OCULAR DISEASE IN THE INTENSIVE CARE UNIT

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KEY POINTS
- Ocular disease is a common manifestation of systemic illness seen in critically ill patients.
- Corneal disease is a possible complication in anesthetized patients, and appropriate preventative therapy should always be provided.
- Identification of ocular disease is accomplished by conducting a thorough ophthalmic examination including indirect ophthalmoscopy, Schirmer tear testing, fluorescein staining, tonometry, and possibly cytologic studies, culture, or biopsy.

Ophthalmic disease can be associated with or secondary to conditions that require a patient to be in a critical care facility. This chapter describes common ocular signs and discusses the appropriate interpretation of these signs and treatment of the ocular disease.

BLEPHAROSPASM

Blepharospasm is a nonspecific sign of ocular pain and may be associated with enophthalmos, elevation of the third eyelid, and spastic entropion. Both surface and intraocular disease can result in blepharospasm. The origin of ocular pain is determined by conducting a thorough ophthalmic examination, including diagnostic tests such as fluorescein staining, Schirmer tear testing, and tonometry. A topical anesthetic (e.g., 0.5% proparacaine) may facilitate examination by eliminating pain related to surface disease.

RED EYE

Veterinary clinicians commonly encounter patients with a "red eye." The redness represents new or congested blood vessels within the episclera, conjunctiva, or cornea. Episcleral vessels are stout, easily identifiable vessels that course perpendicular to the limbus and usually stop before reaching the limbus. Congestion of these vessels is associated most commonly with intraocular disease, specifically uveitis and glaucoma. However, with moderate to severe corneal disease these vessels may become engorged.

Conjunctival blood vessels are extremely fine; without the aid of magnification individual vessels are difficult to identify. When vessels are engorged, a pink-red flush is observable. Mild conjunctival hyperemia may be apparent with intraocular disease, but moderate signs are consistent with surface disease (e.g., conjunctivitis or keratoconjunctivitis). Differential diagnostic considerations for conjunctivitis include infections (canine distemper, feline herpesvirus infection, feline *Chlamydophila* infection, leishmaniasis, onchocerciasis), allergies, postradiotherapy conditions, keratoconjunctivitis sicca (KCS), and exposure. Diagnosis is based on history and the results of Schirmer tear testing, fluorescein staining, cytologic studies, and biopsy.

Conjunctival hyperemia can be easily confused with a conjunctival or subconjunctival infiltrate. This infiltrate may be fluid (chemosis) or cells. Mild chemosis is common with conjunctivitis. Severe chemosis may obstruct visualization of the cornea and intraocular structures. The most common cause of primary chemosis is topical toxicity (from neomycin, atropine, caustic agents). Removing the toxin and treating supportively allows resolution of signs. Rarely, intravenous fluid overload at a rate of two to three times maintenance for a period of 2 days or longer can result in marked chemosis. Tapering the fluid rate allows the chemosis to resolve.

The presence of subconjunctival infiltrates may cause the conjunctiva to appear thickened. The infiltrates may be focal or diffuse. Carefully examining the color of the conjunctiva may help in differentiating an infiltrate from common hyperemia.

A diffuse yellow appearance of the conjunctiva in the absence of thickening is consistent with icterus. This may be the first clinical sign of icterus and should prompt the clinician to pursue further diagnostic tests to assess hepatobiliary status.

Neoplastic cells within the subconjunctiva frequently result in thickening and a yellow to orange hue. Lymphoma is the most common neoplasia presenting in the subconjunctiva, and the subconjunctiva may represent the primary tumor site. Other disorders producing masses in the subconjunctiva include systemic histiocytosis (orange), hemangiosarcoma (red), melanoma (brown), and granulomatous scleritis (pink). A definitive diagnosis can usually be obtained by biopsy. Light sedation and topical anesthesia are typically all that is needed to obtain a diagnostic sample.

Subconjunctival hemorrhage in a critically ill patient, observed as petechiae or ecchymoses, warrants investigation for an underlying coagulopathy. Hemorrhage may be isolated to the subconjunctiva or seen in the anterior chamber (hyphema). Causes of hemorrhage not associated with a coagulopathy include trauma, strangulation (from choke collars), chronic emesis, and rarely constipation.

A blue-green discoloration of the sclera and/or conjunctiva may be noted. This has been observed in dogs receiving mitoxantrone chemotherapy. Signs are temporary and usually resolve within hours to days after cessation of treatment.

TEAR FILM ABNORMALITIES

The tear film is comprised of three layers: an outer lipid layer, a middle aqueous layer, and an inner mucin layer. A deficiency in any of these components may result in decreased tear production or increased tear clearance (evaporation) and may be diagnosed with a Schirmer tear test.

Clinical signs of a tear film abnormality depend on the severity, chronicity, and underlying cause of the tear deficiency. The most consistent and obvious finding is a thick mucoid discharge, commonly accumulated on and around the eyelids. Additional clinical signs include conjunctival hyperemia, a lackluster appearance to the corneal surface, and in chronic cases corneal vascularization and melanosis. Chronic tear film deficiencies lead to thickening of the corneal epithelium, and therefore ulceration is not common. However, the critically ill patient may develop acute KCS resulting in rapid, severe, and potentially globe-threatening corneal ulceration.

There are numerous causes of decreased tear production (KCS) and increased tear clearance. Undoubtedly the most common cause of KCS is immune-mediated destruction of the lacrimal gland and gland of the third eyelid. This will likely be a preexisting disease in critically ill patients, although tear production in canine intensive care unit patients is decreased compared with that in healthy dogs. Treatment with topical cyclosporine or tacrolimus and artificial tear ointments or gels should be initiated to support surface ocular health.

Other causes of decreased tear production include radiation therapy, drug toxicity (sulfonamides, atropine, etodolac), chronic blepharoconjunctivitis, general anesthesia, orbital trauma, neurogenic factors, and congenital lacrimal gland hypoplasia (Yorkshire Terrier, Chinese Crested); rarely, it is secondary to an endocrine disorder (hypothyroidism, diabetes mellitus, hyperadrenocorticism).

In one study megavoltage radiation near the orbit resulted in KCS in 24% of dogs within 1 to 6 months of therapy secondary to direct destruction of glandular tissue.[1] Medical therapy is solely supportive, including the application of artificial tear ointments (petroleum, lanolin, mineral oil base) and gels as frequently as possible.

Sulfa-containing drugs are well known to decrease aqueous tear production, with 65% of patients having decreased tear production, 15% with clinical signs of KCS.[2] Sulfonamides should be used with caution in small breeds, brachycephalic breeds, and those breeds predisposed to KCS. Stopping therapy at the onset of KCS may allow lacrimal function to return in some patients. An idiosyncratic reaction resulting in irreversible, absolute xerophthalmia has been demonstrated in a small percentage (0.0003%) of dogs receiving etodolac. Patients should have a normal Schirmer tear test result before treatment and should be monitored closely during therapy. Any decrease in tear test results warrants cessation of oral therapy and initiation of topical therapy.

General anesthesia, especially with atropine as a premedication, dramatically decreases aqueous tear production, and the decrease may persist for 24 hours.[3] Many patients receive a topical lubricant before anesthesia but rarely afterward. A topical lubricating ointment should be applied at least every 4 hours for 24 hours following anesthesia to decrease ocular surface drying, which may lead to corneal ulceration.

Neurogenic KCS results from disruption of the parasympathetic fibers coursing with the facial and trigeminal nerves to the lacrimal gland. Clinical signs are similar to those of immune-mediated KCS, except that in these cases dysfunction is usually unilateral. If the lesion is near the pterygopalatine ganglion, the caudal nasal nerve will also be affected, and a dry, crusty nose ipsilateral to the dry eye will be noted. Treatment is aimed at stimulating the denervated gland to secrete aqueous tears. Two percent pilocarpine (1 drop/4.4 kg [10 lb] orally [PO] q12h increased slowly to effect) may be effective, although there is a fine line between a therapeutic and a toxic dose. Signs of toxicity include vomiting, diarrhea, and ptyalism. Treatment with topical lubricants and cyclosporine or tacrolimus is also warranted.

Increased tear clearance secondary to evaporation accounts for most cases of dry eye in the intensive care patient. Increased evaporation may be secondary to a tear lipid deficiency, lagophthalmos, or decreased reflex tearing. Meibomianitis, blepharitis, and conjunctivitis damage the meibomian glands or conjunctival goblet cells, which results in instability of the tear film. Treatment with mucinomimetic preparations such as 1% to 2% methylcellulose or sodium hyaluronate helps restore tear film stability.

Lagophthalmos is the inability to completely close the eyelids and may be a conformational disorder (brachycephalic breeds, cicatricial ectropion, eyelid agenesis) or neurologic disorder (facial or trigeminal nerve dysfunction, obtundation). The lack of a consistently complete palpebral reflex is diagnostic. With lagophthalmos, the tear film is exposed and rapidly evaporates, especially in the interpalpebral fissure. Obtunded animals frequently have decreased or absent palpebral reflexes and decreased reflex tearing, which further complicates the tear deficiency. Regardless of the cause, hourly application of an artificial tear ointment or gel is necessary. If the condition is left untreated, progressive corneal ulceration will ensue. Chronic cases (e.g., artificially ventilated patients) may require a lateral temporary tarsorrhaphy.[4]

ABSENT PALPEBRAL REFLEX

Absence of the palpebral reflex is due to either loss of trigeminal nerve function (the afferent arm) or facial nerve paralysis (the efferent arm). Of these, facial nerve paralysis is far more common. In addition to loss of the palpebral reflex, signs associated with facial nerve paralysis in cats and dogs include lowered carriage of the ear, drooping of the eyelids with resultant widening of the palpebral fissure, and increase in scleral visibility on the affected side, as well as "pulling" of the nose toward the normal side (Figure 154-1).

Because the lacrimal nerve runs with the facial nerve over a portion of its course, Schirmer tear test values should be monitored in patients with facial nerve paralysis. Compromise of both facial and lacrimal nerve function can lead to severe corneal disease due to the fact that there is a reduction in both tear production and distribution. This condition is referred to as *neuroparalytic keratitis*.[5] Causes of facial nerve paralysis include trauma, neoplasia, and surgery to the head or neck region. Depending on the cause, the signs of facial nerve paralysis may resolve spontaneously. Because the retractor bulbi muscle is innervated by the abducens nerve, many patients learn to "blink" with their third eyelid by retracting their globes. Facial nerve paralysis can often be managed by frequent application of lubricating ointments (every 4 to 6 hours). However, in severe cases, especially with neuroparalytic keratitis, a partial lateral tarsorrhaphy may be beneficial.

The trigeminal nerve provides sensory innervation to the ocular surface and eyelids. An abnormal palpebral reflex caused by trigeminal dysfunction can be differentiated from that due to facial nerve paralysis by the fact that the patient will blink when menaced.[5] Disruption of this innervation may result in severe keratitis. The most

FIGURE 154-1 Right sided facial nerve paralysis in a dog. Note the lowered carriage of the right ear, widening of the right palpebral fissure, and pulling of the nose to the normal, left side.

common cause of trigeminal nerve loss is orbital trauma.[6] Unlike those with facial nerve paralysis, these patients do not "blink" with their third eyelids because they do not have any sensation of ocular surface dryness. Treatment for ocular problems related to trigeminal nerve compromise is supportive care of the cornea with topical lubricants.

CORNEAL CHANGES

Corneal clarity is sacrificed when there is disruption of the normal organization of stromal collagen lamellae, in-growth of blood vessels, or deposition of pigment or cells. Epithelial cell loss usually results in focal corneal edema, although expansive defects can result in diffuse edema. Corneal endothelial cell dysfunction causes diffuse edema and is secondary to intraocular disease, specifically uveitis or glaucoma. A thorough ophthalmic examination including fluorescein staining and tonometry can aid in differentiating the underlying cause.

The normal cornea is avascular and receives nutrition from the tear film and aqueous humor. Therefore the presence of blood vessels indicates an ongoing pathologic process. Superficial corneal blood vessels appear as fine tree branches and are consistent with superficial disease (superficial corneal ulceration, KCS, exposure keratitis, pannus). Deep corneal blood vessels have the appearance of hedges, with individual vessels difficult to identify. These vessels are present with deep corneal (stromal) or intraocular (uveitis, glaucoma) disease. With disease, vessel in-growth is delayed for approximately 2 to 4 days, and then vessels advance approximately 1 mm per day. Therefore length of the vessels can aid in determining the chronicity of disease.

Corneal ulceration is likely the most commonly encountered primary ophthalmic disease in the intensive care patient. Clinical signs include blepharospasm, conjunctival hyperemia, episcleral congestion, mucoid to mucopurulent discharge, focal to diffuse corneal edema, an observable corneal defect, and potentially vascularization, abscess formation, or malacia. Diagnosis is facilitated by application of fluorescein stain. Stromal loss, cellular infiltrate, moderate vascularization, and progression despite medical therapy indicate a complicated corneal ulcer (Figure 154-2).

The leading cause of complicated ulcers is the use of topical steroids in a patient with a corneal defect. Complicated ulcers require strict monitoring and aggressive medical and potentially surgical therapy. Therefore referral to or consultation with an ophthalmologist is recommended.

When one is dealing with a complicated ulcer, determination of the depth of the defect is the first step. This can be accomplished

FIGURE 154-2 Infected corneal ulcer in a dog. Note the mucopurulent discharge, conjunctival hyperemia, corneal edema, stomal loss, and cellular infiltrate. This patient also has hypopyon with a few red blood cells intermixed in the ventral aspect of the anterior chamber.

using the slit beam on a direct ophthalmoscope and looking at the change in curvature of the light beam. Other hints include the location of corneal blood vessels (see previous discussion) and fluorescein staining characteristics. If stain is observed only along the walls, but not the floor, a descemetocele is present and immediate referral to an ophthalmologist is recommended.

After determination of the depth, samples should be obtained for aerobic bacterial culture and cytologic analysis.[7] Initial therapy should include a broad-spectrum topical antibiotic (dependent on culture and cytology results) at least every 4 hours, topical atropine every 12 hours, and use of an Elizabethan collar. If cellular infiltrate or malacia is present, a more powerful topical antibiotic such as a fourth-generation fluoroquinolone (moxifloxacin or gatifloxacin) should be administered every 2 hours along with topical serum every 2 hours. Serum can be harvested from the patient or a healthy donor of the same species. Serum must be kept refrigerated and a new batch should be harvested once weekly to prevent contamination.

Fortunately, most corneal ulcers are not complicated. A topical triple-antibiotic preparation applied two to four times daily, a single dose of atropine, and use of an Elizabethan collar are usually sufficient. Epithelialization of uncomplicated ulcers occurs within 3 to 5 days. If healing has not occurred within this time, either the ulcer has become complicated or the underlying cause (ectopic cilia, distichiasis, conjunctival or third eyelid foreign body) has not been identified.

Corneal infiltrates other than white blood cells associated with infected ulcers are rare. Notable exceptions include neoplastic cells, mineral, and lipid. Circumferential, severe perilimbal vascularization with a yellow-orange corneal infiltrate along the leading edge of the vessels may represent lymphoma. Cholesterol crystals and lipid may be deposited in an arclike fashion (arcus lipoides) in the anterior corneal stroma. Often this represents a systemic dyslipidemia, and further diagnostic tests to investigate the cause are warranted. Other concurrent signs may include lipemic aqueous or lipemia retinalis. Differential diagnostic considerations include hypothyroidism, diabetes mellitus, hyperadrenocorticism, pancreatitis, and a primary hyperlipidemia.

ANTERIOR CHAMBER ABNORMALITIES

Changes in the appearance of the anterior chamber most often are due to alterations in the composition of the aqueous. The aqueous is essentially modified blood with protein and cells removed in the ciliary body. Under conditions of anterior uveitis, these elements gain entry into the aqueous humor, producing the signs of aqueous flare (protein), keratic precipitates (fibrin and white blood cell aggregates on the posterior cornea), hyphema (red blood cells), and hypopyon (white blood cells) (Figure 154-3).
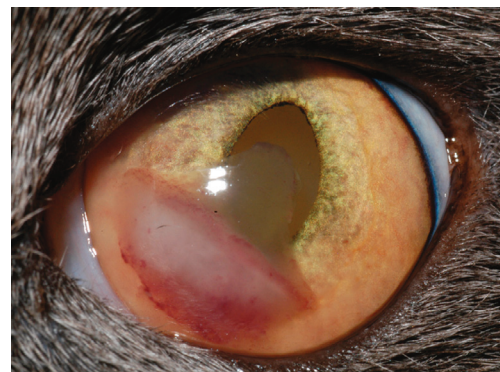

FIGURE 154-3 Fibrin clot in the anterior chamber in a cat with lymphoma.

Although all of these conditions are nonspecific indicators of anterior uveitis, keratic precipitates, hyphema, and hypopyon are seen frequently with certain causes of anterior uveitis. Keratic precipitates are often a sign of feline infectious peritonitis (FIP), lymphoma, or systemic fungal infections. Hyphema frequently is associated with systemic hypertension, coagulopathies, and corneal perforations. Hypopyon is often seen with causes of anterior uveitis that lead to an outpouring of white blood cells, such as systemic fungal or bacterial infections and lymphoma.

Treatment for anterior uveitis should be aimed at both the underlying systemic ailment and the ophthalmic disease. Prednisolone acetate (1% suspension) or dexamethasone (0.1%, either suspension or ointment) administered every 2 to 12 hours depending on severity represent excellent choices for topical therapy. Hydrocortisone is relatively impotent and poorly absorbed when applied topically and is not a suitable antiinflammatory agent for treatment of anterior uveitis.

Topical nonsteroidal ophthalmic medications are also available; namely, flurbiprofen (0.03% solution) and diclofenac (0.1% solution). These are good alternatives to topical steroid preparations if corneal ulceration is present or systemic conditions prevent the use of steroids. Systemically administered steroidal and nonsteroidal drugs reach the anterior uvea and can be helpful adjuncts, especially in severe cases. Topical atropine (1% solution or ointment) may be indicated in the treatment of anterior uveitis to relieve the pain associated with iris sphincter and ciliary body muscle spasms and to prevent posterior synechia. However, it must be used with caution and intraocular pressure must be closely monitored during its use because it can exacerbate glaucoma, especially in dogs.

PUPIL ABNORMALITIES

Pupil abnormalities may or may not be associated with other ophthalmic disease. They are divided into four clinical presentations: anisocoria, miosis, mydriasis, and dyscoria.

Anisocoria

Anisocoria is defined as unequal size of the pupils. Although anisocoria is usually easy to detect, it can be challenging to ascertain which pupil is abnormal. Careful observation of pupil size in ambient light and dim illumination and pupil reaction under stimulation with a bright light source usually allow for this determination. Once it has been determined which is the affected pupil, the next step is to investigate the causes of miosis or mydriasis.

Miosis

Pupil size in mammals is the product of the balance between the parasympathetic tone of the iris sphincter muscle and the sympathetic tone of the iris dilator muscle. Therefore miosis is the result of stimulation of the iris sphincter, loss of sympathetic tone of the iris dilator, or both. Although miosis may be produced with topical medications, such as pilocarpine and latanoprost, there are two clinical conditions that cause miosis: anterior uveitis and Horner's syndrome. Fortunately, these two causes are easily distinguished from one another on the basis of associated ophthalmic signs.

In addition to miosis, signs often associated with anterior uveitis include blepharospasm, epiphora, episcleral injection, 360-degree corneal vascularization, corneal edema, aqueous flare, keratic precipitates, hypopyon, and hyphema. Anterior uveitis is the most common ophthalmic manifestation of systemic disease and is often present in critically ill patients. Common diseases that can cause anterior uveitis in cats and dogs are systemic infectious disease (fungal, bacterial, viral, rickettsial, and algal), primary or secondary neoplasia, blunt or penetrating trauma, and immune-mediated

FIGURE 154-4 Left eye of a cat with Horner's syndrome. Note the narrowed palpebral fissure due to drooping of the upper eyelid (ptosis), miosis, and third eyelid protrusion.

conditions. Treatment for anterior uveitis is covered earlier in the chapter in the section Anterior Chamber Abnormalities.

The signs associated with Horner's syndrome are secondary to compromise of the sympathetic innervation to the eye and consist of a triad of signs in cats and dogs: ptosis, miosis, and third eyelid protrusion secondary to enophthalmos (Figure 154-4). Although ptosis and third eyelid protrusion can mimic blepharospasm, the eyes of patients with Horner's syndrome are comfortable and noninflamed. Horner's syndrome is frequently idiopathic, but it can occur secondary to otitis interna or media, trauma or surgery to the side of the face or neck, or intracranial or thoracic neoplasia.[8,9] Pharmacologic testing can localize the lesion in Horner's syndrome and is described in detail elsewhere.[10,11] Treatment of Horner's syndrome is accomplished by identifying and addressing the underlying cause, if possible. No specific ophthalmic therapy is indicated.

Mydriasis

Mydriasis is due to stimulation of the iris dilator muscle or compromise of the parasympathetic tone of the iris sphincter muscle, or both. As with miosis, mydriasis can be pharmacologically induced with agents such as atropine. However, unlike miosis, mydriasis is associated with many conditions. Highly stressed patients, particularly cats, can have dilated pupils and poor to absent pupillary light responses (PLRs). Likewise, aged patients with iris atrophy may have mydriasis in one or both eyes. Optic nerve or end-stage retinal disease can also lead to mydriasis. For causes of these conditions, see the Blindness section later.

Mydriasis is a consistent sign of glaucoma, and intraocular pressure should be measured in all patients with dilated pupils. The most common cause of glaucoma in critically ill patients is anterior uveitis. Glaucoma is painful and blinding, and steps should be taken immediately to lower intraocular pressure. If the patient's systemic condition allows it, mannitol (20% to 25% solution) administered slowly intravenously (over the course of about 30 minutes) at a dose of 1 to 2 g/kg can produce a dramatic drop in intraocular pressure. Other effective glaucoma medications include methazolamide (5 mg/kg PO q12-24h), dorzolamide solution (q8-12h topically), and latanoprost solution (q12-24h topically).

Mydriasis is also a consistent finding in dysautonomia (Key-Gaskell syndrome), which is most frequently seen in cats,[12] although a similar syndrome has been reported in dogs.[13] In addition to mydriasis, signs associated with this condition include anorexia, depression, weight loss, dehydration, bradycardia, constipation, protrusion of both third eyelids, and decreased tear production. Pharmacologic testing to verify the diagnosis is described elsewhere.[14] Treatment for the ocular component of dysautonomia consists of topical lubrication.
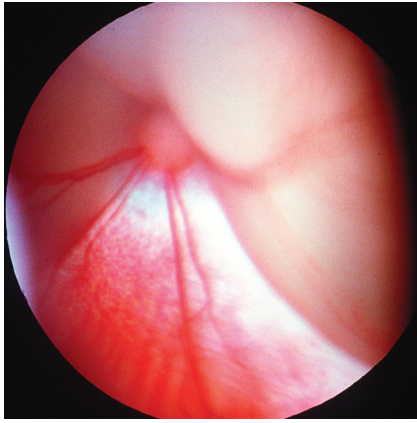
FIGURE 154-5 Exudative retinal separation in a cat.

FIGURE 154-6 Optic neuritis resulting from cryptococcosis in a cat. Note the fuzzy, indistinct borders of the optic disc and the peripapillary hemorrhages.

The unique parasympathetic innervation of the feline pupil can produce a variation in mydriasis, the D-shaped or reverse D-shaped pupil in which only one half of the pupil dilates. This defect is due to lesions involving the medial or lateral short ciliary nerve and is commonly associated with feline leukemia virus infection.

Dyscoria

Dyscoria is defined as an irregularly shaped pupil. It is most commonly secondary to posterior synechiae, a result of anterior uveitis.

BLINDNESS

Visual capability commonly is assessed by eliciting a menace response, observing the patient tracking a cotton ball dropped repeatedly within its visual range, or performing a maze test. Unfortunately, verification of visual status is often problematic for the critical care practitioner because many seeing patients appear to fail these routine tests due to either alterations in mentation or inability to ambulate.

Blindness can occur as a result of disease in one of five anatomic locations: (1) cornea, (2) lens, (3) retina, (4) optic nerve or tracts, and (5) brain. Blindness due to corneal changes is readily apparent, and causes of alterations in corneal transparency are covered elsewhere in this chapter. Causes of cataracts associated with serious systemic disease include diabetes mellitus and anterior uveitis. However, neither of these would likely lead to cataract formation over the period that a patient would be hospitalized in a critical care setting.

A number of systemic conditions can lead to retinal disease and vision compromise. PLRs are often present, even in advanced retinal disease. Therefore normal PLRs do not rule out retinal disease as a cause of vision impairment. Retinal conditions associated with vision compromise secondary to systemic disease are manifested most frequently as retinal separation, retinal hemorrhage, or retinal inflammatory cellular infiltrates. Retinal separations frequently are classified by the nature of the subretinal fluid: serous, hemorrhagic, or exudative (Figure 154-5). The type of fluid under the separation can provide clues as to the underlying cause.

Systemic conditions that may manifest with a serous retinal separation include systemic hypertension (early) and autoimmune disease, such as uveodermatologic syndrome in dogs. Typical causes of hemorrhagic retinal separation and intraretinal hemorrhage include systemic hypertension, rickettsial disease (Rocky Mountain spotted fever, ehrlichiosis), toxic coagulopathies due to rodenticides, vasculitis (FIP), immune-mediated hemolytic anemia, and hyperviscosity syndrome. Exudative retinal separation and intraretinal inflammatory cellular infiltrates are commonly an expression of systemic fungal disease, neoplasia (especially lymphoma), toxoplasmo-
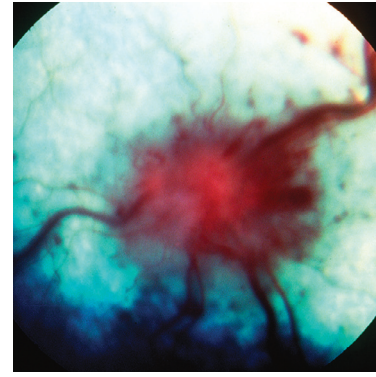
sis, viral diseases (canine distemper, feline leukemia virus infection, feline immunodeficiency virus infection, FIP), and protothecosis. Ivermectin toxicity may cause acute blindness in dogs.[15] In addition to blindness, these dogs may be brought to an emergency facility because of neurologic signs such as depression, hypersalivation, ataxia, and tremors.[16,17] A careful history taking often reveals either exposure to horses being wormed with ivermectin or current treatment with ivermectin for *Demodex* infection. Examination of the fundus is either unremarkable or demonstrates focal "cotton wool" areas of retinal edema near the optic disc. Affected dogs usually have a return to vision over the course of a week. Recently, intravenous administration of lipid emulsion has been shown to rapidly reverse the effects of ivermectin toxicity.[18,19]

In cats, acute blindness has been associated with systemic enrofloxacin therapy at dosages as low as 4 mg/kg PO q12h.[20] This is manifested by diffuse tapetal hyperreflectivity and retinal vascular attenuation without signs of retinal separation or retinal cellular infiltration.

The extent of vision impairment with all of these conditions depends on the extent of retinal involvement. For virtually all of these conditions, there is no specific treatment of the ocular component beyond addressing the underlying systemic cause.

Optic nerve disease can lead to blindness and may be associated with a number of systemic illnesses. Unlike retinal causes of vision impairment, blindness due to optic nerve disease usually is accompanied by loss of PLRs and mydriasis. Optic nerve disease may or may not be manifested by changes in the appearance of the optic disc. When present, ophthalmoscopic signs of optic nerve disease include optic disc swelling, fuzzy and indistinct disc borders, and hemorrhages on the disc or in the peripapillary area (Figure 154-6).

Systemic diseases with the potential for optic nerve involvement include granulomatous meningoencephalitis, canine distemper, lymphoma, systemic fungal infection (especially cryptococcosis), meningioma, and hyperviscosity syndrome. Treatment is aimed at the underlying systemic cause.

Blindness secondary to involvement of the visual center in the occipital cortex is rare in cats and dogs. Affected animals usually have marked neurologic deficits.

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