Topical Review

Acute Blindness

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Sudden loss of vision is an ophthalmic emergency with numerous possible causes. Abnormalities may occur at any point within the complex vision pathway, from retina to optic nerve to the visual center in the occipital lobe. This article reviews specific prechiasm (retina and optic nerve) and cerebral cortical diseases that lead to acute blindness. Information regarding specific etiologies, pathophysiology, diagnosis, treatment, and prognosis for vision is discussed.

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Introduction

Sudden blindness is generally a bilateral condition. Many animals compensate well for the loss of vision in one eye, and only observant owners perceive the more subtle signs of vision deficits that accompany unilateral blindness. Acute loss of vision in both eyes, on the contrary, is easily recognized by clinical signs such as bumping into stationary objects, becoming lost or disoriented in a familiar room, and hesitancy in jumping onto furniture or going up and down stairs.

There are numerous possible causes of acute blindness. Abnormalities may occur at any point within the complex vision pathway, from retina to optic nerve (prechiasm), to the visual center in the occipital lobe. Neuroanatomic localization is an important concept to guide diagnostic recommendations; identifying the underlying cause of blindness is vital to the veterinarian, to counsel owners on prognosis for return of functional vision. Additionally, some conditions that lead to blindness are, in fact, ocular manifestations of systemic diseases that may affect the animal's general health. For this reason, determining the cause of vision loss and whether the problem is of primary ocular or systemic origin should be considered as an emergency.

Neuroanatomically, the vision and pupillary light reflex (PLR) pathways are shared until just before the synapse at the lateral geniculate nucleus (LGN) (Fig 1). Thus, lesions occurring from the eye (retina and optic nerve) to the optic chiasm and optic tracts result in loss of vision (absent menace response) and abnormal (incomplete or absent) PLRs. Conversely, lesions occurring beyond the LGN only affect vision (absent menace response) but do not result in PLR abnormalities (Table 1).

Complete ophthalmic examinations, including detailed evaluation of the ocular fundus (retina and optic disc) and neuro-ophthalmic examination (most importantly, PLRs), are important first steps in establishing a diagnosis in a patient with acute blindness. Some of the diseases discussed in this article, such as retinal detachments or optic neuritis, are likely to show obvious abnormalities in ophthalmic examination results. Others, such as sudden acquired retinal degeneration syndrome, have no obvious associated ocular lesions. Consistent completion of a fundic examination and assessment of PLRs in every patient presenting with blindness is imperative to guide the veterinarian toward the appropriate diagnostic approach.

Diseases of the Retina

Congenital Abnormalities

Retinal Dysplasia

Retinal dysplasia is an atypical differentiation of the retina along with a proliferation of one or more of its layers. In dogs, there is a hereditary basis for many forms of retinal dysplasia. Additionally, viral infections,[1] vitamin A deficiency, drugs, trauma, and radiation[2] are known causes of retinal dysplasia, triggering abnormal retinal differentiation. Histologically, abnormal development of the retina is characterized by aberrant folding of various layers of the neurosensory retina into rosettes around a central lumen.

There are 3 forms of spontaneous retinal dysplasia: (1) focal or multifocal, (2) geographic, and (3) complete retinal dysplasia with detachment. Focal or multifocal and geographic retinal dysplasias are not typically associated with blindness, though geographic retinal dysplasia can result in varying degrees of vision impairment. Retinal folds (Fig 2) are a type of focal or multifocal retinal dysplasia, and do not typically result in noticeable vision impairment. In fact, retinal folds in some dogs are considered to be a
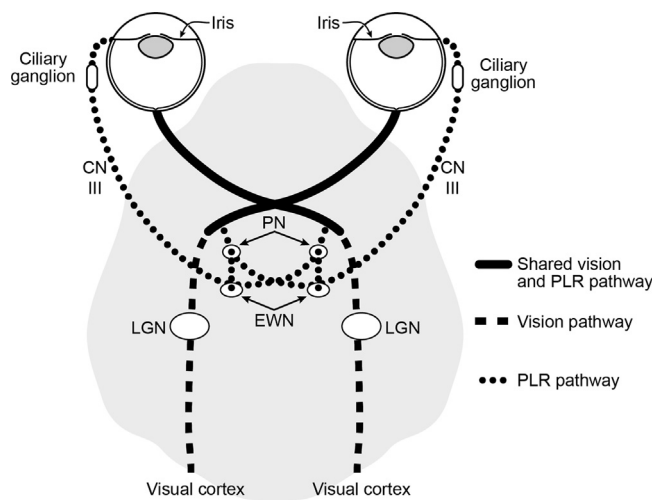
Fig. 1. Schematic diagram of the neuroanatomic pathways for vision and PLR. The vision and PLR pathway is shared before the LGN. The PLR pathway diverges before the LGN to synapse at the PN, then the EWN. The pathway then continues toward the eye with synapse occurring at the ciliary ganglion, and terminates to innervate the iris sphincter muscle. The vision pathway continues on to synapse at the LGN, then travels as optic radiations to converge in the visual cortex of the occipital lobe. Lesions occurring before the LGN, therefore, lead to blindness and abnormal PLRs, whereas lesions after this anatomic landmark affect either vision or PLR. PN, pretectal nucleus; EWN, Edinger-Westphal nucleus (also called the parasympathetic nucleus of CN III); CN III, cranial nerve III.

Fig. 2. Retinal dysplasia in a 1-year-old Airedale dog. Multiple linear to Y-shaped gray retinal folds are clustered dorsal to the optic disc.

developmental problem that disappear as the animal matures, typically by several months of age. Complete retinal dysplasia with detachment is the most severe form of the disease and results in blindness. Owing to the nature of retinal dysplasia as an abnormal differentiation of retinal layers, lesions are congenital and affected animals are diagnosed by funduscopic examination early in life. Affected young puppies are singled out from the litter by evidence of vision impairment, manifested as wandering, bumping into objects and littermates, and other signs of disorientation. Although many breeds have been reported to be affected by retinal dysplasia, some in conjunction with multiple ocular abnormalities or other nonocular defects, the severest forms causing blindness have been identified in Bedlington and Sealyham Terriers,[3] Springer Spaniels,[4] Labrador Retrievers,[5] and Samoyeds.[6] No widely accepted treatment options are available; retinal reattachment surgery, a specialized vitreoretinal surgery for certain types of retinal detachment, is not an appropriate option for complete retinal detachment caused by retinal dysplasia. The retina is inherently malformed due to abnormal differentiation; although anatomic reattachment may be possible, restoration of functional vision is not likely. Efforts are targeted at identifying affected animals by screening ocular examinations and eliminating those individuals from the breeding pool.

Collie Eye Anomaly

Collie eye anomaly (CEA) is an ocular syndrome caused by abnormal mesodermal differentiation that results in defects of the sclera, choroid, optic disc, and retinal vasculature, along with the retina. A region of choroidal hypoplasia lateral to the optic disc is
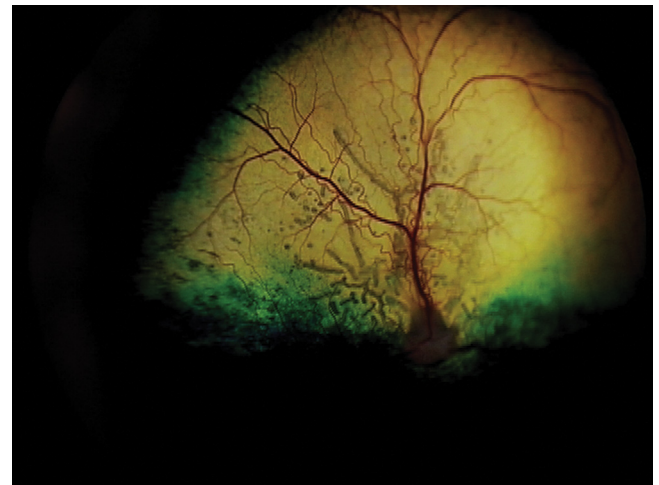
the hallmark clinical sign, and there is a spectrum for severity of lesions ranging from choroidal hypoplasia to optic disc colobomas (Fig 3A), retinal detachments, and intraocular hemorrhage. Retinal detachment occurs in a minority of affected dogs as the severest form of the condition, and is secondary to vitreous abnormalities or optic disc colobomas (Fig 3B). Like many forms of retinal dysplasia, CEA is congenital and inherited; affected dogs are recognized at a young age. Rough and Smooth Collies,[7] Shetland Sheepdogs,[7] Australian Shepherds, Border Collies,[8] Nova Scotia Duck Tolling Retrievers, Lancaster Heelers,[9] and other collie-related breeds are most often affected. Although unilateral retinal detachment is more common, bilateral detachment can occur. It is unlikely that most practitioners face a patient with acute blindness and diagnose bilateral severe CEA, but that scenario is possible. As with inherited forms of retinal dysplasia, no treatment of CEA exists. Recognizing and eliminating every affected animal on the phenotypic spectrum of CEA lesions from breeding programs is the key management strategy.

Retinal Detachments

Retinal detachments occur when the neurosensory retina separates from the underlying retinal pigment epithelium (RPE), and are categorized as rhegmatogenous and nonrhegmatogenous. The term rhegmatogenous describes retinal detachments that occur owing to tears or holes that develop in the retinal tissue, predisposing to leakage of vitreous beneath the retina and subsequent elevation. The term nonrhegmatogenous refers to retinal detachments that occur for 2 main reasons: (1) due to accumulation of fluid in the subretinal space via leakage out of the choroidal blood vessels (exudative) or (2) due to vitreal traction bands that attach to the inner retinal surface and physically "pull" the retina out of place (tractional). In general, rhegmatogenous and nonrhegmatogenous tractional retinal detachments occur owing to primary ocular disease; nonrhegmatogenous exudative retinal detachments are an ocular manifestation of systemic disease. Further, retinal detachments can be focal, multifocal, or complete; generally, significant detachments lead to appreciable vision deficits and blindness, whereas smaller, focal detachments may go unnoticed owing to lack of clinical signs. The more diffusely affected the retina, the more significant the vision deficit; when the neurosensory retina is separated from its underlying choroidal blood supply, it malfunctions and immediately begins undergoing degenerative changes. Identifying a retinal detachment during funduscopy is the most straightforward way to

Table 1
Expected Neuro-ophthalmic Findings Based on Lesion Location

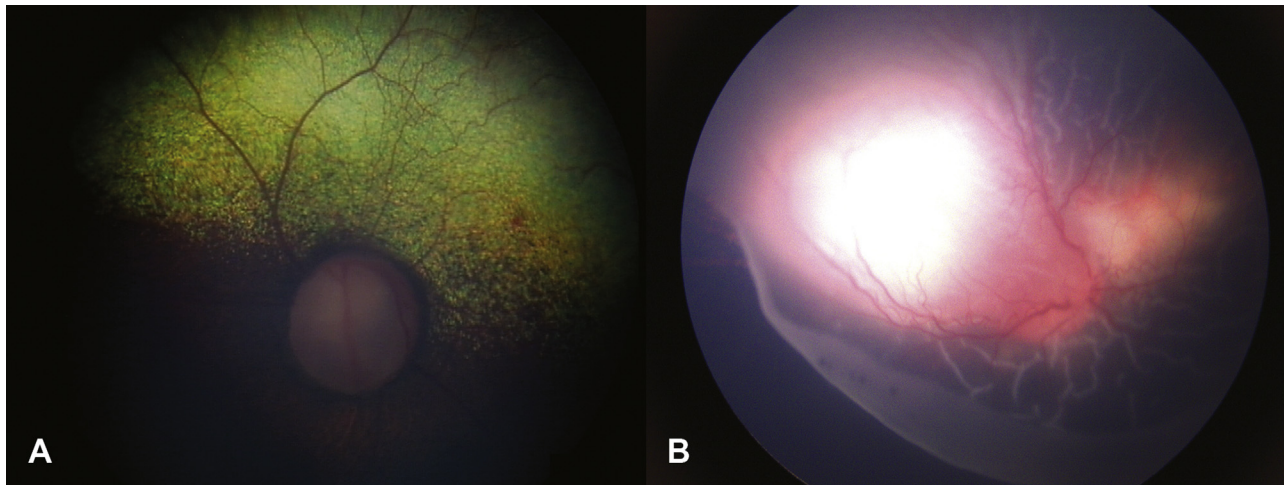| Lesion Location	Menace	PLR—Direct and Consensual
Retina	Absent	Abnormal
Optic nerve	Absent	Abnormal
Occipital (visual) cortex	Absent	Normal

Fig. 3. (A) Large optic disc coloboma in a 9-year-old American Eskimo Spitz dog. (B) Partial retinal detachment due to a peripheral retinal tear (rhegmatogenous) in a dog with CEA. Note the optic disc coloboma laterally and multifocal retinal dysplasia (linear to Y-shaped gray areas of retinal folding) scattered throughout the atapetal ocular fundus. Photo courtesy of Dr. Jonathan Pucket at Oklahoma State University.

diagnose the condition; however, ocular ultrasound can be performed to confirm retinal detachment when the view of the fundus is blocked by opacities in the clear ocular media (e.g., significant corneal disease, hyphema, and cataract).

Rhegmatogenous Retinal Detachments

The most severe form of CEA, mentioned earlier, is an example of rhegmatogenous retinal detachment (Fig 3B). Others include giant retinal tears (Fig 4) caused by retinal and vitreal degeneration, as reported in the Shih Tzu breed,[10] and lenticular diseases such as cataract and lens luxation. An abnormality in the vitreous, primarily or as an indirect connecting point between the lens and the retina, is often the underlying cause for rhegmatogenous retinal detachment.

The vitreous body plays a vital role in support of the retina, acting as a space filler and shock absorber in the large cavity immediately anterior to the retina. In fact, the vitreous is directly attached to the retina at its periphery by fibrillar strands, and anteriorly it is firmly attached to the posterior lens capsule by the hyaloidocapsular ligament. When the vitreous hydrogel becomes liquefied as a part of a degenerative breed-specific developmental

Fig. 4. Giant retinal tear (also called retinal disinsertion) in a 7-year-old Shetland sheepdog. The retina has torn for greater than 180° at its periphery and is now folded in a vertical orientation that obscures the view of the optic disc. White curvilinear condensations of degenerated vitreous are present near the torn retina.

abnormality (e.g., Brussels Griffon, Chihuahua, Chinese Crested, Havanese, Italian Greyhound, Papillon, Shih Tzu, and Whippet), by aging, or after infiltration by inflammatory cells, blood, or organisms, it no longer provides the necessary support to the retina. Rather, the vitreous becomes more mobile, and can cause direct tension on the retina at the attachment points.

Lenticular disease is a relatively common cause of rhegmatogenous retinal detachment in dogs. In a study, retinal detachment developed owing to lens disease in 50% of 46 dogs.[11] Retinal detachment is a well-recognized complication of lens instability[12]; a "pulling" force is exerted on the peripheral retina as the lens moves, causing tension on the vitreoretinal attachments and causing the retina to tear. Surgical removal of an unstable lens (intracapsular lens extraction) is also associated with retinal detachment,[13] though it is often difficult to know whether the detachment occurred as a complication of surgery or if an unrecognized presurgical retinal tear was exacerbated over time.

Cataracts may also cause rhegmatogenous retinal detachment in dogs. As with lens instability, both the disease process and the surgery to correct it can lead to retinal detachment. Several studies have reported a prevalence of 1%-8.4% for retinal detachment after phacoemulsification cataract surgery in dogs.[14-17] A study evaluating the histopathologic features of globes enucleated or eviscerated due to complications after cataract surgery identified 64% of cases with a morphologic diagnosis of retinal detachment, and only 14% of cases were reported from clinical observations before surgical removal of the eye.[18] Of course, this report was biased by the nature of the patient population (dogs with serious postoperative complications necessitating removal of the eye). Identifying a specific mechanism leading to retinal detachment after cataract surgery has remained elusive, though disruption of the vitreous face is the most commonly accepted underlying etiology.

Rhegmatogenous retinal detachments can often be treated with surgery. Depending on the extent of the detachment, surgery goals are aimed either at halting progression of a partial detachment or reattaching the more extensively detached retina. Based on the theory of vitreous instability and traction placed on the peripheral vitreoretinal attachments, holes or tears at the retinal periphery are presumed. However, these lesions are often not identifiable with traditional funduscopy. In cases of partial retinal detachment caused by a peripheral retinal tear, barrier retinopexy can be performed using a diode laser. Laser energy is focused on the retina via a transpupillary approach, where the surgeon wears
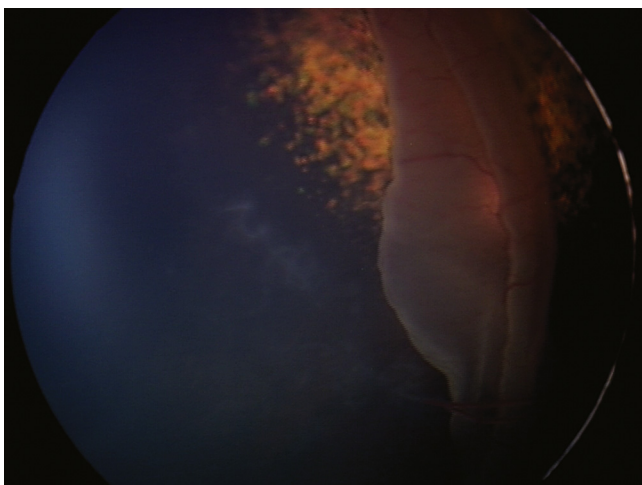
a laser-mounted headset to deliver the laser treatment. The RPE layer absorbs the diode laser energy, causing a focal adhesion to form between the RPE and the overlying neurosensory retina. This "spot-welding" technique is meant to reinforce the retina and provide more anchoring points to minimize progression of a retinal detachment. Retinal reattachment surgery, on the contrary, is a specialized vitreoretinal surgery in which the retina is reattached using perfluoro-n-octane-silicone oil exchange and endolaser retinopexy. Only a few veterinary ophthalmology practices in the United States offer retinal reattachment surgery at this time; the travel and expense associated with choosing this option make it prohibitive for many clients. It appears to be a viable option for canine retinal detachment treatment, despite logistical challenges; a recent retrospective study reported a success rate, as defined by return of vision, of 91.5% at 120 days after surgery.[19]

Nonrhegmatogenous Retinal Detachments

Exudative retinal detachments occur when material such as fluid, cells, or infectious organisms leak from the choroidal vasculature and accumulate in the subretinal space; the neurosensory retina separates in the area(s) where material accumulates, which can be focal, multifocal, or diffuse. Attempts are made to visually identify the subretinal material by funduscopy, as the composition of the material can indicate the disease process. Clear fluid (transudate or modified transudate) is transparent and imparts a bubble-like appearance to the detached retina. Transudate is the most common type of subretinal fluid in hypertensive retinopathy (Fig 5) and in steroid-responsive retinal detachments described in dogs,[20] whereas cloudy yellow-white fluid (exudate) is typical of infectious or inflammatory material beneath the retina, caused by chorioretinitis of various etiologies (Fig 6). Numerous bacterial, fungal, viral, parasitic, and protozoal causes of chorioretinitis have been described in dogs[21-26] and cats.[26-31] The general underlying mechanism is similar for the various types of exudative retinal detachment: disruption of the blood-retinal barrier, by direct vascular damage and leakage of plasma (hypertensive retinopathy) or by inflammation-mediated vasodilation and increased vascular permeability and leakage of cells and organisms (chorioretinitis) into the subretinal space.

Although rhegmatogenous retinal detachments are amenable to surgical treatment, exudative retinal detachments require medical therapy, both symptomatic to lessen the severity of inflammation (systemic anti-inflammatory medications) and specifically targeted at any identifiable cause (e.g., systemic antimicrobials for infectious chorioretinitis). Regardless of the type of retinal detachment, rapid recognition of the lesion resulting in blindness and appropriate diagnostic workup is necessary to
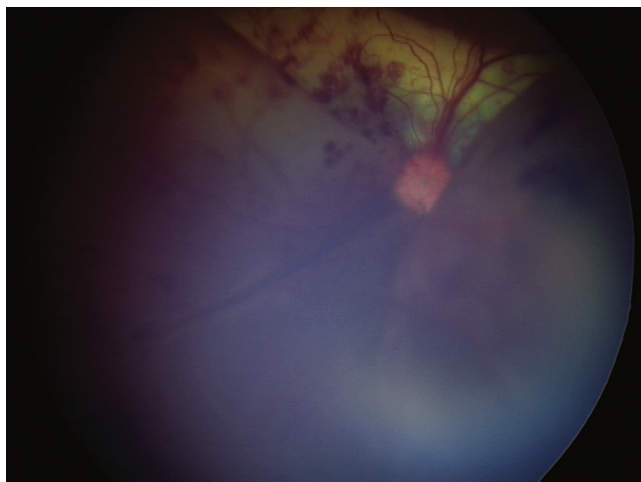
Fig. 6. Examples of nonrhegmatogenous retinal detachment (exudative chorioretinitis). (A) A 10.5-year-old Labrador mix diagnosed with metastatic hemangiopericytoma. Multiple areas of opaque subretinal fluid and hemorrhage of varying size are present in the mid-peripheral fundus. (B) A 2.5-year-old Labrador mix diagnosed with systemic cryptococcosis. Indistinct areas of retinal detachment due to white-yellow subretinal fluid accumulation are present in the tapetal area, along with diffuse hyperpigmented foci scattered throughout the tapetal fundus. (C) A 2-year-old German shepherd dog diagnosed with systemic aspergillosis. Poorly demarcated yellow subretinal material has accumulated in the tapetal area, dorsal to the optic disc. Multiple circumscribed yellow opacities approximately one-quarter of the optic disc's diameter are seen within the pigmented peripheral fundus, away from the primary lesion. Several areas of pre- and intraretinal hemorrhage are also present.

Fig. 5. Hypertensive retinopathy in a dog. The entire ventral retina is detached, with serous fluid distending the subretinal space (bullous detachment). Retinal hemorrhages are present lateral to and above the optic disc.
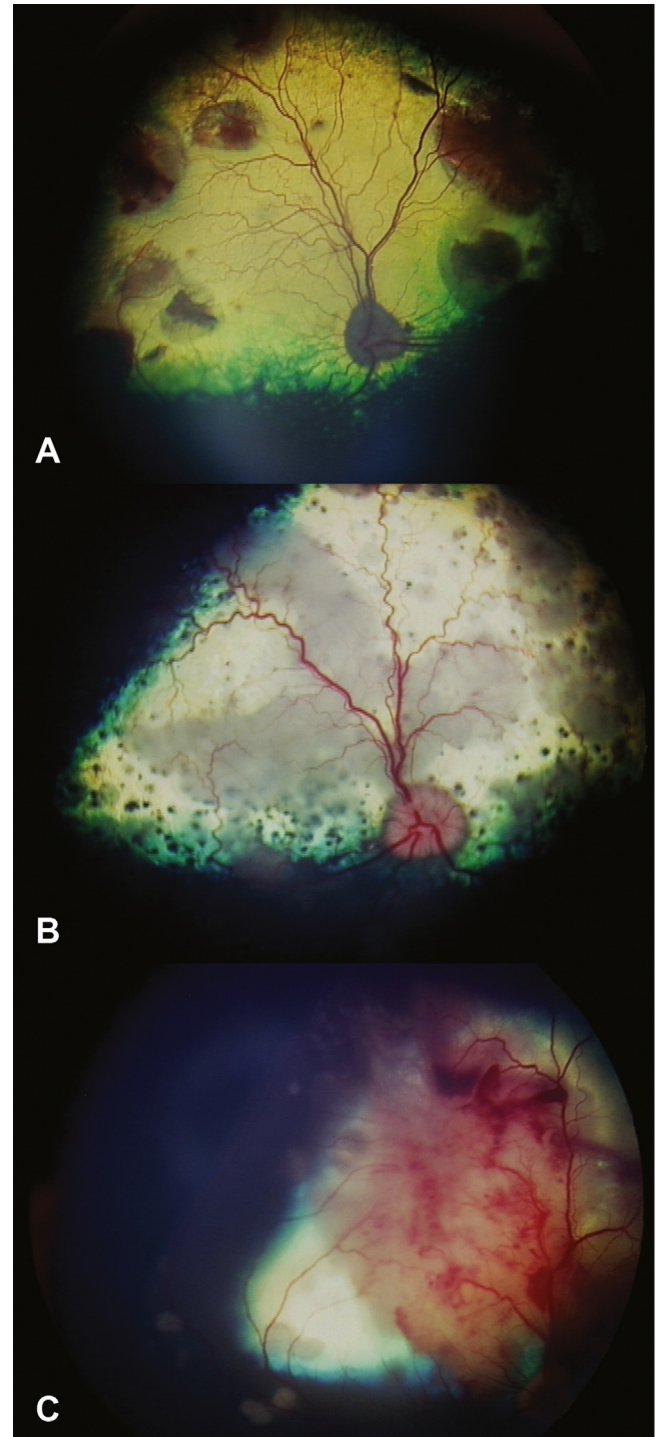
improve prognosis for vision and health in individual animals. Complete physical examination, along with basic clinicopathologic testing, and, when indicated, imaging and infectious disease screening tests, are necessary in cases of exudative retinal detachment suspected to be caused by a systemic infection.

Sudden Acquired Retinal Degeneration Syndrome (Dogs)

Sudden acquired retinal degeneration syndrome (SARDS) is a condition resulting in acute blindness with the absence of funduscopic result abnormalities. The ocular examination result is normal (Fig 7) in the early stages, but affected dogs progress over time to show signs of retinal degeneration (Fig 8) (tapetal hyperreflectivity, retinal vessel attenuation, and optic disc pallor). It was first described more than 30 years ago,[32] but despite extensive investigation, the cause and any consistently successful treatment options have not been identified. Widespread death of the photoreceptor cells within the retina appears to be the underlying abnormality, although the mechanism that causes acute photoreceptor death is unknown. Some research indicates that cell death by apoptosis is one mechanism,[33] and affected dogs often have concurrent clinicopathologic abnormalities such as lymphopenia, lymphopenia with neutrophilia, elevated alkaline phosphatase, and hypercholesterolemia.[34] A subset of affected dogs also exhibit clinical signs consistent with hyperadrenocorticism, including polyuria, polydipsia, polyphagia, and weight gain, though a definitive link between SARDS and this endocrinopathy has not been established.

With the absence of funduscopic result abnormalities (blindness in a "normal" eye), an electroretinogram (ERG) is the gold standard to diagnose SARDS. The ERG response should be extinguished in SARDS animals.[34] Chromatic PLRs can also aid in the diagnosis of SARDS, but are not meant to replace the ERG. A specific instrument that emits colored light of specific wavelengths is used to elicit PLRs. In dogs affected by SARDS, normal pupil constriction should occur when a blue light is used, likely due to stimulation of a photosensitive pigment called melanopsin contained within retinal ganglion cells. Conversely, when a red light is used, the tested pupil should remain unresponsive because light of that wavelength stimulates the photoreceptors.[35]

No widely accepted treatment exists for SARDS, and in general the prognosis for vision restoration is poor. It is important to emphasize quality of life to owners, as many blind dogs can live happy lives with a few adjustments. In the subset of dogs diagnosed concurrently with hyperadrenocorticism, appropriate therapy is suggested.
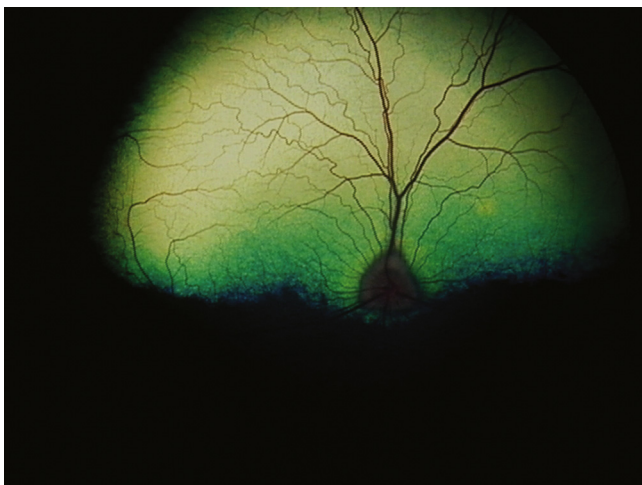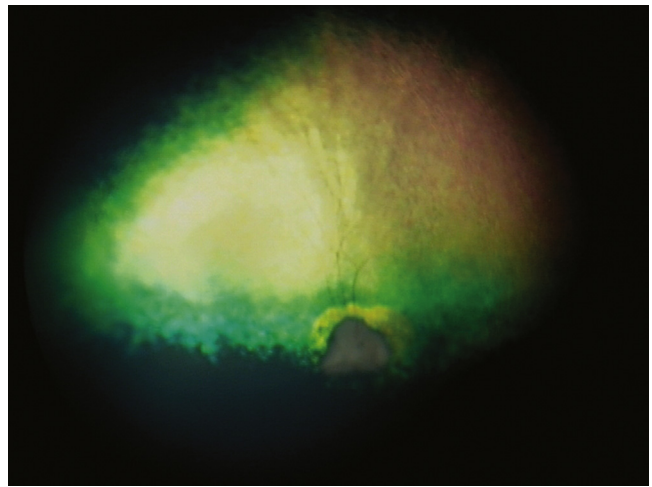

Fig. 8. End-stage retinal degeneration in a dog. Note the tapetal hyperreflectivity, retinal vessel attenuation, and pallor of the optic disc.

Fluoroquinolone Retinopathy (Cats)

Enrofloxacin-associated retinal toxicity is an important ocular drug toxicity that was first recognized almost 20 years ago, when the label dosing for the drug was changed from 2.5 mg/kg every 12 hours to a more flexible range of 5-20 mg/kg as a split or single dose. Soon after this change in dosing recommendation, cases of acute blindness due to severe retinal degeneration (Fig 9) in enrofloxacin-treated cats began emerging. Cats are typically irreversibly blind, though there are sporadic reports of some vision restoration or retention. In a study[36] retrospectively evaluating cats diagnosed with acute blindness after enrofloxacin administration, clinical signs of marked retinal degeneration were reported to develop within a few days of drug administration. Vision loss was usually permanent, and retinal degenerative changes persisted beyond cessation of drug administration; results of ERG responses indicated diffuse outer retina (photoreceptor) disease.[36] Additionally, the dosing range in affected cats was wide, from 4.6 to as high as 27 mg/kg, and the route of administration varied from oral to intravenous.[36] The results of that study prompted the authors to advocate strict adherence to the previously used 5 mg/kg/day dosing recommendation in cats.[36]

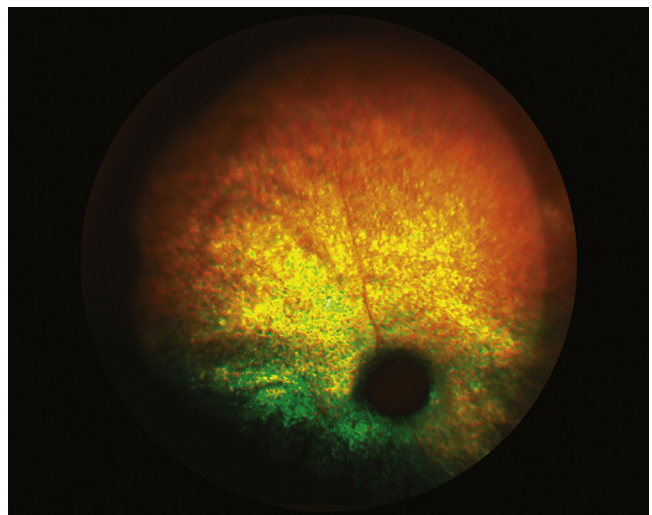**Fig. 7.** Normal funduscopic view of a dog ocular fundus.

Fig. 9. End-stage retinal degeneration in a cat, presumed to be due to enrofloxacin toxicity. Note the tapetal hyperreflectivity, retinal vessel attenuation, and dark optic disc. Photo courtesy of Purdue University Ophthalmology Service.

Although enrofloxacin is the drug most frequently identified in this acute retinal degeneration, all fluoroquinolones must be considered potentially retinotoxic. A study evaluating the safety of a newer fluoroquinolone, pradofloxacin, concluded that this drug caused no retinotoxic effects in cats, as assessed by ERG, at 6 and 10 times the recommended label dose.[37] Despite these findings, it is the author's opinion that the use of any fluoroquinolone antibiotic in cats should be reserved for those cases in which a bacterial susceptibility documents the necessity considering that there are several other choices for empirical antibiotic use.

Diseases of the Optic Nerve

Optic Neuritis

The optic nerve is essentially an extension of the retina; it is composed primarily of the axons of the retinal ganglion cells. Optic neuritis refers to inflammation of the optic nerve, and dogs are affected much more frequently than other species. There are multiple causes of optic neuritis, including idiopathic, neoplastic, immune-mediated, and infectious etiologies; however, most dogs are affected by idiopathic disease. As with many other ophthalmic diseases deemed idiopathic, an immune-mediated etiology is suspected in idiopathic optic neuritis cases due to clinical response to immunosuppressive therapy. Clinical signs of bilateral optic neuritis are acute blindness with fixed and dilated pupils. If only one optic nerve is affected, it is possible for the more subtle signs of unilateral optic neuritis to go unnoticed by the owner. Inflammation can involve the optic nerve at any point, including the intraocular (optic disc), intraorbital, intracanalicular, or intracranial portions. If the optic disc is affected (also termed papillitis), funduscopy allows diagnosis based on visual assessment of an elevated, edematous, disc with blurred margins (Fig 10). If other portions of the optic nerve, collectively called the retrobulbar optic nerve, are affected without concurrent papillitis, the fundic examination result would be normal. In this situation, an ERG is required to distinguish between SARDS and retrobulbar optic neuritis. Because an ERG tests the electrical activity of retinal cells, which should be spared in cases of optic neuritis, the ERG results should be normal in a patient affected by retrobulbar optic neuritis.
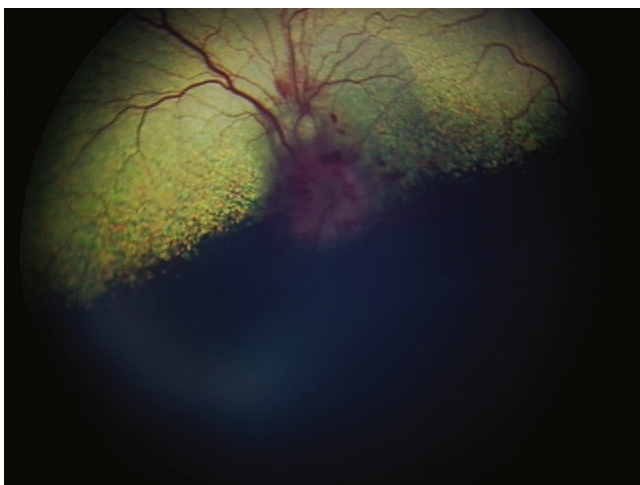

Fig. 10. Optic neuritis (papillitis) of unknown cause (presumed idiopathic or immunity mediated) in a 10-year-old Golden Retriever. The margins of the optic disc are blurred and indistinct, the peripapillary retina is detached, and there are hemorrhages on and around the disc.

Although most cases of optic neuritis in dogs are idiopathic, other causes must first be ruled out before settling on this diagnosis of exclusion. Of the possible immune-mediated diseases affecting the optic nerve, granulomatous meningoencephalitis (GME) is the most common cause of optic neuritis. This disease is characterized by a perivascular proliferation of histiocytes, monocytes, lymphocytes, and plasma cells, with varying numbers of granulocytes and multinucleated giant cells, in the central nervous system (CNS).[38] Nodular (focal), disseminated (multifocal), and ocular forms can occur. Ocular GME can occur as part of focal or disseminated disease, or it can occur alone. Some dogs exhibit ocular signs only initially, and progress to develop signs of more widespread CNS involvement. Any dog can be affected by GME, but it is most common in middle-aged, small-breed female dogs.[39,40] Diagnosis of GME is based on results of magnetic resonance imaging (MRI) and cerebrospinal fluid (CSF) analysis, which can be extremely variable.[38,40] The MRI signs include variable patterns of contrast enhancement, but no disease-specific features.[41] A marginal to marked increase in protein or cellularity in the form of mononuclear cells and macrophages may be present on CSF analysis, but results are highly variable.[40] Immunosuppressive treatment with prednisone and other chemotherapy agents is standard.

Infectious causes of optic neuritis in the dog have been reported, and include viral diseases such as canine distemper virus[42-45] and tick-borne encephalitis virus,[46] ehrlichiosis,[47] mycotic diseases,[23,48-50] and toxoplasmosis.[51,52] Similar to non-infectious causes (e.g., GME), the recommended diagnostic testing plan includes advanced imaging (MRI) and CSF analysis, along with serum titers and other infectious disease screening tests. In cats, a species much less commonly affected by optic neuritis, feline infectious peritonitis,[53,54] mycotic diseases,[55,56] and protozoal disease[57,58] are implicated infectious etiologies.

Most cases of optic neuritis are diagnosed in dogs ultimately as idiopathic, but it is important to rule out possible causes with the appropriate diagnostic tests before instituting therapy. Prognosis for vision is poor in all cases.

Neoplasia

Neoplasia may affect the optic nerve either by direct involvement (primary tumors) or due to invasion of the optic nerve by tumors arising from adjacent structures within the orbit or sinuses. Typically, animals present with unilateral blindness that may not be detectable by the owners, and owing to the generally slow progression of most tumors, vision loss is often gradual. Clinical signs of orbital disease (exophthalmos, elevation of the nictitating membrane, and decreased retropulsion) accompany many tumors of the optic nerve and adjacent structures. Bilateral primary optic nerve or orbital neoplasia is rare, so it is unlikely a patient would be presented for acute blindness due to neoplasia of both optic nerves or orbits concurrently. However, a lesion at the optic chiasm, metastatic neoplasia to the orbit (e.g., lymphoma), or a particularly aggressive nasal tumor affecting both sides of the nasal passages and invading both orbits could result in bilateral blindness. Gliomas (astrocytomas, oligodendrogliomas, and oligoastrocytomas),[59] meningiomas,[60] and teratoid medulloepitheliomas are reported primary optic nerve tumors. Orbital tumors and nasal tumors damage the optic nerve by creating a space-occupying lesion within the limited confines of the bony orbit; compression, inflammation, and, in some cases, invasion by the tumor, follow. In general, primary optic nerve tumors such as gliomas tend to have low metastatic potential. Primary orbital and nasal sinus tumors have a guarded to poor prognosis and treatment such as surgical debulking (orbital exenteration) with follow-up radiation therapy is typically recommended. Survival

time was 10 months in dogs, with a high rate of euthanasia at the time of diagnosis, in a study.[61]

Diseases of the Visual Cortex

Because of the divergence in the neuroanatomic pathway shared by PLR and vision fibers, blindness due to diseases affecting the cerebrum should not be accompanied by abnormalities in PLR (Fig 1). There are many possible causes of cerebral dysfunction leading to blindness, and signalment and history information is vital to narrowing the differential list. Did the animal recently undergo general anesthesia? Is there history of seizures (post-ictal blindness)? Or is the animal older with mentation and behavior changes accompanying the vision loss, beyond what could be explained by adjusting to blindness?

Anesthetic complications such as hypotension, hypercapnia, and hypoxemia occur in dogs and cats, with a published prevalence of 12% in dogs and 10.5% in cats of a study.[62] Impaired blood delivery to cerebral tissues or the delivery of blood with poor oxygen saturation can lead to ischemia and disruption of the visual cortex. Cats seem particularly sensitive to postanesthetic blindness; a recent study describing 20 cats with postanesthetic cortical blindness[63] identified the use of mouth gags as a risk factor for cerebral ischemia and blindness; the authors hypothesized that mouth gags may lead to reduced blood flow to the brain via the maxillary artery, the main vessel responsible for blood flow to the feline brain. This was an interesting theory, as a mouth gag was used in 16 of 20 cases and systemic hypotension was documented in only 7 cats (though blood pressure was not measured in another 7 cats). Necrosis of the cerebral tissues because of ischemic injury is a proposed mechanism of postanesthetic cortical blindness; prognosis for return for vision may be favorable, and 70% of cases in the recent study regained some amount of vision over time.[63]

Infectious and inflammatory conditions of the CNS can affect the visual cortex and lead to cortical blindness. As the optic nerve is an extension of the CNS, many of the diseases already discussed (including GME and infectious optic neuropathies) can lead to central blindness. Neoplastic diseases of the intracranial space often cause sudden blindness; the disease process is really progressive but the gradual onset of more subtle vision deficits is not typically noted by owners. The most common tumor type in dogs and cats is meningioma.[64-66] Blindness as a clinical sign of intracranial neoplasia is variable. In a retrospective study of intracranial neoplasia in 160 cats, only 10% were blind as a presenting neurologic clinical sign.[66]

Surgical resection and fractionated radiotherapy are the current treatment recommendations for brain tumors in dogs and cats. Although these treatments can increase survival time, they are associated with significant morbidity. For treatment of forebrain meningioma, the median postsurgery survival was 7 months in dogs[67] and 24 months in cats.[68,69] The prognosis for dogs with brain tumors treated with palliative therapy alone is poor. A recent study reported a median survival time of 69 days after diagnosis when palliative therapy using corticosteroids and phenobarbital was used.[70] Cats tend to have a more favorable, though still guarded, prognosis. In fact, intracranial tumors were considered an incidental finding in 18.8% of cats in a study.[66] Neurologic signs were completely absent in 21.2% of cases from that same study. In dogs, however, neurologic signs are quite common and the severity of neurologic impairment is related to overall prognosis.[64] A veterinarian's index of suspicion for a central lesion causing blindness should increase based on findings of complete ophthalmic examination and PLR assessment, which should be normal in the face of absent menace response. Signalment and history

information provide additional guidance to then narrow the differential list for causes of cortical blindness.

Conclusions

Neuroanatomic localization is a key concept in the diagnosis of acute blindness. Determining whether blindness is caused by a primary ocular problem or an ocular manifestation of a systemic disease has important implications for the overall health of the animal. Findings of a complete ophthalmic examination, including PLR assessment and funduscopy, provide information regarding the necessary steps in diagnostic workup. Determining the cause of sudden blindness allows the veterinarian to advise the owner on prognosis for vision and general health.

Acknowledgments

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