

Ocular Emergencies:

Palpebral lacerations

Overview: The eyelids and nictitans membrane have an excellent blood supply and therefore will tolerate a greater level of tissue damage and narrow tissue flaps than other areas of the body.

Goals:

1. Anatomical alignment of the lid margin - most critical
2. Maintenance of lid margin
3. Prevention of secondary entropion, ectropion, or trichiasis
4. Control bacterial contamination
5. Pain control

Therapy:

1. Minimal debridement. Even with a bucket handle or thin based flap, give the tissue the benefit of the doubt
2. Figure 8 suture at lid margin - start with opposing the lid margin
3. Accurate dermis apposition to prevent hair follicles being trapped growing the wrong direction and to minimize contractor of the lid results in in entropion or ectropion
4. Deep (tarsconjunctival) sutures? Debatable if actually needed. beware to avoid stiff mono-filament sutures such as PDS as they can cause corneal irritation through the conjunctiva.
5. Topical and systemic antibiotics and NSAIDS and Narcotic pain meds as needed. Avoid systemic or topical corticosteroids as they decrease wound healing and can prevent complete elimination of bacteria from deeper tissues.

Nictitans lacerations

Overview: Most lacerations of the nictitans are “bucket handle” or curvilinear tears. There is not an absolute need for maintaining the leading edge of the nictitans and tears can often be either trimmed or smaller tears can be left to heal as a notch. Surgical repair is needed when the gland is involved or for improved cosmetic appearance.

Goals:

1. Maintain gland
2. Cosmetic appearance
4. Control bacterial contamination
5. Pain control

Therapy:

1. If suturing required, 7-0 or 8-0 vicryl. Make sure knots are on palpebral side and suture does not penetrate bulbar aspect of nictitans. A few horizontal mattress sutures work well.
2. Topical antibiotic for 7 days TID. Systemic Antibiotic if gland was penetrated.
3. Minimal pain - 48 hours of systemic NSAID if needed.

Corneal lacerations

Overview: The main goal with a corneal laceration is to seal the globe and prevent continued fluid leakage. Many lacerations will actually seal on their own within a few hours as fibrin and blood fill the anterior chamber and the cornea becomes edematous and swells at the edge of the laceration. This is particularly true for cat scratch lacerations as they are usually beveled.

Goals:

1. Seal the anterior chamber. Often a temporary loose seal will occur within a few hours if the patient is kept quiet. Corneal sutures are required for long term seal for most patients but this can often wait 24 to 48 hours.
2. Prevent bacterial infection of the cornea and inside the globe
3. PREVENT SECONDARY PHACOCLASTIC UVEITIS AND LOSS OF THE EYE DUE TO GLAUCOMA. If the anterior lens capsule is lacerated during the initial trauma, and lens proteins begin to spill into the anterior chamber. This will cause a severe immune reaction beginning in 7 to 10 days that results in glaucoma and loss of the eye. If the lens has a significant laceration, removal of the lens is required usually within the first week post trauma.
4. Pain Management
5. Minimize corneal scarring and maximize clarity of vision.

Therapy

1. Sedate/Injectable pain meds. Both for pain control but to keep the patient quiet and allow the laceration to seal. Administer topical proparacaine to help improve comfort and make it easier to examine the patient
2. Start topical and systemic antibiotics. Use solution topically not ointment as ointment can prevent sealing of cornea.
3. Atropine? One time dose only. Narcotics and systemic NAIDS provide much more pain relief than Atropine. However, you want to open the pupil during the next 24 hours to allow better evaluation of the lens which is often not visible due to the miotic pupil when the patient presents. BUT do not need to send home with atropine as the secondary

dry eye and long term dilation of the pupil will cause more discomfort. In addition there is a slight risk of glaucoma in some breeds. A better solution would be TID tropicamide, which is short acting and will help dilate the pupil with less risk of glaucoma and less risk of persistent mydriasis.

4. Suture cornea. Small (< 4 mm) beveled lacerations near the limbus often do not require suturing, especially in younger patients. If suturing is required, we often wait 24 to 48 hours to better evaluate the lens (the pupil is usually dilated by this time) provided the anterior chamber is formed. Slow aqueous leakage is tolerated well for a few days, but if the chamber is collapsed and does not reform in 3 to 5 hours, then suturing is best.

IF THERE IS IRIS PROLAPSE: iris prolapse helps seals an acute corneal laceration and often results in reformation of the anterior chamber and improved patient comfort. But, the corneal can not heal with an iris prolapse present. Therefore, lacerations with iris prolapse (above the corneal surface) will require surgical repair. However, they too can often wait 24 to 48 hours provided anterior chamber is sealed.

The key to suturing a cornea is the correct NEEDLE. A spatula needle is required. 9-0 vicryl works well, but 8-0 or 7-0 vicryl is acceptable. Remember to NOT penetrate into the anterior chamber or epithelial down growth into the eye can occur.

5. Once the laceration is bridged with fibroblasts (and usually blood vessels) then topical corticosteroid therapy to minimize scarring (Dex drops BID for 2 weeks) is beneficial. The incision is often bridged in 10 to 12 days

Deep or perforated corneal ulcers

Overview: Unlike corneal lacerations, deep or perforated corneal ulcers are typically round to oval and by definition contaminated with bacteria. They can develop slowly over 10 to 14 days or rapidly over 24 hours depending on type of bacteria.

Goals

1. Eliminate the bacterial infection. :Immediately stopping continued corneal damage from bacterial enzymes is the key to preventing loss of the eye or vision. Even in cats with viral keratitis, the loss of corneal tissue is mediated by secondary bacterial infection.
2. Prevent continued loss of corneal collagen and rupture of the globe (or sealing of an existing rupture). bacterial infection of the cornea and inside the globe
3. Pain Management. Some deep non-perforated ulcers will not be especially painful as most of the nerve ending in the cornea are in the superficial stroma. Ruptured ulcers that are sealed with iris are also much more comfortable than persistently leaking ulcers.

4. Minimize corneal scarring and maximize clarity of vision. This can be greatly influenced by the type of surgical repair required or used, and if surgery is even needed. Healing without surgery, surgical repair using fresh corneal tissue, and surgical repair with conjunctiva and/or xenogenic collagen grafts provide the best to worse vision, respectively.
5. Classify the ulcer:
 - a. Superficial to less than 50% depth and MINIMAL corneal malacia (cloudy, gelatinous cornea around ulcer). These ulcers are less threatening and will likely not require surgical repair.
 - b. Superficial to less than 50% depth and MARKED corneal malacia (cloudy, gelatinous cornea around ulcer) or melting OR marked hypopyon in anterior chamber. These are heavily infected, potentially rapidly progressing ulcers that will require surgical repair if the bacterial infection and corneal damage is not immediately controlled.
 - c. Deep but not perforated ulcer and MINIMAL corneal malacia (cloudy, gelatinous cornea around ulcer). In a young dog, especially if there are vessels at the edge of the ulcer or the ulcer is near the limbus, these may heal with out surgery. In middle aged to older dogs, these will usually require surgical repair. These ulcers are less threatening and will likely not require surgical repair.
 - d. Deep but not perforated ulcer and MARKED corneal malacia (cloudy, gelatinous cornea around ulcer) or melting OR hypopyon in the anterior chamber. These ulcers are highly threatening to the globe and almost always require surgical repair.
 - e. Perforated ulcers. These almost always, except in young animals with small perforations near the limbus, will require surgical repair.

Therapy - Based on Ulcer type

1. Administer topical proparacaine to help improve comfort and make it easier to examine the patient. If quite painful, injectable narcotics.
2. Superficial to less than 50% depth and MINIMAL corneal malacia:
 - a. Moderately aggressive antibiotics (QID for 2 days, TID for 4 days, BID for 7 days). TAB, Tobra and erthromycin, topical Ciprofloxacin all good choices.
 - b. Systemic Antibiotics: Likely no need but can provide some help.
 - c. Anti-collagenase: not needed
 - d. Pain management: Systemic NSAIDs, 24 hours of Narcotic. NO TOPICAL NSAID. One time dose of atropine IF pupil is miotic. If not miotic, skip atropine.

3. Superficial to less than 50% depth and MARKED corneal malacia and/or melting and/or marked corneal edema or hypopyon:

a. Aggressive Topical antibiotics. Q1 hour rotating tobramycin and ofloxacin for 12 hours, then 6 times a day for 48 hours, then 4 times a day for 5 days, then 3 times a day for 5 days. Need to sterilize cornea as quickly as possible.

b. systemic Antibiotics. YES - compliment topicals. Clavamox or cephalexin or even clindamycin good choices.

c. Anti-collagenase: DEFINITELY. Rotate with topical ABs, so AB1 at 9am, AB 2 at 10am, anti-collagenase at 11am, AB 1 12pm, AB 2 1pm, anti-collagenase at 3pm, etc. Use for the first 3 to 4 days. These agents inhibit collagenase released by bacteria and to some degree neutrophils.

serum - any patient's serum, Na EDTA, Bacitracin, Acetyl Cysteine

REMEMBER: collagenase activity will continue for a few days past clearance of the bacteria as the dying bacteria still release enzymes.

d. Pain management: Systemic NSAIDs, 48 to 72 hours of Narcotic. NO TOPICAL NSAID. One time dose of atropine IF pupil is miotic. If not miotic, skip atropine.

e. Surgical repair? Maybe. The ulcer is not deep YET and if we can stop continued loss of collagen before the strength of the globe is compromised, then no repair required. SURGICAL REPAIR IS ALWAYS MORE SUCCESSFUL IF WE CAN FIRST CONTROL THE BACTERIAL INFECTION.

4. Deep but not perforated ulcer and MINIMAL corneal malacia or melting and minimal peri-lesional edema and cells.

a. Moderately aggressive antibiotics (QID for 2 days, TID for 4 days, BID for 7 days). TAB, Tobra and erythromycin, topical Ciprofloxacin all good choices.

b. Systemic Antibiotics: Likely no need but can provide some help.

c. Anti-collagenase: not needed

d. Pain management: Systemic NSAIDs, 24 hours of Narcotic. NO TOPICAL NSAID. One time dose of atropine IF pupil is miotic. If not miotic, skip atropine. These agents inhibit collagenase released by bacteria and to some degree neutrophils.

e. Surgical repair? Maybe. Younger patients (less than 2 years) and lesions near the limbus with good vascular ingrowth may not need surgery. THIS TYPE OF ULCER IS NOT RAPIDLY PROGRESSING, BUT MAY NEED OF SURGICAL REPAIR DUE TO ITS ALREADY SIGNIFICANT DEPTH.

5. Deep but not perforated ulcer and MARKED corneal malacia or melting with peri-lesional cells.

- a. Aggressive Topical antibiotics. Q1 hour rotating tobramycin and ofloxacin for 12 hours, then 6 times a day for 48 hours, then 4 times a day for 5 days, then 3 times a day for 5 days. Need to sterilize cornea as quickly as possible.
- b. systemic Antibiotics. YES - compliment topicals. Clavamox or cephalexin or even clindamycin good choices.
- c. Anti-collagenase: DEFINITELY. Rotate with topical ABs, so AB1 at 9am, AB 2 at 10am, anti-collagenase at 11am, AB 1 12pm, AB 2 1pm, anti-collagenase at 3pm, etc. Use for the first 3 to 4 days. These agents inhibit collagenase released by bacteria and to some degree neutrophils.
serum - any patient's serum, Na EDTA, Bacitracin, Acetyl Cysteine
REMEMBER: collagenase activity will continue for a few days past clearance of the bacteria as the dying bacteria still release enzymes.
- d. Pain management: Systemic NSAIDs, 48 to 72 hours of Narcotic. NO TOPICAL NSAID. One time dose of atropine IF pupil is miotic. If not miotic, skip atropine.
- e. Surgical repair? Almost always. The ulcer is already deep and despite therapy some tissue will be lost during the next 48 hours. **SURGICAL REPAIR IS ALWAYS MORE SUCCESSFUL IF WE CAN FIRST CONTROL THE BACTERIAL INFECTION.**

6. Perforated ulcer.

- a. Topical antibiotics. If marked corneal edema, cells or malacia adjacent to ulcer then treat as in number 5 above. if a quite eye with minimal edema around a firm looking hole, treat as in number 4.
- b. systemic Antibiotics. Same criteria as above
- c. Anti-collagenase: Same criteria as above
- d. Pain management: Same criteria as above. **AVOID** atropine if perforation sealed by iris. **WE WANT** the seal to remain until surgical repair. Do not pull the nail form a tire until you are ready to repair the rubber.
- e. Surgical repair? Almost always. The ulcer is already perforated and despite therapy some tissue will be lost during the next 48 hours. **SURGICAL REPAIR IS ALWAYS MORE SUCCESSFUL IF WE CAN FIRST CONTROL THE BACTERIAL INFECTION.**

Globe Proptosis

Overview: Globe proptosis indicates that the equator of the globe has been pushed out beyond the orbital ligament. The orbital ligament is like a rubber band around the globe that attaches to the bony margin of the orbit. Once the globe is pushed out, the ligament unfortunately hinders the globe returning to a normal position. In addition, marked tissue hemorrhage and occasionally free hemorrhage occurs behind the globe during the first few hours further pushing out the globe.

Trauma at the temporal orbital rim, just above and back from the lateral canthus is the most common cause of a proptosis.

Vision loss occurs from either immediate damage to the optic nerve (stretched and compromising blood flow or completely severed) or from corneal opacification over days to weeks from exposure.

Globe deviation is common due to rupture of medial and ventral rectus muscles making the globe turn out and up. This is often more noticeable after replacement of the globe and resolution of orbital swelling.

Corneal desensitization for 3 to 5 months is common due to ciliary nerve damage. This leads to reduce tear production, reduce blink frequency, and chronic central keratitis.

Goals

1. Assess globe damage and potential for site. This can affect therapy.
2. Protect surface of globe and reposition as best as is possible
3. Reduce peri-ocular swelling to facilitate globe returning to a normal position
4. Control secondary bacterial contamination (these are often bite wounds)
5. Pain management. Many brachycephalic dogs with proptosis will not exhibit significant pain due to rupture of ciliary nerves.

Therapy

1. Administer topical proparacaine, sedate the patient to assess damage to eye and facial bones (zygomatic fracture, mandibular or maxillary fractures, frontal bone fractures).
2. Evaluate globe. There are many recommendations based on small versus large pupil, but standing pupil size not particularly useful. Miotic is better prognosis than mydriatic. Detectable PLR, direct or indirect, or a dazzle reflex are great signs for preservation of vision. Blood in the anterior chamber, any displacement of the lens (globe rupture likely), marked globe deviation laterally, severe corneal ulcer or corneal drying (prolapsed for awhile) or a dark area in the fundus instead of a myelinated optic nerve head are poor prognostic signs.

3. Lubricate the surface of the eye every few hours until surgical repair is possible.
4. Surgically reposition globe into orbit as best as possible.
 - a. Under GA (avoid ketamine due to increased muscle tone and BP) and preferentially neuromuscular blockade.
 - b. Using 3 horizontal mattress sutures, slowly push the globe back into the orbit while pulling over the eyelids. Mild proptosis with minimal blood will simply pop back in place. But significant proptosis will not completely go back into orbit. LEAVE SMALL AREA AT MEDIAL CANTHUS OPEN TO ALLOW FOR DRAINAGE AND APPLICATIONS OF TOPICAL ANTIBIOTICS
 - c. A peritomy (incise conjunctiva and Tenon's capsule 360 degrees) can help replace the globe and occasionally allow the escape of free blood DO NOT SEVERE THE RECTUS MUSCLES.
 - d. Surgical repair of rectus muscles is also helpful.
 - e. If no vision and has several of the poor prognostic indicators, then enucleation may be best therapy. However, can always replace and if no vision recurs enucleate at a latter date.
5. Topical antibiotics. TAB drops, or tobramycin good choices. TID for 5 days then BID while sutures in place.
6. Systemic Antibiotics
7. Anti-inflammatory meds? Systemic NSAID an excellent choice. Corticosteroids work better but increase risk of corneal ulceration, fungal keratitis, or orbital abscess. But if no evidence of bite wound and cornea in good shape, could use corticosteroids systemically instead of NSAID.
8. Pain management, Narcotics for 48 to 72 hours.
9. Long term care. Leave the eyelids closed 3 WEEKS or more unless there is marked ocular discharge or evidence of persistent discomfort. The swelling and hemorrhage around the globe will require 6 to 8 weeks to completely resolve in severe cases. Watch for KCS and exposure keratitis. Many patients will require ocular lubrication for 4 to 6 months.

Acute Hyphema

Overview: There are many causes of acute bleeding in the eye. In order of most common occurrence they include: systemic hypertension (dogs and cats), trauma, vasculitis (neoplastic and infectious such as *E. canis*), bleeding disorders, namely thrombocytopenia (may be unilateral), intraocular tumors, and vitreal degeneration and spontaneous retinal detachments

Acute hyphema is not an emergency. However, it can cause glaucoma which is an emergency and can be a sign of a systemic such as thrombocytopenia which is an emergency.

Goals

1. Assess globe damage and intraocular pressure (IOP). Rule out penetrating injury, corneal laceration, and secondary glaucoma.
2. Treat any secondary ocular problems such as glaucoma
3. Search for underlying systemic cause!
4. Pain Management? Hyphema can cause photosensitivity but in itself is minimally painful.

Therapy

1. Assuming no direct trauma to the globe, management clearly depends in the underlying cause.
2. Control Glaucoma.
 - IOP < 25 mmHg - simply monitor IOP
 - IOP 25 to 35 mmHg - Start topical carbonic anhydrase inhibitors such as Azopt or Trusopt TID and recheck IOP in 24 hours
 - IOP > 35 mmHg. Use Azopt or Trusopt every 15 minutes for 8 treatments. If IOP < to 35 mmHg or less, TID and recheck in 24 hours. If still above 35 mmHg, try topical prostaglandin such as Xalatan or Travatan ONE TIME and recheck IOP in 2 hours. Also, sedate patient with narcotic or ACE as this will lower IOP associated with hyphema.
3. Topical Corticosteroids (pred acetate) if cornea normal to help minimize secondary inflammation in the eye. This also reduces photophobia.
4. NO ATROPINE. Even if IOP is LOW and you feel that there is marked uveitis, LEAVE THE BOTTLE IN THE DRAWER.

Acute Primary Glaucoma

Overview: The majority of cases of increased intraocular pressure will be due to primary glaucoma. Many breeds have inherited glaucoma but order of most common presentation to our clinics these include: American Cocker spaniel, Bassett Hounds, Artic Circle breeds, Labrador retrievers, Terriers (especially Jacks and Rats), Shar peis and Chow Chows, Dalmatians, and Shih Tzus.

GLAUCOMA IS A MEDICAL EMERGENCY BUT TYPICALLY A SURGICAL DISEASE. The emergency is to lower the pressure in the eye ASAP. An eye will tolerate slowly rising glaucoma of moderate pressure ($I < 40$ mmHg) for days and some times weeks or months. But a rapidly rising pressure over 40 mmHg can easily cause permanent loss of vision within days.

Goals

1. Immediately lower IOP and attempt to preserve vision
2. Resolve the severe pain associated with acute glaucoma
3. With chronic glaucoma (no vision, large globe, lens sublimation) the goal is simply pain management.

Therapy - Acute glaucoma

1. Pain management. Administer narcotics as this will decrease pain AND help lower IOP. NSAIDs help with pain BUT can increase IOP so avoid. Remember, lowering IOP provides the best pain control
2. Lower IOP FAST
 - a. Topical Prostaglandin (Xalatan or Travatan) ONE TIME
 - b. Topical Carbonic anhydrase inhibitor (Trusopt or Azopt) every 15 minutes for 8 treatments
 - c. Oral carbonic anhydrase inhibitor (methazolamide) 1 mg/lb (BID)
 - d. Check IOP in 2 hours. If still > 35 mmHg, consider aqueous paracentesis with 27ga to 30 Ga. needle. ONLY need 1 drop out of hub DO NOT ASPIRATE FLUID.
3. Keep IOP controlled with Travatan or Xalatan BID, Trusopt or Azopt (or could use co-opt) TID and methazolamide BID. May stay down 24 to 48 hours, may stay down for month. WILL NOT STAY CONTROLLED INDEFINITELY.
4. If there is vision then strongly recommend laser cycloablation and glaucoma filtering implant for most breeds.

5. NO ATROPINE. Some acute glaucoma patients will have a red steamy eye and look just like uveitis. They may even have a faint ulcer from rubbing at the eye. NEVER give atropine to a patient without knowing the IOP. It will blind a dog with glaucoma.

Therapy - Chronic glaucoma

1. Pain management. Administer narcotic such as tramadol. Can use an NSAID although it can raise IOP. Since no vision, the comfort from the NSAID is more important. Remember, lowering IOP provides the best comfort.
2. Lower IOP - No need to be as aggressive as with acute glaucoma.
 - a. Topical Prostaglandin (Xalatan or Travatan) BID
 - b. Topical Carbonic anhydrase inhibitor (Trusopt or Azopt or Cosopt) TID
 - c. Oral carbonic anhydrase inhibitor (methazolamide) 1 mg/lb (BID)
 - d. Check IOP in 24 hours.
3. Majority of patients will require surgical therapy (enucleation, intravitreal gentocin injection, or evisceration) to obtain long term control of glaucoma.

Acute Vision loss

Overview: There are many causes of acute vision loss. It is clinically helpful to breakdown acute vision loss into two groups:

Acute vision loss with Anatomically Normal eyes (or at least no reason for loss of vision).

1. Sudden acquired loss of vision. Middle aged dogs, any breed but Dachshunds, and Pomeranians over represented, MOST have a history of increased appetite and/or weight gain the months prior to vision loss, many present with Cushing's like signs. Eyes will look normal and usually still have sluggish PLRs.
2. Central vision loss. Any age or breed. Frontal lobe neoplasm, meningoencephalitis (GME, Rickettsial, Fungal, Toxoplasma), post seizure, Hepatic encephalopathy, vascular ischemia, head trauma, POST ANESTHESIA: Especially in cats post Telazol and less commonly ketamine induction even if intubated.
3. Syncope. Not really vision loss but may present as vision loss

Acute vision loss due associated with Anatomic ocular change

1. Retinal detachment. Systemic hypertension, Chorioretinitis from vasculitis (Ehrlichiosis, Babesiosis, Bartonella sp., bacteremia, systemic fungal diseases, Prototheca sp.), Immune mediated Chorioretinitis (Young Golden retrievers, GME in brachycephalic breeds, post vaccination), associated with inherited vitreal degeneration (Shih Tzus,

Lhasas, Italian Greyhounds, Dachshunds, COLLIES, Labradors with chondrodyplasia), Trauma, Bleeding disorder, and Intraocular tumors.

2. Acute onset cataracts - Diabetes mellitus and inherited cataracts in dogs less than 2 years of age.
3. Glaucoma. Usually second eye.
4. Anterior uveitis - Sever forms. Infectious vasculitis (Ehrlichiosis, Babesiosis, Bartonella sp., bacteremia, systemic fungal diseases, Prototheca sp.), neoplastic vasculitis (LSA), Immune mediated.
- 5, Acute Hyphema - see above for causes.
6. Lipiod aqueous. Schnauzers or diabetes mellitus patients.

Goals

1. Determine if any anatomical changes are present in both eyes to account for loss of vision. If you can see in, the patient should be able to see out. MEASURE IOP.
2. Look for other systemic changes (CBC, Serum biochem profile, evidence of recent seizure, evidence of hepatic shunt, neurologic deficits besides vision, cardiovascular disease).
3. Treat ophthalmic and non-Ophthalmic disease as needed

Therapy

1. If no ocular changes present to account for loss of vision and no other systemic changes, the next step is an electroretinogram to determine retinal function and rule out SARDs.
2. If electroretinogram reveals normal retinal function, then MRI and CSF tap are the next step to try and determine the cause of the central blindness.
3. Start therapy for any ocular changes detected
 - a. Retinal detachment. Treat underlying cause. No specific therapy for detachment. Diuretics WILL NOT help. If a primary problem (vitreal degeneration) surgical attachment is rewarding.
 - b. Acute cataracts - start topical pred acetate to help control the uveitis associated with acute cataracts.
 - c. Glaucoma - immediately lower IOP - see above.
 - d. Anterior uveitis - Start Topical pred acetate and oral doxycycline until etiology can be established or at least specific causes ruled out.

e. Acute hyphema - treat underlying cause. Control IOP, topical Pred acetate for mild inflammation associated with hyphema.

f. Lipoid aqueous. Low fat meals for 48 hours and most lipoid aqueous cases will resolve. Evaluate patients diet, thyroid status, and rule out diabetes mellitus.