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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.

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

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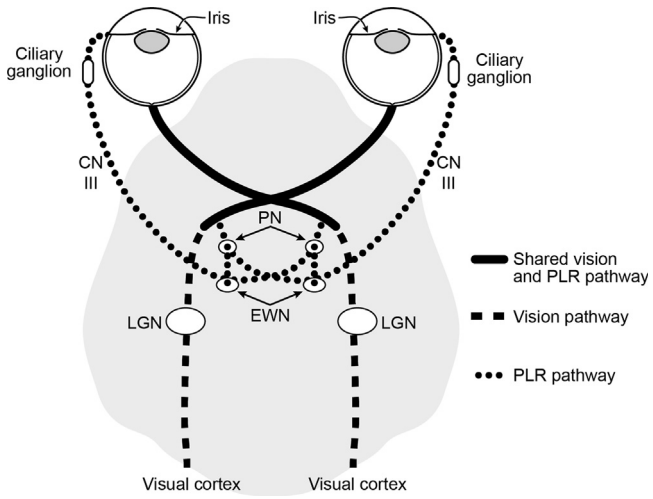

Fig. 1. Schematic diagram of the neuroanatomic pathways for vision and PLR. The vision and PLR pathway is shared before the LGN. The PLR pathway diverges before the LGN to synapse at the PN, then the EWN. The pathway then continues toward the eye with synapse occurring at the ciliary ganglion, and terminates 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.

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

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

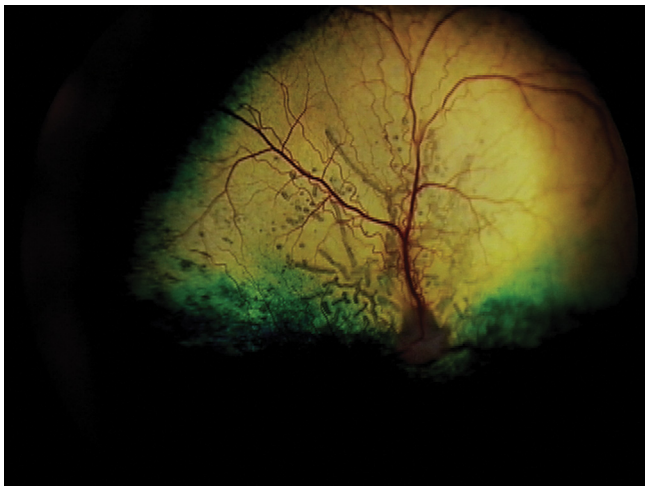

Fig. 2. Retinal dysplasia in a 1-year-old Airedale dog. Multiple linear to Y-shaped gray retinal folds are clustered dorsal to the optic disc.

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