneal epithelium result in severe pain when the epithelium is disrupted. This is manifested by blepharospasm, enophthalmos, photophobia, and lacrimation.

If only the epithelium is abraded, the outer glyco-protein layer of the stroma is not disrupted and therefore the cornea does not take on much edema and remains relatively clear. Superficial ulcers may go undiagnosed unless stained with fluorescein. If the outer stroma is disrupted, the cornea imbibes tears and becomes cloudy. Next, the cells in the stroma become pyknotic or swell and burst. In 24 h

C
O
L
O
R

Fig. 17. Variations of corneal ulcers: (a) indolent superficial; (b) acute melting, caused by *Pseudomonas* infection; (c) chronic mycotic, caused by *Aspergillus;* (d) perforating chronic, with iris prolapse.

polymorphonuclear cells invade the cornea, resulting in more opacity. In some infections this cell infiltration may result in a corneal abscess. If infectious organisms such as *Pseudomonas* spp. that are capable of producing collagenase or similar proteolytic enzymes are present, rapid destruction of the collagen framework of the cornea will result in the melting ulcer. When this occurs, progression of the ulcer to Descemet's membrane will result in a Descemetocele or perforation. Intrinsic collagenase also produces budding, endothelial cells, leukocytes, and regenerating epithelial cells. It is during this phase of collagenase activity that corticosteroids are contraindicated because they increase the activity of collagenase by 14 times.

Corneal ulceration with stromal involvement will result in a secondary anterior uveitis, plasmoid aqueous, and circumcorneal injection. Deep ulcers frequently stimulate a severe leukotactic response that results in hypopyon. If anterior chamber centesis is performed, this exudate will invariably be sterile.

As previously mentioned, the first step in successful ulcer management is to determine the cause. A thorough examination including culture with sensitivity testing and cytology may be necessary.

Severe ocular pain will necessitate chemical restraint and topical anesthesia before a complete examination can be performed. It is not unusual in horses with a *Pseudomonas* ulcer to have a sterile sample collected from the conjunctival sac when a pure culture of *Pseudomonas* is isolated from the ulcer. Cytology may be collected with a swab or spatula or as an impression smear.

After the cause has been identified, an appropriate treatment may include systemic and topical medications. Systemic flunixin meglumine 1 mg/kg q 24 h will control pain and reduce secondary anterior uveitis. After 5–7 days, aspirin 30 mg/kg q 24 h is usually adequate for maintenance until topical medication is discontinued.

An appropriate topical treatment may require drugs that are antimicrobial, anticollagenase, pain relieving, and mydriatic. If bacterial infection is suspected, broad-spectrum antibiotics, such as tobramycin, gentamicin, or chloramphenicol, or triple antibiotic mixtures can be used until sensitivity results are available. Tobramycin is the best if *Pseudomonas* spp. is suspected. Miconazole 1% parenteral solution is an excellent presumptive choice for mycotic ulcers. If not available, Clotrimazole 1% dermatological solution or Thiabendazole 14%
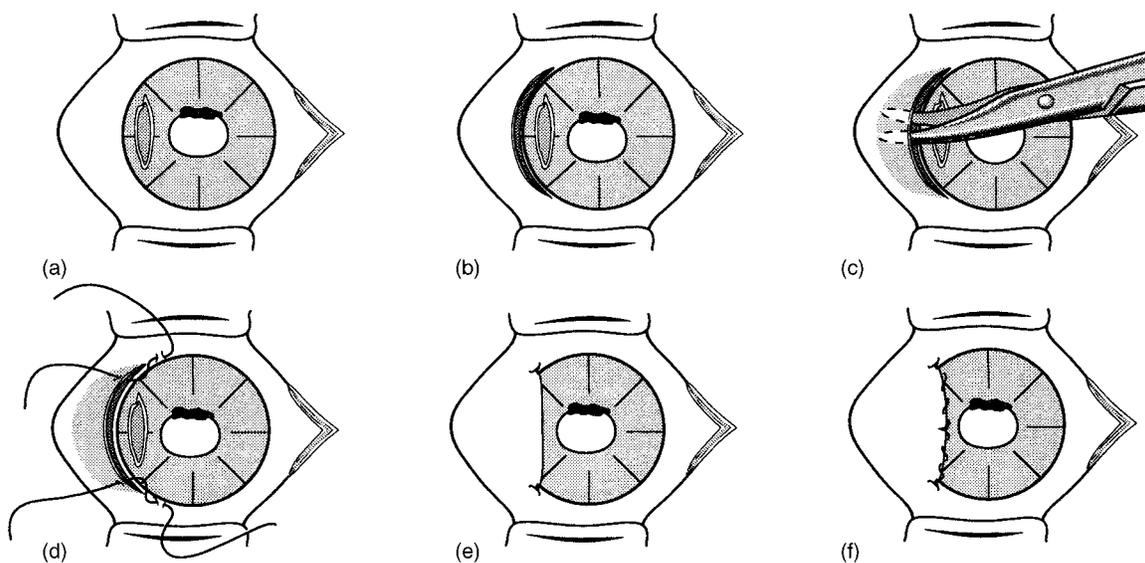

Fig. 18. Peripheral conjunctival flap for a corneal lesion near the limbus: (a) Appearance of a peripheral ulcer is shown. (b) Perilimbal conjunctival incision is made adjacent to the wound. (c) Conjunctiva is undermined with blunt scissors. (d) Sutures are placed in the sclera at the limbus and along the edge of the undermined conjunctiva. (e) Sutures are tied at the limbus. (f) Flap is shown with margins sutured to the cornea.

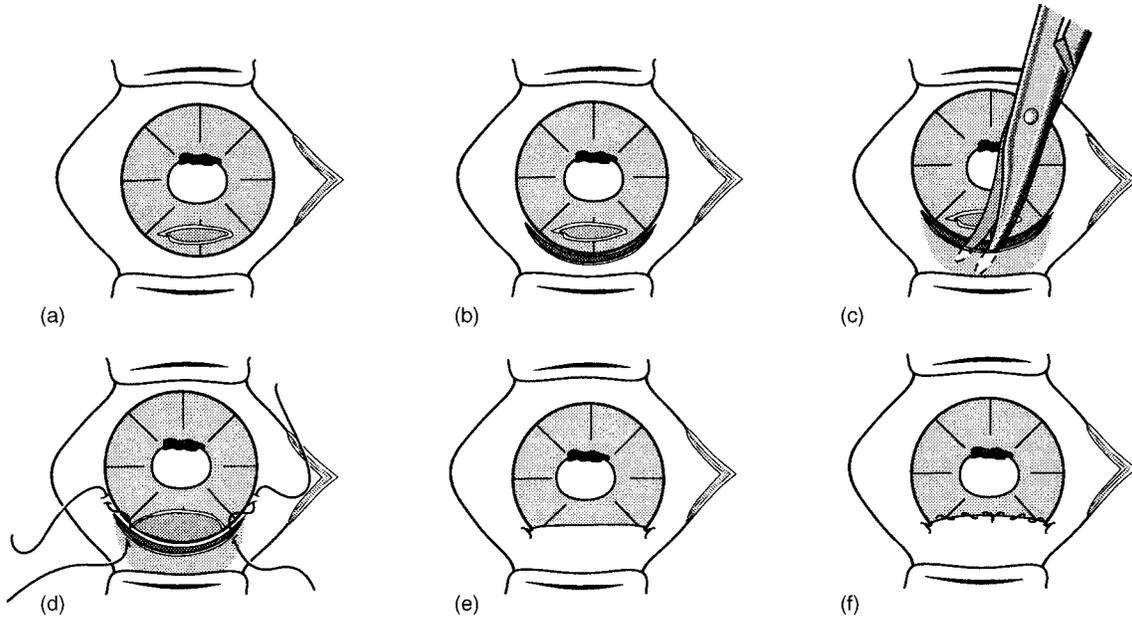Proceedings of the Annual Convention of the AAEP 1998

Fig. 19.   Peripheral conjunctival flap for a corneal lesion due to facial paralysis: (a) Appearance of a peripheral ulcer is shown.
(b) Perilimbal conjunctival incision is made adjacent to the wound.   (c) Conjunctiva is undermined with blunt scissors.   (d) Sutures
are placed in the sclera at the limbus and along the edge of the undermined conjunctiva.   (e) Sutures are tied at the limbus.   (f) Flap is
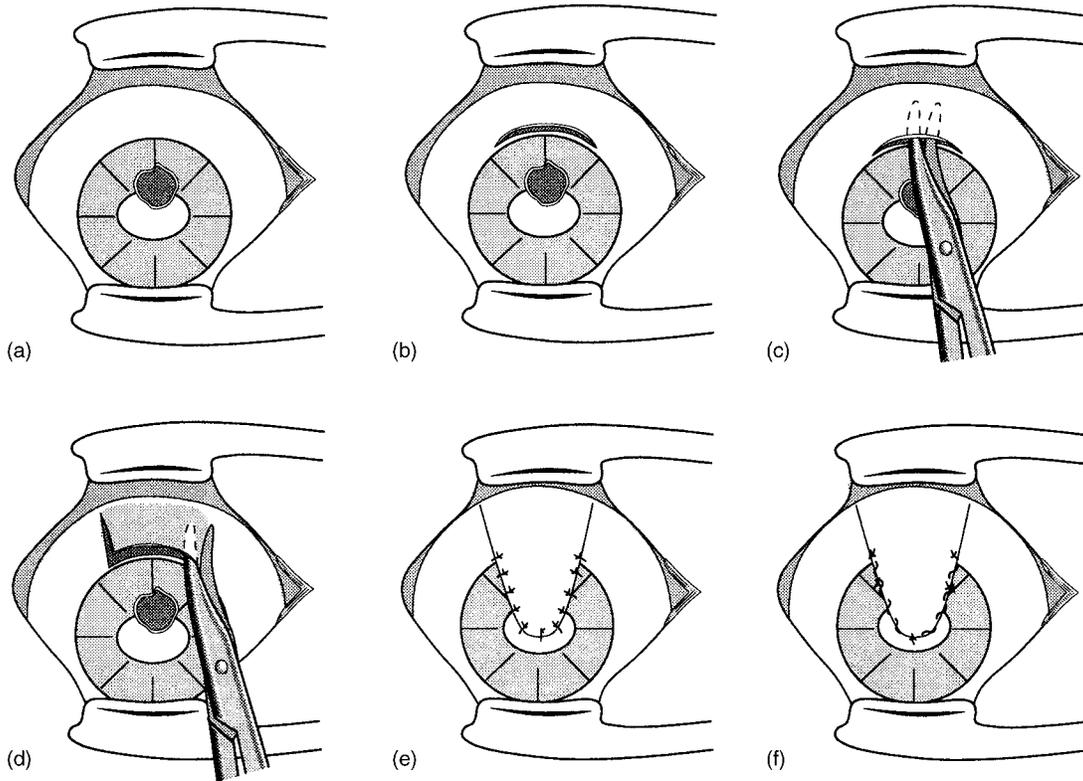shown with margins sutured to the cornea.

Fig. 20.   Advancement (pedicle) conjunctival flap: (a) Appearance of a corneal ulcer is shown.   (b) Conjunctival incision near the
limbus has been extended to anticipated width for the flap.   (c) Subconjunctival pocket is made with blunt scissors (corneal,
strabismus).   Care should be taken to stay above Tenon's capsule.   (d) Incisions are made in the conjunctiva perpendicular to the
limbus.   (e) Conjunctival flap is extended over the ulcer and sutured with absorbable suture, using an interrupted suture pattern.
Suture size depends on species and degree of corneal edema: small animals, 8-0, healthy cornea, 7-0, marked edema; large animals,
7-0, healthy cornea, 6-0, marked corneal edema.   (f) Conjunctival flap sutured with continuous pattern is faster but presents a greater
risk of flap dehiscence.   Several anchor sutures are recommended.

paste could be temporarily used. Mycotic keratitis should be concurrently treated with an antibiotic until the cornea is negative to fluorescein. Acetyl cysteine 5% is an effective anticollagenase agent in ulcers with severe collagenase breakdown. Atropine 1% is indicated for mydriasis and cycloplegia. In severe ulcers these medications should be given every few hours. This is virtually impossible to do unless they are compounded into a single solution. For a simplified administration, a compounded mixture referred to as an ulcer solution is sometimes beneficial. An ulcer solution can be prepared with fresh serum as a diluent for an antibiotic and, if indicated, atropine. Serum has anticollagenase activity from α_2-macroglobulins, as well as healing

stimulation from endogenous epithelial growth factors. Medication delivery systems are indicated if the patient is difficult to treat or if a melting cornea requiring frequent medication is presented.

All patients with severe ulcers are candidates for subconjunctival injection with antibiotics, mydriatic–cycloplegic agents, and anticollagenase preparations as supplements to topical medications. The antibiotics used frequently are chloramphenicol or gentamicin. Tetanus antitoxin is the preferred anticollagenase source. The antibiotic can be combined with an equal amount of tetanus antitoxin, and then 0.75–1.5 ml of this mixture can be injected. A subconjunctival injection combination of atropine and phenylephrine (Table 1) is a potent mydriatic–
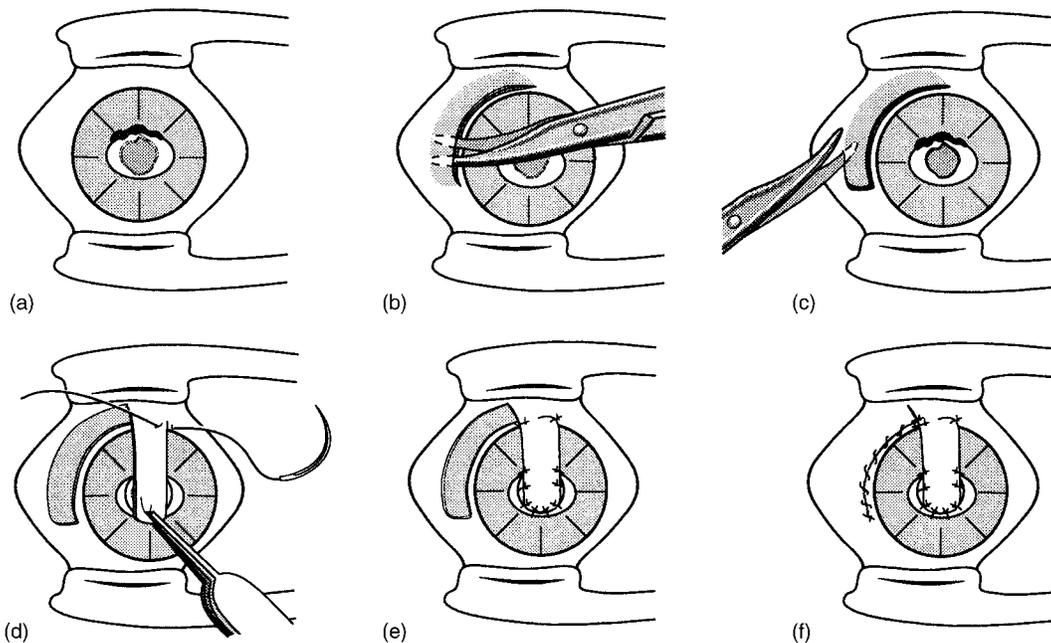

Fig. 21.   Conjunctival rotation flap: (a) Central corneal ulcer is shown.   (b) Small conjunctival incision is made near the limbus and extended as the conjunctiva is undermined with blunt scissors.   The length of the limbal incision determines the length of the flap. If possible, the incisions should be made so that when the flap is rotated it will be perpendicular to eyelid motion.   (c) Undermined conjunctiva is cut to form the rotation flap.   (d) Conjunctival flap is rotated over the ulcer and a fixation suture is placed before the edge is trimmed to fit the ulcer.   (e) After trimming, the flap is sutured to the ulcer edge and anchor sutures are recommended where the flap crosses the limbus.   (f) Suturing the conjunctival donor site is optional.
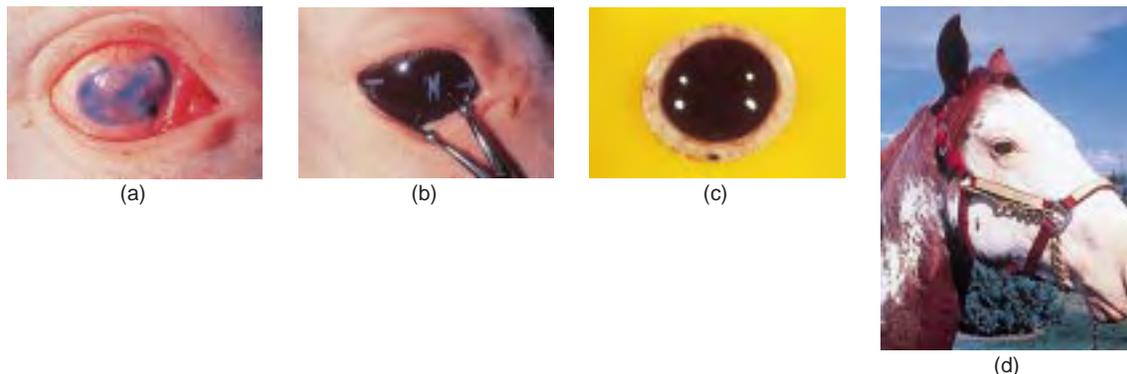

Fig. 22.   Phthisis bulbi with severe corneal changes: (a) phthisis eye 3 months after injury; (b) a cast of the eye is made by using a template filled with alginate; (c) the completed prosthesis is ready for fitting; (d) the prosthesis is positioned over the phthisical eye.

cycloplegic combination. Mydriasis will begin in 5–10 min and be complete in 1 h. Subconjunctival injections can be repeated as needed.

The last phase of ulcer management, if indicated, is corneal support and protection. The cornea can be protected and supported with eyelid suturing, a nictitating membrane flap, a conjunctival flap (Figs. 18–21), an extended wear contact lens, corneal adhesives, corneal suturing, corneal transplant, and corneoscleral transposition. Treatment should be continued until the ulcer is epithelialized and infection has been controlled. Hypertonic sodium chloride 5% ointment or a 2–5% solution two or three times daily promotes epithelial reattachment and should be used 3–4 weeks after epithelialization.

15. Phthisis Bulbi

Phthisis bulbi is a blind shrunken eye that is due to irreversible ciliary body damage. It is usually not painful but results in an unsightly eye and, if severe, ocular discharge. Treatment is not needed unless secondary problems exist. If there are problems, several treatment options are available. The best treatment from the patient's standpoint would be the removal of the blind eye and positioning of an orbital implant in the orbit before the wound is closed.

If cosmetic appearance is a factor, the owner may choose a corneoscleral prosthesis. This prosthesis is placed over the phthisical eye, thereby filling the anterior orbit and providing support to the eyelids (Fig. 22). The prosthesis is removed periodically for cleaning and then replaced immediately.

Severe phthisis bulbi requires a large prosthesis, which because of its size would be difficult to keep in the orbit. An alternative procedure would be to enucleate the eye, position a subconjunctival implant, and later fit the eye with a corneoscleral prosthesis. This procedure is also indicated for animals with intraocular tumors.

References and Footnotes

1. Ball M, Rebhun W, Gaarder J, et al. Evaluation of itraconazole-dimethyl sulfoxide ointment in the treatment of keratomycosis in nine horses. *J Am Vet Med Assoc* 1997; 211:193–203.

2. Figure 8 is modfied from Slatter D. *Fundamentals of veterinary opthalmology,* 2nd ed. Philadelphia: Saunders, 1990.

[a]Mila International, 510 W. 6th St., Covington, KY 41018.
[b]Jorgensen Laboratories, 1450 N. Van Buren St., Loveland, CO 80538.
[c]Pilopine HS Gel, Alcon Laboratories, Fort Worth, TX 76161.
[d]Equron, Solvay Animal Health, Inc., Mendota Heights, MN 55120-1139.
[e]Hyalovet, Ft. Dodge Laboratories, Ft. Dodge, IA 50501.
[f]Synacid, Schering, Kenworth, NJ 07033.
[g]Silicone Implant, Jordan Plastics Research Corp., 17100 W. 12 Mile Rd., Southfield, MI 48076; 313-424-8560.
[h]Maxitrol, Alcon Laboratories, Fort Worth, TX 76161.
[i]Betadine, Purdue Frederick Co., Norwalk, CT 06850-3590.