Diagnosis and management of ophthalmic emergencies can be challenging, however diagnosing the source of a red or painful eye, ocular clouding or vision loss can be simplified with an organized, strategic approach and a few basic diagnostic tests. Some common ocular emergencies and treatment options will be outlined below.

**Ocular proptosis**
Ocular proptosis is often the result of blunt periorbital trauma or increased pressure applied to the posterior periorbital tissues. It occurs most commonly in brachycephalic breeds as a result of their shallow orbits and lagophthalmos. Unfortunately, the prognosis for vision in any propptosed eye is guarded. In a 1995 retrospective study of 84 cases of proptosis, only 27% of eyes were visual at follow-up evaluation. Positive prognostic indicators for vision noted in that study included a brachycephalic conformation, intact direct pupillary light reflex (PLR) or consensual PLR to the contralateral eye at presentation, intact vision at presentation, and normal funduscopic exam findings. Negative prognostic indicators for vision included hyphema, a non-brachycephalic facial conformation, absent vision and PLR, facial fractures and visible optic nerve damage on funduscopy. Interestingly, pupil size was not related to outcome in the study. The recommended treatment for proptosis almost always involves either reduction of the proptosis with placement of temporary tarsorrhaphy sutures or enucleation. Prior to surgery, owners should be counseled that proposed globes are at risk of lateral strabismus, keratoconjunctivitis sicca, reduced corneal sensation/neurotrophic keratitis and vision loss. The decision to attempt to save a propptosed eye should be made with the above considerations in mind. Propptosed globes with 3 or more avulsed extraocular muscles should be enucleated. Proptosis in the cat is almost universally blinding and enucleation is recommended in this species. Reduction of the proptosis is usually performed under general anesthesia. The eyelids are retracted away from the posterior aspect of the globe, either with the use of atraumatic forceps or pre-placed sutures, and gentle pressure is applied to the globe to reposition the globe into the orbit. Once the proptosis is reduced, non-absorbable suture (generally 4-0) is used to place tarsorrhaphies. Horizontal mattress and simple interrupted patterns are commonly used. Stents may be placed per surgeon’s preference. Usually a small opening is left adjacent to the nasal canthus to allow for application of topical medication. The patient should be discharged with systemic anti-inflammatories, topical lubricating agents and generally, systemic antibiotics. The tarsorrhaphies should stay in place for 2 weeks, or until the periorbital swelling has resolved. Tear production should be evaluated periodically.

**Exophthalmos-inflammatory orbital disease/orbital cellulitis**
Retrolublar disease is generally either inflammatory, infectious or neoplastic in nature. The most common sign of retrolublar disease is exophthalmos, which may be combined with third eyelid elevation, globe deviation, reduced/absent retropulsion and corneal exposure. Orbital cellulitis is a general term for retrolublar inflammatory disease and is generally acute in onset, is often associated with pain on manipulation of the mandible, may be associated with pyrexia, and is often unilateral. Close evaluation of the soft tissue caudal to the ipsilateral second maxillary molar may reveal a notable swelling in the case of an associated abscess. The underlying cause is often unidentified, though an underlying penetrating trauma or foreign body, dental disease, infection of the zygomatic salivary gland, and hematogenous seeding of bacteria have been proposed. *Staphylococcus* spp, *E. coli, Pasteurella multocida, Bacteroides* spp and *Clostridium* spp have all been identified on culture of orbital abscesses, although bacterial cultures are occasionally negative. A tentative diagnosis can be reached through clinical examination and confirmed with orbital ultrasound, CT or MR. Treatment includes transoral lancing (+/- antibiotic lavage), broad spectrum systemic antibiotics, systemic anti-inflammatory and topical lubrication. In a 2009 retrospective study evaluating aerobic culture and sensitivity results of material isolated form orbital abscesses, mixed infections were common. In dogs, antibiotic sensitivity to amikacin, ceftiofur, gentamicin, imipenem, ticarcillin and trimethoprim–sulfamethoxazole was high, while sensitivity to ampicillin, clindamycin, erythromycin and penicillin was low. Bacteria isolated form the orbit of cats did not exhibit a high degree of antibacterial resistance (Wang, et al, 2009). A temporary tarsorrhaphy is often recommended to prevent exposure keratitis while the swelling resolves. Inflammation caused by an unidentified tooth root abscess or foreign body is likely to recur. The prognosis is generally good, although vision loss can result from optic nerve damage caused by severe exophthalmos. Exophthalmos related to neoplasia is often slowly progressive and non-painful. Advanced imaging is often necessary to evaluate the retrolublar space in these patients.

**Deep corneal ulcer/malacia/laceration**
Deep or penetrating ulcers or corneal lacerations generally require surgical intervention and should be referred to a veterinary ophthalmologist. Topical and oral broad spectrum antibiotics, systemic analgesics and use of an Elizabethan collar are recommended in the interim. Systemic anti-inflammatories are also often of benefit to treat secondary uveitis. Administration of a topical mydriatic
agent (atropine or tropicamide) may prevent iris prolapse in an impending rupture. In general, topical solutions (rather than ointments) are recommended in ruptured corneas as ointments can be toxic to the corneal endothelium. Malacic ulcers should be treated temporarily with broad spectrum oral and topical antibiotics and serum/plasma q2-6 hours depending on the severity of stromal loss and associated malacia.

**Glaucoma**

Common clinical signs of acute glaucoma include blepharospasm, third eyelid elevation, episcleral congestion, corneal edema, mydriasis and vision loss. Acute glaucoma generally presents as a unilateral disease but may be bilateral, especially if secondary to other intraocular disease. A breed predisposition for primary glaucoma has been described in a number of breeds, including the Cocker Spaniel, Basset Hound, Chow, Beagle, Boston Terrier and many more. Emergency management of acute glaucoma can involve intravenous, oral and/or topical treatment.

Osmotic agents such as mannitol and glycerin are commonly used in emergency management of glaucoma due to rapid efficacy. Mannitol is administered IV at doses ranging from 1-2 g/kg over 30 minutes. The reduction in IOP generally begins within 30 minutes-1 hour with effects lasting from 6-10 hours. Mannitol is not metabolized and therefore can be administered to diabetic patients. It should be administered through a filter given its propensity to form crystals. Glycerin is easy to administer, inexpensive and does not require intravenous access or special storage. It is administered orally at a dose of 1-2 g/kg (for a 99% mannitol solution I generally administer 0.75mL/pound). A reduction in IOP should be observed within an hour of administration and can last as long as 10 hours. Administration may result in vomiting. Use of hyperosmotic agents is contraindicated in uveitic eyes due to increased blood-ocular-barrier permeability of inflamed eyes. They should not be administered with fluids (and water should be withheld for ~2 hours post-administration). Due to the expected increase in intravascular volume associated with these agents, hyperosmotics should not be administered in patients with significant cardiovascular disease.

Carbonic anhydrase inhibitors inhibitors inhibit formation of bicarbonate in the ciliary body that is necessary for production of aqueous humor. Commonly used topical agents include dorzolamide (Trusopt®) and brinzolamide (Azopt®). Oral CAIs include methazolamide and acetazolamide. Dorzolamide is available as a generic and is fairly cost effective. Topical CAIs can be administered 2-3 times daily. Maximum efficacy may take 4-5 days to achieve but decreased aqueous humor production occurs within 30 minutes – a few hours of dosing. The topical CAIs can be used in dogs and cats and are effective in both species. They can be used in all types of glaucoma, have no effect on pupil size, and do not contribute to intraocular inflammation. These drugs have a slower, less dramatic effect on IOP and therefore should not be used alone as management for high pressure acute glaucoma.

Prostaglandin analogs appear to be the most effective drugs in the treatment of canine glaucoma. These drugs increase aqueous outflow (with no effect on aqueous production). The mechanism of action is mediated through binding to prostanoid FP receptors. The most commonly prescribed prostaglandin analog in veterinary medicine is latanoprost (Xalatan®), which is now available as a generic. Other available PG analogs include bimatoprost and travaprost. In the cat, latanoprost and other PG analogs are ineffective because activation of prostanoid EP receptors is required for similar effects in this species. The prostaglandin analogs are generally administered q12h in dogs (once daily in humans). In the dog, PG analogs result in maked miosis and because they work through activation of inflammatory mediators, should not be used in cases of secondary glaucoma caused by anterior lens luxation or chronic uveitis.

**Uveitis**

Clinical signs of dogs and cats with uveitis include blepharospasm, corneal edema and miosis. Intraocular pressure may be decreased or elevated, depending on the chronicity of the inflammation. Uveitis can present as either unilateral or bilateral disease. In cats and blue-eyed dogs, a change in iris color may be appreciated due to vascular congestion of the iridal blood vessels. Causes of uveitis are numerous, and include idiopathic, inflammatory/auto-immune, infectious, neoplastic and traumatic etiologies. Baseline bloodwork (CBC/Chem) and infectious disease screening for tick-borne, fungal, bacterial and, in cats, viral diseases endemic to your area are recommended. Treatment involves topical (ie pred acetate q6h) and oral (steroids or NSAIDs, depending on your index of suspicion for infectious disease, the overall health of the animal and the severity of the inflammation) anti-inflammatories until the patient can present to an ophthalmologist. Additional screening may include thoracic radiographs, abdominal ultrasound, and in cases of suspected ocular trauma, radiographs of the head. Clients should be counseled that the workup and treatment of uveitis can be protracted, especially if rapid response to medications does not occur.

**Hyphema**

Like uveitis, hyphema can be the result of local (ie intraocular) or systemic disease. Causes of intraocular disease include intraocular neoplasia, retinal detachment, blunt or sharp trauma, and underlying intraocular vascular anomalies (more likely in a young animal). Systemic causes include hypertension, coagulopathies, and causes of severe uveitis including neoplastic or infectious diseases. Baseline diagnostics include a CBC/Chemistry and systolic blood pressure. An ocular ultrasound may be performed to evaluate for the presence of intraocular neoplasia and determine the status of the retina. The intraocular pressure should be evaluated as hyphema
can result in both short- and long-term elevations in IOP. Treatment is dependent on the underlying cause, however topical anti-inflammatory agents (ie pred acetate/neo-poly-dex/dex SP q6h) and, if the intraocular pressure is acceptable, use of a short-acting mydriatic agent (ie tropicamide q12h), may be of benefit in preventing secondary glaucoma.

Acute-onset blindness
Determining the source of acute bilateral vision loss can be complicated, but in a basic sense vision loss occurs from dysfunction of one of three structures: the eye, the optic nerve or the brain. Ocular sources of acute vision loss include glaucoma, cataract development, retinal detachment/retinal hemorrhage, or sudden acquired retinal degeneration syndrome (SARDs). Primary glaucoma does not generally develop in both eyes simultaneously but can present as bilateral vision loss if the ‘first’ eye has gone undiagnosed. Although glaucoma itself is a complex disease, its diagnosis generally is not (ie it can be ruled in or out via estimation of intraocular pressures). Cataracts, retinal detachment and retinal hemorrhage should be visible on ophthalmic exam. If the ophthalmic evaluation is unremarkable the primarily differential for an ocular source of vision loss is SARDs. Patients with SARDs generally have resting mydriasis with slow PLRs. The diagnosis of SARDs is confirmed with an electroretinogram, which evaluates the electrical activity of the retina. Patients with SARDs exhibit flat line retinal function, while non-retinal sources of vision loss should have a normally appearing ERG waveform. Unfortunately, no consistently effective, safe treatment for SARDs is available, and the cause remains unknown. Referral to an ophthalmologist should still be recommended so that the disease can be confirmed. Non-ocular causes of acute vision loss include optic nerve disease (ie optic neuritis), or central nervous system disease (infectious, inflammatory, neoplastic, etc). If the ERG findings are within normal limits, referral to a neurologist will be recommended.

References
Funduscopic examination is an integral part of the complete ophthalmic evaluation and can be considered an important component of any physical exam. Direct ophthalmoscopy is the most common method for evaluation of the fundus, namely because the equipment is readily available in most practices. Indirect ophthalmoscopy requires minimal equipment, namely a light source and a focusing lens. The panoptic ophthalmoscope is a newer device that is becoming more commonly used and in many ways combines the advantages of the other two. For best visualization of the fundus, pupillary dilation and a dark environment are recommended.

Direct ophthalmoscopy- The direct ophthalmoscope provides a highly magnified view of the fundus and can be used for assessing the depth or length of depressed or raised fundic lesions, respectively. The image is real and upright. Disadvantages include the required close proximity to the patient and the high degree of magnification, which can make generalized evaluation of the fundus more difficult.

Indirect ophthalmoscopy- When compared with direct ophthalmoscopy, indirect ophthalmoscopy has the advantages of a longer working distance and larger field of view. Using a light source (penlight or transilluminator) and a converging lens, indirect ophthalmoscopy provides an inverted, reversed view of the fundus (i.e., a lesion that appears ventrolateral through the indirect lens is actually dorsonasal). The degree of magnification of the converging lens is inversely related to the dioptic power of the lens (i.e., a 28D lens provides a less magnified view than a 20D lens, and will therefore also provide a larger field of view). Another thing to keep in mind is that the larger the eye, the less magnification any given lens will provide, which is why an indirect lens that is appropriate for a dog or cat (i.e., 22-28D) may be too ‘zoomed out’ for a horse, for whom a 14-20D lens is more appropriate.

Panoptic ophthalmoscope (Welch Allyn)- The Panoptic provides a real, upright view of the fundus with a magnification strength somewhere between the direct and indirect methods. It provides an intermediate working distance from the patient as well.
Uveitis in its basic sense represents a breakdown of the blood-ocular barrier. In a healthy eye, the blood ocular barrier acts as a protective mechanism that regulates the constitution of intraocular contents. The blood-ocular barrier consists of a set of anterior chamber (blood-aqueous barrier) and posterior segment (blood-retinal barrier) vascular endothelial and epithelial tight junctions in the iris and ciliary body blood vessels, retinal capillaries and retinal pigment epithelium. Tissue injury, either of immune-mediated, infectious, neoplastic, etc etiology, results in release of inflammatory mediators (histamine, prostaglandins, kinins, plasmin, complement, peptide growth factors) that causes breakdown of these barriers. The breakdown of intracellular tight junctions allows for protein and inflammatory cell leakage and migration into the globe. This results in the classic clinical sign of anterior uveitis, known as ‘flare’. The aqueous humor in health is clear, such that a slit beam of light crossing the anterior chamber will reflect off of the cornea and lens capsule, with no evidence of light transit through the anterior chamber. With flare, the beam of light reflects off of the proteins and cells that have accumulated in the anterior chamber, resulting in a visible light beam within the chamber. This effect is known as the ‘Tyndall’ effect. The classic example of the Tyndall effect is the light of an old movie projector bouncing off of dust in a theater. Breakdown of the blood-retinal barrier results in similar protein, fluid and cell accumulation posterior to the retina, resulting in subretinal fluid accumulation and possibly retinal detachment.

Common clinical signs of anterior uveitis include blepharospasm, epiphora, episcleral congestion, corneal edema, keratic precipitates and miosis. In blue eyed dogs and most cats, the iris may also appear discolored as result of iridal vascular congestion. The anterior chamber can exhibit flare, hyphema, hypopion, fibrin or all of the above. Adhesions may be present between the iris and anterior lens capsule (‘posterior synechiae’). The intraocular pressure may be either low or high, depending on the chronicity of the inflammation. Uveitis can be either unilateral or bilateral regardless of the underlying cause. Unfortunately, the cause of the uveitis is only rarely evident based on the appearance of the eye. The majority of cases of uveitis are idiopathic and many will respond nicely to appropriate treatment. Unfortunately, however, uveitis can also be caused by almost any infectious, inflammatory or neoplastic systemic disease.

Infectious (taken from Gelatt, see references)
Canine- algal (Prototheca), fungal (Histo, Blasto, Cocci, Crypto, Aspergillus, Candida), bacterial (Brucella, Borrelia, Leptospira), rickettsial (Ehrlichia, Rickettsia), parasitic (ophalmomyiasis/Dipteria sp, Dirofilaria, Angiostrongylus, Toxocara, Balisascaris) protozoal (Leishmania, Toxoplasma, Neospora, Trypanosoma), viral (Adenovirus, Herpesvirus)
Feline- viral (FeLV, FIV, FIP), fungal (Cryptococcus, Cocci, Blasto, Candida, Histo), bacterial (Bartonella), protozoal (Toxoplasma), parasitic (Diptera)

Neoplastic
Any primary or secondary neoplasm (lymphosarcoma most common), histiocytic disease, feline diffuse iris melanoma in cats

Immune mediated
Idiopathic, Uveodermatologic syndrome (canine only), immune-mediated vasculitis, immune-mediated thrombocytopenia, lens-induced, secondary to scleritis, traumatic, reflexive, radiation-induced, general toxemia or sepsis

The diagnostic workup for any patient with uveitis should include a complete physical exam with evaluation of peripheral lymph nodes, etc. Baseline bloodwork (CBC/Chemistry) and infectious disease screens should be submitted based on current location and recent travel history. Thoracic radiographs and abdominal ultrasound may also be recommended based on the index of suspicion for systemic disease.

With the exception of reflexive uveitis caused by ulcerative keratitis, treatment should consist of topical anti-inflammatories (generally corticosteroids q4-6h), plus or minus oral anti-inflammatories based on the systemic health of the patient, index of suspicion of systemic infectious disease and degree of inflammation. Posterior segment inflammation (ie chorioretinitis) requires systemic treatment in all cases. If an underlying cause is identified, specific treatment should be initiated in conjunction with anti-inflammatories. In general these cases should be referred to an ophthalmologist and rechecked within the first week of treatment. Long term sequelae including secondary glaucoma, cataract development, corneal degeneration and/or retinal degeneration from retinal detachment can be vision threatening. Treatment for the uveitis can be protracted depending on response to treatment and anti-inflammatories should, as a rule, be tapered slowly over weeks to months to prevent recurrence once the inflammation is controlled.
References
