Redness of the eye is a warning signal common to many diseases. Minor conjunctival inflammation caused by superficial infections, inadequate tear production, and allergies can mimic more serious eye diseases such as uveitis and glaucoma, especially when only a cursory ocular examination is performed. How can one be sure not to overlook these more serious, potentially blinding diseases? Assume the worst if your patient demonstrates one or more of these eight criteria: vision loss, pain, ocular opacity, pupillary irregularities, redness concentrated at the limbus, a decrease in intraocular pressure, a history of previous intraocular inflammation, or failure to respond to therapy.

Examination
Examination of the eye should be performed with proper equipment in a systematic manner. Remember that the diffuse light omitted by most direct ophthalmoscopes and disposable penlights provides inadequate penetration and illumination in the presence of opaque ocular media. Because choroidal inflammation may precede anterior uveal involvement in diseases such as the systemic mycoses, assessment of the posterior globe is critical in both eyes. The choroid and retina in an inflamed or cloudy eye are better visualized using an indirect ophthalmoscopic technique, as with the Welch-Allyn PanOptic. Tonometry is indicated in any patient with a red, painful eye and to objectively assess response to therapy in patients with uveitis. Normal IOP ranges from 8 to 18 mmHg, with no more than 5 mmHg difference between the two eyes. A thorough physical examination should be performed on all patients in which uveitis is diagnosed, especially if the uveitis is bilateral. Careful assessment of the major organ systems may reveal abnormalities related to the observed ocular signs, leading to additional clinicopathologic and/or radiographic studies. Serology may be dictated by preliminary laboratory data or as part of a routine screen for regionally endemic diseases.

Clinical Signs
By far the most important abnormality of the canine and feline anterior uvea is inflammatory disease, i.e., anterior uveitis or iridocyclitis. Early recognition and treatment are imperative, as the structural and functional abnormalities that accompany uveitis may result in blindness. More significantly, uveitis may be a component of systemic disease that could ultimately compromise the patient’s general health.

Although the most sensitive indicators of intraocular inflammation are the presence of aqueous flare and a decrease in intraocular pressure, clinical signs of uveitis can include nonspecific indicators of pain such as tearing, blepharospasm, enophthalmos, and even lethargy. Hyperemia results from vasodilation of conjunctival and episcleral vessels. Ciliary flush refers to a rose-red coloration around the limbus that reflects the dilation and congestion of the uveal vasculature. Corneal opacification results from edema produced when toxins, inflammatory mediators, and/or debris damage the endothelium. Vessels may invade the deeper corneal layers while inflammatory cells often settle on the inner surface (keratic precipitates, or KPs). Aqueous turbidity results from breakdown of the blood-aqueous barrier, increasing the protein within the aqueous (flare) or allowing inflammatory cells and fibrin to enter the eye. The iris appears swollen and dull in color due to edema and cellular infiltrates. The pupil is classically constricted and sluggish in its reactions due to tissue edema and stimulation of the iridal sphincter by inflammatory mediators. Abnormal pupillary shape may result from adhesions of iris to lens known as posterior synechiae.

Decreased intraocular pressure is due to ciliary body dysfunction and reduced aqueous production.

Changes in the eye associated with chronic or recurrent anterior uveitis include those previously mentioned, as well as permanent pigmentary changes in the iris; cataracts secondary to synechiae or toxic influences on the lens epithelium; lens luxations due to degeneration of the ciliary processes and zonules; glaucoma, arising from impaired aqueous flow through the pupil due to posterior synechiae or impaired outflow through the iridocorneal angle due to peripheral anterior synechiae; and phthisis bulbi—a shrunken, hypotonic, nonfunctional globe.

 Conjunctival hyperemia and corneal edema are typically more subtle in cats when compared to dogs with anterior uveitis. This may explain why cats are presented later in the course of their intraocular inflammation, often when secondary complications such as glaucoma have developed. The more lightly colored feline iris demonstrates vascular congestion and cellular infiltration more readily than the darkly pigmented canine iris. The shape of the feline pupil may also reduce the likelihood of 360-degree iris-to-lens adhesions that would otherwise trap aqueous behind the iris (iris bombe).
Etiology

Primary Ocular Disease
Blunt contusion, perforating injury, and corneal ulceration represent the more common traumatic events leading to anterior uveitis. Wounds affecting pericocular sites may support a traumatic etiology if history is incomplete. Blunt trauma is often associated with generalized corneal edema and hyphema. Perforating injury causes release of prostaglandins that mediate blood-aqueous barrier breakdown and miosis. Traumatic perforation or rupture of the lens capsule results in an immunologic response to lens protein. Corneal ulceration causes pupillary constriction and inflammation through a trigeminally mediated axonal. Secondary bacterial infection of corneal ulcers potentiates uveitis by chemotaxis of inflammatory cells and elaboration of toxins and inflammatory mediators into the anterior chamber.

Primary ocular neoplasms are relatively uncommon in dogs and cats. The differential diagnosis should include inflammatory infiltrates, uveal cysts, extraocular neoplasms with intraocular extension, and perforating wounds of the globe with uveal prolapse. Uveal melanomas are the most common primary uveal tumor in both dogs and cats, although clinical signs and prognosis differ markedly between the two species. Nodular growth is more typical of canine uveal melanoma, while diffuse infiltration and iris color change characterize that of the cat. The rate of metastasis from the canine eye is approximately 4%, while that in cats is 63%. Other primary canine tumors include ciliary body adenoma and adenocarcinoma, medulloepithelioma, hemangiosarcoma, and leiomyosarcoma.

Posttraumatic sarcoma has been documented in cats with a history of perforating ocular injury. Lens-induced uveitis results when insidious or sudden leakage of lens material breaches the level of “immunotolerance” of uveal tissues to lens material. Dogs are more likely to develop lens-induced uveitis due to the frequency with which rapidly developing or hypermature cataracts are seen when compared to cats. The reaction may occur in either species following traumatic rupture of the lens capsule with liberation of large quantities of lens protein.

Idiopathic disease may ultimately be diagnosed if an etiology cannot be documented. Please consider this category as one of exclusion, first ruling out the other possibilities.

Secondary Ocular Disease
Uveitis is often a clinical sign of a more widespread disease process. The uvea’s vascular component provides ample opportunity for hematogenous spread of infectious, immunologic, and neoplastic diseases. The globe has no lymphatic drainage, and the uvea subserves this function by acting as an accessory lymph node. Virtually any antigen that has potential to incite an immune response has the potential to cause uveitis.

Metabolic Disease: The rapid cataract development accompanying diabetes mellitus often leads to lens-induced uveitis as protein leaks through microscopic breaks in the expanding lens capsule. A striking consequence of blood-aqueous barrier breakdown in the diabetic is an influx of lipid into the aqueous. The dense gray-white opacity of the aqueous often obscures intraocular detail and may be misinterpreted as corneal edema.

Infectious Disease: A growing number of infectious agents have been implicated in the etiology of anterior uveitis in dogs and cats. Direct replication or migration of organisms, stimulation of immune-mediated inflammation, and development of secondary opportunistic infections are possible disease mechanisms. The list of differential considerations often varies according to geographic locale. As a general rule, anterior uveal signs predominate with viral-induced inflammation. Myotic infections are more often characterized by posterior segment inflammation, with anterior uveitis developing in the later stages of the infection. Bacterial, rickettsial, and protozoal infections often demonstrate anterior and posterior uveitis concurrently. The following summarizes selected infectious agents documented or suspected to cause uveitis in the dog and cat.

A history of intermittent or relapsing uveitis characterizes Brucella canis infection. Hypopyon is a common ocular feature. Concurrent clinical signs may include testicular enlargement, scrotal dermatitis, and diskospondylitis. Prognosis for effective therapy is guarded owing to the intracellular nature of the organism. Other bacterial diseases implicated in canine uveitis include borreliosis and leptospirosis. Bartonella spp. has been implicated in feline uveitis. Any bacteremia/septicemia has the potential to cause anterior uveitis. Endocarditis, pyometra, dental disease, and deep pyoderma have all be associated with intraocular inflammation.
Ocular disease associated with canine adenovirus usually develops unilaterally from 1 to 3 weeks following infection. Postvaccinal uveitis is rarely seen with contemporary CAV-2 vaccines, but occasional reactions still occur. Clinically the uveitis is accompanied by secondary corneal edema and the classical “blue eye.”

Although a classical retrospective study concluded that cats infected with feline leukemia virus have a twofold greater risk of developing eye disease than noninfected animals, the study also stated that feline leukemia virus (FeLV) appears to cause little in the way of ocular disease with the exception of its role in lymphosarcoma. Cats with ocular lymphosarcoma initially present with mild uveitis and/or subtle iridal masses. As the disease progresses, infiltrating tumor cells distort the iris and may compromise aqueous outflow by invading the iridocorneal angle.

Approximately 40% of cats with feline infectious peritonitis (FIP) will demonstrate ocular signs. Ophthalmic lesions are more likely to occur in the nonneffusive form of the disease. The uveitis is classically exudative owing to an underlying vasculitis, with large keratitis precipitants and a fibrinous exudate in the anterior chamber of young cats.

Feline immunodeficiency virus (FIV) has been linked with chronic anterior uveitis, especially in cats over 5 years of age. Inflammatory cells in the anterior vitreous (i.e., pars planitis) occur with some frequency in affected cats. In a study of 54 clinically ill cats infected with FIV, 19 had ocular disease and 76% of these were also positive for *Toxoplasma gondii*. Long-term use of topical and/or oral corticosteroids may be necessary to control the uveitis, with limited improvement in patients with pars planitis.

Inflammatory disease caused by *Toxoplasma gondii* can affect both the anterior and posterior uvea. A low-grade anterior uveitis may be present alone or in combination with small multifocal retinal granulomas or optic neuritis. Ocular lesions are more commonly seen in cats than dogs. In a histologic study of 100 cats with confirmed toxoplasmosis, 22 had ocular involvement. Definitive diagnosis is often complicated by the absence of systemic disease. Laboratory assessment of both *T. gondii*-specific IgM and IgG are recommended, as the IgM will rise and fall for 3 to 4 months after infection, whereas the IgG class may rise more slowly and remain elevated for years after exposure.

The most common presentation of Ehrlichia-associated ocular disease is a low-grade inflammatory process characterized by fine keratic precipitates, progressive secondary corneal edema, and subtle iridocyclitis. Acutely infected dogs may demonstrate retinal vessel engorgement and perivascular edema. *Ehrlichia canis* has recently been implicated etiologically in 3 cats with fever and polyarthritis. None of the cats demonstrated anterior uveitis, but one presented with bilateral retinal detachments and subretinal fluid suggestive of choroiditis. In dogs, the acute stages of ehrlichiosis and Rocky Mountain Spotted Fever (RMSF) are difficult to differentiate on the basis of ocular and clinical signs. Anterior uveitis is generally mild in RMSF, with or without subconjunctival and intraocular hemorrhage. The ocular prognosis is more favorable with RMSF than with ehrlichiosis.

Systemic fungal infections are less common in the cat than the dog. Fungal organisms generally enter the body through the respiratory tract and spread hematogenously to the eye, affecting the choroid before the anterior uveal tract. In endemic areas, dogs with progressive/nonresponsive anterior uveitis, vision loss associated with marked vitreous opacification, or those with pronounced scleral inflammation should be suspected of blastomycosis. *Cryptococcus neoformans* is the most commonly reported feline mycotic infection. Chorioretinitis, anterior uveitis, and optic neuritis have been reported in both dogs and cats. Similar lesions are reported in *Histoplasma capsulatum* infections. The posterior segment lesions of histoplasmosis are often characterized by areas of abnormal pigment proliferation within the tapetal fundus. Retinal detachments and uveitis are also described in *Coccidioides immitis* infections and in disseminated aspergillosis.

**Immunologically Mediated Disease:** Canine uveodermatologic (VKH-like) syndrome is characterized by immune-mediated destruction of uveal and dermal melanocytes. Susceptible animals include the Akita, the Samoyed, and the Siberian husky. Bilateral panuveitis is accompanied by mucocutaneous depigmentation and whitening of the haircoat. Long-term therapy is necessary to control disease recurrences and prevent sight-limiting postinflammatory sequelae. Prognosis for vision is poor due to secondary cataract formation, retinal degeneration, and glaucoma.

**Neoplasia:** Intraocular bleeding of undetermined cause should always alert the clinician to a possible neoplasm. Lymphosarcoma is the most common metastatic tumor of the eyes of dogs and cats. Unilateral or bilateral uveitis with hyphema in dogs greater than 4 years of age may be the first clinical suggestion of lymphosarcoma. Cats usually demonstrate aqueous flare, iris swelling, changes in iris color, and iris vascularization. Hypopyon may also
be a presenting sign in either species. The ocular disease is treated symptomatically, with its outcome dependent upon overall disease remission. Other reported metastatic neoplasms include transmissible venereal tumor, hemangiosarcoma, squamous cell carcinoma, fibrosarcoma, and adenocarcinoma.

**Treatment**
As a general rule, therapy should be initially aggressive, tapering frequency of administration as clinical signs subside. Anti-inflammatory therapy should be continued for at least 2 weeks beyond resolution of clinical signs. Resolution of inflammation should be determined ideally by objective data such as return to normal IOP measured by tonometry. Recurrences of inflammation following cessation of treatment suggest the need for prolonged if not indefinite maintenance therapy. Such long-term therapy is commonly required to successfully manage feline uveitis and prevent secondary complications such as glaucoma in that species.

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**Therapeutic Plan for Anterior Uveitis**

**Initial Topical Therapy**

1% prednisolone acetate q 2–4 hours
1% atropine to effect

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Reevaluate after 24 hours

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**If No Improvement**

Recheck for corneal ulceration
Continue initial regimen
Add topical flurbiprofen q 8 hrs
Consider subconjunctival steroids
Reevaluate in 24 hours

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**If Improved**

Reduce topical frequency to q 4–6 hrs
Reevaluate in 72 hrs

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**If No Improvement**

Reevaluate diagnosis
Continue above regimen
Consider phenylephrine for mydriasis
Consider outside consultation / referral

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**If Improved**

1% prednisolone acetate q 6 hrs
Reduce or discontinue atropine
Continue 10–14 days past resolution of clinical signs

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References available upon request.