

## Article Concerning Terrier Eye Health

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### The Eyes Have It!

#### Fighting Ocular Disease and Preserving Terriers' Sight

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Your terrier's eyes are its windows on its exciting, vermin-infested world; they are two of your dog's most precious tools. Healthy eyes are crucial to its life as an alert, playful, energetic purebred terrier. These magnificent animals need to avoid eye injuries, infections, and genetically transmitted conditions. Breeders, owners, and handlers do their best to prevent eye injury and infection. But we do need to know more about controlling serious and inherited disease. We need to be able to recognize symptoms of serious conditions, to seek proper treatment if they occur, and, most important to the breed, we need to prevent these conditions.

In this brief review, we list conditions that affect most terriers, we give descriptions of procedures of eye examinations conducted by a specialist in veterinary ophthalmologist (VO), including some terms to help you speak your VO's language, we include a summary of the current treatments for common conditions, and we offer some thoughts on breeder responsibility.

#### Eye Conditions That Frequently Threaten Terriers

Ocular diseases found in terriers can attack structures in the front-and-center of the eye (cornea and lens), the perimeter (eyelid-conjunctiva area), and the interior-rear (vitreous, retina, and optic nerve). The Figure below summarizes the structure of the canine eye.

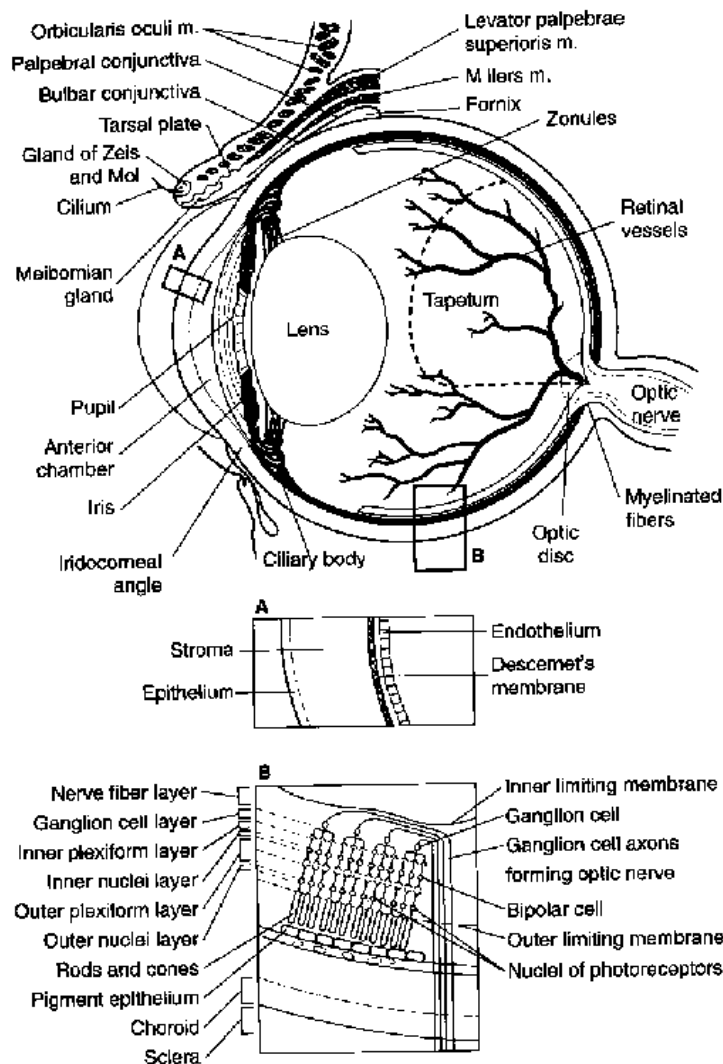


Fig. 1.3 Schematic anatomy of the canine and feline eye.

Terriers, as most other breed groups, are commonly associated with cataracts (these have been reported in 15 of the 26 American Kennel Club-recognized breeds of terriers), progressive retinal atrophy (14 breeds), and lens luxation (12 breeds).<sup>2, 3</sup> Several less common conditions, but equally serious (especially if they occur in your animals) can also appear. These conditions include retinal dysplasia (reported in eight terrier breeds), persistent pupillary membranes (eight breeds), entropion (seven breeds), glaucoma (five breeds), optic nerve hypoplasia (one breed), and optic nerve coloboma (one breed).<sup>2, 3</sup>

These conditions and others represent disorders of nature that rob your animals and their human families of joy. These diseases represent the Enemy. Your first line of defense is the annual VO's examination.

#### A Veterinary Ophthalmologist's Examination

At Canine Eye Registry Foundation (CERF) examinations for your pups or adults, your VO will take a history, asking the owner about signs noted prior to the examination, and will examine both eyes of each animal, first in available, "ambient" light and then in relative darkness.<sup>1</sup> The "ambient light" exam will likely begin with a general assessment of the terrier's level of health and pain, eye size and structure, swelling, and discharges including normal and abnormal tearing or "lacrimation." If signs call for ("indicate"), the VO can examine tear fluid production (test for "dry eye") by performing a Schirmer Tear Test or "STT." The VO uses a 2x to 4x magnification glass ("loupe") with a light to examine the interior of the eye. Pupillary light reflexes (PLRs) and blinking reflexes, both direct and "consensual" (response in the other eye) are assessed next, remembering that a flinching or "menace" reflex usually does not occur in very young pups. After reflex testing, the VO examines eyelids, especially the third eyelid

between the lower eyelid and the eye, the conjunctival lining inside the eyelids, the cornea, iris, and, looking through the pupil, the lens.

Visual examination of eye drainage pathways is conducted through a gonioscope. Gonioscopy is especially important for glaucoma patients, and it is conducted before eye dilation or "mydriasis" with one or two drops of 1% solution of tropicamide 15 to 20 minutes before the second phase of the eye examination.

During the second, "darkroom" phase of the eye exam, after the dog's eyes are dilated, the VO assesses the lens, the internal vitreous fluid, and the rear of the eye ("ocular fundus") that contains the retina and optic nerve head. Using a transilluminator (bright light source) or an ophthalmoscope, the VO can examine the terrier's lens and vitreous for clouding or "opacities" and the retina for dysfunction, bleeding, and malformations. The direct ophthalmoscope can magnify the structures in the eye 15 times. An indirect ophthalmoscope, a type of ophthalmoscope most commonly used by VOs, provides a much wider field of view. This allows the VO to scan the back of the eye and to identify abnormalities rapidly.

When further visualization or tests are indicated, the VO can use diagnostic stains. Fluorescein is used on terriers who present with ocular pain and corneal lesions. It is used to detect lesions and faulty tear drainage. Rose bengal and lissamine green stains are used on the cornea and conjunctiva to indicate damaged cells or the effects of herpesvirus infections. Samples of cells from any part of the eye can also be taken for microscopic and chemical analysis. Intraocular pressure (IOP) in both eyes is assessed through tonometry, gently applying the tonometer's plunger to the cornea. The retinoscope provides a beam of light that allows the VO to "refract" the eye, to see if images are in focus on the retina or not. Finally, radiographs, computed tomography (CT) scans, magnetic resonance imaging (MRI), ultrasound, electroretinography (ERG), and scanning laser ophthalmoscopy (SLO) are available to the VO for in depth examination and definitive diagnoses.

### **How Eye Diseases Attack Our Dogs**

Although many eye conditions exist that can affect terriers, this section discusses the group's most common ones. Genetically transmitted eye diseases, or eye diseases that are presumed to be genetically transmitted, can attack any part of your terrier's eyes. Most symptoms concern abnormal appearance, visual impairment, orbital (socket) and ocular pain, or ocular discharge, or any combination of these.

Because eye function relies totally on the health and transparency of the pupil, lens, and vitreous, as well as the health of the retina, any departure from an optimal state is serious and important to catch early. A non ocular disease or injury might leave scarring on a terrier's leg, head, or torso, and not affect his or her function, but even the slightest injury or disease of a cornea, lens, vitreous, or retina might lead quickly to impaired vision or even blindness.

**The Big Three: cataracts, progressive retinal atrophy, and lens luxation.** These eye conditions are the three most commonly found in the 26-breed terrier group.

**Cataracts** involve the lens. Although the lens does not contain blood vessels, it does grow elongated cells that sometimes swell, thicken, and become opaque, thus affecting the lens' transparency. Areas of these opaque fibers can partially or totally block vision. Cataracts in terriers are most commonly congenital (present at birth and likely to be autosomal recessive). Cataracts can also develop in adult dogs or result from trauma. Cataracts can also arise from infection and diabetes. 4 Treatment for cataracts, aside from atropine eyedrops to dilate the pupils in cases of smaller and centrally located cataracts, can involve several forms of surgical extraction of the clouded lens, often with implantation of a prosthetic lens. Surgery can involve several months of careful post-operative medical treatment and risk of complication.

**Progressive Retinal Atrophy (PRA)** involves several conditions (with varying age of onset) with degeneration of the retina's photoreceptors (see inset B in Figure 1). Typical ages of onset vary, but the range is usually from puppyhood to 6 years of age, with early development associated with more rapid retinal degeneration. Across terrier breeds, PRA always affects both eyes (bilateral), and always leads to blindness. Signs of PRA include initial loss of night vision, followed by a general loss of vision. Before these symptoms, however, your VO might detect brown or gray coloring of the interior of the eye, shrinkage of vessels, and altered coloring of affected areas of the retina. No treatment currently exists for the condition, and prevention, through knowledge-based breeding, is the only weapon against this disease.

**Lens luxation** (dislocation or displacement) results from abnormalities of the fibers that hold the lens in place and when it occurs, it is most often found in middle-aged terriers. During the VO examination, early luxation can be detected because the lens is not firmly held in place and may wobble as the eye moves. If the lens luxates anteriorly (comes forward), it can contact the cornea, cause a buildup of fluid in the cornea (edema) and within the eyeball itself and cause secondary (caused by the first condition) glaucoma. Because the structures and processes of the eye are interdependent, primary glaucoma (with increased IOP) can also cause secondary lens luxation. Lens luxation is often accompanied by pain and, because the optic nerve head is often damaged, blindness. Treatment involves rapid medical or surgical reduction of IOP and removal of the lenses of both eyes.

### **Less Frequently Encountered Conditions in Terriers**

The following conditions are reported to occur less frequently in terriers, but if any affect your dogs, they are no less serious than more common diseases.

**Retinal Dysplasia (RD)** is a form of malformation of the retina present at birth. The only "treatment" is prevention: that is, not using affected animals, or even known carriers, in future breeding. The disorder involves abnormal formation, or even detachment, of the retinal nerve cells. RD can occur focally (one location) or multifocally (many locations) and it is commonly bilateral. In severe cases, usually with severe RD concentrated in a single location, the dog becomes blind. Secondary glaucoma and bleeding can also occur within the eye.

When puppies have severe focal RD in both eyes, euthanasia is recommended. Multifocal RD usually does not cause blindness, and affected dogs can live close-to-normal lives. Infections, prenatal trauma, vitamin A deficiency, and radiation are associated with secondary RD.

**Persistent Pupillary Membrane (PPM)** and **Persistent Hyaloid Artery (PHA)** involve eye structures that normally begin shrinking in size just before birth and are virtually absent by the time a puppy is 4 weeks old. If the membranes or arteries abnormally "persist" in the eye, they can cloud vision. With PPM, the pupillary membrane that cover the fetus' pupil is retained and, with PHA, the hyaloid artery within the eye remains.

PPM occurs as filaments that reach from the pupil forward to the cornea or rearward to the lens, blocking vision. Some PPM appears as spots of pigment on the lens. Vision is usually not severely affected. PPM is congenital and not itself accompanied by pain. Affected animals and known carriers should not be used in breeding programs.

**Entropion** is a relatively common problem in which the eyelid turns inward so that the external hair rubs on the cornea. Abrasion, irritation, pain, and the likelihood of cornea scarring result. In contrast to other conditions discussed in this review, entropion is relatively easily treated, but it must resolve or be fixed to be considered "minor." The condition can develop due to abnormal anatomy, most often in puppies who have pronounced skin folds before their growth tightens the skin around the eyes. In addition to anatomical causes, the condition can be secondary to eye disease. Treatment might require only temporary pulling of the eyelid, with stitches, from the eye. More permanent fix, usually when symptoms occur in adult dogs, might include cutting away a minimal amount of eyelid tissue.

**Glaucoma**, although not occurring as frequently as other conditions, is arguably the worst of all eye-related nightmares a dog owner can experience. Glaucoma can arise from either a chronic or rapid increase in fluid pressure (that is, IOP). Pressure increases due to faulty drainage of eye fluid in the eye and leads to eye damage and blindness. With pressure, especially rapidly increasing pressure, there can be considerable pain. Fluid is continuously produced in the eye and must be continuously drained through microscopic drainage pathways that empty into blood vessels.

Glaucoma is due to a drainage problem of an anatomical cause, such as narrow drainage structures (primary glaucoma), or from malfunctioning drainage caused by another condition, an outlet plugged by pigment (a warning sign of glaucoma known as ocular melanosis that can occur in cairn terriers), bleeding, a luxating lens, or by a tumor (secondary glaucoma). The condition often occurs in one eye and then, months or years later, in the other. Enlarged eyes, half-dilated and fixed pupils, and even luxated lenses are symptoms of real trouble. Later symptoms include frequent blinking, eye rubbing, lethargy, refusal to eat, and yelping in pain. Acute glaucoma, with almost total blockage of fluid, can progress from rapidly increasing IOP, to breakdown of eye structure and pain, to blindness, all in a matter of hours!

***Acute glaucoma is a serious medical emergency that requires immediate and aggressive medical and/ or surgical treatment to decrease the IOP.***

Treatment can involve aggressive medication (diuretics to decrease fluid production), treatment of other conditions that may be causing the glaucoma, and surgery to improve drainage, although frequently the glaucomatous eye is simply removed. The remaining eye is treated medically and followed for symptoms in an effort to postpone its glaucoma. If the remaining eye becomes severely affected, it may also have to be removed.

The condition is hereditary and affected animals and known carriers should not be used in breeding.

**Optic Nerve Hypoplasia** occurs somewhat infrequently in the terrier group, although recently detected in cairn terriers. It is a congenital malformation of the optic nerve; there are a decreased number of nerve fibers and the nerve itself has a reduced diameter. Optic nerve hypoplasia can be unilateral or bilateral. Unilaterally affected dogs are usually minimally impaired and live with virtually normal visual function. Bilaterally affected dogs are usually blind. Signs are constantly dilated eyes and the absence of a reflex to light. Affected dogs, and known carriers, must not be used in breeding.

**Optic Nerve or Posterior Segment Coloboma** is, at this point, rarely found in terriers, but it is one characteristic of collie eye anomaly. "Coloboma" refers to a "pitted" or "notched" area. It is considered hereditary, and affected dogs and known carriers should not be bred. The "pitted area" in posterior segment colobomas occurs in the optic nerve disc. If the pitted and non functioning area of the disc is relatively small, vision is usually minimally affected. Larger areas of pitting or total involvement in the optic nerve are associated with blindness. On examination, colobomas appear as gray or whitish areas in the retina-optic nerve area. Extensive colobomas sometimes occur with detached retinas, in which parts of the retina loosens from the back of the eye, floats in the vitreous, and obscures vision. This symptom usually happens, if it does at all, in the first year of the affected puppy's life.

## Summary

Table 1 summarizes the main facts about these diseases.

### **Table 1. Most Frequently Encountered Eye Conditions in Terrier Breeds**

| Condition            | Symptoms                            | Treatment Options a | Prevention b |
|----------------------|-------------------------------------|---------------------|--------------|
| 1. Cataracts         | Clouded lens                        | Medication/ surgery | Neuter/spay  |
| 2. PRA               | Retinal degeneration                | No treatment        | Neuter/spay  |
| 3. Lens luxation     | Dislocated lens                     | Medication/ surgery | -----        |
| 4. Retinal dysplasia | Abnormally developed retina         | No treatment        | Neuter/spay  |
| 5. PPM/ PHA          | Retained membrane or hyaloid artery | No treatment        | Neuter/spay  |
| 6. Entropion         | Inward eyelid                       | Medication/ surgery | -----        |
| 7. Glaucoma          | Increased IOP                       | Medication/ surgery | Neuter/spay  |
| 8. ON Hypoplasia     | Narrowed nerve                      | No treatment        | Neuter/spay  |
| 9. ON Coloboma       | Pitted nerve disc                   | No treatment        | Neuter/spay  |

Notes: a. Either or in combination

b. Neuter/spay affected animals and known carriers

### **Overcoming Inertia and Denial: Welcoming The Truth**

Most people have heard of some of these conditions, often occurring in humans and other mammals, but the mere mention of these diseases can send shivers up the spine of any breeder of purebred terriers. Some breeders who have decades of experience and success are among

those who have the most to lose by spaying or neutering a valued animal. They are understandably fearful of the diseases themselves, fearful of losing a reputation for high-quality animals, and at times also fearful of even knowing what conditions, if any, their dogs have. For some breeders, ignorance is bliss. The anxiety behind this position clarifies our problems: breeders must fight not only genetically transmitted diseases themselves but their sometimes paralyzing fear of them, too.

By maintaining the health of eyes in each of our animals and in our lines, breeders will be protecting their dogs, present and future, from the misery of ocular pain, visual dysfunction, and even blindness. Further, with testing (annual examinations conducted by a VO), registration of clear dogs with CERF (<http://www.vmdb.org/history.html>), and thoughtful breeding programs, breeders will control genetic transmission of eye conditions. Following these regimens, breeders also protect their show and pet families from the heartbreak that accompanies the chronic illness of a family member.

Overcoming the fear of knowing might be easier if we consider the immense cost of doing nothing. We are in a series of long-term protracted wars against these diseases. The time for ignorance and gossip is past. These diseases will not disappear by themselves.

We will fight these diseases with the assurance that, one DNA marker at a time and one clear litter at a time, we and our beloved terriers will cease being their victims.

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Note: The authors are both Cairn Terrier breeder-handlers and co-chairs of the Greater Twin Cities Su-Mac Cairn Terrier Club's Ocular Melanosis and CERF Toto clinics held October 27, 2001 and on 26 October, 2002, in St. Louis Park, MN. The clinic is one of several sponsored by the Cairn Terrier Club of America and the CTCA Foundation to support research to identify markers for ocular melanosis and glaucoma in Cairn Terriers. Data, blood samples, and pedigrees of any affected Cairn Terriers are forwarded to principal investigator Dr. Simon Petersen-Jones of Michigan State University, East Lansing, MI. The authors thank Sue Ellen Bell, RN, PhD, and Simon Petersen-Jones, DVetMed, PhD, MRCVS, for their clinical reviews of this material and their helpful suggestions. We thank Wendy Schadewald for her skilled edit of the copy.