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Chapter 11

UVEA

Paul E. Miller

The uvea plays an important role in ocular physiology, and disorders of this tissue are common in veterinary practice. The iris controls the amount of light entering the eye, and the ciliary body alters the focal power of the lens, produces aqueous humor that supplies nutrition to ocular structures, and aids in regulating intraocular pressure (IOP). Together they also form a blood-aqueous barrier so as to maintain the clarity of the aqueous humor and vitreous. The choroid plays a major role in providing nutrition to the retina. Because of these diverse roles, uveal disorders are frequently associated with alterations in vision and IOP.

ANATOMY AND PHYSIOLOGY

The eye consists of the following basic layers (Figure 11-1):

- Fibrous (outer) layer—the sclera and cornea
- Vascular (middle) layer—the uvea, or uveal tract
- Neuroectodermal inner layer—the retina and optic nerve

The *uveal tract* has three parts: the *iris* and the *ciliary body,* which together form the *anterior uvea,* and the *choroid,* which is also known as the *posterior uvea.*

Iris

The *iris* controls the amount of light entering the eye by varying the size of the pupil. Reduction in the size of the pupil also increases the depth of field for near objects and reduces certain optical aberrations. To accomplish this goal, the iris has two sets of muscles:

- *Musculus constrictor pupillae:* A circular band of muscle fibers concentric with the pupil. These fibers have predominantly *parasympathetic* innervation (Figure 11-2).
- *Musculus dilator pupillae:* Radially oriented fibers passing from near the root of the iris toward the pupillary margin. These fibers have predominantly *sympathetic* innervation.

Viewed from the anterior surface, the iris has two zones, the *pupillary zone* (Figures 11-3 and 11-4) and the *ciliary zone.* A variable thickening of the iris at the junction of these two zones is called the *collarette.* The anterior surface of the iris is covered by a modified layer of stromal cells, the *anterior border layer* (Figure 11-5). The remaining parts of the iris are the *stroma* and *sphincter muscle,* the *anterior epithelium* and *dilator muscle,* and the *posterior pigmented epithelium* and *pigment ruff.* The posterior pigmented epithelium is continuous with the nonpigmented epithelium covering the ciliary body and eventually with the retina.

The bulk of the iris is stroma, which consists of fibrous connective tissue with bundles of collagen, pigmented and nonpigmented cells, and blood vessels in a mucopolysaccharide matrix. Variations in iris color are due to variations in pigmentation of the stroma and posterior pigmented epithelium and in the arrangement of the anterior border layer (Figure 11-6).

The *temporal and nasal long ciliary arteries* enter the iris near its root (see Figure 11-3) and form the major arterial circle, which may be incomplete. The vascular supply of the iris of domestic animals greatly exceeds that of the human iris. Therefore surgical procedures near the iris root in animals often result in profuse hemorrhage if the major arterial circle is transected.

The dilator pupillae muscle extends as a continuous sheet in front of the anterior epithelium (see Figure 11-4) and is intimately related with it. The constrictor pupillae muscle is a flat ring of smooth muscle surrounding the pupil in the posterior iris stroma (see Figure 11-5).

In horses, cattle, sheep, and goats, which have a horizontally elliptical pupil, black masses suspended from the superior and occasionally the inferior rim of the pupil are termed *corpora nigra* (e.g., in horses) or *granula iridica* (e.g., in ruminants). These masses aid in further control of light entering the pupil and should not be mistaken for tumors or cysts.

Ciliary Body

The *ciliary body* lies immediately posterior to the iris. On its posterior surface the ciliary body has numerous folds known as the *ciliary processes* (Figures 11-7 and 11-8). This area of the ciliary body, termed the *pars plicata* (folded part), merges posteriorly into a flat area *(pars plana),* which joins the retina. The *zonular fibers,* which support the lens, originate from the pars plana and between the ciliary processes (Figures 11-9 and 11-10).

Viewed in section, the ciliary body is triangular, with one side joining the sclera, one side facing the vitreous body, and the base giving rise to the iris and *iridocorneal angle* (Figure 11-11). The ciliary body is covered with two layers of epithelium, the inner layer of which is nonpigmented and the outer layer of which is pigmented. It is continuous with similar epithelium on the posterior surface of the iris and the pigment epithelium of the retina (Figure 11-12). The smooth muscle fibers of the *ciliary muscle* (parasympathetic innervation) together with blood

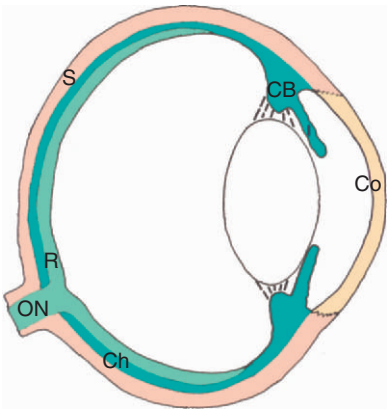

Figure 11-1. The three layers of the eye. *CB,* Ciliary body; *Ch,* choroid; *Co,* cornea; *ON,* optic nerve; *R,* retina; *S,* sclera. (Modified from Fine BS, Yanoff M [1972]: Ocular Histology. Harper & Row, New York.)

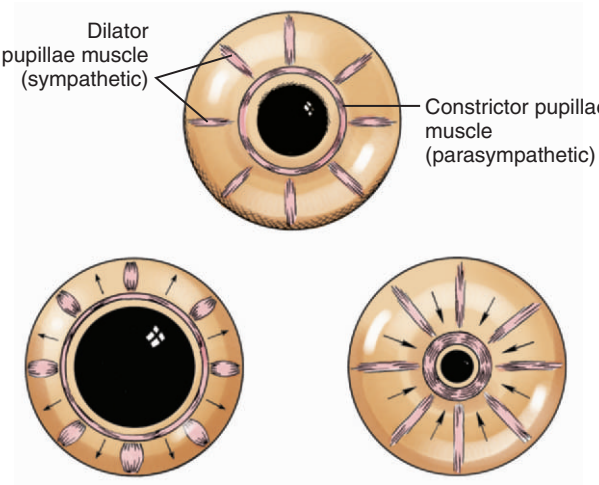

Figure 11-2. Control of pupil size. The arrangement of the constrictor fibers varies among domestic species, but the principles are similar.

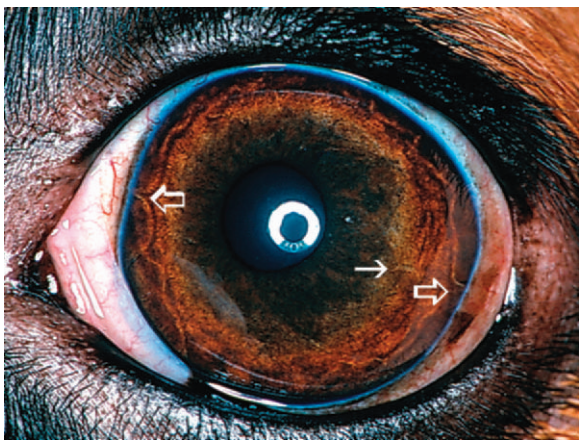

Figure 11-3. Clinical anatomy of the iris. The pupillary zone of the iris is typically darker than the surrounding, lighter-colored ciliary zone. The junction between the two zones is termed the iris collarette *(solid arrow)*. Persistent pupillary membranes, if present, typically originate at the iris collarette region. The sinuous posterior ciliary artery enters the iris near the limbus at the 3 and 9 o'clock position *(open arrows)*. From there it divides into superior and inferior branches to form the major vascular circle of the iris.

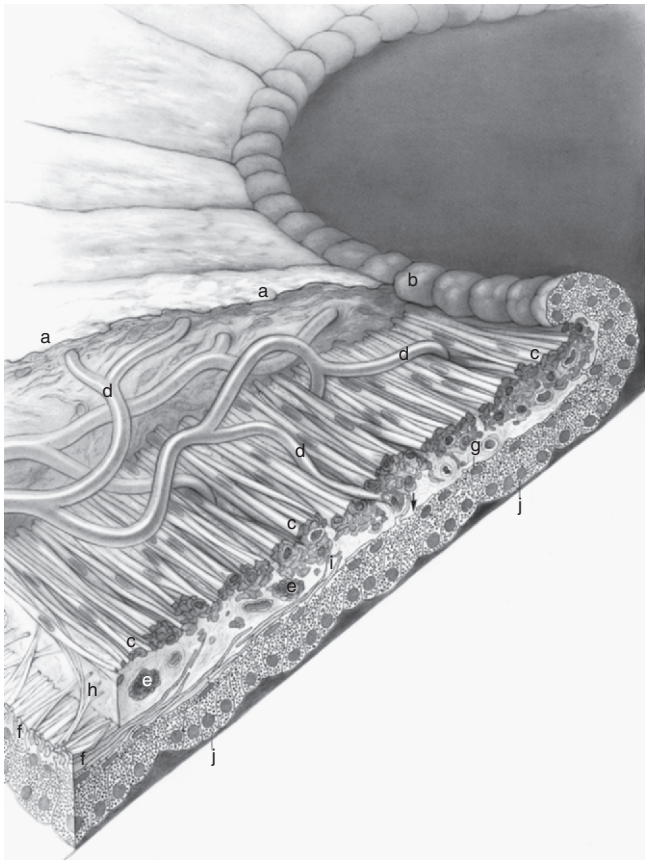

Figure 11-4. Pupillary portion of the iris. The dense, cellular anterior border layer *(a)* terminates at the pigment ruff *(b)* in the pupillary margin. The sphincter muscle is at *(C)*. The arcades *(d)* from the minor circle extend toward the pupil and through the sphincter muscle. The sphincter muscle and the iris epithelium are close to each other at the pupillary margin. Capillaries, nerves, melanocytes, and clump cells *(e)* are found within and around the muscle. The three to five layers of dilator muscle *(f)* gradually diminish in number until they terminate behind the midportion of the sphincter muscle *(arrow),* leaving low, cuboidal epithelial cells *(g)* to form the anterior epithelium to the pupillary margin. Spurlike extensions from the dilator muscle form Michel's spur *(h)* and Fuchs's spur *(i)* (these spurs are not commonly described in domestic animals). The posterior epithelium *(j)* is formed by columnar cells with basal nuclei. Its apical surface is contiguous with the apical surface of the anterior epithelium. (From Hogan MJ, et al. [1971]: Histology of the Human Eye. Saunders, Philadelphia.)

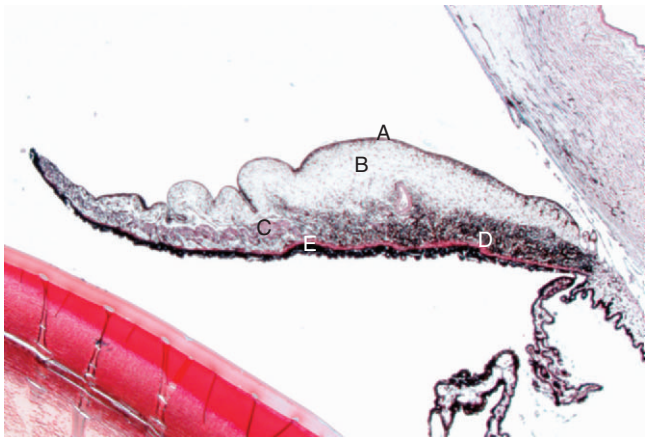

Figure 11-5. Structure of the iris. *A,* Anterior border layer; *B,* stroma; *C,* constrictor muscle; *D,* dilator muscle; *E,* posterior epithelium. (Courtesy Dr. Richard R. Dubielzig.)

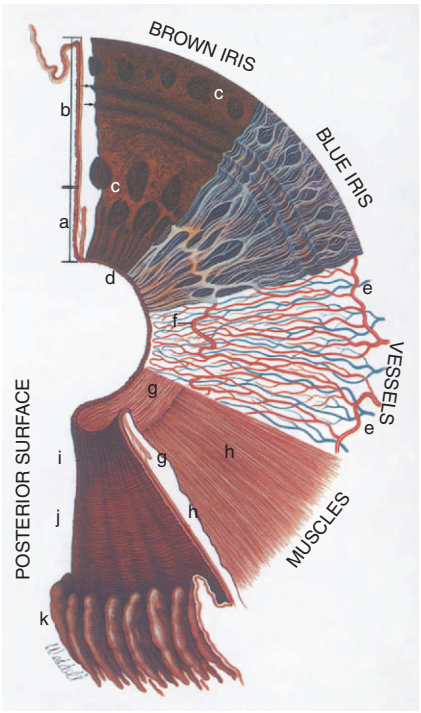

Figure 11-6. Surfaces and layers of the iris. Clockwise from the top the iris cross-section shows the pupillary *(a)* and ciliary *(b)* portions, and the surface view shows a brown iris with its dense, matted anterior border layer. The blue iris surface shows a less dense anterior border layer and more prominent trabeculae. Arrows indicate circular contraction furrows. *c,* Fuchs's crypts; *d,* pigment ruff; *e,* major arterial circle. Radial branches of arteries and veins extend toward the pupillary region. The arteries form the incomplete minor arterial circle (*f*), from which branches extend toward the pupil, forming capillary arcades. (*Note:* The incomplete minor arterial circle is variable or absent in many animals.) *g,* Circular arrangement of the sphincter muscle; *h,* radial processes of the dilator muscle; *i,* radial contraction furrows; *j,* structure folds of Schwalbe; *k,* pars plicata of the ciliary body. (Modified from Hogan MJ, et al. [1971]: Histology of the Human Eye. Saunders, Philadelphia.)

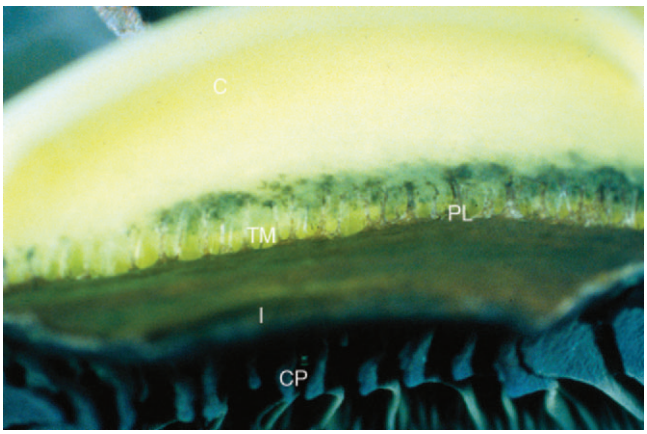

Figure 11-7. Dissecting microscope view of the relationship between the iris, ciliary body, and iridocorneal angle. *C,* Endothelial surface of the cornea; *CP,* ciliary processes; *I,* iris at pupil margin; *PL,* pectinate ligament; *TM,* trabecular meshwork. (Courtesy Dr. Mitzi Zarfoss.)

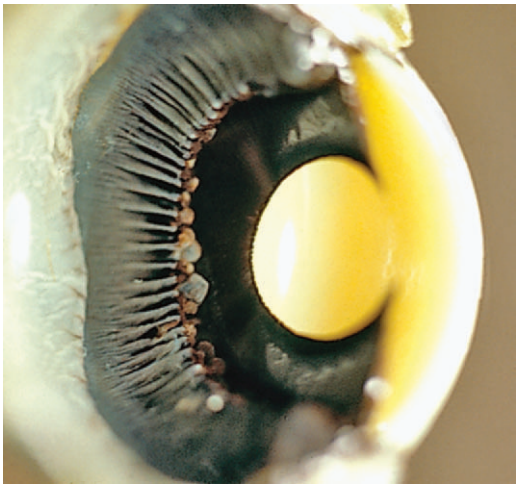

Figure 11-8. Posterior aspect of the canine iris and ciliary body (with the lens removed) showing the arrangement of the numerous bladelike ciliary processes. In this golden retriever multiple small ciliary cysts are also present at the tips of these processes. (Courtesy Dr. Richard R. Dubielzig.)

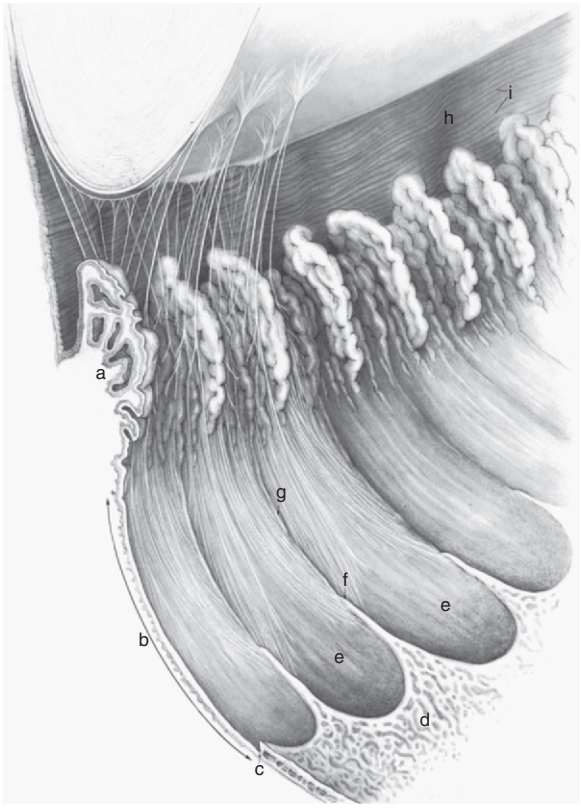

Figure 11-9. Posterior aspect of the ciliary body, showing pars plicata *(a)* and pars plana *(b)*. The junction between ciliary body and retina is at *c,* and the retina at *d.* In primates this junction is scalloped with bays *(e),* dentate processes *(f),* and striae *(g)* (ora serrata), but in most domestic species it is a straight line (ora ciliaris retinae). (From Hogan MJ, et al. [1971]: Histology of the Human Eye. Saunders, Philadelphia.)

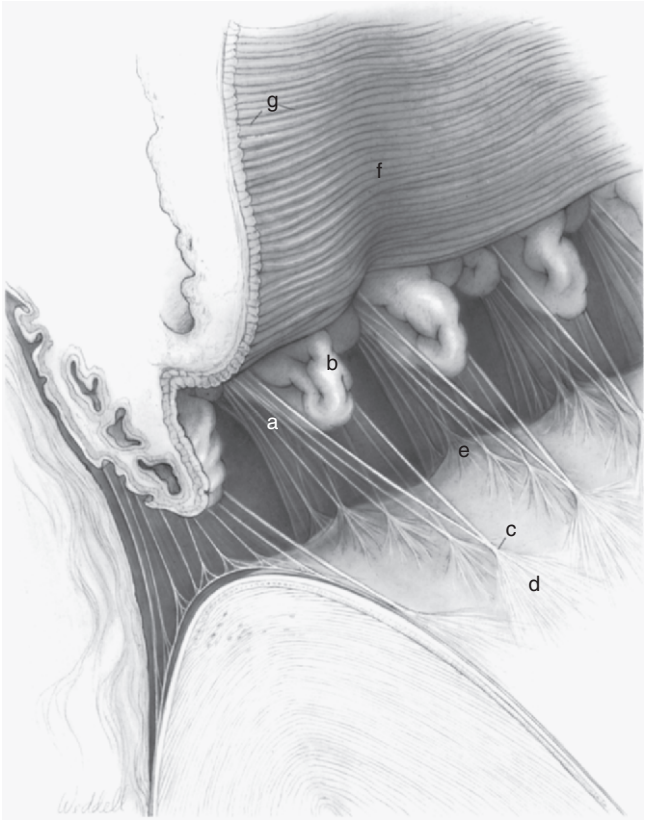

Figure 11-10. Anterior view of ciliary processes showing zonules attached to the lens: *a,* lens zonules; *b,* ciliary process; *c, d,* and *e,* attachment of zonules to lens capsule; *f,* radial folds in iris; *g,* circular folds in iris. The precise arrangement of the lens zonules with the lens capsule varies considerably among species. (From Hogan MJ, et al. [1971]: Histology of the Human Eye. Saunders, Philadelphia.)

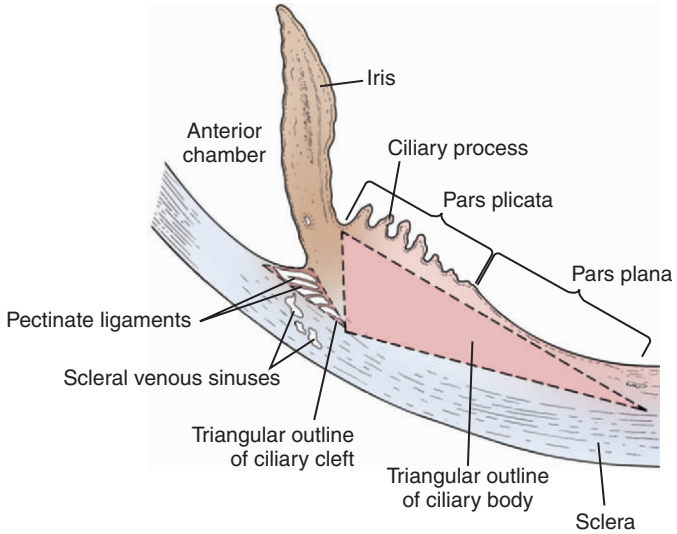

Figure 11-11. Parts of the ciliary body.

vessels, connective tissue, and nerves occupy a large portion of the ciliary body (Figure 11-13). The muscle fibers originate near the apex of the triangle and insert into the region of the ciliary cleft and trabecular spaces of the iridocorneal angle. *Contraction* of the ciliary muscle causes the following:

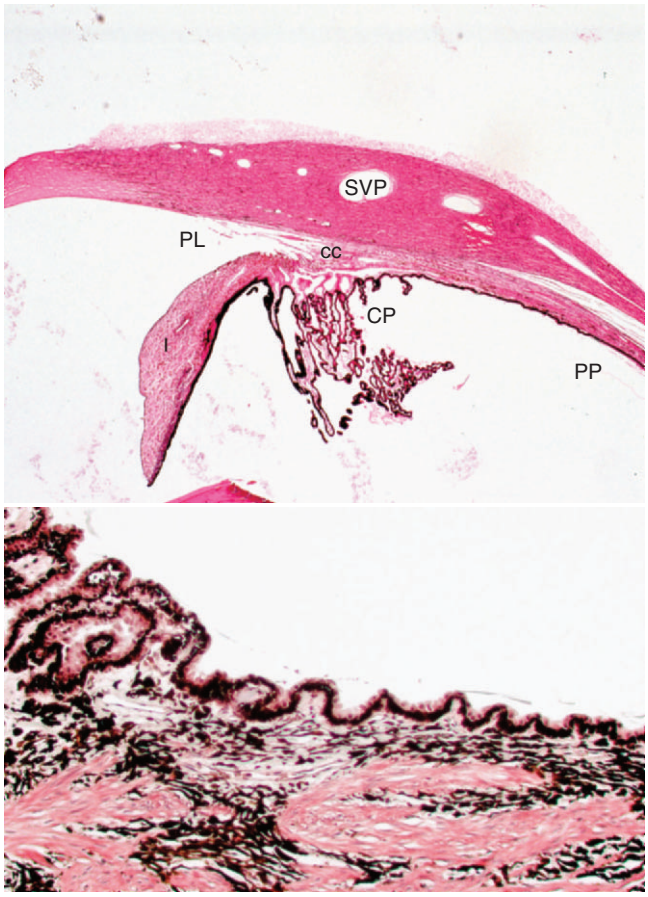

Figure 11-12. A, Normal ciliary body of a cat: *CC,* region of the ciliary cleft; *CP,* ciliary processes; *I,* iris; *PL,* pectinate ligament, *PP,* pars plana; *SVP,* scleral venous plexus. **B,** The ciliary body epithelium is bilayered, with the innermost layer being nonpigmented and the outer layer containing pigment. (Courtesy Dr. Richard R. Dubielzig.)

- Relaxation of lens zonules and change in shape or position of the lens to allow for near vision
- Increased drainage of aqueous via the trabecular meshwork

Inflammation of the ciliary body often leads to spasm of the ciliary muscle, which in turn causes ocular pain. Pain relief may be achieved by use of a *cycloplegic* drug (e.g., atropine), which relaxes the ciliary body. Although drugs that dilate the pupil (mydriatics) may also relax the ciliary muscle (atropine), not all do so (e.g., epinephrine).

Choroid

The *choroid* is a thin, variably pigmented, vascular tissue forming the posterior uvea. It joins the ciliary body anteriorly and lies between the retina and sclera posteriorly. The choroid is extremely vascular, with its capillaries arranged in a single layer on the inner surface to nourish the outer retinal layers (Figure 11-14). In species with limited retinal vasculature (e.g., horse, rabbit, guinea pig) the retina depends to a large extent on the choroidal blood supply. The choroidal stroma typically contains numerous melanocytes, which form a dark optical background to the retina. In most domestic mammals except the pig, a reflective layer—the *tapetum lucidum*—lies within the inner capillary layer. In large animals the tapetum is penetrated by

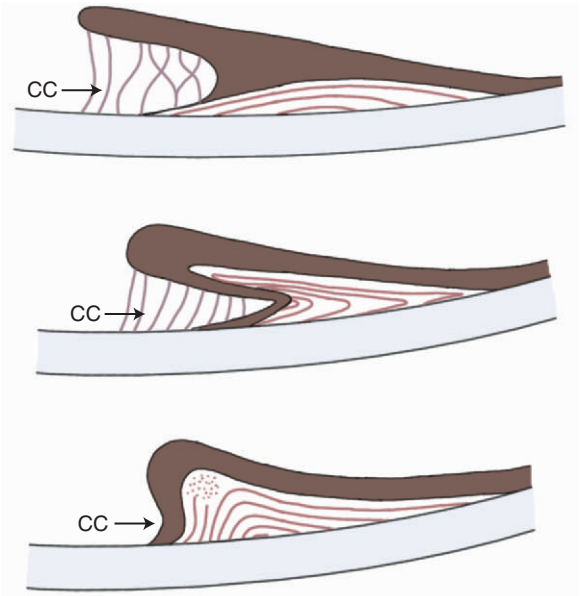

Figure 11-13. Degree of development of the ciliary body musculature among mammalian iridocorneal angles in the ungulate *(top),* carnivore *(middle),* and ape *(bottom).* Development is most pronounced in primates (ape) and least pronounced in herbivorous species (ungulate), with carnivore development between. The size of the iridocorneal angle and its ciliioscleral cleft or sinus *(CC)* is inversely large or most pronounced in the ungulate. (Modified from Samuelson DA [1999]: Ophthalmic anatomy, in Gelatt KN [editor]: Veterinary Ophthalmology, 3rd ed. Lippincott Williams & Wilkins, Philadelphia, p. 77; which was drawn after Duke-Elder S [1958]: System of Ophthalmology, Vol 1: The Eye in Evolution. Henry Kimpton, London.)

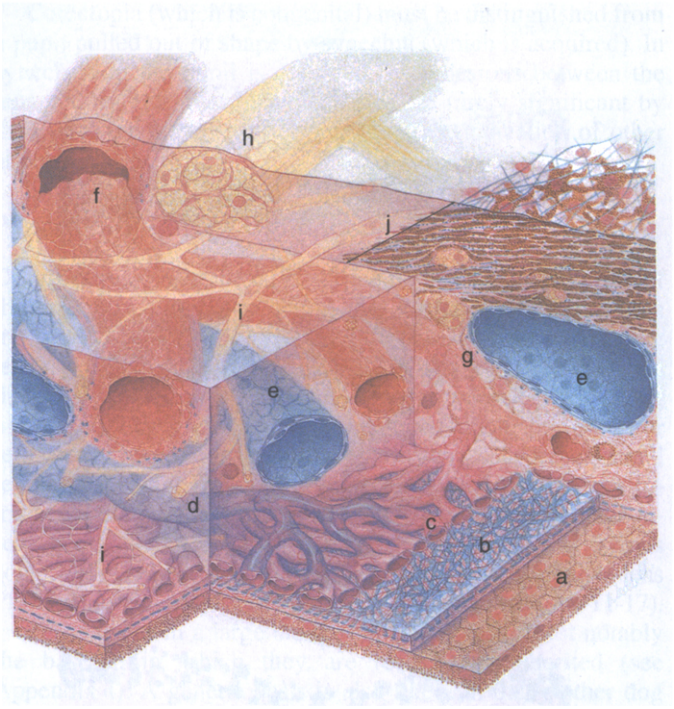

Figure 11-14. Choroidal blood supply and innervation, and Bruch's membrane. The retina is located at the bottom and the sclera at the top of the drawing. The retinal pigment epithelium *(a)* is in close contact with Bruch's membrane *(b).* The choriocapillaris *(c)* forms an intricate network along the inner choroid. Bruch's membrane is very thin in some domestic species. In the superior fundus the tapetum lies between the branching vessels in the choroid and the single layer of the choriocapillaris under the retina. Venules *(d)* leave the choriocapillaris to join the vortex system *(e).* The short ciliary artery is shown at *f,* before its branching *(g)* to form the choriocapillaris. A short ciliary nerve enters the choroid at *h* and branches into the choroidal stroma *(i). j,* Superchoroidea. (Modified from Hogan MJ, et al. [1971]: Histology of the Human Eye. Saunders, Philadelphia.)

numerous small capillaries, which appear as small focal dark spots (the *stars of Winslow*) when viewed end-on with the ophthalmoscope. The arteries and nerves to the anterior parts of the eye pass forward through the choroid. The choroid receives its main arterial supply from the following vessels:

- *Short posterior ciliary arteries,* which penetrate the sclera around the optic nerve
- *Long posterior ciliary arteries,* which enter near the optic nerve and branch near the ora ciliaris retinae and lead back into the choroid
- *Anterior ciliary arteries,* which send branches back into the choroid after penetrating the anterior sclera

Histologically the choroid consists of the following layers (see Figure 11-14):

- *Suprachoroidea*: avascular, pigmented connective tissue lying adjacent to the sclera
- Large-vessel layer: typically also contains numerous melanocytes
- Intermediate-vessel layer: also contains the tapetum in the superior fundus
- *Choriocapillaris*: a layer of capillaries adjacent to Bruch's membrane and the retina

In herbivores the tapetum is fibrous in nature *(tapetum fibrosum),* whereas in carnivores the tapetum is cellular and composed of reflective crystals *(tapetum cellulosum)* (Figure 11-15). The reflective properties of the tapetum, and not the presence of pigments, causes the distinctive color of the fundi of different animals and is the reason an animal's eyes "shine" in the dark. This color varies with thickness of the tapetum, breed, age, and species. Reflecting light through the retina a second time improves the animal's ability to function in dim light.

Blood-Ocular Barrier

The uveal tract plays a key role in maintaining the blood-ocular barrier (Figure 11-16). Diseases involving the uveal tract frequently cause a breakdown of this barrier, which leads to exudation of excessive amounts of proteins or cells into the aqueous humor, vitreous, or subretinal space. The blood-ocular barrier is composed primarily of a blood-retinal barrier and a blood-aqueous barrier. The blood-retinal barrier is formed at the level of the retinal capillary vascular endothelium, which is nonfenestrated and has tight junctions, and the retinal pigment epithelium, which also has tight junctions and separates the relatively leaky choroidal blood vessels from the overlying retina. The blood-aqueous barrier is formed by tight junctions at the level of the nonfenestrated iridal vascular endothelium and between cells constituting the nonpigmented ciliary body epithelium. Most large molecules, especially proteins, are unable to pass through or between the cells in this barrier system. The exact anatomic location of the barrier is probably different for different substances (e.g., capillary endothelial cells, endothelial basement membrane, and intercellular junctions). By limiting the amount of

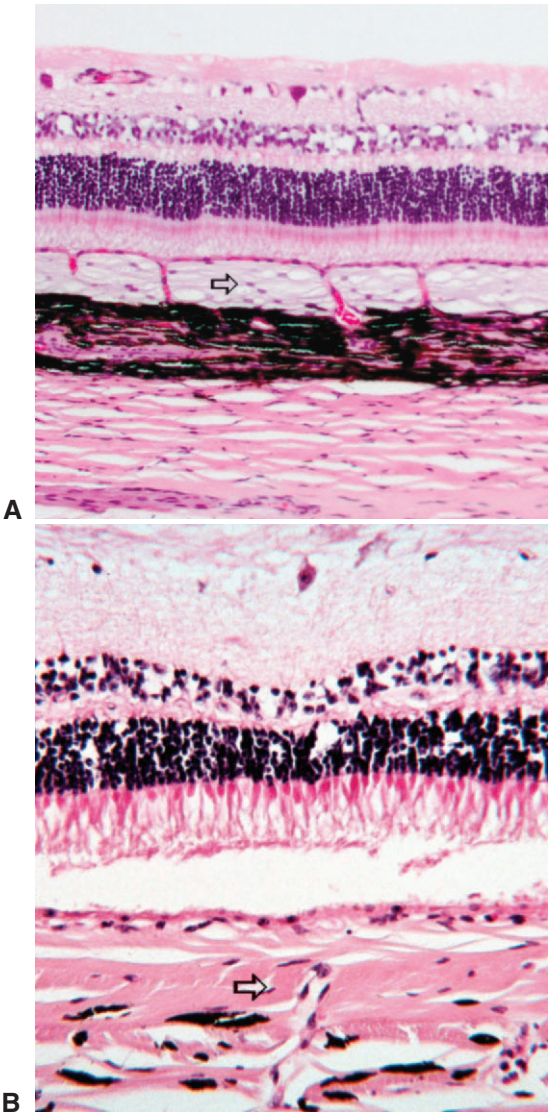

Figure 11-15. A, Normal canine tapetum cellulosum *(arrow)*. It is located between the choroid and the photoreceptor layer and pierced by the choriocapillaris. **B,** Normal tapetum fibrosum of a bovine *(arrow)*. (Courtesy Dr. Richard R. Dubielzig.)

protein and other large molecules that may scatter light in the aqueous and vitreous humor, these barriers serve to create a more optically perfect media. They are, however, frequently disrupted by inflammation or other disease processes.

PATHOLOGIC REACTIONS

Definitions

Although the uvea exhibits the same range of reactions as other tissues, inflammation is the most important. The following terms describe inflammation of the various parts of the uveal tract:

- *Uveitis:* inflammation of the uvea
- *Iritis:* inflammation of iris
- *Cyclitis*: inflammation of ciliary body
- *Iridocyclitis:* inflammation of iris and ciliary body
- *Choroiditis:* inflammation of choroid

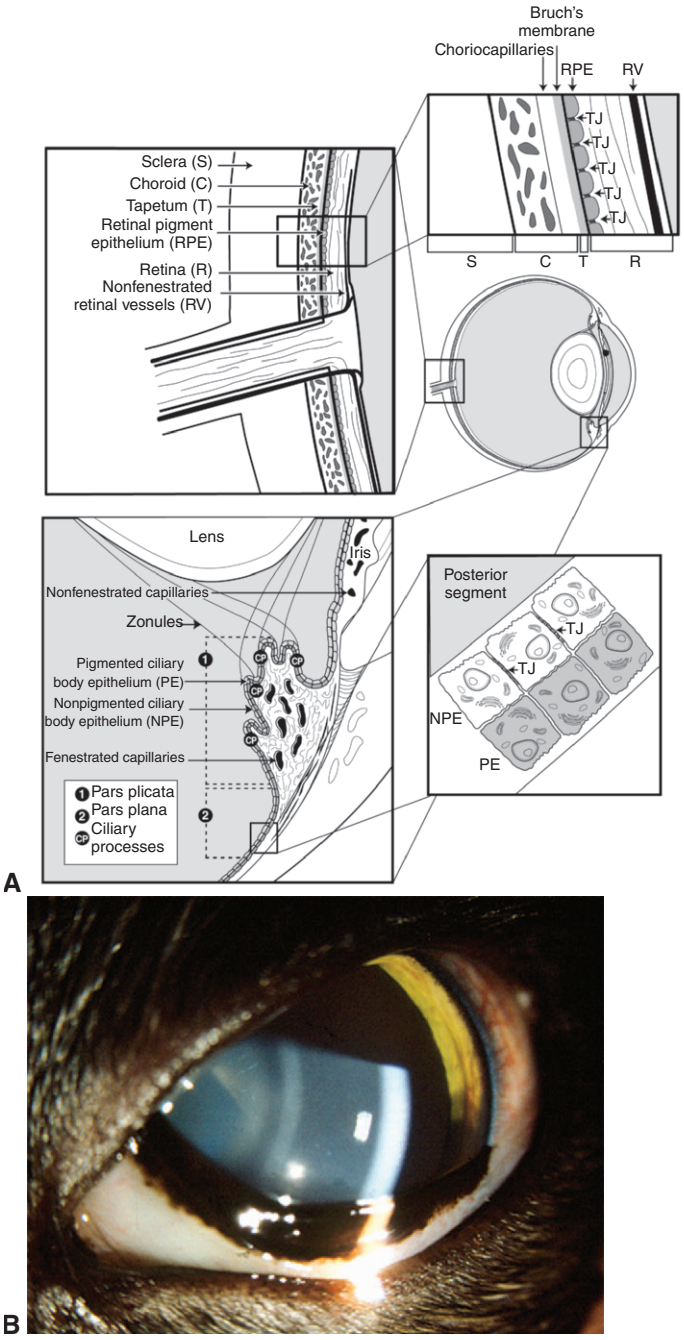

Figure 11-16. A, Blood-ocular barrier. The barrier normally prevents large molecules and cells from leaving the blood vessels and entering the eye, thereby maintaining clarity of the aqueous humor and vitreous. **B,** Aqueous flare in a cat with uveitis. This finding, which represents breakdown of the blood-aqueous barrier, is a hallmark of anterior uveitis. (**A** from Gilger B [2005]: Equine Ophthalmology. Saunders, St. Louis.)

Because of the continuity between the various parts of the uvea, aqueous humor, and vitreous, however, uveal inflammation often involves many ocular structures. The retina and choroid are adjacent, with no major barriers between, so they are frequently inflamed together. Consequently, the following terms are often preferable:

- *Anterior uveitis:* inflammation of iris and ciliary body
- *Posterior uveitis:* inflammation of choroid

- *Chorioretinitis:* inflammation of choroid and retina with primary focus in choroid
- *Retinochoroiditis:* inflammation of choroid and retina with primary focus in retina
- *Panuveitis:* inflammation of all uveal components

Immune Mechanisms

The uvea is an immunologically competent tissue that behaves as an accessory lymph node. Intraocular antigens may enter the systemic circulation and stimulate distant lymphoid organs. In 5 to 7 days sensitized B and T lymphocytes migrate toward the antigen within the eye, enter the uvea, and engage in antibody formation or cell-mediated immune reactions, which may create intraocular inflammation. Subsequent exposure to the same antigen results in a faster and greater (anamnestic) response.

The uvea is often secondarily inflamed when other parts of the eye are inflamed (e.g., secondary anterior uveitis frequently accompanies keratitis). Although such reactions are commonly beneficial in resolution of the primary disease (e.g., production of immunoglobulins and sensitized lymphocytes), excessive secondary uveitis may irreparably damage the eye.

Autoimmune phenomena also occur in the uvea. Preceding tissue damage (e.g., previous inflammation) releases tissue-specific retinal or uveal antigens that are normally intracellular or otherwise immunologically isolated. Hence one cause of uveal inflammation (e.g., trauma, infection by various organisms) may subsequently lead to a secondary, immune-mediated mechanism that results in persisting or recurring inflammation. Such a response may be involved in recurrent equine uveitis. Immune-mediated inflammation may also occur after exposure to lens proteins that have been immunologically isolated by the lens capsule before birth (e.g., lens-induced uveitis) or in response to antigens associated with uveal melanocytes (e.g., uveodermatologic syndrome).

Autoimmune diseases may originate with, or be perpetuated by, the following processes, which may also lead to recurrent episodes of uveitis:

- *Molecular mimicry:* Externally derived antigens (bacterial, viral, other) mimic host antigens, thereby directly stimulating T cells to attack sequestered host antigens.
- *Bystander damage:* An agent (viral or otherwise) damages tissue, releases sequestered antigens, and re-stimulates resting autoreactive T cells.
- *Epitope spreading:* The immune response spreads from one autoantigenic molecule to another (intermolecular) or from one site on the same molecule to another (intramolecular).

CONGENITAL UVEAL ABNORMALITIES

Abnormalities of the Pupil

Pupillary abnormalities are as follows:

- *Dyscoria:* abnormally shaped pupil
- *Corectopia:* eccentrically placed pupil
- *Polycoria:* more than one pupil
- *Aniridia:* lack of iris
- *Coloboma:* sector defect in iris (see later)

Corectopia (which is congenital) must be distinguished from a pupil pulled out of shape by synechia (which is acquired). In synechiation the pupil is distorted by adhesions between the lens and iris. Pupillary abnormalities are rarely significant by themselves, but they may be an important indication of other abnormalities.

Persistent Pupillary Membrane

During development the pupillary membrane (anterior portion of the tunica vasculosa lentis) spans the pupil from one portion of the iris collarette to another and supplies nutrients to the developing lens (see Chapter 2). In dogs this membrane is usually resorbed during later fetal development and the first 6 weeks of life, leaving a clear pupillary aperture. It is not uncommon, however, for remnants to remain for several months or longer. In general small remnants spanning from one portion of the iris to another (iris-to-iris persistent pupillary membranes [PPMs]) have no visual consequences, although visual impairment may occur if strands contact the cornea (iris-to-cornea PPMs) or lens (iris-to-lens PPMs) and create an opacity within the visual axis (Figure 11-17).

PPMs occur in a large number of dog breeds, most notably the basenji, in which they are recessively inherited (see Appendix I). A genetic basis is also likely in many other dog breeds, but the mode of inheritance is probably not simply mendelian. PPMs may span from one region of the iris to another (sometimes crossing the pupil) or they may extend to the cornea or lens, creating opacities in these structures. PPMs can usually be differentiated from inflammatory anterior or posterior synechia on the basis of their origin near the iris collarette region (versus an origin at the pupillary margin for synechia) and their presence at birth. It usually is possible to see the membrane extending from the iris collarette region to the cornea or lens, although occasionally the membrane may have broken free and the cornea or lens opacity (often pigmented) is all that remains. Therapy is not typically required or possible. The best method of preventing the disorder is to examine breeding stock and breed only animals that are free of PPMs. Slit-lamp biomicroscopy is essential for the examinations.

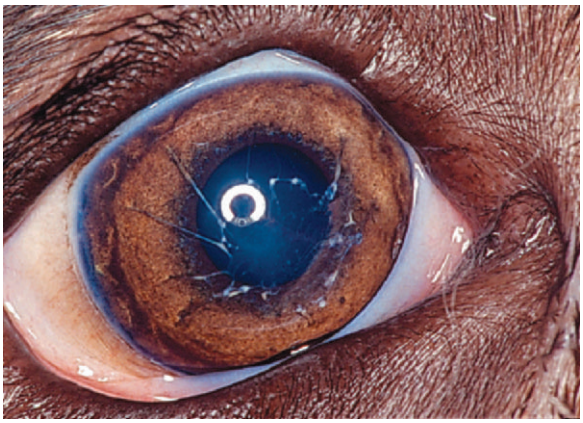

Figure 11-17. Persistent pupillary membranes (iris to cornea) in a young Saint Bernard dog. Unlike postinflammatory anterior synechia, these iridal strands originate near the iris collarette region. Anterior synechia would originate at the pupillary border or in the far periphery of the iris, near the iridocorneal angle. (Courtesy University of Wisconsin–Madison Veterinary Ophthalmology Service Collection.)

Coloboma

A *coloboma* is a defect in the eye resulting from incomplete closure of the embryonic fissure. Typical colobomas occur in the inferomedial portion of the iris or choroid or adjacent to the optic disc (Figure 11-18). Colobomas of the sclera also occur in the collie eye anomaly. Although the embryonic fissure is not involved, *coloboma* is also applied to lid defects and to sector defects in the iris and lens.

Anterior Segment Dysgenesis

Anterior segment dysgenesis is an autosomal recessive trait in the Doberman pinscher characterized by variable degrees of microphthalmia, corneal opacity, lack of anterior chamber, undifferentiated iris and ciliary body, hyaloid artery remnants, absence of or rudimentary lens, retinal dysplasia and separations, and congenital blindness. There is no treatment for this disorder.

Anterior segment dysgenesis syndrome occurs frequently in Rocky Mountain horses and has two distinct ocular phenotypes: (1) large cysts originating from the temporal ciliary body or peripheral retina (Figure 11-19) and (2) multiple anterior segment anomalies, including ciliary cysts, iris hypoplasia, iridocorneal adhesions and opacification, nuclear cataract, and megalocornea (Figure 11-20). This condition may be codominantly inherited, so that ciliary cysts are seen in heterozygous animals and multiple anterior segment anomalies are seen in homozygous animals.

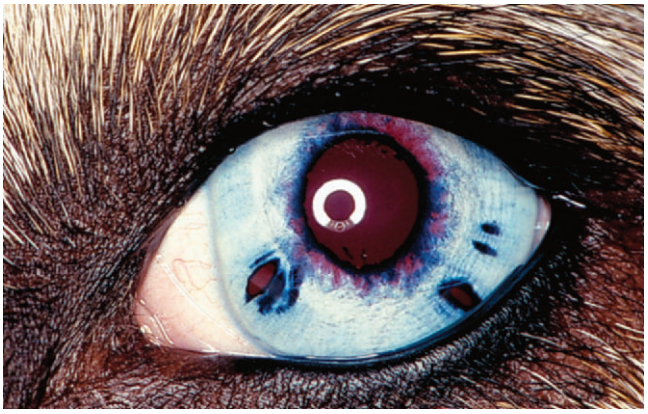

Figure 11-18. Several small iris colobomas are visible as full-thickness defects in the iris in this Australian shepherd dog.

Figure 11-19. A temporally located cyst involving the posterior iris, ciliary body, and peripheral retina in a Rocky Mountain horse presumed to be heterozygous for the responsible gene.

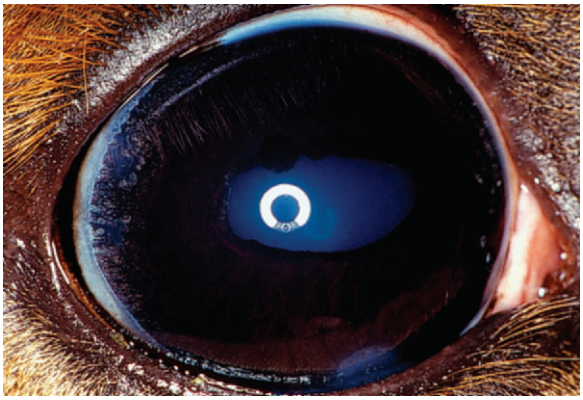

Figure 11-20. Anterior segment dysgenesis in a Rocky Mountain horse presumed to be homozygous for the responsible gene. The iris is smooth, dark, and histologically hypoplastic. The pupil resists dilation, presumably owing to defects in the iris musculature. This horse had other anterior segment anomalies, including ciliary cysts, iris hypoplasia, iridocorneal adhesions and opacification, nuclear cataract, and megalocornea.

Disorders of Pigmentation

Partial albinism (subalbinism) refers to reduction in ocular pigmentation. Part or all of the iris may lack pigment and appear blue. In a true albino the iris is pink.

Heterochromia

Heterochromia refers to variations in iris coloration. Both eyes, one eye only, or only part of an iris may be affected, and often there are concurrent variations in coat color (Table 11-1). *Heterochromia*

Table 11-1 | **Breeds Affected by Heterochromia Iridis**

SPECIES	BREED	CHARACTERISTICS
Cat	Siamese	Subalbinism
	Burmese	Variable iris hypopigmentation
	Abyssinian	Variable iris hypopigmentation
	Persian	Variable iris hypopigmentation
Dog	Australian cattle dog	Dappling
	Australian shepherd	Merling
	Boxer	White coat
	Collie	Merling (autosomal dominant)
	Great Dane	Harlequin coat (autosomal dominant)
	Long-haired dachshund	Harlequin coat (autosomal dominant)
	Dalmatian	Dappling (autosomal dominant)
	Malamute	Dappling
	Old English sheepdog	Heterochromia iridis
	Siberian husky	Dappling (autosomal dominant)
	Weimaraner	Iris hypopigmentation varies
Horse	Pinto, appaloosa, white and gray horses	Variable heterochromia
Cattle	Hereford, shorthorn	Albinism, subalbinism

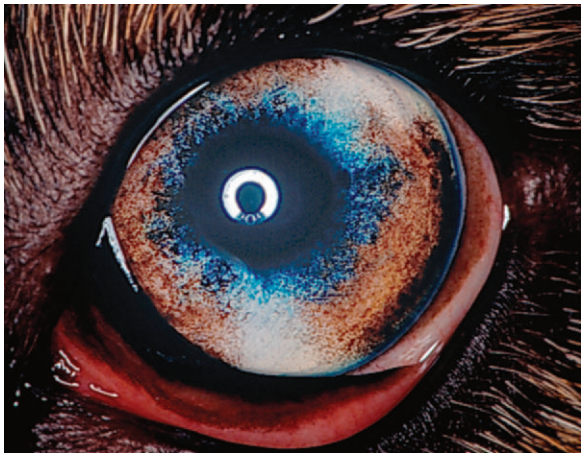

Figure 11-21. Heterochromia iridis (blue and brown iris) in an otherwise normal Australian shepherd dog.

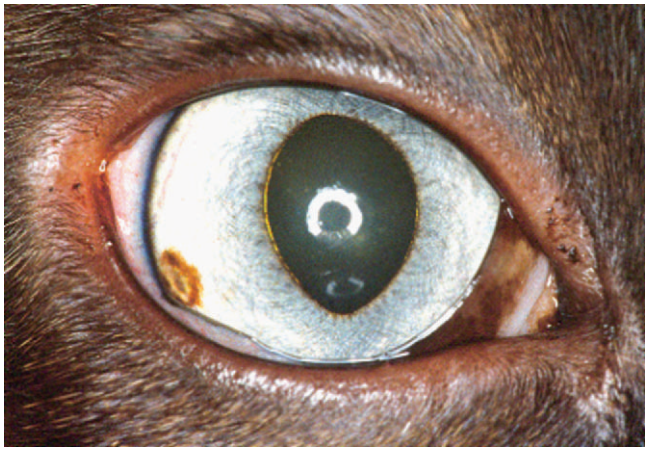

Figure 11-22. Iris nevus (freckle) in a cat. Such lesions should be regularly monitored for signs of progression.

iridis refers to variations in pigmentation of different regions of the iris in the same eye (Figure 11-21), and *heterochromia iridium* refers to variations in coloration between the two eyes of the same animal. Although heterochromia may be normal, blue iridal tissue has also been associated with iris hypoplasia, iris coloboma, and corectopia as well as with absence of or a small tapetum and lack of pigmentation of the nontapetal fundus. An association between congenital deafness and heterochromia has also been recognized in blue-eyed white cats and in the Dalmatian, Australian cattle dog, English setter, Australian shepherd, Boston terrier, Old English sheepdog, and English bulldog.

Lay terms for heterochromia are as follows:

- Wall eye: blue and white iris or part of an iris
- China eye: blue iris or part of an iris
- Watch eye: blue and yellow/brown iris or part of an iris

In dogs, heterochromia is due to incomplete maturation or absence of pigment granules in the iris stroma or anterior pigmented layer. Heterochromia iridis is proposed to be due to decreased availability of tyrosine hydrolase, necessary for the synthesis of melanin, as follows:

L-tyrosine → L-dihydroxyphenylalanine → melanins

tyrosine

hydrolase

In most species heterochromia is of no clinical significance. In cattle ocular albinism has been further subdivided (Table 11-2).

Table 11-2 | **Ocular Albinism in Cattle**

TYPE OF ALBINISM	FEATURES
Partial	Iris blue and white centrally, brown peripherally Hair color normal
Incomplete	Iris light blue, gray, and white Hair color white Some brown sectors in iris, and some colored hair patches Nontapetal fundus incompletely pigmented and choroidal vasculature visible
Complete	Iris very pale blue or white Hair pure white Variable fundus colobomas and tapetal hypoplasia

Iris Nevus

Iris nevi (Figure 11-22) are most commonly observed in cats and dogs. They may consist of focal spots of hyperpigmentation. They must be differentiated from neoplasms that require surgical treatment. Iris nevi do not protrude above the surface of the iris and do not enlarge. Nevi have a low malignant potential and show an increase in the number of cells or greater pigmentation of existing cells. They must be observed carefully for changes, especially in cats, in which they may transform into the early stages of diffuse malignant iris melanoma.

Waardenburg's Syndrome

Waardenburg's syndrome consists of deafness, heterochromia iridis, and white coat color. Although this hereditary syndrome occurs most commonly in blue-eyed white cats, it also occurs in dogs (especially the Australian cattle dog, Great Dane, and Dalmatian), mice, and humans. Not all blue-eyed white cats are affected. In the cat, the syndrome is inherited as a dominant trait with complete penetrance for the white coat and incomplete penetrance for deafness and blue irides.

UVEITIS

Clinical Signs

The detection of uveitis depends on familiarity with the clinical signs. In general the clinical signs of uveitis are similar regardless of cause. Signs of ocular discomfort are as follows:

- Photophobia and blepharospasm
- Pain (may manifest as anorexia or depression)
- Epiphora

Clinical signs more specific for uveitis are as follows:

- Aqueous flare
- Inflammatory cells free in the anterior chamber or adherent to the corneal endothelium (keratic precipitates)
- Hypopyon or hyphema
- Episcleral vascular injection or circumcorneal ciliary flush
- Corneal edema
- Miosis

- Resistance to mydriatics
- Lowered IOP
- Anterior or posterior synechiae
- Swollen or dull appearance of the iris
- Increased pigmentation of the iris
- Vitreous haze or opacity
- Retinal edema, exudate, or detachment
- Aqueous lipemia, which may be seen if circulating lipid levels are high

Aqueous flare is due to breakdown of the blood-aqueous barrier with increased permeability of vessels in the iris and ciliary body, resulting in release of protein into the aqueous. *Keratic precipitates* (KPs) are accumulations of inflammatory cells (neutrophils, lymphocytes, or macrophages) that adhere to the corneal endothelium. In large numbers these cells form a white layer in the anterior chamber called *hypopyon* (Figure 11-23). KPs may be small and scattered (in feline infectious peritonitis) or large and yellow ("mutton-fat" KPs) in granulomatous diseases. Miosis may be due to iridal edema or spasm of the iridal sphincter muscle. As the inflammation subsides, synechiae may form, causing an irregularly shaped pupil (Figure 11-24) or a scalloped appearance on dilation, with pigment remnants on the anterior lens capsule. If posterior uveitis is present, the vitreous may become hazy, and retinal edema, exudates, or detachments may be seen.

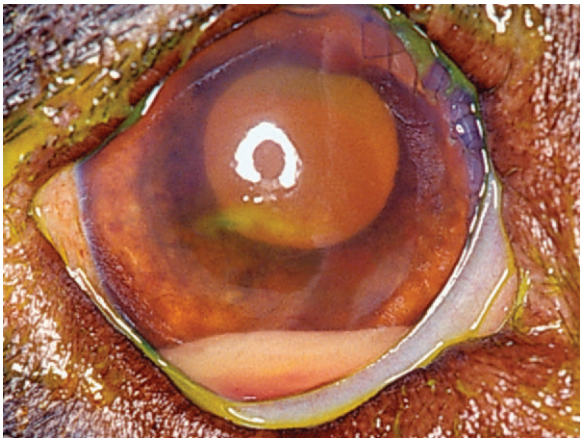

Figure 11-23. Hypopyon in the ventral anterior chamber in a dog that had suffered a penetrating ocular injury. Unless the cornea has been perforated, the anterior chamber is usually sterile in most patients with hypopyon.

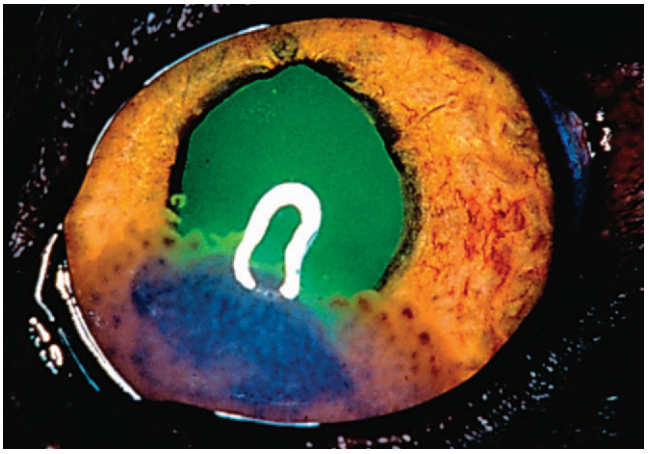

Figure 11-24. Dense "mutton fat" keratic precipitates with admixed blood in a cat with chronic anterior uveitis. The pupil is irregularly shaped (ectropion uveae) owing to small posterior synechia.

Sequelae of Uveitis

Posterior Synechiae

Posterior synechiae occur when fibrinous adhesions form between the lens and iris, with fibrovascular organization occurring later (see Figure 11-24). Formation of synechiae is more likely when aqueous protein content is high. If synechiae form around the entire circumference of the pupil, *iris bombé* occurs, preventing aqueous flow to the anterior chamber, and secondary glaucoma almost invariably follows. An irregularly shaped pupil is frequently caused by synechiae. If blood or exudate organizes in the anterior chamber, a connective tissue membrane may occlude or obliterate the pupil.

Peripheral Anterior Synechiae

Adhesions may form between the iris and trabecular meshwork or between the iris and cornea. Swelling, iris bombé, and cellular infiltrates may reduce drainage of aqueous through the iridocorneal angle early in uveitis, but once peripheral anterior synechiae have formed, an alternative route for drainage must be provided, because the angle is held closed by the synechiae.

Cataract

Cataract (opacity of the lens) occurs frequently after uveitis. It is probably caused by altered composition of the aqueous that interferes with lens nutrition. When an animal with a cataract and signs of uveitis is examined, determination must be made as to whether the cataract caused the uveitis or the uveitis caused the cataract.

Glaucoma

IOP is usually *lowered* during uveitis because an inflamed ciliary body makes less aqueous humor and endogenous prostaglandins may increase uveoscleral outflow. If IOP is *normal* or *increased* in the presence of active inflammation, it is likely that aqueous humor outflow via the trabecular meshwork is impaired in one of the following ways:

- Blockage of the angle with inflammatory cells, debris, or neovascular membranes
- Peripheral anterior synechiae
- Occlusion of the pupil by posterior synechiae

Eyes with normal IOP and active uveitis may have impaired aqueous humor outflow and should be monitored carefully for glaucoma.

Intractable secondary glaucoma due to lens-induced uveitis is a common entity, especially in dogs. This condition may be seen after penetrating injuries to the lens, in patients with long-standing cataracts undergoing lens resorption, and sometimes after cataract extraction.

Retinal Detachment

Exudation and cellular infiltration from the choroid may cause retinal detachment.

Atrophy

The iris and ciliary body atrophy as the stroma is replaced by fibrous tissue. Defects may appear in the iris. Atrophy of areas of the choroid frequently results in atrophy of the overlying retina, which is visible ophthalmoscopically. Severe atrophy of the ciliary body causes *hypotony* (lowered IOP). In some animals the color of the iris becomes *darker* after uveitis. In severe cases the entire globe may shrink, a condition called *phthisis bulbi*.

Preiridal Fibrovascular Membranes

In some animals with chronic anterior uveitis new blood vessels and fibrous membranes form on the anterior surface of the iris. These may result in eversion of the pupillary margin, called *ectropion uveae*, or glaucoma as they cover the trabecular meshwork.

Cyclitic Membranes

A *cyclitic membrane* is a band of fibrovascular tissue extending from the ciliary body across either the pupil or the anterior face of the vitreous. It consists of fibrous tissue and blood vessels and may severely obstruct vision.

Sympathetic Ophthalmia

Sympathetic ophthalmia is a *rare* immune-mediated disorder in humans, and perhaps in animals, in which unilateral intraocular inflammation liberates previously immunologically isolated antigens. The resulting immune response to these ocular antigens leads to damage of the previously normal other eye.

Diagnosis of Uveitis

Anterior uveitis is distinguished from conjunctivitis, superficial keratitis, and glaucoma, the other causes of the red-eye syndrome (Table 11-3). The uvea is involved in numerous systemic disorders (Table 11-4). Such diseases usually affect other parts of the eye in addition to the uvea and are discussed in Chapter 18. Once uveitis is detected, every effort should be made to identify a specific cause of the inflammation so that the most effective therapy may be started. A thorough history and complete physical examination are essential for the proper diagnosis as to the cause of the inflammation in a given patient.

Numerous uveitis classification schemes have been proposed, including those based on the tissues affected (anterior uveitis, posterior uveitis, panuveitis), on the presumed histologic nature of the disorder (suppurative, nonsuppurative, granulomatous, nongranulomatous), on whether the cause starts inside the eye or from its surface (endogenous versus exogenous), and on a specific etiology (see Table 11-4). Although each of these schemes has its own advantages and disadvantages, classification into granulomatous or nongranulomatous and then by specific etiology is probably the most useful method in a clinical setting, because it also helps guide specific therapy (Table 11-5). This scheme, however, is plagued by the presence of a large percentage of patients having idiopathic uveitis in which the cause remains obscure and therapy can be only nonspecific and directed at controlling inflammation and preventing further damage to the eye. Presumably, most of these cases are immune-mediated or involve microorganisms that are not yet recognized as pathogenic. It is hoped that over time the percentage of patients with idiopathic uveitis will decline as our understanding of the causes of this disorder improves.

Although classification as granulomatous or nongranulomatous uveitis is based on a histologic classification scheme, the criteria in Table 11-5 can also be used to make reasonable clinical inferences about the histologic nature of the inflammation and to allow for prioritization of the diagnostic tests to be performed. Most cases of granulomatous uveitis are associated with microorganism or foreign material stimulation of a chronic immune response, whereas nongranulomatous uveitis is often associated with physical, toxic, or allergic causes. After determining whether a specific animal has granulomatous or nongranulomatous uveitis, the clinician should consider specific tests to try to determine the exact cause (e.g., serum titer measurement for *Toxoplasma*). In general the following specific categories of uveitis should be considered:

- Infectious associated—algal, bacterial, fungal, viral, protozoal, parasitic

Table 11-3 | **Differential Diagnosis of Ocular Inflammations**

PARAMETER	ANTERIOR UVEITIS	CONJUNCTIVITIS	SUPERFICIAL KERATITIS	GLAUCOMA
Conjunctiva	Variably thickened	Thick; folded	Variably thickened	Not thickened
Conjunctival vessels	Episcleral; not movable with conjunctiva, infrequently branch	Superficial, diffuse, extensive branching	Superficial, diffuse, extensive branching	Episcleral, not movable with conjunctiva, infrequently branch
Secretion or discharge	None to serous	Moderate to copious, serous to purulent	Moderate to copious, serous to purulent	None to serous
Pain	Moderate	None to slight	Moderate to severe	Moderate to severe
Photophobia	Moderate	None	Severe	Slight
Cornea	Clear to steamy	Clear	Clouded to opaque	Steamy
Pupil size	Small, sluggish, irregular, or fixed	Normal	Normal to small	Dilated, moderate to complete, and fixed
Pupillary light response	Variable	Normal	Normal	Absent
Intraocular pressure	Variable: may be normal, elevated, or diminished	Normal	Normal	Elevated

Modified from Lavignette AM (1973): Differential diagnosis and treatment of anterior uveitis. Vet Clin North Am 3:504.

Table 11-4 | **Causes of Uveitis**

CAUSE	MOST COMMONLY AFFECTED SPECIES
NEOPLASTIC/PARANEOPLASTIC	
Lymphosarcoma	Any
Melanoma	Dog, cat
Histiocytic proliferative disease	Dog
Hyperviscosity syndrome	Dog
Granulomatous meningoencephalitis	Dog
Miscellaneous primary intraocular tumors	Any
Miscellaneous metastatic tumors	Any
METABOLIC	
Diabetes mellitus (lens-induced uveitis)	Dog
Systemic hypertension	Cat, dog
Hyperlipidemia	Dog
Coagulopathies	Any
IDIOPATHIC	Any
IMMUNE-MEDIATED	
Cataracts (lens-induced uveitis)	Any
Lens trauma (phacoclastic uveitis)	Any
Immune-mediated thrombocytopenia	Any
Immune-mediated vasculitis	Any
Uveodermatologic syndrome (Vogt-Koyanagi-Harada–like syndrome)	Dog
INFECTIOUS	
Algae	
Geotricha spp.	Dog
Prototheca spp.	Dog
Bacteria	
Septicemia/endotoxemia due to any cause	Any
Leptospira spp.	Dog, horse
Bartonella spp.	Dog, cat
Borrelia burgdorferi	Dog, horse
Brucella spp.	Dog, horse
Escherichia coli	Cattle, horse
Streptococcus spp.	Horse
Rhodococcus equi	Horse
Listeria monocytogenes	Sheep, cattle
Haemophilus spp.	Cattle
Tuberculosis	Cattle, cat
Protozoa	
*Toxoplasma gondii**	Any
Leishmania donovani	Dog
Ehrlichia canis or *Ehrlichia platys*	Dog
Rickettsia rickettsii	Dog
Yeasts and Fungi	
Aspergillus spp.	Chickens, turkeys, cat
Blastomyces spp.	Dog, cat
Coccidioides immitis	Dog
Cryptococcus spp.	Dog, cat
Histoplasma capsulatum	Dog, cat
Pseudallescheria boydii	Dog
Viruses	
Canine adenovirus types 1 and 2 (immune-mediated)	Dog
Canine distemper virus	Dog
Coronavirus (feline infectious peritonitis)	Cat
Feline leukemia virus	Cat
Feline immunodeficiency virus	Cat
Herpesvirus (Marek's disease)	Chickens, turkeys
Herpesvirus	
Feline herpesvirus 1	Cat
Canine herpesvirus 1	Dog
Equine herpesvirus 1 and 2	Horse
Ovine herpes virus 2 (MCF)	Cattle
Alcelaphine herpes virus 1 (MCF)	Cattle
Rabies virus	Dog
Equine influenza	Horse
Equine viral arteritis	Horse
Parainfluenza type 3	Horse
MCF	Cattle
Parasitic	
Taenia multiceps	Sheep, dog
Echinococcus granulosis	Horse (rare)
Angiostrongylus vasorum	Dog
Dirofilaria immitis	Dog
Setaria spp.	Horse
Onchocerca cervicalis	Horse (equine recurrent uveitis)
Strongylus	Horse
Diptera spp. (ophthalmomyiasis interna)	Various
Toxocara spp., *Baylisascaris* spp. (ocular larval migrans)	Dog, cat
Trypanosoma sp.	Sheep/Goats, cat
Elaeophora schneideri	Sheep/Goats
TOXIC	
Drugs	
Pilocarpine, carbachol other parasympathomimetics	Any
Prostaglandin derivatives (latanoprost)	Any
Sulfamethazine/trimethoprim (immune-mediated)	Dogs
Endotoxemia from any systemic source	Any
Infectious keratitis with bacterial toxin production	Any
Radiation therapy	Any
TRAUMA	
Blunt or penetrating injuries	Any
Corneal foreign bodies	Any
REFLEX UVEITIS	
Ulcerative keratitis of any cause	Any
Deep necrotizing or nonnecrotizing scleritis	Dog
Episcleritis	Dog

MCF, Malignant catarrhal fever.
**Neosporum caninum* has been found responsible for some cases of dogs previously diagnosed with *T. gondii* infection. The clinical significance is undetermined.

- Immune-mediated
- Neoplastic or paraneoplastic
- Metabolic
- Traumatic
- Toxic
- Reflex
- Idiopathic

Differential diagnosis of the cause of uveitis often requires specialist assistance, notably when potential zoonotic diseases may be involved or the cause remains unclear.

A few generalizations may be made. Uveitis associated with KPs is often associated with intraocular neoplasia, feline infectious

Table 11-5 | **Classification Criteria for Anterior Uveitis**

NONGRANULOMATOUS	GRANULOMATOUS
Acute onset	Gradual onset
Short course	Chronic or recurrent
No keratic precipitates	Keratic precipitates/greasy exudate on lens surface
No synechiae	Posterior synechiae
No iris nodules	Iris nodules may be present
Primarily anterior uveitis	Posterior uveitis may also be present

These criteria are useful but not absolute and are interpreted along with other clinical signs.

peritonitis, deep fungal agents, and intraocular foreign bodies. Severe uveitis that involves the anterior and posterior segments is often associated with a deep fungal agent, lymphosarcoma, or uveodermatologic syndrome. The last is also commonly associated with loss of pigment in the uveal tract, skin, or hair. Uveitis with hemorrhage is often associated with systemic hypertension, intraocular neoplasia, coagulopathy, or a tick-borne disorder.

General Therapeutic Principles

1: Make an Etiologic Diagnosis

The clinician must make a concerted attempt to find a cause for the uveitis. Although not all such attempts are successful, idiopathic uveitis is a diagnosis of exclusion. Often, if a specific cause is identified, more effective therapy may be instituted (e.g., removal of an abscessed tooth, treatment for deep mycosis, control corneal infection, chemotherapy for lymphosarcoma). Routine hematologic analysis and serum chemistry profiles are useful in indicating the presence of inflammatory disorders and concurrent systemic disease (see Table 11-4). In endemic areas appropriate serologic tests are indicated (e.g., for toxoplasmosis, coccidioidomycosis, blastomycosis, cryptococcosis). Blastomycosis is found most frequently in the central United States east of the Mississippi River, and coccidioidomycosis is found in Arizona, Nevada, and the central valley of California.

2: Control Inflammation

CORTICOSTEROIDS. Corticosteroids may be given via the topical, systemic, or, occasionally, subconjunctival route. These agents inhibit cell-mediated immune reactions, decrease antibody production, and stabilize lysosomal membranes, reducing release of intracellular proteolytic enzymes. If corticosteroids are administered via the topical or subconjunctival routes the cornea must not retain fluorescein stain. Additionally, immunosuppressive therapy should not be instituted if active infectious diseases, such as a deep fungal agent, have not been ruled out. In general the following approach is helpful:

- For mild uveitis (mild conjunctival hyperemia, no obvious or only minimal aqueous flare, hypotony, with/without miosis):
  1. Topical corticosteroids—0.1% dexamethasone or 1% prednisolone acetate q6-12h
- For moderate uveitis (moderate conjunctival hyperemia, readily detected aqueous flare, normal or decreased IOP, with/without miosis):
  1. Topical corticosteroids—0.1% dexamethasone or 1% prednisolone acetate q4-6h
  2. Systemic prednisone 0.25 mg/kg PO in dogs and cats; in horses a systemic nonsteroidal antiinflammatory drug (NSAID) should be used instead
- For severe uveitis (marked conjunctival hyperemia, marked aqueous flare/fibrin/hypopyon, with/without miosis):
  1. Topical corticosteroids—0.1% dexamethasone or 1% prednisolone acetate q1-4h
  2. Systemic prednisone 1.0 mg/kg PO in dogs and cats; in horses a systemic NSAID should be used instead
  3. Consider triamcinolone acetonide 1-2 mg per eye administered subconjunctivally.

NONSTEROIDAL ANTIINFLAMMATORY DRUGS. Significant protein leakage from uveal vessels during inflammation is mediated by prostaglandins. Inhibition of prostaglandin production decreases the amount of antibody present to engage in immunologic reactions and also decreases fibrin, which reduces synechia formation. Because endogenous prostaglandins also contribute to miosis by a mechanism that is not blocked by atropine, an NSAID may facilitate pupillary dilation with atropine. In general topical and systemic NSAIDs are not as potent as corticosteroids in the treatment of immune-mediated uveitis but may approximate or exceed the efficacy of corticosteroids in traumatic uveitis. Topical NSAIDS include flurbiprofen, suprofen, and diclofenac. These drugs are administered every 6 to 12 hours in most species. Systemic NSAIDS are typically dosed at levels recommended for the species being treated.

IMMUNOSUPPRESSIVE AGENTS. Topical 0.02% to 2.0% cyclosporine, oral cyclosporine, or oral azathioprine may be used in select cases of nonresponsive uveitis. Typically these agents require periodic laboratory evaluations for systemic side effects, especially those involving the bone marrow, liver, and kidney. Azathioprine has been suggested at 1 to 2 mg/kg/day for 3 to 7 days, followed by tapering to as low a dosage as possible.

3: Prevent Undesirable Sequelae

MYDRIATICS/CYCLOPLEGICS. Pupillary dilation (mydriasis) can help reduce synechiae formation and the likelihood of iris bombé with secondary glaucoma. Relaxation of the ciliary muscle *(cycloplegia)* can help lessen ocular pain. In general the dose required to dilate the pupil is somewhat lower than that necessary to induce cycloplegia and provide pain relief. One percent atropine ophthalmic ointment or solution is a parasympatholytic agent with potent mydriatic and cycloplegic activity, whereas 0.5% to 1.0% tropicamide solution is a shorter-acting parasympathomimetic with relatively potent mydriatic effects but milder cycloplegic effects. Sympathomimetics, such as 10% phenylephrine given every 8 to 12 hours, can boost the mydriatic effects of atropine and tropicamide, but these drugs afford no meaningful cycloplegia. On rare occasions mydriasis can compromise the drainage angle, leading to rises in IOP, or reduce tear production, especially in animals with keratoconjunctivitis sicca. In general atropine is used one to three times per day or to effect.

ANTIGLAUCOMA DRUGS. IOP is typically low in uveitis because an inflamed ciliary body makes less aqueous humor and endogenous prostaglandins increase uveoscleral outflow. If IOP is normal or elevated in the presence of inflammation, the drainage angle is probably compromised and the clinician must be concerned about impending glaucoma. It is essential that

irreversible glaucomatous damage not be allowed to occur while antiinflammatory therapy works to clear the drainage angle. In general a topical or systemic carbonic anhydrase inhibitor (dorzolamide or methazolamide), a topical β-blocker (timolol), or an adrenergic agent (dipivefrin) is preferred to a parasympathomimetic (pilocarpine, demecarium bromide) or a prostaglandin derivative (latanoprost, travoprost), either of which may exacerbate intraocular inflammation.

4: Relieve Pain

The cycloplegic action of atropine relaxes the ciliary muscle and helps reduce ocular pain in uveitis. The patient may also be placed in a darkened room or stall to alleviate photophobia. Topical or systemic NSAIDs can provide pain relief as well as aid in controlling inflammation. For severe pain a systemic analgesic, such as butorphanol, morphine, or oxymorphone, may be used.

Specific Forms of Uveitis

Infectious Uveitis

The infectious causes of uveitis are summarized in Table 11-4. Many of these agents are located in specific geographic regions, a feature that helps narrow the list of possible causes in a given patient. Not all patients with infectious uveitis have living organisms within the eye. Uveitis may occur as a result of intraocular infection or in response to bacterial toxins generated within or outside the eye, or may stem from an immunologic response to the organism, which may be within the eye or elsewhere in the body. It is well recognized that uveitis may be associated with infection outside the eye, including prostatitis, endometritis, gingivitis and tooth root abscess, mastitis, metritis, navel ill, and pneumonia. In these cases uveitis may result from shedding of bacteria into the circulation, the uveitis being secondary to previously sensitized lymphocytes in the uvea, or may be due to bacterial toxins released from the primary site. Often the uveitis is recurrent in these cases, and hematologic examination or blood culture may be of value in arriving at a definitive diagnosis.

Blastomycosis, ehrlichiosis, histoplasmosis, and coccidioidomycosis are important causes of uveitis in dogs, as are cryptococcosis, toxoplasmosis, and feline infectious peritonitis in cats. If uveitis is present in association with lesions of lungs, bone, lymph nodes, skin, or testicles or if the animal is located in an area endemic for any of these organisms, appropriate serologic, radiographic, and cytologic tests are indicated.

Immune-Mediated Uveitis

Immune-mediated uveitis may be the result of a primary reaction to a foreign antigen, an autoimmune phenomenon directed against self-antigens, or a combination of the two. It is believed that the majority of idiopathic cases of uveitis are actually immune-mediated. Often the diagnosis is made through exclusion of all known causes of uveitis. In some cases specific clinical signs (depigmentation) or historical events (a complete cataract preceding the inflammation or cat-scratch injury involving the lens) support the diagnosis of immune-mediated uveitis, and a detailed evaluation is not required.

UVEODERMATOLOGIC SYNDROME. Synonym: Vogt-Koyanagi-Harada–like syndrome.

Uveodermatologic syndrome affects certain breeds more commonly than others—Akita, Old English sheepdog, golden retriever, Siberian husky, and Irish setter. It is a spontaneous autoimmune disease apparently directed against melanin that affects the anterior and posterior uvea, frequently resulting in blindness from retinal detachment or glaucoma. Antiretinal antibodies to previously sequestered retinal antigens may also be present. Presumably the antibodies develop after the initial insult has severely damaged the retina and may represent epitope spreading. Depigmentation of the mucocutaneous junctions, eyelids, and hair coat may precede or follow the ocular signs. Histologic examination of a biopsy specimen from the mucocutaneous junction (especially the lips), even if the tissue appears grossly normal, can be useful in the diagnosis of this disorder if results of a systemic evaluation are otherwise noncontributory and the animal has severe anterior and posterior uveitis. Neurologic signs are associated with the syndrome in humans but are rare in dogs. In some geographic regions the onset of the disease has a definite seasonal incidence (e.g., February to May in southern California).

Vigorous early antiinflammatory therapy with topical and systemic steroids, NSAIDs, and azathioprine is often necessary to save vision. Recurrences of the disease can be expected, with maintenance therapy using appropriate medications between recurrences. Given the severe and relentless nature of the uveitis, the *immediate* assistance of a veterinary ophthalmologist should be sought in the handling of dogs affected with uveodermatologic syndrome.

LENS-INDUCED UVEITIS. The embryology of the lens is such that the lens capsule essentially isolates the lens proteins immunologically from the immune system before birth. Therefore if the lens capsule ruptures or leaks, lens proteins may enter the aqueous and elicit an immune-mediated uveitis that may be acute or chronic. The most common causes of lens-induced uveitis are liquefaction of cataractous lens proteins that escape through an intact lens capsule, swelling of a cataractous lens with increased "porosity" of an otherwise intact lens capsule, small tears in the lens capsule from rapidly forming cataracts and lens swelling (diabetes mellitus), and traumatic disruption of the lens capsule (cat scratch, penetrating injuries).

Leakage through the Intact Lens Capsule. The most common form of lens-induced uveitis is caused by leakage through intact lens capsule, which is most frequently seen in conjunction with the advanced stages of cataract (complete on resorbing). It should be suspected in every animal in which a complete or resorbing cataract precedes the onset of a "red eye," or in animals with a "red eye" and a cataract. It may be differentiated from uveitis-induced cataract by the fact that in the latter, the "red eye" uveitis precedes the cataract. Lens-induced uveitis should be anticipated in all eyes with cataract, although it does not always occur. In this form of the disease the lens capsule becomes permeable, allowing liquefied cortex to leak into the aqueous and creating an immune-mediated uveitis and, possibly, secondary glaucoma. Without tonometry and biomicroscopy, this inflammation may not be evident, and many such eyes exhibit a dilated pupil—not a miotic pupil as would be expected in uveitis. Affected eyes, however, do typically exhibit at least some conjunctival hyperemia. Eyes with lens-induced uveitis before cataract surgery have a greater risk for many postoperative complications (glaucoma, retinal detachment) than eyes without it.

Therapy with topical corticosteroids or NSAIDs, often for relatively long periods, may be needed to control lens-induced uveitis. In particularly severe cases systemic antiinflammatory agents may be required. Corticosteroids, even those administered

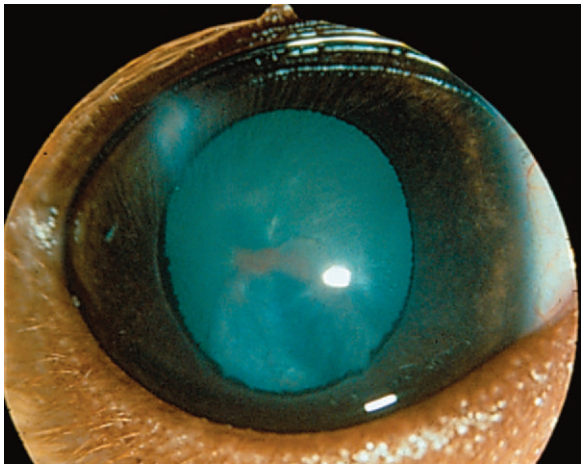

Figure 11-25. Chronic lens-induced uveitis in a basenji puppy after a cat claw injury. The lens capsule has been ruptured by the nail.

topically, should be used with caution in dogs with poorly regulated diabetes mellitus, cataract, and lens-induced uveitis so as to avoid worsening the glycemic control.

Lens-induced uveitis should be suspected in all red eyes in which cataract preceded the conjunctival hyperemia. Glaucoma should be ruled out in these cases.

Failure to recognize and treat lens-induced uveitis when cataracts are first diagnosed is a *very* common cause of lower success rates of cataract surgery in dogs. Medical therapy for lens-induced uveitis should be implemented as soon as the diagnosis is established.

Penetrating Lens Injuries. Penetrating injuries to the lens often quickly progress to endophthalmitis with secondary glaucoma (Figure 11-25). Bacteria are commonly inoculated during the injury, resulting in a mixed purulent inflammation with numerous neutrophils. Early lens extraction may offer the greatest chance for saving the eye, although large case studies to support this aggressive method of treatment are lacking. In many older dogs, medical treatment after lens capsule rupture cannot prevent loss of the eye through uncontrolled inflammation and secondary glaucoma. In young dogs (less than 12 months), much of the lens cortex may be resorbed, with less inflammation than in older animals, provided that infection is controlled. Nevertheless the long-term prognosis remains guarded in these animals.

Penetrating injury and lens capsule rupture are common causes of uveitis and endophthalmitis in dogs and cats.

UVEITIS ASSOCIATED WITH DENTAL DISEASE. Untreated gingivitis, periodontitis, and tooth root abscesses are very common causes of severe uveitis in dogs. Treatment of dental disorders is essential before any intraocular surgery is undertaken as well as for the patient's general health.

PIGMENTARY UVEITIS IN GOLDEN RETRIEVERS. In pigmentary uveitis in golden retrievers, pigment is dispersed in the anterior chamber, the iris becomes dark and thickened, and clumps of pigment may be seen on the lens capsule and corneal endothelium. Aqueous flare, posterior synechiae, cataract, and glaucoma may also occur. The cause of the disorder is undetermined, although some workers believe it to be immune-mediated and others have associated it with uveal cysts.

FELINE UVEITIS. Causes of uveitis in cats include feline infectious peritonitis, lymphosarcoma caused by feline leukemia virus, feline immunodeficiency virus, toxoplasmosis, cryptococcosis, histoplasmosis, blastomycosis, and coccidiomycosis. For details of the ocular manifestations of specific disorders, see Chapter 18. A specific type of nongranulomatous anterior uveitis described as *lymphocytic-plasmacytic uveitis* has been recognized as a common precursor to *glaucoma* if the uveitis is uncontrolled. It is also a common cause of glaucoma in cats. Idiopathic lymphocytic-plasmacytic uveitis occurs in both diffuse and nodular forms, with the nodular form being more commonly unilateral, and the diffuse form bilateral.

A minimum laboratory evaluation for cats with either unilateral or bilateral uveitis consists of the following procedures:

- Complete blood count
- Serum biochemical profile
- Urinalysis
- Thoracic radiography
- Serologic tests relevant to the geographic location: *Toxoplasma* (immunoglobulin [Ig] G and IgM), feline leukemia virus, feline immunodeficiency virus, *Cryptococcus, Blastomyces* spp., *Histoplasma* spp., and *Coccidioides* spp.

Uncontrolled or unobserved *idiopathic lymphocytic-plasmacytic uveitis* is a common cause of feline glaucoma.

In 93 cats with endogenous uveitis in Colorado in which a specific agent was identified, the following seroprevalence of infection was found: *Toxoplasma gondii*, 78.5%; feline immunodeficiency virus, 22.9%; feline leukemia virus, 4.95%; and feline coronavirus, 27%. The combination of topical corticosteroids and clindamycin hydrochloride (25 mg/kg, divided, twice daily) was beneficial in cats with uveitis associated with toxoplasmosis (Chavkin et al., 1992). It is highly probable that the various causes of feline uveitis vary greatly by geographic region.

EQUINE RECURRENT UVEITIS. Synonyms: "moon blindness," periodic ophthalmia.

As in other species, the horse may exhibit a single episode of uveitis due to any one of a multitude of causes. In addition to this form of uveitis, horses also frequently have apparently spontaneously recurring episodes of uveitis (equine recurrent uveitis [ERU]) that are presumably immune-mediated. ERU, however, is not a single disease as the name would imply but instead is a group of diseases united only by a clinical pattern of recurrent bouts of uveitis. With each subsequent uveitis attack, cumulative damage occurs to the ocular tissues, and blindness may result. The long-term prognosis is guarded, but with therapy, vision may be retained for a prolonged period in many animals.

History and Geographic Distribution. ERU has been recorded for millennia and is the most common cause of vision loss in the horse. As with many ancient disorders, the proposed causes and treatment have varied greatly over the years, and the disease has often been shrouded in folklore, ignorance, and misconceptions. For example, the term "moon blindness" has two origins: (1) the frequent recurrences were once thought coincident with the phases of the moon and (2) the cataract that often accompanies chronic ERU looked like a small moon in the eye.

The disease is worldwide in distribution, although distinct regional differences in frequency occur. It is more common in North America than in Australia, the United Kingdom, or

South Africa. An incidence of up to 12% has been recorded in eastern areas of the United States, and some investigators believe it is more prevalent in low-lying areas with high rainfall. There is no age or sex predilection. The Appaloosa breed appears to be at higher risk for development of recurrent uveitis, suggesting a genetic predisposition to ERU.

Etiology. There is no single cause of ERU (see Table 11-4). The most commonly held explanation is that the uveitis is an autoimmune phenomenon in which IgG antibodies and autoreactive T cells specific for retinal antigens are present. A cell-mediated immunity to uveal antigens has also been demonstrated in horses with ERU. The association between ERU and previous or current infection with *Leptospira* has been studied in greater detail than many of the other known etiologies of ERU. This organism appears to be capable of immunologically cross-reacting with the equine cornea and lens, and in horses with ERU, leptospiral antisera is also cross-reactive with the equine iris pigment epithelium and retina. In Europe leptospiral strains have been isolated from the ocular fluids of horses with chronic ERU, and it is postulated that persistent intraocular leptospiral infections by certain strains of the organism cause ERU. Many horses with ERU in the United States, however, do not appear to be infected with leptospiral organisms; also, potent immunosuppressive therapy with drugs such as intravitreal cyclosporine does not exacerbate the disease as would be expected with an active infectious process. Therefore the relative importance of the direct effects of the organism on the eye, locally produced antibodies against *Leptospira interrogans*, and autoantibodies against retinal autoantigens (retinal S-antigen and interphotoreceptor retinoid-binding protein) remain unclear in the pathogenesis of ERU. In any event, it is clear that ERU is a highly complex disorder with multifactorial causes related to the genetic constitution of the animal and that it is strongly immune-mediated. Common causes of ERU are *Leptospira*-associated uveitis and uveitis associated with migrating microfilariae of *Onchocerca cervicalis*.

Leptospira-Associated Uveitis. Although both experimental infections and natural outbreaks of leptospirosis have been associated with ERU, clinically apparent uveitis does not develop in most adult horses until 1 to 2 years after infection. Several reports have described isolation of *L. interrogans* from various ocular fluids, especially the vitreous, in horses with chronic ERU. The organism is difficult to culture, however, and results of polymerase chain reaction testing for leptospiral DNA are typically positive in many animals that are culture-negative, suggesting that the organism may be more prevalent than once thought. Serum antibody titers greater than 1:400 are suggestive of previous infection, although lower serologic titers may be found in many infected horses. In fact, negative serologic titers do not necessarily rule out leptospirosis as a possible cause, because the organism or its DNA is occasionally identified in the intraocular fluids of horses with negative serologic titer results. Interpretation of serologic test results may be further confounded by the occurrence of positive serologic titer results for *Leptospira* in horses without uveitis. Vitreal titers for *Leptospira* may also be elevated, although again the value of this test remains questionable.

Numerous serologic studies have shown widespread exposure (up to 30%) of the equine population to a variety of serotypes of *Leptospira* in North America, Britain, Europe, and Australia. Serotypes associated with the disease include *pomona, bratislava, autumnalis, grippotyphosa, canicola, icterohemorrhagiae, hardjo,* and *sejroe.*

There are at least two main theories as to the role of *Leptospira* in ERU. In the first theory, ERU after infection with *Leptospira* is primarily an immune-mediated disorder in which the organism is no longer present. In this scenario autoimmune inflammation tends to "burn out" as antiinflammatory regulatory cells get the upper hand in an active attack, leading to a clinically quiescent period. Recurrent active periods may be the result of the autoimmune response shifting from one site to another on the same autoantigen (intramolecular spreading) or to another entirely different autoantigen (intermolecular spreading). This theory is supported by the responsiveness of the disease to immunosuppressive therapy, which, if viable organism were to be present in the eye, would be expected to ultimately result in an exacerbation of the inflammation. Alternatively, it has been theorized that persistence of *L. interrogans* in the vitreous humor of horses with ERU can induce and maintain an autoimmune uveitis. During the periods between overt episodes, the number of leptospiral organisms may decline to such a level that overt inflammation is not clinically detectable, and antibody titers decline. When the antibody titer falls below a certain threshold, bacterial numbers may increase, resulting in a resurgence of antibodies that cross-react with host antigens, leading to greater inflammation, damage to adjacent tissues, and, perhaps, recognition of new antigenic epitopes. This theory is supported by the observation that infusion of antibiotics into the vitreal cavity in conjunction with a surgical vitrectomy may greatly reduce the frequency of recurrent episodes.

Clinical Signs. Clinical signs vary with the phase of the disease (Figure 11-26).

Active Phase. Clinical signs in the active phase are as follows:

- Marked blepharospasm
- Photophobia
- Lacrimation
- Pain
- Protrusion of the third eyelid
- Corneal edema
- Scleral injection
- Aqueous flare (with/without hypopyon)
- Miosis
- Thickened, infiltrated iris
- Anterior and posterior synechiae
- Fibrinous clots in anterior chamber
- Decreased IOP (occasionally increased)
- Depigmented butterfly lesions near optic disc (Figure 11-27)
- Any of the quiescent signs

In an animal with onchocerciasis, the following may be seen in addition to the typical ocular lesions of ERU:

- Focal dermatitis on the head, ventral thorax, and neck
- Vitiligo affecting the scrotum, lateral canthus, or lateral conjunctival limbus

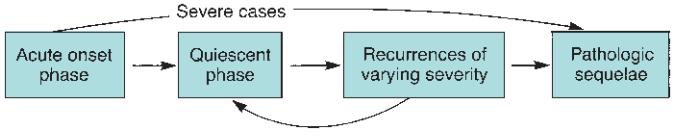

Figure 11-26. Clinical course of equine recurrent uveitis.

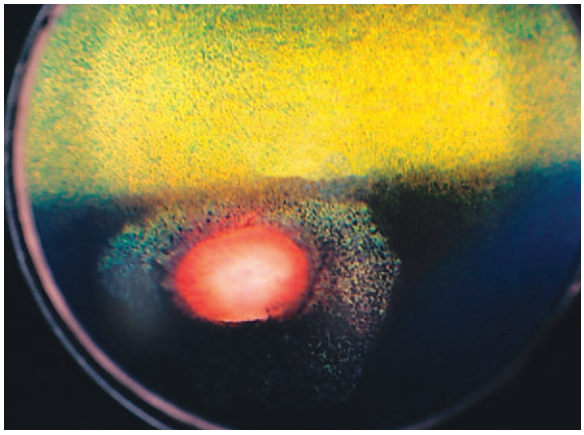

Figure 11-27. Wing-shaped hypopigmented lesions nasal and temporal to the optic disc ("butterfly lesions") are suggestive of previous uveitis. (Courtesy University of Wisconsin–Madison Veterinary Ophthalmology Service Collection.)

- Focal corneal opacities at the lateral limbus
- Hyperemia and chemosis of the perilimbal temporal conjunctiva

In the active phase, rapid intensive treatment is mandatory to prevent severe complications (e.g., synechiae, cataract, retinal detachment). Most active periods last several days to weeks.

Quiescent Phase. Typically an active period is followed by a quiescent phase of variable duration. Although inflammation may be clinically minimal or undetectable in the quiescent phase, histologic signs of inflammation and altered vascular permeability continue. During the quiescent phase immunologically active cells and cytokines also persist, and new antigenic epitopes or autoantigens may be recognized—prompting a resurgence of inflammation (Figures 11-28 and 11-29). It is not uncommon for horses in the quiescent phase to be offered for sale by unscrupulous individuals who represent the horse as "sound" or by those who are unaware of a horse's past history.

Clinical signs most likely to be seen during clinical examination of horses in the quiescent phase are as follows (Figures 11-30 and 11-31):

- Corneal opacity
- Pigment on anterior lens capsule
- Anterior and posterior synechiae
- Blunted and rounded corpora nigra
- Occluded pupil
- Iris atrophy
- Cataract (poor surgical candidates)
- Vitreous bands and opacities
- Butterfly lesions or retinal detachment
- Phthisis bulbi
- Partial or complete loss of vision

The presence of inflammatory sequelae in an equine eye indicates the possibility of ERU.

Treatment. In general the number of medications and frequency of the therapy are adjusted in accordance with the severity of the clinical signs. Mild disease may be treated with topical therapy alone, whereas more severe inflammation typically demands systemic therapy as well. Initial therapy usually includes the following measures:

1. Attempt to establish a definitive etiologic diagnosis, and specifically address the cause if possible.
2. Ensure good husbandry practices: Place the horse in a dark stall to relieve photophobia. Prevent ocular trauma by mowing pastures and removing sharp objects from the environment. Reduce contact with cattle and wildlife that may harbor leptospirosis, prevent access to ponds and swampy areas, and ensure good insect and rodent control. Minimize stress, ensure a good diet, and employ an optimal deworming schedule. Vaccinations should be optimized for each patient and based on the horse's use and specific needs. Multiple vaccinations should be spaced at least 1 week apart so as to avoid excessive antigenic stimulation and potential exacerbation of the disease.
3. Atropine ointment (1%) applied 1 to 4 times a day. This medication reduces pain by relaxing the ciliary muscle, aids in the prevention of synechia, and may help stabilize the blood-aqueous barrier. Atropine should be discontinued or reduced in frequency if the horse shows reduced gut motility and/or colic. Resistance to pupillary dilation is an indicator of the severity of the uveitis, the presence of synechia, or both. Once the uveitis is controlled, the pupil may remain dilated for days to weeks, especially if the drug was used frequently during an acute attack.
4. Systemic NSAIDs (listed here in order of potency—use only 1 at a time):
   - Flunixin meglumine 0.25 to 1.0 mg/kg q12h, IV, IM, or PO for 5 days; then, if required by the severity of the inflammation and if patient is appropriately monitored for gastric and renal side effects, 0.25 mg/kg PO q12-24h on a more long-term basis. If after 5 days systemic antiinflammatory therapy is still required, many ophthalmologists switch from flunixin meglumine to phenylbutazone. Flunixin meglumine may also facilitate pupillary dilation by atropine because endogenous prostaglandins can induce miosis by directly acting on the iris sphincter muscle; this action is blocked by NSAIDS but not by atropine.
   - Phenylbutazone 1 g per adult horse (or up to 4.4 mg/kg) q12-24h IV or PO. This drug typically is used after a 5-day course of flunixin meglumine if additional systemic antiinflammatory therapy is required. On occasion, with appropriate monitoring for gastric and renal toxicity, it is used as long-term therapy in an effort to reduce the frequency and severity of acute episodes, especially if aspirin is ineffective at such reductions.
   - Aspirin 25 mg/kg PO q12-24h (12.5 g/500 kg). Typically this agent is used in horses in which long-term topical antiinflammatory therapy cannot prevent recurrent outbreaks and long-term systemic NSAID therapy is required.
   - Consider ranitidine (6.6 mg/kg q8h) and sucralfate (20 mg/kg PO q8h) or omeprazole (4 mg/kg PO q24h) for gastric ulcer prophylaxis in foals.
5. Topical corticosteroids (e.g., 0.1% dexamethasone ointment, 1.0% prednisolone) applied every 1 to 6 hours, depending on severity. Long-term therapy is often required, and it is generally advisable to treat an acute episode for at least 2 weeks after the apparent resolution of all signs of

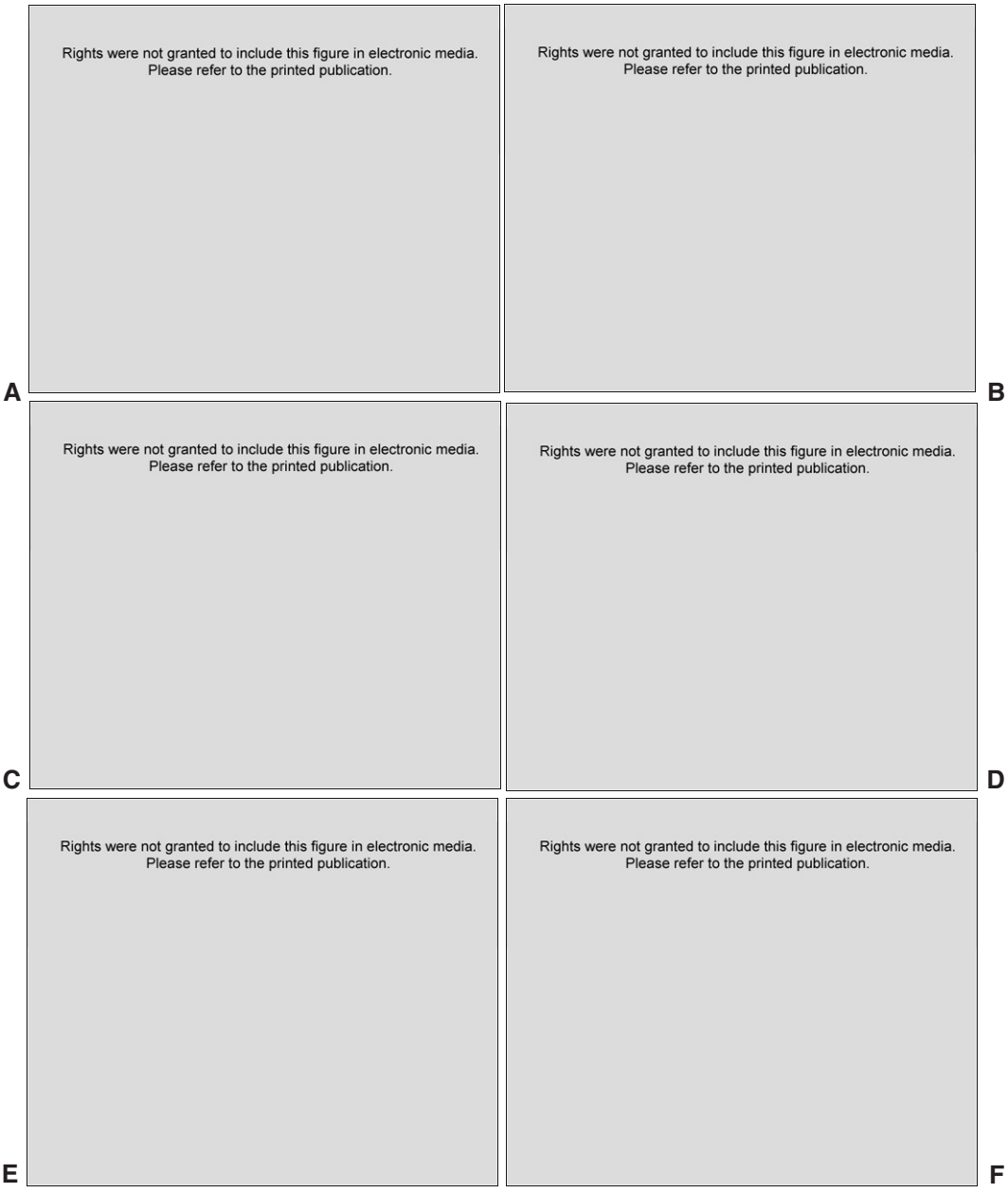

Figure 11-28. Histologic appearance of eyes from horses with experimental equine recurrent uveitis. **A,** Normal equine retina. *Bar,* 25 µm. **B,** Normal equine retina stained with antibodies to retinal S-antigen (S-Ag). Photoreceptor outer segments were clearly labeled *(red, *). Bar,* 20 µm. **C,** Affected horse with complete destruction of retinal architecture associated with immune-mediated disease directed against retinal S-Ag. CD3+ T cells *(brown)* are infiltrated around retinal neuronal cells. Leftover retinal pigment epithelial cells *(RPE)* and neovascularization were visible in the retina *(*). Bar,* 25 µm. **D,** Retinal infiltration by T cells (CD3+; *brown, arrows*). Destruction of photoreceptor outer segments with some remaining cells from the inner or outer nuclear layer and formation of epiretinal gliosis *(EG). NL,* Nuclear layer; *bar,* 15 µm. **E,** Severely destroyed retina in affected horse. Infiltration of CD3+ T cells *(brown, arrows)* in the nuclear layer *(NL)* of the remaining photoreceptor cells (visualized by red staining for S-Ag) and in the neuronal cell layer at the borderline to a severe epiretinal gliosis *(EG). Bar,* 40 µm. **F,** Subconjunctival lymphoid follicle (CD3+ cells stain red). **,* Sclera; *bar,* 120 µm. (From Deeg CA, et al. [2004]: The uveitogenic potential of retinal S-antigen in horses. Invest Ophthalmol Vis Sci 45:2286.)

active inflammation. In many patients long-term topical corticosteroid therapy is required to reduce the frequency and severity of subsequent attacks.

Additional approaches that can be used in unusually severe cases or cases refractory to the preceding approaches are as follows:

1. Topical NSAIDs (e.g., flurbiprofen 0.03%, 0.1% diclofenac, or another topical NSAID applied every 6 hours): These agents are not as potent as topical corticosteroids in ERU therapy, but in severe cases they may be used in addition to topical corticosteroids. Alternatively, they may be used long term, either alone or with topical corticosteroids in an effort to prevent recurrent

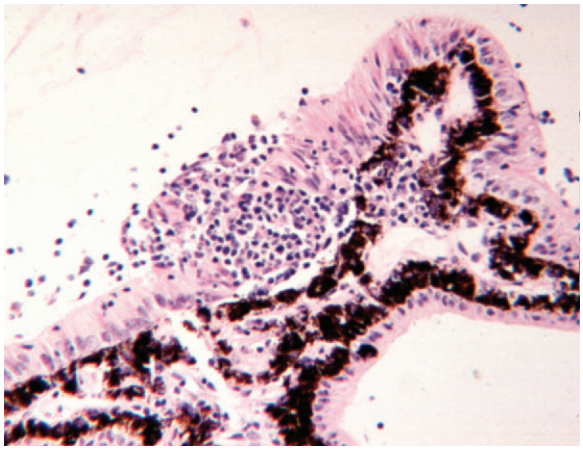

Figure 11-29. Lymphocytic inflammation of the ciliary body of a horse with chronic equine recurrent uveitis. (Courtesy Dr. Richard R Dubielzig.)

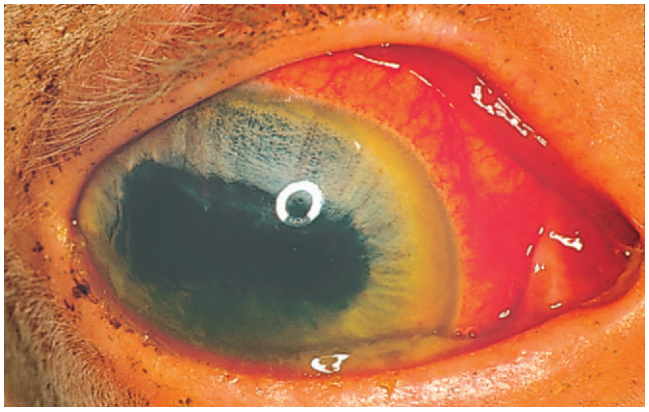

Figure 11-30. Acute equine recurrent uveitis. Note the extensive conjunctival hyperemia, miosis, and blue-green hue to the iris. The yellow serum of horses often makes a blue iris appear green.

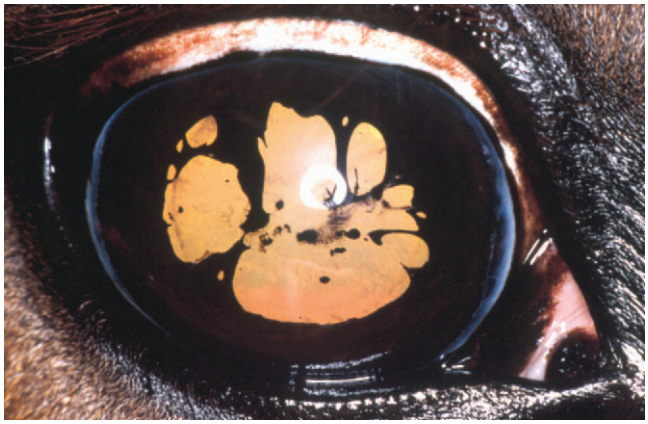

Figure 11-31. Chronic equine recurrent uveitis. The iris is hyperpigmented. Note also the numerous posterior synechiae and early cataract formation.

episodes. Topical NSAIDs can slow corneal epithelialization.

2. Cyclosporine A: Topical 0.2% cyclosporine ophthalmic ointment or 2% cyclosporine in oil applied every 6 to 12 hours has been suggested to be of value in the treatment of ERU. Because of the relatively limited intraocular penetration of this compound when applied topically, however, its efficacy appears to be somewhat less than that of topical corticosteroids. Experimentally, an intravitreal sustained-release insert containing cyclosporine has shown considerable promise in the treatment of ERU.
3. Subconjunctival corticosteroids (triamcinolone acetonide): Reported dosages for triamcinolone acetonide vary greatly from 1 to 2 mg per eye, to 20 mg per eye, to 40 mg per eye as often as every 1 to 3 weeks. Usual duration of action is 7 to 10 days. The major concern with this drug is that it creates a strong predisposition for bacterial and fungal keratitis and that, unlike topically applied corticosteroids, it cannot be withdrawn if the disease should occur. Therefore it is typically used as an adjunct to topical corticosteroids in the acute phase in especially severe cases or when the owner has difficulty medicating the horse as often as required. *Note:* The sustained-release vehicle in methylprednisolone acetate may result in an unsightly plaque and irritating granuloma.
4. Systemic corticosteroids (e.g., dexamethasone 5 to 10 mg/day PO or 2.5 to 5.0 mg daily IM or oral prednisolone 0.5 mg/kg q24h): In general, because of frequent adverse effects, systemic corticosteroids are used only as a last resort in the treatment of ERU. They can be considered in unusually severe cases or when the inflammation is refractory to systemic NSAIDS and topical corticosteroids. Side effects include laminitis and gastrointestinal upset.
5. Antibiotic therapy in horses with presumed leptospiral-associated uveitis: The efficacy of this therapy remains speculative, and side effects are not uncommon. Drugs that have been suggested include streptomycin (11 mg/kg IM q12h) and a 4-week course of oral doxycycline (10-20 mg/kg q12h). In one study by Gilmour et al., however, doxycycline at 10 mg/kg q12h orally did not result in appreciable drug concentrations in the aqueous humor or vitreous of normal eyes. Some researchers believe that the efficacy of vitrectomy for this disorder is due to the use of gentamicin in the irrigation fluid as much as the procedure itself. This theory has prompted some ophthalmologists to give a single intravitreal injection of 4 mg of gentamicin in an effort to prevent or eliminate recurrent episodes in severely affected eyes. Gentamicin injections, however, should be made with extreme caution because the drug may cause retinal degeneration, cataract formation, intraocular inflammation, endophthalmitis, and irreversible vision loss.
6. Surgical vitrectomy via a pars plana approach has been advocated by some workers to reduce the frequency and severity of attacks of ERU. The rationale for its use is based on the hypothesis that persistent organisms within the vitreal cavity (and perhaps the uveal tract) are capable of perpetuating an immune-mediated uveitis. Controlled clinical trials have yet to demonstrate the efficacy of this procedure, and cataracts are a common postoperative complication.
7. Vaccination is controversial. No approved vaccine is available for horses. The cross-reactivity of leptospiral antigens with normal constituents of the equine eye suggests that vaccination may actually cause the disease in some animals. Vaccination of seronegative horses with a multivalent bovine vaccine, with appropriate informed consent, may help suppress a herd outbreak. Vaccination as an adjunctive therapy in horses with ERU, however, failed

to slow the progression of the disease in one study (Rohrbach et al., 2005).
8. Enucleation is, on occasion, the only means of effectively treating a blind, painful globe.

Onchocerca Uveitis. *O. cervicalis* lives in the ligamentum nuchae of the horse. The microfilariae released by these adults migrate to the skin and ocular region and are transmitted by midges of the genus *Culicoides* and mosquitos. Ocular lesions are associated with the migration of the microfilariae from the ligamentum nuchae to the skin, some entering vessels of the bulbar and palpebral conjunctiva. The microfilariae are most readily found in the conjunctiva adjacent to the temporal limbus and in the corneal stroma adjacent to this area.

In 1971 Cello described the corneal lesions as "superficial subepithelial fluffy or feathery white opacities 0.5 to 1.0 mm in diameter, located 1 to 5 mm from the temporal limbus." The adjacent conjunctiva was hyperemic and chemotic, but biomicroscopic examination was required to demonstrate the corneal lesions.

The ocular lesions of onchocerciasis alone, including conjunctival vitiligo, are insufficient to indicate the presence of microfilariae. Unilateral ocular infestations with microfilariae may also occur.

ERU is said to be caused by the dead microfilariae or to be mediated by immunopathologic mechanisms involving IgE. Diethylcarbamazine stimulates IgE antibody responses. This feature, rather than a reaction by the host to killed microfilariae, may explain the inflammation seen after its administration.

Microfilariae are demonstrated by removing, under local anesthesia, (1) a small piece of conjunctiva from the affected area or (2) a piece of skin from the ventral thoracic midline. The tissue is minced with scissors and placed in 5 mL of saline at 37° C for 30 to 50 minutes (e.g., in a small vial in the clinician's pocket). The supernatant is centrifuged and examined for motile microfilariae. Alternatively, the tissue may be examined in saline on a slide immediately after collection. Interpretation of such slides must be made in association with other clinical findings, because many horses without ERU have microfilariae.

Microfilaricides must not be used during acute uveitis.

Treatment. The treatment is the same as that for *Leptospira*-associated uveitis. After the inflammation has subsided, ivermectin 0.2 mg/kg may be administered systemically. A single dose of ivermectin 0.2 mg/kg was found to be very effective in eliminating microfilariae from the skin of horses afflicted with dermatitis due to *O. cervicalis.* Alternatively, diethylcarbamazine 4 mg/kg daily is administered in the food for 21 days. At the first sign of recurrent inflammation during treatment, corticosteroid therapy is begun. In endemic areas prophylactic feeding of diethylcarbamazine and aspirin is recommended throughout the season when vectors are present. Aspirin may also be used continuously. The routine use of ivermectin and other highly effective anthelmintics appears to have substantially reduced the incidence of onchocercal uveitis in the United States.

UVEITIS DUE TO *Dirofilaria immitis*. Mature and immature adult dirofilaria are infrequently reported in the anterior chamber of dogs. Treatment is surgical removal. If adulticides are used while adult worms are present in the anterior chamber, severe uveitis and endophthalmitis may result. Severe endophthalmitis has also been observed when microfilariae are present in the eye and an adulticide is administered.

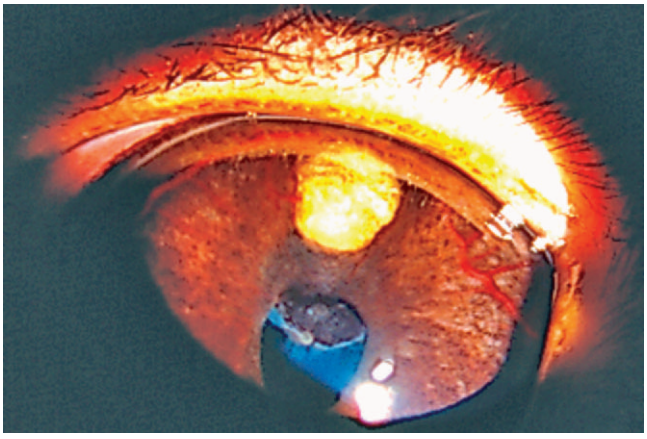

Figure 11-32. Uveitis and focal lens capsule rupture with cataract formation associated with the protozoan *Encephalitozoon cuniculi* in a rabbit.

PHACOCLASTIC UVEITIS IN RABBITS. An unusual form of uveitis associated with apparent spontaneous lens capsule rupture, phacoclastic uveitis, occurs in the rabbit and frequently results in enucleation (Figure 11-32). Organisms believed to be *Encephalitozoon cuniculi* have been identified in affected lenses. Clinical signs of infection include a white or yellowish uveal or anterior chamber mass that progresses to severe uveitis and glaucoma that is usually refractory to treatment. Early lens removal has been suggested as a method of treatment to prevent development of uveitis.

TOXIC UVEITIS. The eye is exquisitely sensitive to bacterial endotoxins, and amounts as small as a few nanograms are capable of inducing substantial uveitis. Other toxic agents are pilocarpine and other topical parasympathomimetics as well as topical prostaglandins used in the treatment of glaucoma (e.g., latanoprost). Ethylene glycol poisoning has been associated with anterior uveitis in dogs. Sulfa-containing drugs and those associated with thrombocytopenia or coagulopathies have also been associated with uveitis (usually associated with hemorrhage).

TRAUMA

Traumatic Uveitis

Trauma is a common cause of uveitis in domestic animals.

Uveitis may result from either blunt or sharp trauma to the globe or may occur after intraocular surgical procedures. Therapy is the same as that for other forms of uveitis, although topical corticosteroids should be avoided if a corneal erosion or ulceration is present; in this case topical NSAIDS may be used, although they, too, may impair corneal epithelialization and there is some potential for topical NSAIDS to elicit a corneal melt. Topical and systemic NSAIDs are also typically avoided if significant intraocular hemorrhage is present. If the corneal epithelium is not intact, a topical antibiotic such as neomycin–polymyxin B–bacitracin combination product applied every 6 to 8 hours should be used prophylactically. If the globe has been penetrated, the wound may require suturing and systemic antibiotics in addition to topical therapy. Traumatic uveitis is aggressively treated in

the horse because a traumatic breakdown of the blood-aqueous barrier may increase the risk of recurrent episodes of uveitis.

In severe ocular trauma, early and vigorous treatment is required to prevent permanent ocular damage and, perhaps, repeated episodes of uveitis.

In many cases the long-term prognosis of traumatic uveitis is determined more by the nature of the injury than by the therapy that was chosen.

Common uveal injuries are as follows:

- *Iris prolapse:* Protrusion of a portion of the iris through a corneal or scleral perforation
- *Hyphema:* Hemorrhage into the anterior chamber
- *Staphyloma:* A weakened or protruding lesion in the cornea or sclera into which a portion of the uvea protrudes from the inside; the uveal tissue usually adheres to the cornea or sclera
- *Concussion*
- *Iridodialysis:* Tearing of the iris from the ciliary body at its root. This condition is uncommon in domestic animals. Iris prolapse and hyphema are discussed in greater detail later.

Iris Prolapse

Iris prolapse is a common sequela to penetrating corneal wounds or ruptured corneal ulcers. The iris is carried forward into the corneal defect by escaping aqueous. Emergency treatment of such injuries is described in Chapter 19. When iris passes through such a corneal defect, its vascular supply is usually compromised, resulting in venous congestion and edema. This changes the appearance of the protruding mass so that it commonly looks like uvea-colored mucus adhering to the cornea.

Signs

Clinical signs of iris prolapse are as follows:

- The color of the prolapsed portion becomes lighter than the remaining iris.
- The protruding iris tissue forms a mound on the cornea.
- The tissue has a gelatinous mucoid appearance and frequently attracts adhering strands of conjunctival mucus.
- The pupil is eccentric as a result of traction of the protruding iris tissue.
- The corneal wound is often obscured by the edematous iris tissue. Protrusion of the ciliary body occurs most commonly in horses as a result of scleral rupture posterior to the limbus after blunt trauma.

Treatment

If the corneal wound is small, iris prolapse may be treated temporarily with a third-eyelid flap and topical and systemic antibiotic solutions until specialized assistance is available. In larger wounds requiring immediate repair, an attempt is made to replace the iris with an iris spatula before the cornea is sutured. If this is not possible, the protruding piece may be carefully excised with the use of an electrosurgical unit. The cornea is sutured, and the anterior chamber reconstituted with balanced salt solution or an air bubble. *Caution:* If the major arterial circle of the iris is transected, profuse intraocular hemorrhage can result. Enucleation or evisceration and intrascleral prosthesis are alternative therapies if the eye is blind.

Visual Outcome and Ocular Survival after Iris Prolapse in Horses

Iris prolapse is usually associated with a ruptured corneal ulcer or full-thickness corneal laceration. In a review of 32 cases, combined medical and surgical therapy (primary closure with or without a conjunctival graft) was successful in saving vision of 40% of eyes with perforating corneal disease (ulcers or stromal abscesses) and 33% of eyes with perforating lacerations (Chmielewski et al., 1997). Complications resulting in blindness included phthisis bulbi, extensive keratomalacia, and endophthalmitis. A favorable visual result was more likely in horses presented for specialist care with ulcers of less than 15 days' duration or corneal lacerations smaller than 15 mm.

HYPHEMA

The emergency treatment of hyphema is discussed in Chapter 19.

Etiology

Hyphema may be idiopathic or may result from many factors, such as the following:

- Traumatic disruption of a uveal blood vessel: sharp or blunt trauma, severe pressure around the neck as in choking or increased intrathoracic pressure in severe traumatic compression of the chest or dystocia
- Fragility of vessel walls, especially preiridal fibrovascular membranes that form in response to chronic disorders causing intraocular hypoxia (e.g., inflammation, glaucoma, retinal detachments, neoplasia, or after intraocular surgery)
- Clotting disorders, platelet disturbances, and blood dyscrasias
- Highly vascularized tumors
- Severe uveitis
- Retinal dysplasia with rupture of vessels
- Systemic disease (e.g., tropical canine pancytopenia, Rocky Mountain spotted fever)

Erythrocytes released into the anterior chamber undergo phagocytosis by the cells lining the trabecular meshwork. The surface of the iris provides fibrinolysin, which aids in resolving clots in the anterior chamber. The sequelae of hyphema often have a greater impact on the ultimate visual outcome than the hemorrhage itself (Figure 11-33).

Most hyphemas are small and are resorbed spontaneously in a few days.

Treatment

The treatment of hyphema is controversial because of conflicting experimental results with different drug regimens in different species. In the vast majority of patients surgical drainage of the hyphema is not useful because rebleeding is frequent. The

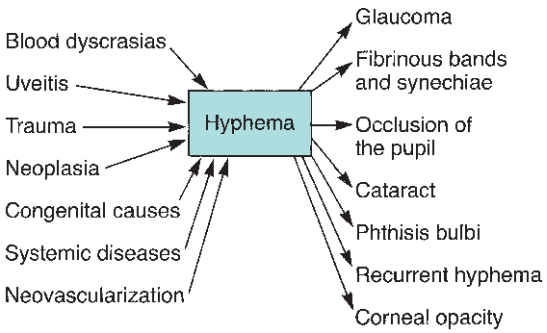

Figure 11-33. Causes and effects of hyphema. (Modified from Blogg JR [1980]: The Eye in Veterinary Practice. Saunders, Philadelphia.)

procedure may be considered, however, in patients with glaucoma secondary to blood in the anterior chamber in which the cause of the bleeding has been controlled. Although different methods are used, the aims are to:

- Identify the cause
- Prevent recurrent bleeding
- Control uveitis
- Limit the sequelae of uveitis

Surgical removal of clots from the anterior chamber is generally not an effective therapy.

The following treatment is recommended for hyphemia:

1. Prevent further trauma by immediate and enforced cage or stall rest.
2. Administer corticosteroid drops (dexamethasone 0.1%, prednisolone 1%) three times daily. NSAIDs are not used because of their effects on platelets and blood clotting.
3. Intracameral tissue plasminogen activator may be beneficial in select patients.

Additional Therapy for Mild Hyphemia

1. If the hyphema is not secondary to uveitis, administer 1% to 2% pilocarpine drops three times daily and attempt to dilate the pupil every second day with phenylephrine (10%) to prevent synechia formation.
2. If the hyphema is secondary to uveitis, dilate the pupil with 1% atropine every 8 to 12 hours.
3. Monitor IOP.

Additional Therapy for Severe Hyphemia

1. Instead of pilocarpine, use 1% atropine ophthalmic ointment or solution three times daily to relieve pain if present.
2. Administer systemic corticosteroids (e.g., prednisolone or dexamethasone) in appropriate systemic dosages.
3. Monitor IOP twice daily.
4. If glaucoma is incipient, use a topical or systemic carbonic anhydrase inhibitor or topical dipivefrin.

Recurrent Hyphema

If the hyphema is recurrent, a complete laboratory examination, including measurement of complete blood count, platelets, and clotting parameters, is indicated. In the absence of specific indications, use of vitamins C and K is not advised, nor are such agents as proteolytic enzymes or carbonic anhydrase inhibitors. Recurrent hyphema, especially with glaucoma, should prompt the clinician to rule out intraocular neoplasia as the cause of the bleeding.

UVEAL CYSTS AND NEOPLASMS

The uvea may be affected by cystic disorders that mimic neoplasia or by both primary and secondary neoplasms. Intraocular tumors frequently are also accompanied by glaucoma, intraocular hemorrhage, or chronic unresponsive uveitis.

Uveal Cysts

Uveal cysts are fluid-filled, ovoid to spherical structures that originate from the posterior pigmented epithelium of the iris or the ciliary body. Although they may represent a recessively inherited, congential uveal defect (especially in Great Danes and golden retrievers), they are often not seen until adulthood. They also are commonly seen in Boston terriers and occasionally in cats and Rocky Mountain horses. A second type of cyst in horses may be seen within the iris stroma at its base in lightly pigmented irides. Uveal cysts may also occur secondary to inflammation. The cysts either remain attached or break free and float into the anterior chamber, either singly or in groups (Figure 11-34). In the anterior chamber they may float free or adhere to the iris or corneal endothelium, occasionally obstructing the visual axis and the pupil. Deflated cysts appear as patches of pigment adherent to the corneal endothelium. In rare circumstances large numbers of cysts may push the iris root forward, causing secondary closed-angle glaucoma. Uveal cysts may be differentiated from a pigmented neoplasm or iris nevus by the ability of the cyst to be transilluminated with a bright focal light, although this feature may be sometimes difficult to appreciate in horses or very heavily pigmented cysts. In such cases ultrasonography may be required to differentiate a cyst from neoplasia.

Removal of a uveal cyst is rarely indicated but should be considered in the following circumstances:

- The pupil is obstructed, impairing vision.
- Glaucoma is impending or present owing to anterior displacement of the iris by large numbers of cysts, or multiple cysts are present and the debris liberated by their collapse may obstruct the trabecular meshwork.

Figure 11-34. Causes and effects of hyphema. (Modified from Blogg JR [1980]: The Eye in Veterinary Practice. Saunders, Philadelphia.)

- The cyst is contacting the corneal endothelium, causing corneal edema.

Cysts may be removed by aspiration under microsurgical control or deflated by laser photocoagulation.

Cystic Corpora Nigra in Horses

Corpora nigra generally occupy the central portions of the upper and lower pupillary margins. Cystic corpora nigra appear as large, smooth structures at the pupillary margin. They may obstruct the pupil enough to cause visual impairment or blindness, manifested as decreased jumping performance or head shaking.

The differential diagnosis for such cysts is as follows:

- Cystic dilation of the iris stroma (blue or lightly pigmented irides)
- Free-floating iris cysts
- Pigmented neoplasms, such as melanoma
- Hypertrophic corpora nigra
- Inflammatory nodules

Cystic dilations of the iris stroma and free-floating iris cysts in horses rarely require treatment. Cystic corpora nigra must be distinguished from neoplasms, but they do not transilluminate readily. Cystic corpora nigra have a smooth appearance, whereas melanomas and hypertrophic corpora nigra have a roughened surface. Ultrasonography may be used to distinguish cystic corpora nigra from melanoma or hypertrophic corpora nigra. Cystic corpora nigra may be removed by aspiration under microsurgical control or with laser therapy.

Primary Tumors

Of the primary uveal tumor types listed in Box 11-1, adenoma, adenocarcinoma, and melanoma are the most common. Iris nevi were discussed earlier.

Box 11-1 | Classification of primary tumors

Melanocytes
Acquired

Iris nevus
Melanocytoma (benign)
Melanoma (potentially malignant)
Diffuse iris melanoma (feline)

Ciliary Epithelium
Congenital

Benign medulloepithelioma
Malignant medulloepithelioma
Benign teratoid medulloepithelioma
Malignant teratoid medulloepithelioma

Acquired

Nonpigmented:
  Adenoma
  Adenocarcinoma
Pigmented:
  Adenoma
  Adenocarcinoma

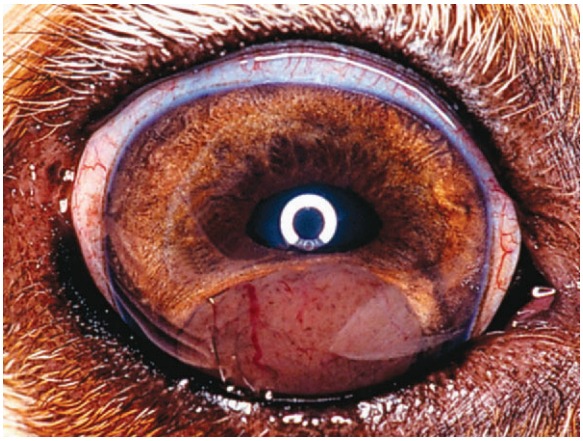

Figure 11-35. Ciliary body adenoma extending from the ciliary body through the iris and into the anterior chamber of a dog. (Courtesy University of Wisconsin–Madison Veterinary Ophthalmology Service Collection.)

Adenocarcinoma and Adenoma

Neoplasms of the ciliary epithelium are occasionally observed in dogs. Such a lesion usually appears as a single mass protruding from behind the iris into the pupil (Figure 11-35). The mass may be pigmented or unpigmented, depending on whether it arose from pigmented or unpigmented ciliary epithelium, and must be distinguished from melanocytoma or potentially malignant melanoma of the same site. The neoplasms infrequently infiltrate anteriorly into the drainage angle and iris, elevating IOP. The extent of the lesion may be outlined by transillumination and reflected light from the tapetum, and by ultrasonography. Treatment consists of removal of the tumor and adjacent ciliary body (iridocyclectomy), frequently including replacement of the defect with a scleral graft, laser cyclodestruction, or, if the tumor is extensive, enucleation. Provided that the tumor has remained within the globe, the prognosis for survival is good.

Melanocytoma and Melanoma

Although the vast majority of uveal melanomas are benign, malignant tumors arising from the iris, ciliary body, or, less commonly, the choroid do occur. They are most common in dogs and cats and less common in horses and cattle. Mitotic index is a more useful indicator of behavior and prognosis in dogs than the histologic criteria used for human ocular melanomas. The potential for metastasis is present, but different studies demonstrate wide variation in observed rates, making generalizations difficult. Intraocular and palpebral melanomas in cats have a greater tendency to metastasize and are more malignant than those in dogs, with higher rates of mortality and metastasis.

Melanoma in dogs and cats, unlike that in humans, occurs more frequently in the iris and ciliary body (Figure 11-36) than in the choroid and has a reasonable prognosis for survival if the eye is enucleated before the tumor has penetrated the sclera. Penetration may occur via ciliary arteries, veins, or nerves, by direct extension, or via the optic nerve. In a study of feline ocular melanomas by Patnaik and Mooney (1988) 10 of 16 uveal melanomas had metastases before enucleation. On the basis of tumor behavior in only three animals in the same study, feline palpebral melanomas may have a high rate of metastasis.

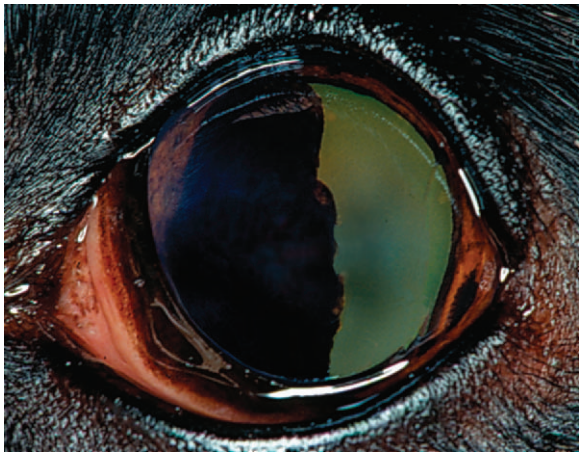

Figure 11-36. Ciliary body tumor (melanoma) in a dog. The mass is posterior to the iris but extends through the iris nasally. (Courtesy University of Wisconsin–Madison Veterinary Ophthalmology Service Collection.)

Figure 11-37. Epibulbar melanoma in a German shepherd. This benign tumor must be differentiated from an intraocular tumor that has broken through the sclera.

Epibulbar melanomas occur in dogs, cats, and horses (Figure 11-37). In dogs the average age of onset is 6 years, the most common site is the superior limbus, and the German shepherd has a higher incidence. These tumors grow slowly and are treated with local surgical excision, cryotherapy, or laser photoablation. The resulting defect may require structural support with a graft of autogenous or heterologous tissue or a synthetic material. Small, slow-growing tumors in older animals or animals with limited life span may simply be observed.

CLINICAL SIGNS. Melanomas usually cause the following clinical signs:

- Change in color or visible mass in the iris
- Uveitis or endophthalmitis due to necrosis of the tumor; cornea is often opaque
- Hyphema
- Secondary glaucoma

Melanomas often cause secondary glaucoma.

TREATMENT. Treatment of melanomas and melanocytomas consists of the following measures:

1. If the tumor is small or localized, local excision with *iridectomy* or *iridocyclectomy,* or alternatively with diode or neodymium:yttrium-aluminum-garnet (Nd:YAG) laser photocoagulation may be considered. By the time clinical signs are present, many tumors are too large for this treatment.
2. Enucleation of the globe is often mandated by the presence of intractable glaucoma, uveitis, or hyphema.

The prognosis for the animal's survival after enucleation is good; in one study, only 7 of 129 canine uveal melanomas had confirmed metastases. If there is any indication of scleral penetration, *orbital exenteration* is performed in an attempt to remove tumor cells. Frequent postoperative examinations (every 3 months for a year, then annually) are advisable, with special attention given to the submandibular, retropharyngeal, and bronchial lymph nodes. Adjunctive chemotherapy or radiation therapy may be used, although the efficacy of these treatments is unclear.

Primary Feline Ocular Sarcomas

Posttraumatic sarcomas of the feline eye have been reported to occasionally occur months to years after severe ocular trauma. Although the vast majority of cats with primary ocular sarcomas have a history of penetrating trauma that damaged the lens and/or other intraocular structures, a few cases have been described in which there is no history of trauma, infection, or ocular surgery. In addition to clinical signs consistent with the injury, signs of ocular sarcoma are chronic, relatively unresponsive uveitis, glaucoma with buphthalmos, and a previously phthisical eye that is now enlarging. Metastasis or local recurrence after enucleation is common. Because it lines the inner surface of the globe, the tumor commonly extends into the orbit via the optic nerve. Metaplastic bone has also been observed in ocular sarcomas. Although some researchers have suggested removal of all traumatized or phthisical feline eyes to prevent development of this rare tumor, the value of this approach remains to be determined.

Feline Diffuse Iris Melanoma

The diffuse iris melanoma seen in cats has specific features that differentiate it from other anterior uveal tumors. The tumor is often *very* slowly progressive, arising from pigmented areas on the anterior surface of the iris (see Figure 11-22) and perhaps eventually involve the iridocorneal angle, causing secondary glaucoma (Figure 11-38). In some cats, however, the tumor is rapidly progressive and quick to metastasize. Although the tumor is potentially malignant, the risk for metastasis in the majority of cats appears to be relatively low. Cats with this disorder should be regularly evaluated by a veterinary ophthalmologist to ascertain whether or when enucleation may be indicated. In single or multiple early iris melanomas that are progressing, tumor ablation with laser therapy may be useful. Because the disease is slowly progressive, the clinical conflict is whether to simply observe the patient or enucleate a functional eye if persistence of such an eye may present a risk to the animal's life through metastasis. Many affected cats, however, even those with metastatic disease, may live for long periods with few ill effects.

All pigmented iris tumors in cats should be referred to a veterinary ophthalmologist for evaluation and long-term therapy.

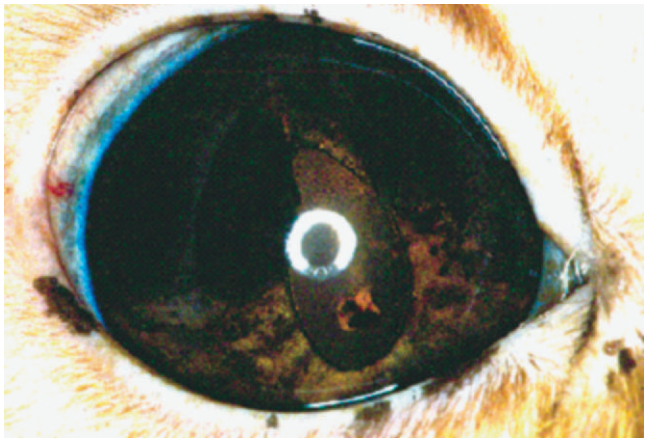

Figure 11-38. Diffuse iris melanoma in a cat. The iris is diffusely infiltrated, and the pupil is dyscoric. Enucleation is advised.

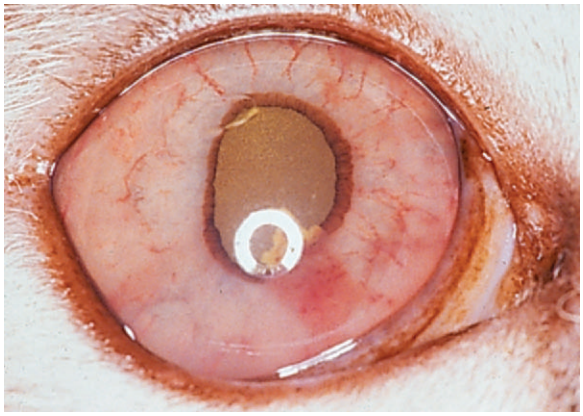

Figure 11-39. Lymphosarcoma of the iris of a cat. The iris is diffusely thickened with neoplastic lymphocytes. (Courtesy University of Wisconsin–Madison Veterinary Ophthalmology Service Collection.)

Criteria for considering enucleation of an eye with a progressively enlarging hyperpigmented iridal lesion are as follows:

- Noticeable thickening of the iris stroma with distortion of the pupil or its mobility
- Involvement of the ciliary body
- Extension into the sclera
- Secondary glaucoma
- Intractable uveitis

Secondary Tumors

With the exception of lymphosarcoma, tumors metastasizing to the uvea are uncommon. Although any metastatic tumor may potentially spread to the eye, the most common tumors to do so in dogs are mammary carcinoma, hemangiosarcoma, thyroid, pancreatic and renal carcinomas, malignant melanoma of the skin, seminoma, and rhabdomyosarcoma. Any metastatic tumor may spread to the eye, however.

Lymphosarcoma

Ocular manifestations of lymphosarcoma occur in the dog, cat, cow, and horse (see Chapter 18). In the dog ocular manifestations are clinically similar to those of uveitis and endophthalmitis; they include iridal swelling, hyphema, aqueous flare, retinopathy and retinal detachment, conjunctivitis, keratitis, noninflammatory chemosis, corneal edema with vascularization, KPs, intrastromal corneal hemorrhage, miosis, hypotony, ciliary injection, and secondary glaucoma. Approximately 40% of dogs with lymphosarcoma show some ocular signs. Histologically, the iris and ciliary body are more frequently affected than the choroid. Dogs with ocular signs may have a poorer prognosis for long-term remission and response to chemotherapy. Detailed consideration of chemotherapy for lymphosarcoma is beyond the scope of this book; however, animals blinded by lymphosarcoma *may* recover vision once chemotherapy with one of the standard regimens is begun. Adjunctive topical therapy with corticosteroids, atropine, and antiglaucoma drugs should be considered in animals with uveitis secondary to lymphosarcoma.

In cats similar but less common ocular lesions occur in lymphosarcoma, myeloproliferative disease, reticuloendotheliosis, feline immunodeficiency virus infection, and feline leukemia virus infection (Figure 11-39). Older male cats are more frequently affected with ocular lymphosarcoma; ocular signs were the initial presenting sign in more than 50% of affected cats in a retrospective pathologic study by Corcoran et al. (1995). In cattle ocular lesions in lymphosarcoma are restricted to infiltration of orbital tissues, often resulting in exophthalmos with exposure keratitis. Up to 10% of cattle with lymphosarcoma may have exophthalmos.

Lymphosarcoma should be considered in cattle with exophthalmos.

In poultry, infiltration of the iris and uveal tract with a change in color to bluish gray ("pearly eye") is seen in Marek's disease; it is called *epidemic blindness.*

MISCELLANEOUS DISORDERS

Iris Hypoplasia

In congenital iris hypoplasia in color-dilute, albinotic, and subalbinotic animals, the iridal holes may progress over time, leaving large spaces in the iris.

Iris Atrophy

Several types of iris atrophy occur, as discussed here.

Primary Iris Atrophy

A slowly progressive iris atrophy in previously normal adults occurs in dogs and cats. Spaces and holes develop in the iris, often leading to dyscoria, and are especially visible on retroillumination, in which light is reflected from the tapetum back toward the examiner. The condition is especially seen in Siamese cats, miniature schnauzers, poodles, and Chihuahuas but may occur in any breed. Although the disorder is not typically associated with obvious clinical signs, anisocoria may be present, and the pupillary light reflex may be diminished or, occasionally, absent.

Secondary Iris Atrophy

Atrophy of the iris may occur after the following conditions:

- Chronic glaucoma
- Chronic recurrent uveitis
- Severe ocular trauma

Senile Iris Atrophy

Senile iris atrophy occurs in older animals of all species and is characterized by irregular pupillary margins, spaces in the iris, and sluggishness or absence of pupillary reflexes. The condition must be distinguished from secondary iris atrophy. It is common in toy and miniature poodles, miniature schnauzers, and Chihuahuas and is significant in the evaluation of patients with cataract or visual impairment (Figure 11-40).

SURGICAL PROCEDURES

Surgical procedures for primary diseases of the iris are rarely performed, even in specialty practice. Examples are as follows:

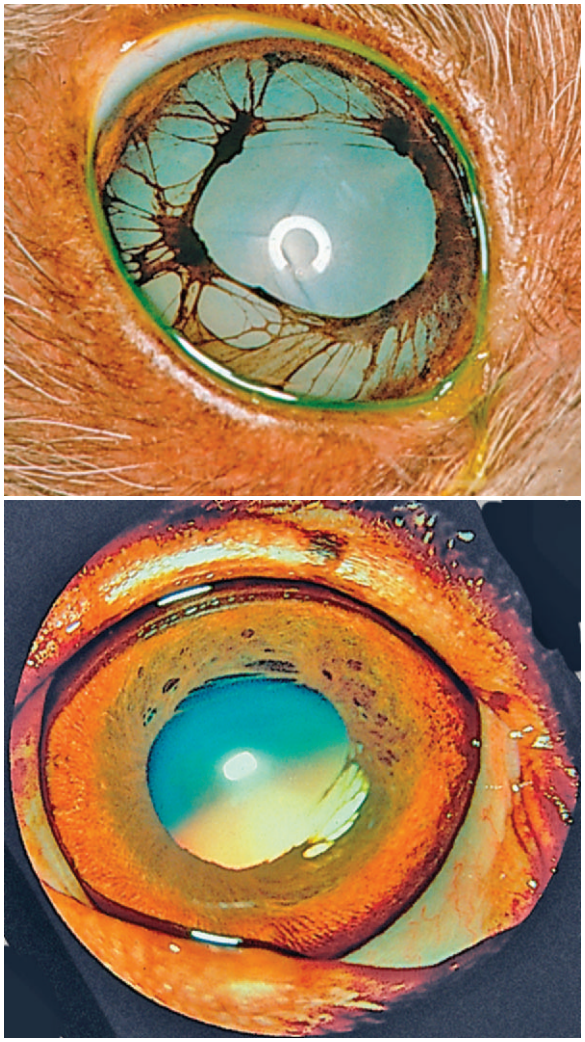

Figure 11-40. **A,** Marked iris atrophy in a miniature poodle. Full-thickness holes in the iris are readily visible. **B,** Milder form of iris atrophy. Note that the pupil is dyscoric and that the pupillary light reflex in such cases is often reduced.

- *Iridectomy* (removal of part of the iris): For focal circumscribed melanomas of the iris. Such lesions may also be treated by diode or Nd:YAG laser photocoagulation.
- *Iridocyclectomy* (removal of a portion of iris and ciliary body): For neoplasms of the ciliary body. Many neoplasms are too advanced at presentation for this procedure, but for those in the early stages the technique, although demanding, allows removal of affected tissue and salvage of the eye and vision. For larger neoplasms infiltrating the sclera, the scleral defect may be replaced with an autogenous graft.
- *Sphincterotomy* (incision of the sphincter): Performed occasionally during cataract surgery. The sphincter is cut in one or more places, if mydriasis is poor, to allow access to the lens and to reduce the chance of a small pupil postoperatively. Since the advent of NSAIDs the technique is rarely necessary. The use of iridectomy and iridencleisis for canine glaucoma has been superseded by cyclocryotherapy and laser cyclotherapy (see Chapter 12).

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