These notes are a **'quick-support'** source of information for emergency eye cases seen by general practice veterinarians and emergency veterinarians alike. These notes are not meant to replace textbooks in veterinary ophthalmology. A variety of textbooks exist for use in general practice including the BSAVA Manual of Canine and Feline Ophthalmology (English), Ophthalmology for the Veterinary Practitioner (Dutch or English) and Slatter's Fundamentals of Veterinary Ophthalmology (English), to name a few. Every practice should have at least one such book for extended consultation.

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA

The purpose of these notes is to offer veterinarians in first-line practice general guidelines regarding some of the most important aspects of ocular emergencies.

No patient-specific advice is given here for obvious reasons. Veterinarians must decide what category / categories a patient falls into and what to do to help while a referral appointment to an ophthalmologist is made (i.e., if referral is necessary).

The goals of emergency care for ophthalmic cases include what these notes refer to as the '**SPP minimum'**: **Stabilize/Pain control /Protect-Prevent**, this is:

A. Stabilize the eye (and the patient (i.e., non-ophthalmic care, if needed)
B. Treat for ocular / periocular pain
C. Protect the eye while minimizing the possibility of self-harm and infection

Most practices should strive to have access to the following (logically, the degree of care will vary depending on the experience of the veterinarian):

- **Medication / treatment support:**
  - **Topical antibiotics** of a 'wide spectrum' (e.g., topical chloramphenicol and gentamicin, which may be combined)
  - **Ocular lubricant** for topical use that is non-medicated, and, if possible, preservative-free, and viscous (i.e., a variety of veterinary ocular lubricants exist)
  - **Oral antibiotics** (e.g., amoxicillin/clavulanic acid and clindamycin)
  - **Oral NSAIDs** (i.e., tablet and fluid preparations exist)
  - **Antiglaucoma drops** (see page 12 for more information)
  - **Atropine drops** (see page 4, section 1 for more information)
  - **Topical anesthetic drops** (e.g., proparacaine, others too)
  - **Sterile saline** that owners can take home (e.g., small plastic bottles)
  - **Elizabethan collars** of a variety of sizes for protection

- **Instrumentation (diagnostic aids):**
  - **A bright source of light** (i.e., ideally a direct, hand-held ophthalmoscope) that allows for examination of the anterior part of the eye and visualization of the fundus, though the bright light of an otoscope can also be very helpful for the former.
  - **Schirmer tear test–1 strips** (i.e., to measure tear production)
  - **Fluorescein** (i.e., strips that are moistened with sterile saline for use)
  - **A tonometer** (i.e., to confirm if the intraocular pressure is elevated at that moment). Tonometry training helps avoid falsely elevated pressures due to a 'handler effect'. A diagnosis of glaucoma and the creation of a treatment plan goes far beyond tonometry, requiring referral for gonioscopy, when possible.
  - **Systemic blood pressure measurement** capabilities

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA

General knowledge points regarding the **application / use of eye treatments**:

- One drop is all the ocular surface can maximally hold (i.e., even a single drop will have some waste; there is no need to apply more than 1 drop at a time).
- Wait 20 minutes between different medications (i.e., when more than one type of medication is used) to avoid a diluting effect between drops.
- Apply water-containing drops before applying ointment.
- Shake suspensions before use (i.e., medication settles in the bottom).
- Veterinarians must become familiar with appropriate dosing, onset of action and duration of action of each ophthalmic drug in their medicine cabinet.

Assessment of eye health includes **the following skills** for every veterinarian:

- **Menace response test** (i.e., this crudely determines if the eye can see).
- **Pupillary light reflex test** (PLR, direct and indirect (i.e., consensual))
    - **Direct PLR:** the eye assessed is the one in which the light is shone.
    - **Indirect PLR**: assesses the opposite eye to where the light is shone.
- (Schirmer tear test-1 for a tear reading is rarely vital in emergency cases and is best avoided with descemetocele at risk of rupture, or with an obviously wet eye. It is invalidated by the application of topical eye medication).
- **Fluorescein testing** to see if there is corneal damage. A drop of fluorescein is dropped onto the eye (i.e., by wetting dry strips with saline, if not already available in a pre-prepared form). The eye is then flushed with saline. The examiner then looks at the eye with a blue light that makes the dye fluoresce.
- **Additional skills:** tonometry, funduscopy, distant direct ophthalmoscopy.

List of ocular emergencies: this varies depending on the degree and type of damage sustained by the eye, the risk the problem poses for vision (or for recovery of vision if vision has been lost), and patient discomfort. There is a certain degree of subjectivity in every list of ocular emergencies, though most would probably include the following broad categories covered here:

1. Blunt trauma to the eye (excluding globe proptosis)
2. Globe proptosis
3. Retrobulbar abscess / cellulitis (including when caused by a foreign body)
4. Corneal damage
    a. Deep corneal damage
    b. Penetrating corneal damage
    c. ‘Extensive’ corneal damage (i.e., corneal melting, large bullae)
5. Glaucoma (i.e., primary and secondary forms, excluding lens luxation)
6. Lens luxation
7. Acute vision loss

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA

1. Blunt trauma to the eye

Ocular trauma may be direct (e.g., eyes hit by a ball) or occur in conjunction with head trauma (e.g., animals that were hit by car), while palpebral (i.e., eyelid) and conjunctival damage often accompany different types of blunt trauma to the eye. A report of eyes that underwent blunt trauma (Rampazzo *et al*, Vet. Ophth. 2006) described they often presented with hyphema, subconjunctival hemorrhage and conjunctival as well as eyelid swelling, while intraocular pressure of the affected eye was usually ½ of the pressure of the unaffected eye. Posterior scleral rupture was suspected in ocular ultrasound when eyes had poorly defined scleral margins and significant posterior segment changes (i.e., echodense structures suggestive of hemorrhage and retinal detachment). Small scleral ruptures heal on their own with time, but ocular damage is often so extensive and prognosis for recovery of vision is so poor that enucleation is often recommended soon after the trauma has occurred. The role of the emergency veterinarian is not to provide a prognosis for the long term, though the emergency veterinarian may wish to comment on this.

The main role of the emergency veterinarian is to offer the '**SPP minimum'**:

- **Stabilize**: is there ocular exposure? If so, lubricate
- **Pain control**: administer appropriate cover for the time being (i.e., an oral NSAID, and consider topical atropine for iris/ ciliary body muscle relaxation). Remember this is used ***'to effect'*** (i.e., 2x day for 2-5 days often suffices). It causes temporary ocular dryness. It tastes terrible, sometimes causing ptyalism.
- **Protect-Prevent**:
    - Is there corneal ulceration? If so, start prophylactic topical antibiosis.
    - Is there a suspicion of ocular rupture (anterior or posterior)? If so, consider starting with an oral antibiotic.
    - Is there a possibility of self-trauma (i.e., accidental and/or from rubbing)? If so, start using a collar.
    - Is there globe proptosis? If so, see the globe proptosis section.
    - Is there anterior uveal exposure? If so, see the penetrating corneal damage section.

Poor prognostic indicators for blunt ocular trauma:

- Eye exam with light:
    - Absence of a menace response in the affected eye.
    - Absence of an indirect pupillary light reflex (see 'PLR' in page 3).
- Intraocular pressure (IOP) reading (if possible): the IOP of the affected eye is at least half of that of the unaffected eye.
- US examination of the eye (if available / indicated): significant posterior segment changes on ultrasound examination of the eye

Further reading: Rampazzo A., Eule C., Speirs S., Grest P., Spiess S. Scleral rupture in dogs, cats and horses. *Veterinary Ophthalmology* 2006; 9(3): 149-156.

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA

2. Traumatic proptosis of the globe

This problem is relatively common in animals with shallow orbits and large palpebral apertures (e.g., Shih-Tzu, Pug, Pekingese, etc.) Proptosis develops when there is rapid swelling of the retrobulbar area (i.e., sudden bleed associated with blunt trauma), which quickly displaces the eye anteriorly. This is followed by eyelid spasm and conjunctival swelling, and a globe that is unable to return to its normal position.

The **potential damage** encountered is diverse:

- The acute stretch of the **optic nerve and extraocular muscles** may damage these structures. Extraocular muscle rupture at the tendinous insertion of the muscle to the sclera occurs commonly and it usually affects the shorter muscles, such as the medial rectus. Medial rectus rupture will lead to a downward and laterally deviated eye (i.e., the 'down and out eye'). Other extraocular muscles may also rupture. Extraocular muscle rupture is often the cause of the subconjunctival hemorrhages seen in these patients.

- The **pupil** may be miotic due to inflammation in the eye or it may be dilated and unresponsive due to optic nerve damage. The latter, in the author's experience, carries a poor prognosis for vision but a miotic pupil may still be present with significant optic nerve damage. Independent of the size of the pupil of the affected eye, a lack of indirect (i.e., consensual) pupillary light reflex (see page 3 for a definition) usually carries a poor long-term prognosis for vision of the affected eye.
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  The **cornea** may have been traumatized during the same initial event or may have occurred later due to exposure and dissecation of the ocular surface. Corneal ulcers are common in globe proptosis.

- The **intraocular structures** might also be affected during proptosis, and this could also negatively affect long term prognosis (i.e., vitreal hemorrhage, retinal detachment).

The main role of the emergency veterinarian is to offer the '**SPP minimum'**:

- **Stabilize**: is there ocular exposure? If so, lubricate with a preservative-free viscous gel and/or consider applying a 'lateral and a central temporary tarsorrhaphy', which will require general anesthesia. A tarsorrhaphy is a horizontal mattress suture that will keep the eyelids closed. Eyes with proptosis may be difficult to reposition and a lateral incision (i.e., a lateral

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA

canthotomy) may be necessary in some cases. This is not a surgery text. Please, refer to surgery and/or ophthalmology textbooks for this.

- **Pain control**: administer appropriate cover for the time being (e.g., oral NSAID, topical atropine - see comments on page 4, section 1).
- **Protect-Prevent**:
    - It is always recommended to start using a protective E-collar to avoid self-trauma (i.e., accidental and/or from rubbing).
    - Is there corneal ulceration? If so, start prophylactic topical antibiotics
    - Is there a suspicion of ocular rupture (anterior or posterior)? If so, consider starting with oral antibiotics. Otherwise, they are unnecessary. If unsure about posterior rupture, do not start them yet.
    - Is there anterior uveal exposure? If so, see the penetrating corneal damage section.

Brief comments on the lateral tarsorrhaphy procedure:

Eyes can be reassessed at a later date to see if further treatment or enucleation is warranted. In the acute stage, unless enucleation is clearly indicated (i.e., the eye is dangling from the conjunctiva). The globe may be manoeuvred back into the orbit under a general anesthetic.

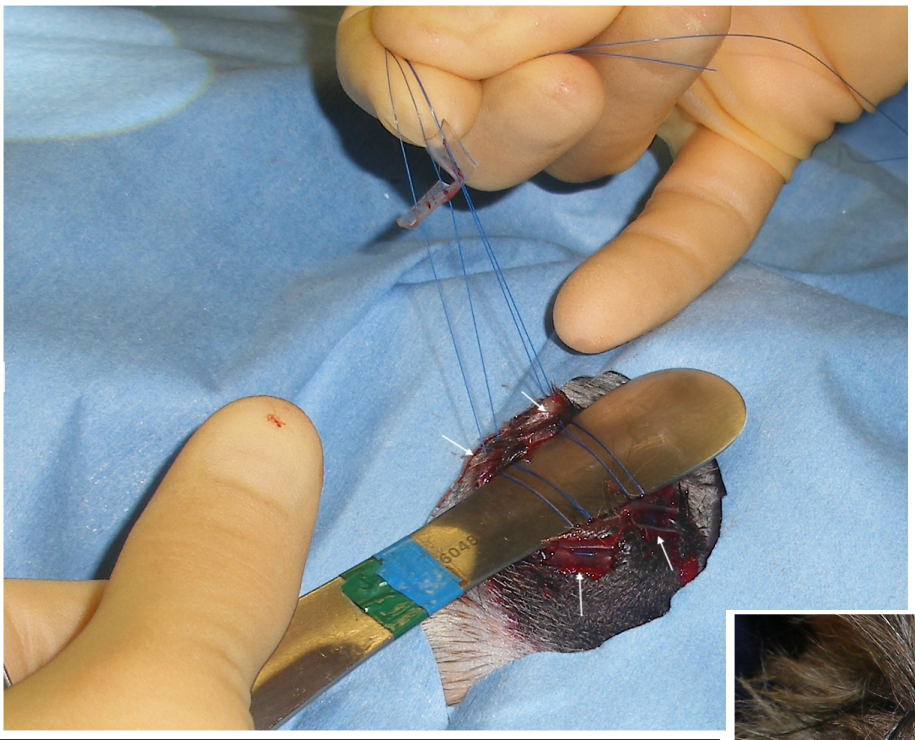
The globe should be irrigated with sterile saline solution first to remove all debris from the ocular surface and conjunctival fornices, while remembering that material might be trapped between the globe and the eyelids. The corneal surface may be assessed for damage with fluorescein dye once the eye is clean. The eye must be kept well lubricated immediately after that at all times. An aqueous based lubricating jelly that is safe to be used on the eye (e.g., KY jelly) is appropriate, as are others.

Most proptosed globes require 2 tarsorrhaphies, as a single lateral tarsorrhaphy is often not enough to counteract the pressure of the proptosed globe. A tarsorrhaphy is a simple interrupted or horizontal mattress suture placed in the eyelid margins. The 4-0 nylon sutures should engage the skin and tarsal plate but should not engage the conjunctiva underneath. The suture may exit at or in front (i.e., anterior to) the meibomian gland openings, but not behind to avoid sutures from contacting the globe. The surgeon must ensure the sutures are passed through 'stents' placed at every spot where the suture enters and exits the eyelid skin (e.g., small pieces of IV tubing cut in half work well for this purpose). Stents dissipate the pressure that the suture puts on the eyelid skin; this pressure could cause eyelid damage (note: sometimes damage of the eyelid is still possible due to the retrobulbar pressure that develops when the proptosis occurs). Before closing the tarsorrhaphy /-phies, gentle pressure is carefully applied to the lubricated ocular surface using a flat non-abrasive surface (i.e., a wooden or plastic tongue depressor, or the flat handle of a

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA

scalpel, etc.) while the eyelids are still open. The sutures are then tied over the eye and are left in place for 2 to 4 weeks while the swelling behind the eye disappears. A gap of about ¼ to 1/3 the eyelid length should be left open medially for topical medication to be administered and for tears and mucus to exit.

The eye / face should be protected by an E-collar and oral and topical medications should be given as deemed necessary. It may be difficult to assess the extent of extraocular muscle damage at the time of the emergency and even more so to surgically locate the position of an extraocular muscle that has been torn. Suturing a torn extraocular muscle is very challenging, if not impossible due to extensive tissue damage in the acute patient and should not be attempted. When placing 2 tarsorrhaphies the central one may be opened later as the swelling subsides, while the second one (i.e., the lateral one) may be left in place for longer.



Two tarsorrhaphies have been pre-placed in the left eye of this patient (as viewed from above in the first image). The patient is under anesthesia while laying on its back. The position of the stents is indicated with white arrows. The surgeon is using a surgical eyelid plate to gently push on the lubricated eye before tying the sutures. The second image shows the sutures have already been tied, though mainly the central suture and stents are visible in this image, while the lateral suture and stents are difficult to spot in this example.

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA

3. Retrobulbar abscess cellulitis

This is relatively common in small animals and affects any breed and all age groups. The causes vary but include hematogenous spread, infection in a tooth root, and entry of a foreign body through the conjunctiva or the oral cavity that later migrates. Other routes may exist, though they are arguably less common, and the exact cause is unknown in the majority of cases.

There is rarely a foreign body that requires surgical removal. An obvious open wound and/or purulent ocular discharge could, arguably, have a higher chance of harboring a foreign body. A clearly demarcated abscesses that may be drained through the pterygopalatine fossa (in the mouth) is occasionally present.

The clinical presentation is nearly always one of anterior (often dorsolateral) displacement of the globe in the presence of concomitant third eyelid protrusion (i.e., there is a mass occupying effect in the orbit). The most common differential diagnoses include neoplasia of the orbit and salivary gland adenitis / mucocele (i.e., of the zygomatic salivary gland), though salivary gland adenosis is a rare condition that may also be included. Retrobulbar abscess / cellulitis is often associated with marked pain when opening the mouth. This is caused by the forward movement of the ramus of the mandible into or near the swollen area.

Clinical approaches vary though, as before, **the main role of the emergency veterinarian** is to offer the '**SPP minimum'**. Medical therapy may be chosen first and this may be followed by imaging if there is failure of treatment, or imaging may be chosen first, followed by medical therapy. Surgeon's preference often dictates the preferred approach, while the certainty there is a foreign body based on a witness account or an obvious presenting sign is an indication for surgery.

A simple approach is treating the condition medically to see if there is a favorable response to treatment. This includes an oral antibiotic and NSAID (if the latter is tolerated). Treatment may start with an injectable form of each. If there is no rapid resolution (or if there is partial resolution) in 4-5 days, there may be bacteria present that are not responsive to the antibiotic selected (typically this is amoxicillin/clavulanic acid) and a second or alternative antibiotic (e.g., clindamycin or metronidazole) is recommended, or there may be a foreign body. If there is further resolution of clinical signs when adding a second antibiotic (or changing it), there is no need to consider further steps, such as imaging.

Another approach is imaging prior to starting medical treatment. Some surgeons advocate the use of CT first (occasionally followed by ultrasonography), and others opt to start with ultrasonography. An advantage of using imaging first is that if a drainable abscess is present, it may be identified and lanced, which may increase patient comfort. The disadvantages of imaging are access and cost.

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4. Corneal damage

This may be superficial, deep, localized, multifocal, or extensive and it may present in any combination of the above. Unless there is a history of trauma (e.g., cat claw injury is a typical example), the cause of the corneal damage is often unknown. The emergency veterinarian must remember foreign bodies under the third eyelid (i.e., commonly a grass seed) are common and its removal often leads to quick resolution of the problem. Deep corneal ulceration may be seen with chronic conditions such a dry eye but also ectopic cilium, which are typically found under the central upper eyelid. Interestingly, brachycephalics may have spontaneous corneal ulceration due to a complex confluence of physiological factors (i.e., shallow orbits, large palpebral opening, unstable tear film, and in some cases additional factors such as trichiasis and/or distichiasis, in addition to dry eye or ectopic cilium). Lastly, small foreign bodies may become lodged in the cornea. Some may be flushed out with saline, and others might sit deeper and need surgical intervention by an ophthalmologist.

The main role of the emergency veterinarian is to offer the '**SPP minimum'**:

- **Stabilize**: In addition to what is mentioned below, consider the use of sterile saline to irrigate the eye and remove foreign bodies from the ocular surface to help assess the cornea. The use of a topical anesthetic can be of great help. Keeping calm, avoiding busy, noisy rooms, and taking time might prove to be essential to minimize the response to pain, which can be challenging.
- **Pain control**: administer appropriate cover for the time being (i.e., injectable / oral NSAID, and topical atropine for iris/ ciliary body muscle relaxation).
- **Protect-Prevent**:
    - Always use a protective E-collar to avoid self-trauma (24/7 use).
    - Start prophylactic topical antibiotic use (see page 2)
    - If there is penetrative corneal trauma, start an oral antibiotic too (e.g., amoxicillin/clavulanic acid)
    - Start on an oral NSAID to help control/prevent inflammation (if tolerated by the patient)
    - Consider the use of an anticollagenolytic agent (see further below, page 11, section d)
    - Consider applying a preservative-free, viscous ocular lubricant for further protection
    - The veterinarian should inform the owners the eye should be examined by an ophthalmologist for possible surgery if there is a deep ulcer (see page 10, section d), a penetrating corneal wound, or iris prolapse.

Comments of a variety of severities of corneal damage:

a. Superficial corneal ulcers

These are painful but they are not an ocular emergency. An example is a canine SCCED (a spontaneous chronic corneal epithelial defect). This is a

superficial ulcer that may or may not be painful and that has loose epithelial edges; these should be referred for treatment to an ophthalmologist unless the referring veterinarian feels competent to treat them in-house. These ulcers rarely become deep. However, superficial ulcers can also be the start of what might rapidly become a deep ulcer; something we must be very careful with, especially in brachycephalic animals. These ulcers often start with a small, and very painful gray to whitish dot that often occurs in the central cornea and may or may not be accompanied by hypopion (i.e., a white line of white blood cell accumulation in the ventral anterior chamber). When they occur in brachycephalics they can quickly deteriorate (i.e., can become deep or even perforate in as little as 2 to 7 days). All superficial ulcers require the emergency veterinarian offers at least the '**SPP minimum** for the patient, as detailed above.

b. Deep corneal damage

This is a deep ulcer or laceration that has not penetrated the anterior chamber. There may be hypopion (i.e., a white line in the bottom of the anterior chamber caused by the accumulation of white blood cells in the gravity-dependent part of the eye), but there should be no blood or fibrin in the anterior chamber and no pupil distortion other than that caused by posterior synechiae (i.e., adhesions of the iris to the anterior lens capsule) or by miosis (i.e., a small pupil caused by intraocular inflammation).

Often, there is confusion about the depth of an ulcer. The cornea is only 0.5mm thick (it may be 0.6 mm in larger animals, while cornea edema also increases corneal thickness). This means that **a visible 'crater'** or 'pot-hole' **must be deep**. Assessment of the eye by an ophthalmologist for possible surgery is indicated if a wound has reached 50% corneal depth. This means **all ulcers with a visible 'crater'** or 'pot-hole' **have an indication for surgery and should be assessed by an ophthalmologist.** However, they do not all necessarily need immediate attention. As before, **the main role of the emergency veterinarian** is to offer the '**SPP minimum** for the patient.

Some ophthalmologists are surgery inclined and others are medical therapy-inclined; all options are valid so long as the problem has been assessed by a veterinary ophthalmologist and the prognosis as well as risks of each option are discussed with the owner. The risk of not operating a deep ulcer is perforation and vision loss. Surgical outcomes are **always** best (i.e., for vision and saving an eye) when the eye has not perforated.

c. Penetrating corneal damage

This is a deep ulcer or laceration that has penetrated the anterior chamber. There often is blood and/or fibrin in the anterior chamber. If there is iris and/or lens damage there may be pupil distortion, which is obvious if there

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is iris prolapse (i.e., the extrusion of part of the iris into the corneal wound), which may adapt a tan color if there is fibrin over the exposed iris, a black color if the posterior pigment of the iris is exposed, and/or a red color, if there is bleeding from the vessels of the iris. Posterior synechiae (i.e., adhesions of the iris to the anterior lens capsule) are also possible, as is miosis (i.e., a small pupil caused by intraocular inflammation).

Penetrating corneal damage is nearly always best corrected surgically to re-establish an anterior chamber. Fibrin extrusion (fibrin arises mostly from the inflamed iridal vasculature) can plug the hole and might result in fibrosis and healing without surgery. However, it carries a risk that corneal edema will open the wound after 2-3 days leading to further wound breakdown. Therefore, this should always be assessed by an ophthalmologist to determine if surgery is indicated / advised. There is evidence that lens damage may heal without surgical intervention though this is very difficult to foresee as it depends on a several factors. Small lens wounds can potentially lead to cataract formation, which can cause serious lens induced uveitis, while a larger lens wound might heal with minimal cataract formation. However, the state of the lens should not worry the emergency veterinarian, who should offer the '**SPP minimum** for the patient, as detailed above.

d. 'Extensive' corneal damage (i.e., corneal melting, large bullae)
Corneal melting (i.e., collagenolysis) occurs when there is an initial lesion that has deteriorated due to infection and/or inflammation. This always requires the emergency veterinarian offers at least the '**SPP minimum** for the patient, as detailed above. Corneal melting may be stopped medically or through surgical intervention. Serum eye drops from the patient's own blood is potentially the most comprehensive form of anticollagenolytic agent we have access too, while oral doxycycline is widely available and can be of help and may be given at the same time, and topical acetylcysteine is used by some. **Feline Acute Bullous Keratopathy** deserves particular attention. A diagnosis is made with acute onset of severe corneal edema and/or obvious corneal stroma bulla formation. It may be unilateral or bilateral and can affect cats of all ages, but the cause is poorly understood. There is rapid deterioration and referral within 24 hours, when possible, is strongly recommended. The best form of treatment has not been described, though it is thought that a third eyelid flap used for approximately 21 days may become the treatment of choice (i.e., the pressure exerted by the flap presumably reduces the edema while the eye has time to heal). Third eyelid flaps are difficult to place for the inexperienced veterinarian and their correct positioning is vital to avoid further corneal damage.

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA

5. Glaucoma (primary and secondary forms – here, the latter excludes lens luxation)

This is a group of diseases (i.e., 'the glaucomas') associated with periods of elevated intraocular pressure (**IOP**) that causes damage of the retina and optic nerve. Repeated spikes in IOP or a sustained high IOP, leads permanent vision loss. A normal IOP is about 12-24 mmHg (or 11- 25 mmHg). Types of glaucoma:

- **Primary:** a group of hereditary problems of the filtration apparatus that can no longer allow the aqueous humor to leave the anterior part of the eye. There are further subdivisions. Prognosis is guarded to very poor, as nearly all hereditary glaucomas end in blindness due to poor response to treatment over time and partial response to surgery.
- **Secondary:** caused by trauma to the eye or diseases that lead to blockage of the filtration apparatus through inflammatory cells, blood, fibrin, a tumor, or tumor cells, or of the pupil through a luxated lens or vitreous. Prognosis depends on the cause and the stage of the disease.

Clinical signs associated with increased IOP offer **important clues** and include:
- Corneal edema: usually diffuse
- A mid-dilated, nonresponsive pupil and a lack of indirect PLR (see Page 3).
- Congestion of the conjunctival vessels (typically branching) and episcleral vessels (typically larger and meandering with almost no visible branching)
- Other signs (note: not all possible signs have been listed):
    - Blindness
    - Globe enlargement (i.e., buphthalmia, usually an 'end-stage' globe)

The focus of the emergency veterinarian is to determine if there is a high IOP, treat it if possible, and offer the '**SPP minimum** for the patient. The emergency veterinarian does not need to determine the type of glaucoma, but it is important to rule out anterior lens luxation, as this will affect the medical options (see the lens luxation section, page 13).

Medications commonly used in glaucoma (not all medications are included)**:** **Carbonic anhydrase inhibitors**: brinzolamide (almost neutral pH, may be used in cats though blood K should be monitored) and dorzolamide (more acidic, it is contraindicated in cats due to possible fatal hypokalemia). Decrease IOP by up to 30% used 1-3x per day (maximal effect achieved at 3x day after 2-4 days). **Betablockers:** Timolol has a relatively low effect on IOP. Sometimes combined with dorzolamide. **Prostaglandin analogues**: latanoprost, travaprost, etc. Decrease IOP by up to 50% and may be used 1-4x per day. Cause miosis and inflammation. Contraindicated in anterior lens luxation. Does not work in cats though it causes miosis. **Pilocarpine** is best avoided as it causes inflammation and does not affect IOP to a significant degree clinically. Other medications are rarely used including **IV mannitol**, which causes temporary dehydration (i.e., little use if drops do not work).

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6. Lens luxation

This is a hereditary condition in dogs (mainly terriers) that have an inherent weakness of the zonular fibers (**PLL mutation**), though some dogs with lens luxation do not have this mutation. It may also occur in cats, secondary to **uveitis**.

The lens may be fully luxated and may move into the anterior chamber in what is called **'anterior luxation'**, which creates a risk for acutely elevated intraocular pressures (IOPs), or it may fall into the vitreous in what is called **'posterior luxation'**, during which the risk of elevated IOPs is less but still exists. Interestingly, some dogs and most cats, have a large enough anterior chamber, or a small lens compared to the size of the anterior chamber (or both), and do not develop **acute pupil block glaucoma**. As the lens may move anteriorly and then posteriorly, several episodes of pupil block glaucoma may occur over time. The lens may become trapped in the anterior chamber or adhered to the iris during anterior luxation, making the spontaneous movement of the lens into the vitreous difficult or impossible.

Long-term treatment options include surgical removal of the lens (this assumes the risks of surgery) followed by medical therapy, or medical therapy without lens removal (this assumes the risks of anterior lens luxation and lens induced uveitis). The choice made will depend on the patient's/owner's needs and the treatment bias of the ophthalmologist.

Signs associated with lens subluxation / luxation:

- **Iridodonesis and phacodonesis**: subtle movements (wobbling) of the iris and lens that happen together during eye movement.
- **Anterior presentation of the vitreous:** white, thin strand/s arising from the pupillary edge into the anterior chamber, floating in front of the iris.
- **Anterior chamber depth changes**: when the iris loses lens support.
- **Aphakic crescent**: breaks of the zonules and development of a gap or space between the pupil and the edge of the lens, as the lens moves to one side.
- **Intraocular pressure changes**
- **Central corneal edema**: when the lens has contacted the cornea (anterior luxation) it causes central edema. If the lens returns to the vitreous (posterior luxation) the edema often persists.

The focus of the emergency veterinarian is to determine if there is a high IOP, to refer promptly if there is, and always offer the '**SPP minimum** for the patient. 'Transcorneal reduction' of an anteriorly luxated lens may be achieved under general anesthesia or sedation, after the application of a topical anesthetic with a moistened cotton bud, by indenting the cornea and pushing the lens to a posterior position behind the iris. This risks corneal damage and will not work if the lens is adhered to the iris. The use of a prostaglandin analogue, such as **latanoprost** 2x day will make the pupil small enough to keep the lens in the vitreous, though this may fail and risk anterior luxation. Prostaglandin analogues are contraindicated in anterior luxation.

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7. Acute vision loss

There is a long differential diagnosis list for acute vision loss (see below, though this is not exhaustive). The challenge for the emergency veterinarian is to rule out diseases that are causing pain (e.g., glaucoma) or that may need to be promptly address and should not wait (e.g., systemic hypertension in cats, steroid responsive retinal detachment in dogs, or inflammatory optic nerve or central nervous disease in dogs). The prognosis for most causes of sudden onset blindness is guarded to poor, though a few etiologies might improve with prompt diagnosis and treatment.

It is important for the emergency clinician to obtain a thorough history and examine the direct / indirect pupillary light reflex (see page 3), and determine if there is:

- systemic hypertension, especially in an older cat
- an elevated IOP (and if it is caused by anterior lens luxation, see other section)
- a retinal detachment (in a dog or cat)
- intraocular bleeding

Acute vision loss may be caused by one of the following:

- **Acute glaucoma** (see previous section)
- **Intraocular bleeding** (in the anterior chamber = hyphema, vitreous, retina)
    - Hyphema due to uveitis, systemic hypertension, or trauma
    - Vitreal and/or retinal bleeding (same causes as above)
- **Retinal detachment**
    - Steroid responsive retinal detachment in dogs (always bullous)
    - Breed-predisposition (i.e., Shih-Tzus), bullous or rhegmatogenous (i.e., when a hole or tear of the retina is present)
    - Systemic hypertension of older cats (see also intraocular bleeding)
- **Acute non-detachment retinal disease in dogs**
    - SARD(s) (Sudden Acquired Retinal Degeneration (Syndrome))
    - IMR (immune mediated retinopathy or retinitis)
- **Retinal toxicities** (rare; with high enrofloxacin dose previously described in cats before official dose adjustment, and ivermectin toxicity also described)
- **Optic nerve and central nervous system disease**
    - Optic neuritis (unilateral, bilateral, or of the chiasm)
    - Optic nerve and /or optic chiasm tumors
    - Meningitis of Unknown Origin (MUO), sometimes referred to as Meningitis of Unknown Etiology (MUE) and before referred to as Granulomatous Meningoencephalitis (GME)
    - Post-operative blindness (secondary to brain hypoxia)
- *PRAs* (progressive retinal atrophies) are a slowly developing condition. However, owners, occasionally do not notice the progressive blindness and are surprised to find their dog with a perceived 'sudden onset blindness' that is in fact chronic (this may also happen in cats, but it is an even rarer presentation history (i.e., in the acute form) for feline patients).

By Rick F Sanchez BSciBiol, DVM, DipECVO, CertVetEd / FHEA