Exploration of the Ocular Fundus

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While examination of the ocular fundus can be the most intimidating component of the ocular exam, it can often be the most revealing. Evidence of systemic disease is often found in these important structures of the eye, including the retina, choroid and optic nerve.

The examination begins with a thorough history and behavioral testing to assess vision. Vision tests involve covering each eye and observing responses in both bright and dim light settings. Vision tests include the menace response (reaction to a threatening hand motion without air current) and response to objects (i.e. maze testing, tossing of quiet objects such as cotton balls, and placement of feet on table edge).

Pupillary light reflex (PLR) requires normal retinal function and normal conduction to the brainstem. Blindness caused by a tumor in the occipital cortex visual center should have a normal PLR since the retina and the pathways to the subcortical brainstem are intact. In contrast, the absence of PLRs suggests complete retinal degeneration, glaucoma or a lesion along the reflex pathway (i.e. optic chiasm.) Typically, a dog with progressive retinal disease will have a decreased or absent PLR. However, the requirement for stimulation is low, meaning that even in stages where a dog is functionally blind but the retina has not yet atrophied completely, a dog will often retain a slow or diminished PLR.

Examination tools

There are three main tools for examining the ocular fundus: the direct ophthalmoscope, the indirect light source with a handheld lens, and the panoptic. Since the panoptic offers features of both direct and indirect, it can be a useful tool. However, indirect examination with a handheld lens offers the least expensive option with the widest field of view making it the preferred method for routing exam screening. The indirect lens image offers 2-5 times magnification and provides an inverted, reversed image. Should an abnormality be detected with an indirect exam, the direct ophthalmoscope will then be helpful for detailed investigation (upright image of 15x magnification).

Ocular ultrasound is useful for evaluating the structures in the back of the eye when direct visualization is obstructed. For instance, if the eye is filled with blood or a complete cataract is present, ultrasound can quickly provide information to rule out a retinal detachment or mass in the posterior segment of the eye. Using topical proparacaine and sterile lubrication, useful images can be obtained with a standard ultrasound machine using a 10-12mgHz probe on placed directly on the cornea.

Electroretinography (ERG) records the retina’s electrical response to a light stimulus. The response is measured using an active contact lens electrode, a ground and a reference
electrode. The retinal response is transmitted as a waveform to the computer display for both photopic (cones) and scotopic (rods) analysis. The ERG is useful to assess the health of the retina when visualization is obscured by cloudy medium such as a cataract. It is also useful in blind patients if the ophthalmoscopy changes are not obvious (i.e. SARDS) to verify that the retina, rather than the brain, is responsible for vision loss. Advanced ERG techniques can also be used to specifically test rod and cone function in breed-related retinal degeneration.

Anatomy
Primary structures evaluated in a fundic exam include the retinal blood vessels, the colored tapetum lucidum and the pigmented non-tapetal zone within the choroid, and the optic nerve. In the normal tapetal fundus, the overlying retina lacks pigment in the Retinal Pigmented Epithelial (RPE) cells so that the brightly colored and reflective tapetum is visible. The non-tapetal fundus comprises the largest area of the fundus peripherally and ventrally and is typically dark brown or grey.

The retina arterioles and venules should be evaluated for size, length, tortuosity and integrity. The thickness of the retina can be interpreted with a hyperreflective tapetum denoting a thin, overlying retina or blurred, grey changes consistent with a thickened, edematous retina.

The most significant difference between the dog and the cat is found in the optic nerve. The visible nerve fibers in the canine optic nerve are wrapped in myelin, giving it a thickened, white appearance. In contrast, the portion of the optic nerve visible through ocular exam in the cat is not myelinated so the optic nerve head will appear dark and flat. Changes in the optic nerve due to glaucoma, optic neuritis or atrophy are therefore more easily detected in the canine optic nerve as the quality of myelination will be altered.

While best illustrated in the photographs, there are several variations of normal that are commonly difficult to interpret. Many variations in tapetal color can be seen and may vary with age and breed. During normal maturation, a dog may have a blue tapetum until 5-7 weeks of age when it begins to acquire its yellow coloration. The tapetum can be vast in large dogs and sight hounds who rely heavily on the increased light that the tapetum provides. The tapetum may be small or absent in small breed dogs. The subalbinotic fundus, seen in dogs and cat who lack pigment in their iris (blue) or choroid, will appear significantly (and often alarmingly) red as one looks directly at the underlying choroidal blood vessels radiating out from the optic nerve. Due to the lack of brown pigment in the choroid and the retina (normally in the RPE), the white scleral wall will often be visible between the thick, brick-red choroidal vessels. In addition, it is often difficult to distinguish or interpret the quality of the retinal vessels in these patients as the retinal vessels blend into the underlying choroidal vessel background.

Age-related degeneration can make interpretation of other abnormalities more challenging, so a diagnosis of retinal disease should be made in conjunction with history and signalment. For instance, the index of suspicion for pathology should be higher in a middle age, pure breed dog with high risk for inherited retinal disease such as a poodle, spaniel or retriever. Age-related retinal degeneration is often limited to thinning of the retinal vessels whereas changes that represent pathology could include significant thinning of the retina, giving the appearance of tapetal hyperreflectivity.
**Progressive Retinal Atrophy**

Progressive retinal atrophy (PRA) describes a large class of inherited degenerative diseases leading to blindness in the dog. The classification has been further divided based on the type of cell responsible for the vision decline such as the photoreceptors (rods and cones) or the retinal pigmented epithelium (RPE).

Many breeds demonstrate specific types of PRA that vary with age of onset and rate of progression. Common patients include miniature and toy poodles, Labrador and Golden retrievers and Cocker spaniels. The most common form, prcd-PRA, is inherited in an autosomal-recessive pattern and affected dogs should not be used in breeding programs.

Clinical signs include a gradual decline in vision over months beginning with navigation difficulty in dim lighting (as rods degenerate first). Loss of vision is often masked by familiarity with the home setting, so a trip outside of the home often prompts the recognition of a problem. Patients present with dilated pupils, sluggish PLRs, decreased to absent menace response and poor performance in a maze test, particularly in dimly lit settings.

Ophthalmic exam of the retinas reveals evidence of retinal degeneration including a decrease in retinal vessel length and width as well as hyperreflectivity of the tapetal fundus (i.e. increased eye shine.) In advanced stages, one can observe complete absence of retinal blood vessels, pallor of the optic disc and patchy depigmentation in the non-tapetal fundus. If the retinal exam alone does not provide sufficient evidence of the disease, an electroretinogram (ERG) can be used to demonstrate a decrease in retinal function in response to flashing light. Further testing for specific gene markers is available for many breeds.

Cataracts can result from PRA as the degenerating retina releases glutamate which acts as a toxin to the lens fibers. When a patient presents for cataract surgery evaluation with complete cataracts obstructing the view of the retina and a decrease in PLR is noted, the suspicion for PRA should be high. Moving forward without performing an ERG to confirm this suspicion prior to cataract surgery would lead to marked disappointment when vision was not restored once the cataracts were removed surgically so a pre-surgical ERG is always recommended.

Unfortunately, there is no treatment for retinal degeneration to date and gene therapy and retinal transplantation remain in developmental stages. Owners can be assured that PRA is a painless disease and most dogs adjust very well to being blind. Websites such as [www.blindtails.com](http://www.blindtails.com) can also provide supportive information. In addition, vitamin supplements are available for pets based on the same principles of lutein and antioxidant supplementation in people with macular degeneration and other retinal disease.

**Sudden Acquired Retinal Degeneration**

Sudden Acquired Retinal Degeneration (SARDs) is a non-painful, acute disease of the retina that most often results in sudden and complete blindness. This is common in middle aged female Dachshunds and in approximately 30% of the cases, one finds an association with Cushing's disease or signs of Cushing's (polydipsia, polyuria, polyphagia, weight gain).
SARDs is not completely understood but the most plausible theory involves an immune-mediated attack on the retina that is complete and permanent.

Owners will report an acute loss of vision occurring over days, and ophthalmic exam will reveal decreased vision and absent light reflexes. However, the retina often appears entirely normal so an electroretinogram (ERG) can be performed to demonstrate a severe decrease in retinal function.

Unfortunately, there is no reliable treatment for SARDs at this time. Experimental trials using IVIG to combat a potential immune-mediated process show a possible pathway for the future, but results are not reliable and risks of complications are high.

Collie Eye Anomaly
Collie eye anomaly (CEA) is a congenital, autosomal-recessive inherited syndrome seen in up to 97% of the Collies in the United States. The most common finding is an area of choroidal hypoplasia lateral to the optic disc. More advanced forms include colobomas (holes) around the optic nerve (34%), retinal detachments (10%) and intraocular hemorrhage. Choroidal hypoplasia appears as a large white area of exposed sclera, often with tortuous blood vessels. While the more mild forms of choroidal hypoplasia do not significantly affect vision, the optic nerve and retina changes can have devastating effects.

This disease presents a breeding dilemma for a number of reasons. In light of the high prevalence, breeding only homozygous normal would limit the gene pool deleteriously. In addition, this syndrome demonstrates a “go normal” phenomenon in which puppies with evidence of CEA when examined at 5-7 weeks will appear normal by 3 months as more pigment accumulates with maturation. A “normal” misdiagnosis leads the breeder to assume that the dog is merely a carrier for CEA or even homozygous normal. A genetic test is now available for CEA through Optigen.com. To select away from CEA-affected dogs in the future, the recommendation for breeding would be to ensure that at least one of the breeding pair is homozygous normal.

Retinal dysplasia
Retinal dysplasia describes pattern of folds in the canine retina. This disease is commonly bilateral and while viral infections, toxins and vitamin A deficiencies are amongst the causes, inherited forms are the most common etiology. The appearance of retinal dysplasia can be focal grey streaks, large geographic patterns, or a complete retinal detachment. Retinal dysplasia is found in many breeds including Rottweilers, Labrador retrievers, Cavalier King Charles Spaniels and Chow Chows. The significance for vision varies with the severity of the disease.

Retinal detachment
A retinal detachment occurs when the neurosensory retina containing the photoreceptors separates from the retinal pigmented epithelium below. Causes range from congenital defects to systemic hypertension to infection. If not an early onset congenital defect, detachment is divided into 3 major classifications: rhegmatogenous, traction and exudative.
A rhegmatogenous tear is a focal tear or hole that allows fluid from the vitreous to leak under the retina. The cause is often breed related (i.e. Italian Greyhound). A traction detachment occurs when scar tissue from previous inflammation, hemorrhage or surgery causes contraction of scar tissue that pulls the retina off of the posterior wall. An exudative detachment describes fluid or cells under the retina that push the retina forward occurring in systemic hypertension or lymphosarcoma.

Patients present with a variety of clinical signs depending on the size of the tear and the underlying cause. A patient with a small rhegmatogenous tear may have no signs at all, and the finding may be an incidental discovery with a thorough ocular exam. Conversely, a patient with a large detachment may present with sudden blindness and exam may reveal fixed, dilated pupils with floating retinal blood vessels evident with a focal light source.

The workup for a retinal detachment is similar to the search for the cause of uveitis including common reasons for infection (UTI, tick-borne), immune-mediated (uveodermatologic disease) or neoplasia (intraocular mass or lymphosarcoma). Treatment depends upon finding the underlying cause. In cases of systemic hypertension in cats, vision can return within two weeks following a complete retinal detachment if the blood pressure is controlled quickly and consistently. The recommended starting dose for amlodipine is typically 0.625mg orally once daily with increases as needed.

Retinal surgery options range from diode laser retinopexy around a small rhegmatogenous tear to complex vitreoretinal surgery using fluorocarbon fluids and silicone oil to tamponade the retina into place. The success of retinal surgery depends on the severity of the original detachment, the underlying cause and the duration of the detachment. The ideal candidate has been detached for less than 4 weeks. Success is rated on both anatomical correction (80%) and visual return (70%).

**Chorioretinitis and Optic neuritis**

Chorioretinitis describes inflammation that manifests as edema, exudates and often hemorrhage within and beneath the retina. In optic neuritis, the optic nerve will appear raised and edematous with possible focal hemorrhage and the patient will be blind. The inciting cause of inflammation is typically elsewhere in the body and further workup is required. After a complete physical exam, CBC, Chemistry, urinalysis, chest radiographs and abdominal ultrasound may be recommended. Infectious titer recommendations could include tick-borne rickettsial diseases (Ehrlichiosis, Rocky Mountain Spotted Fever), fungal infections (i.e. Blastomycosis, Cryptococcosis), protozoal disease (i.e. Toxoplasmosis) and Bartonella. Treatment depends on finding the underlying case and initiating appropriate systemic management, as topical medications will not penetrate to the retina, choroid and optic nerve.

**References**


Genetics Committee of the ACVO. *Ocular Disorders presumed to be Inherited in Purebred Dogs*, 2nd ed, 1996