



# Pet-vision

Ophthalmology Referral Practice

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# Newsletter

**Welcome to our second newsletter. The format is slightly different as we highlight some interesting cases instead of breezing through statistics about what was more or less commonly referred during the past 12 months. Corneal disease and cataract however remained the most commonly presented conditions.**

## Aberrant tissue

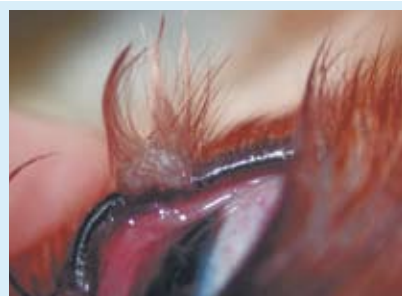
*Dermoids* are relatively uncommon in the dog (or cat). They represent tissue in an abnormal location and may contain elements such as fat, glands, hair



D1. Epibulbar or limbal dermoid.



D2. Lower eyelid dermoids in a CKCS pup.



D3. View of the larger dermoid.

follicles and hair. In ophthalmology we see these mostly around the limbus (D1) with long hairs from the dermoid often resulting in irritation. They are removed by sclero-keratectomy. Eyelid dermoids are, at least in my experience, rare. These may result from isolated ectoderm and are therefore perhaps not true dermoids but just overgrowth of jumbled up tissue. Multiple (2) dermoids over the right lower eyelid (D2,D3) causing severe irritation and epiphora were found in an 11 week old CKCS pup.



D4. 6 weeks postoperatively.

They were removed using wedge resections, somewhat modifying this technique near the medial canthus because of the close proximity of the medial dermoid

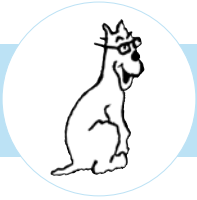
to the lower lacrimal punctum and cannaliculus. Re-examination 4 weeks later showed a good cosmetic end result (D4).

## Glue it

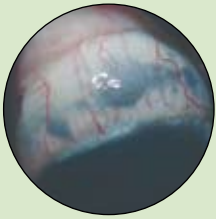
In several cases we used *tissue glue* (N-butyl cyanoacrylate adhesive) to reinforce wounds following earlier corneal surgery. The benefit of glue is that it provides an instant (temporary) seal but it may also help with wound healing by stimulating inflammation and fibrovascular repair. Studies have shown that it creates a barrier against polymorphonuclear leucocytes and their enzymes, decreasing stromal melting and that it also has anti-infectious properties. Care should be taken though to apply small amounts because the adhesive expands on polymerisation. Glue has been used for a long time in treatment of corneal ulcers and several papers describe clinical applications (R1).

## Pigment!

In man there is a form of glaucoma, caused by slowly progressive accumulation of pigment granules in the eye (pigmentary glaucoma). A similar condition exists in the dog. This was first described in the Cairn Terrier (R2) but has also since been documented in the Labrador Retriever and Boxer. In these cases there is a slowly progressive but relentless pigment cell proliferation within both eyes eventually resulting in secondary glaucoma due to obstruction of the drainage angle and scleral venous plexus. The pigment invades the iris, ciliary body, iridocorneal angle and to a lesser extent the choroid, sclera and episcleral tissues.



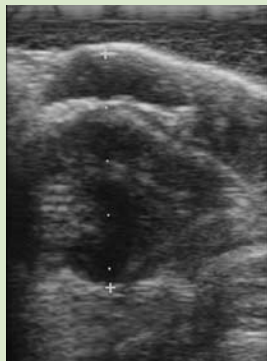
**P1. Ocular melanosis.**  
Advanced pigmentary changes in a Cairn Terrier – glaucoma stage.



**P2. Less advanced case in a Cairn Terrier.** IOP 19 mm. Hg and visual eye.



**P3. Unilateral ocular melanosis** affecting the left eye in a WHWT. Diffuse unilateral pigment infiltration. Note the band of pigment over the limbal endothelium.



**P4. Thickening of the iris,** cornea and sclera.

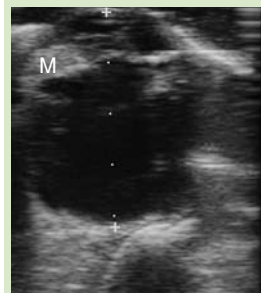


**P5. Normal right eye of the** same dog.

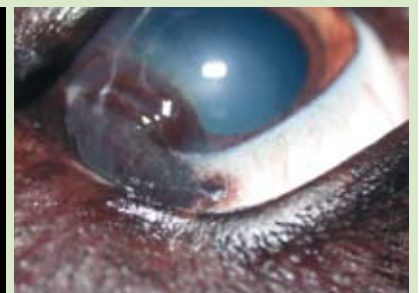
There is no absolute consensus as to whether this condition is a (benign) neoplastic process (diffuse melanoma) or proliferation of a primitive melanocytic cell type. The condition is commonly referred to as *ocular melanosis* or *melanocytic glaucoma*.

The most striking sign on presentation is the increase of pigmentation over the episclera and limbus with a pigment band extending along the limbal cornea (P1). The cases I have seen have over the years have always been bilateral including a very recent one in a Cairn (P2). We diagnosed unilateral melanosis however in a WHWT (P3, P4, P5) and again this raised the question whether we were dealing with melanosis or a diffuse type of (benign) iris melanoma (and whether perhaps this is one and the same condition after all). Because it was felt that the pigment proliferation was more than likely benign, prophylactic glaucoma treatment (Travatan drops) was started and the eye was monitored closely before glaucoma developed 11 months later. Ocular melanosis or melanocytic glaucoma was confirmed by histology.

The more common presentation of canine (uveal) melanocytic neoplasm is not the pigment explosion as seen with the above cases but an increase in iris pigmentation or pigmented lesion (mass), usually relatively well demarcated (P6) with in some advanced cases inflammation or overt glaucoma. These tumours are almost invariably benign and have a low metastatic potential but they can be locally invasive.



**P6. Iris melanoma in a Pekingese.** Focal iris mass (M).



**P7. Epibulbar melanoma in a GSD** with considerable extension into the cornea.

Further on the subject of benign pigment, we have seen the occasional *epibulbar melanoma* (P7). These tumours are typically benign and may extend slowly into the cornea, sclera and conjunctiva although intraocular extension is possible. German Shepherds and Labradors seem more commonly affected. In the older dog the tumours tend to be more stationary. The difficulty sometimes is establishing whether they are truly epibulbar (episcleral) tumours or an extension of intraocular (uveal) melanoma. A recent retrospective study of 30 cases (R3) concluded that on histology a minority (n = 4) showed some features of malignancy (small amount of cells with malignant appearance, features such as tumour necrosis). It also demonstrated that in the Golden and Labrador Retriever there is some evidence that iris melanoma, diffuse ocular melanosis and epibulbar melanoma have a hereditary basis, because of some affected dogs being traced back to a single male dog on both the maternal and paternal sides of their pedigrees.



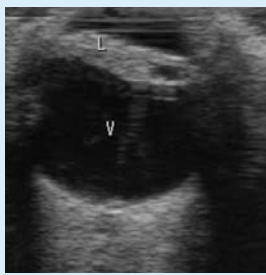
**C1. Retinal Dysplasia with total** retinal detachment (RD) in a young English Springer Spaniel. The detached retina is thickened.

### Congenital trouble

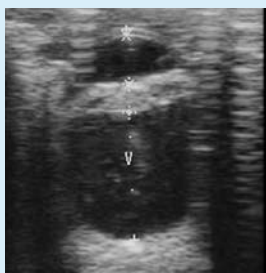
Retinal dysplasia is condition where the retina has not developed properly. This is an inherited bilateral condition in some pure breeds and the lesions can vary from a few insignificant folds to total retinal detachment (C1). Dogs may have one eye with minor lesions and one severely affected eye and it

is interesting to see how little there is in these dog's behaviour that alerts the owner to any problems. Some eyes with total retinal detachment go on to develop glaucoma. Gonioscopy in the contralateral eye is often normal.

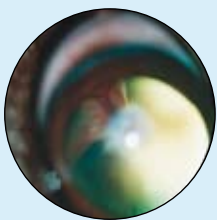
Two young dogs (Molly and Peppa, both Labradors) were seen with a severe expression of abnormal vitreous development and incomplete regression of



**C2.** Young Labrador, right eye. Persistent hyaloid artery seen as a moderately echodense strand in the central vitreous. Hypermature cataract with lens collapse.



**C3.** Different image of the same eye. V = vitreal strand representing persistent hyalooid.



**C4.** Persistent hyaloid and hyperplastic tunica vasculosa lentis in a young Labrador. Tissue band with blood vessels running from the fundus (optic nerve area) through the vitreous and attaching (white opacity) to the back of the lens.

the hyaloid system and tunica vasculosa lentis (vascular tunic surrounding the lens) that is present during the very early development of the eye. In Peppa's case the abnormal tissue could be readily seen (C4). In Molly's case there was also total cataract and lens induced uveitis. A persistent hyaloid was first suspected because of blood traces found within the lens. The persistent vessel was just about noticeable on ultrasound (C2,C3). We operated successfully on Molly, using cautery to seal off the vessel ends over the posterior lens capsule. On postoperative examination another abnormality (retinal dysplasia) then came to light but Molly today enjoys good vision from this eye and the biggest reward to us was to hear that she has become more confident and adventurous.

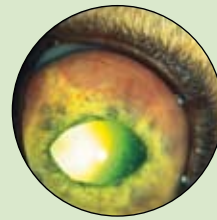
### Blood pressure

I have always heavily relied on our Doppler monitor to evaluate *blood pressure* and it seems generally accepted that this is the most reliable form of assessing (systolic)

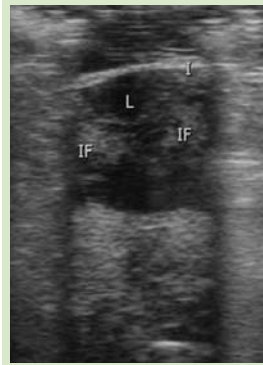
blood pressure, more so than by oscillometric method. After testing multiparameter vital signs monitors we found that a dedicated single purpose blood pressure monitor outperformed blood pressure assessment by multiparameter machines. Our machine has been very consistent in giving reliable results and I was encouraged to see that in some cases these matched those obtained by colleagues using the "gold standard" Doppler method on the same patient.

### Uveitis

Chronic (anterior - intermediate) uveitis in cats can be an enigma. Some of these patients are generally well but present with persistent iritis (U1) sometimes resulting in lens luxation or secondary glaucoma. Serology (including *Toxoplasma gondii*) is often



**U1.** Chronic iritis. Iris nodules and neo-vascularisation over the iris.



**U2.** Vitreous infiltrate (IF) in a DSH with chronic bilateral anterior - intermediate uveitis. L = lens, I = iris.

unremarkable and these cases may be classified under "idiopathic lymphocytic-plasmacytic" uveitis. They do respond well to topical steroid treatment but need ongoing treatment and careful monitoring. White - grey "snowy" infiltrates in the vitreous are commonly seen and these infiltrates make it difficult to examine the fundus. The infiltrates are also easily demonstrated on ultrasound (U2).

### So where is the lesion located?

Neuro-ophthalmology cases are always challenging. And when we feel we have finally learned something from staring at complex illustrations of visual and pupillary light

reflex pathways we are quickly reminded by the next patient that text-book cases do not exist. Some of the cases based on history (rapid onset blindness) and findings from examination (i.e. absence of any fundus abnormalities) could be classified as SARDS (Sudden Acquired Retinal Degeneration). The absence of a pupillary light reflex is often described as a prerequisite for a diagnosis of SARDS but is this always "black and white"? In some of these cases a response is still detectable (sometimes only using a very bright light stimulus) but the electroretinogram is consistent with abnormal retinal function. In two of these cases hyperadrenocorticism was diagnosed and there does appear to be a (though somewhat tenuous) link between this condition and SARDS.

*Parotid duct transpositions* are a last resort for cases where medical treatment of keratoconjunctivitis sicca is not possible and most patients are much better off. The intensity of saliva flow cannot be predicted however. Many patients progress well (PDT1 - see overleaf) but in some cases there are complications from heavy saliva flow, mineral deposition (PDT 2 - see overleaf) and irritation from slightly alkaline saliva. Reducing saliva flow by partially ligating the transposed duct may be the only option in those cases. EDTA solutions, tetracycline treatment, special diets, adding yoghurt and Vitamin C to the diet and drugs to



**PDT 1.** Cross - right eye. 2 months after PDT. Low levels of saliva flow.



**PDT 2.** WHWT - left eye. 2 weeks after PDT. Heavy saliva flow with saliva sediment accumulation over the cornea, eyelids and periocular area.

reduce saliva flow (unfortunately these also have side effects) have all been described as remedies following PDT. Constricting the duct however could in the long - term lead to sialolith formation (R4).

### Penetrating or perforating injuries

can be very destructive making repair or successful treatment impossible.

There is often severe uveitis with intraocular haemorrhage,

substantial loss of corneal tissue (sometimes because of rapid stroma break down from proteolysis), prolapse of ocular contents (iris) or lens damage (PI 4). It may be very difficult to recognise intraocular structures

(PI 1). Intraocular foreign bodies are rare and they may only be diagnosed following enucleation (PI 2) because of severe uveitis and intraocular opacity at the time.

In cases of traumatic proptosis there is often irreversible damage and blindness, although globes can sometimes be repositioned and preserved following intense conservative treatment. Intraocular assessments are not an option in many of these cases (e.g. because of hyphaema) and although perhaps uncommon, globe rupture (PI 3) should be ruled out.

Nearly all our patients were dogs and cats; we saw the occasional exotic and horse. Severe keratitis from what appeared to be a KCS like



**PI 1.** Perforating injury in a YT. Severe intraocular haemorrhage surrounding the lens. There is also retinal detachment.

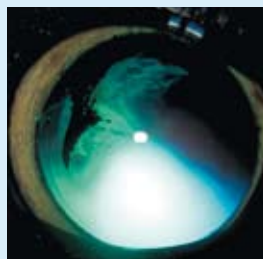


**PI 2.** Labrador, right eye. Foreign body found lodged in the lens following enucleation for endophthalmitis and glaucoma.

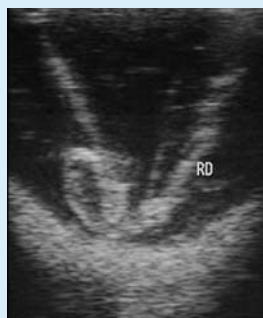
condition was diagnosed in a marmoset. KCS in this species apparently is very rare. One of the horses, Billy Red, presented with suspected impaired vision in the right eye but the history was obscure. He was diagnosed with unilateral blindness and early physical changes, a common complication in the horse.



**PI 3.** DSH. Rupture of the globe following proptosis. The lens is identified and there is severe haemorrhage in the vitreous extending into the retrobulbar space.



**PI 4.** Sequel to lens perforation in a DSH. Pigment deposit over the lens capsule and cataract formation.



**H1.** Total retinal detachment in the horse with fragment of coiled up detached retina.



**H2.** Granula Iridicae in the horse.

Most of the findings were consistent with damage from chronic inflammation. The iris was darkened and a large brown spherical pigmented mass (cyst) was found in the ventral iris. There was cataract and lens luxation and on ophthalmoscopy retinal detachment was suspected. This was confirmed by ultrasound (H1). I could not resist leaving out the image of the granulae iridicae (H2).

As you can see this newsletter seems to be mostly about anarchistic cell behaviour and destructive intraocular disease. Next time we will focus on something a little more different.

A year ago and somewhat prematurely I planned to have an open day and I apologise to all those who showed interest to come along and kindly returned forms. With some walls to be knocked out in the next few months it looks as if we have to put the plans on ice again but I promise to make work of it in the very near future. In the mean time thanks to all practices and surgeons for sending us cases !

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