ABSTRACTS

for

BrAVO

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A Miniature Lens-free Direct Ophthalmoscope

Roger H Armour
Retired Consultant Surgeon, Lister Hospital, Stevenage

INTRODUCTION

I here describe the performance of a new, uncomplicated ophthalmoscope.

METHOD

The original model was home-made in June 2000 from black card, a reflector and a pen torch (Fig 1). It is now produced as the 14cm long, 45g Optyse™ that works on two AAA batteries, has only one light and no lenses (Fig 2). Observers who normally wear glasses keep their glasses on.

TESTS AND RESULTS

Bristol Eye Hospital Trial (observer wearing spectacles). The trial ophthalmologist wore his glasses (RE+4.00D, LE+3.500D) when examining 24 patients and had a sharply focused view in them all.

Ease of use At a charity club meeting in Cardiff, 26 consecutive delegates who had not used an ophthalmoscope before all saw my retinal vessels through my tropicamide-dilated pupil.

Auto-ophthalmoscopy (Fig 3). Using the ophthalmoscope and two plane mirrors, I have seen my own discs and retinal vessels. Unexpectedly, I also saw two microaneurysms due to diabetes (Fig 4). An ophthalmologist colleague confirmed the findings.

Observations on animals (Fig 5, 6). The instrument has not had a formal veterinary trial. I looked at the fundi of a variety of healthy mammals, and of frogs, tortoises, turtles, snakes and fish, and had a sharply focused view except in rabbits, guinea pigs and some fish. In the case of these exceptions, monocular indirect ophthalmoscopy with the instrument may give a focused image.

CONCLUSIONS

The clarity of view and ease of use of this pen-sized ophthalmoscope make it a handy pocket companion, always there when needed for students to practise, as I have, on family, friends and pets, and for practitioners to help their patients. It appears to merit veterinary trials.

Figure 1: The home-made ophthalmoscope

Figure 2: The Optyse™ ophthalmoscope

Figure 3: Auto-ophthalmoscopy

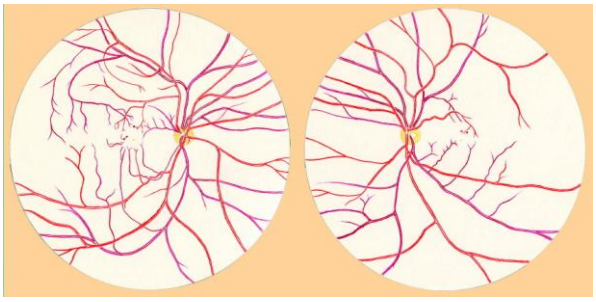

Figure 4: Unexpected results

Figure 5: Direct ophthalmoscopy

Figure 6: Indirect ophthalmoscopy

To Applanate or to Rebound?
A preliminary comparison of the Tonopen and Tonovet tonometers.

David Williams MA VetMB PhD CertVOphthal MRCVS and Matt Green BA VetMB MRCVS

Department of Veterinary Medicine, University of Cambridge,
Madingley Road, Cambridge CB3 0ES

Introduction

For many years the Tonopen has been the standard, hand-held tonometer for veterinary use, providing an electronic measurement of intraocular pressure widely accepted throughout veterinary ophthalmology. It has some shortcomings, particularly when used in small eyes such as those of laboratory rodents, where the footplate is just too wide for accurate tonometry. More than 60 years ago, Obbink invented a totally different concept in tonometry,[1] where a small probe is fired at the corneal surface and its rebound velocity is measured, this correlating with intraocular pressure. Dekking improved the technique thirty years later [2] but it was not brought into widespread use until the beginning of this decade when Kontiola and colleagues developed a system for use in mice.[3] The device has been marketed for the veterinary market as Tonovet (Figure 1).

Comparison of this rebound or dynamic tonometer with the Tonopen applanation tonometer has been reported in the literature but only to date investigating animals with normotensive eyes.[4] We have undertaken both *in vitro* and *in vivo* studies of the device but here we provide some preliminary *in vivo* evidence comparing the two tonometers in both normotensive and glaucomatous canine eyes.

Materials and Methods

50 dogs of varying breeds with and without ocular pathology were included in the preliminary study. Dogs were examined ophthalmologically with direct and indirect ophthalmoscopy and slit lamp biomicroscopy. Measurements of intraocular pressure were performed with both tonometers. Topical anaesthetic (Amethocaine Minims, Chauvin) was applied to each eye 30 seconds prior to measurement with the Tonopen tonometer. Order of use of the Tonovet rebound tonometer and the Tonopen applanation tonometer was assigned randomly.

Results

Four measurements were taken with each tonometer and the mean value was used as the intraocular pressure measurement for that eye. This mean value for intraocular pressure as measured with the Tonovet was plotted against the averaged readings for to Tonopen for each eye (Figure 2). In 12 dogs the mean value for one or other tonometer has a standard deviation greater than 10%, normally associated with a less than placid temperament, and these figures are shown as blue data points in figure 2. There was good correlation between the readings obtained with the Tonovet rebound tonometer and the Tonopen applanation tonometer (r=0.96) but as can be seen from

figure 2, the Tonovet apparently over-estimated the intraocular pressure in severely glaucomatous eyes by around 2mmHg at 30mmHg, 5mmHg at 50mmHg and 7mmHg at 70mmHg.

Discussion

Both tonometers were easy to use; the Tonovet had the advantage of not requiring local anaesthetic but the disadvantage of requiring horizontal use while the Tonopen can be used in any position. The good correlation between the two tonometers is encouraging although, as can be seen from figure 2 for each individual set of readings there can be significant variation between mean values. The higher values for the Tonovet compared with the Tonopen, especially in glaucomatous eyes is to be noted, although it is, of course, impossible from these readings to say which tonometer is providing the more accurate readings compared with the true pressure within the eye. We have performed experiments comparing Tonovet and Tonopen pressure readings in post-mortem eyes with the true intraocular pressure as determined by a U-tube manometer and will present these at a future meeting. This study shows that the Tonovet tonometer is a valuable tool in veterinary ophthalmology, useful for measuring the intraocular pressure in the canine eye.

References

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[2] Dekking HM, Coster HD (1967) Dynamic tonometry. *Ophthalmologica* 154: 59–74
[3] Kontiola AI, Goldblum D, Mittag T (2001) The induction/impact tonometer: a new instrument to measure intraocular pressure in the rat. *Experimental Eye Research* 73: 781–785
[4] Leiva M, Naranjo C, Pena MT (2006) Comparison of the rebound tonometer (ICare®) to the applanation tonometer (Tonopen XL®) in normotensive dogs. *Veterinary Ophthalmology* 9: 17-21

Figure 1: The Tonovet Rebound Tonometer

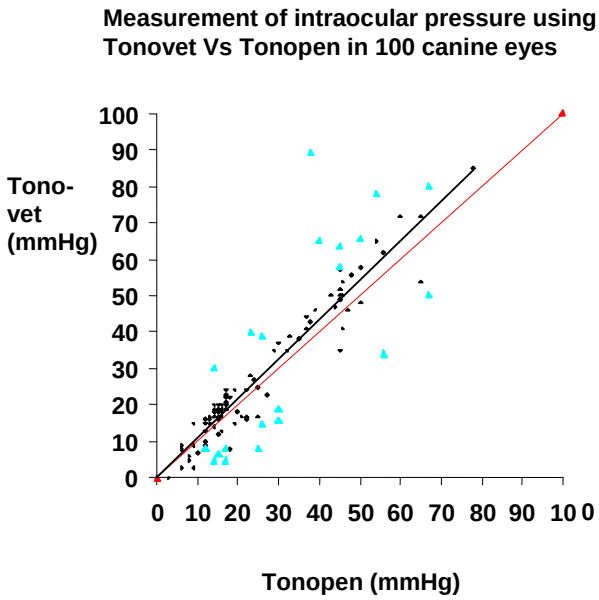

Figure 2: Graphical representation of Tonovet and Tonopen measurements

Bilateral Intraocular Tuberculosis in a Domestic Short Haired Cat

P.D.Hanlon BVSc (Syd) MRCVS
Milton Keynes Veterinary Group, Fyfield Barrow,
Walnut Tree Milton Keynes MK7 7AN

Case History: "C.J.", a seven and half year old, neutered female tortoiseshell DSH had, 15 months previously, had a case of undiagnosed uveitis in her left eye which had left her with extensive cataractous changes but had otherwise resolved.

Clinical signs: "C.J." presented with an extensive, fleshy growth filling the dorsal half of the left anterior chamber. The right pupil was dilated and non-responsive with retinal detachment and evidence of a large choroidal lesion. Biochemistry and haematology were unremarkable, she was FeLV/FIV negative, and other than the ocular signs appeared fit and well. As the left popliteal lymph node was enlarged, a FNAB was performed, the consistency of which felt quite gritty.

Proper History: 2 years previous to the uveitis "C.J." had been treated for a growth below the right eye which initially had been thought to have been a cyst but subsequently turned out to be mycobacterial pyogranuloma. This had been un-typed but she had had a 6 month course of triple therapy antibiotics which had finished 4 months prior to her bout of uveitis.

The FNAB revealed "mycobacterial consistent" organisms within some macrophages so C.J. was admitted for chest radiographs and biopsy of the affected lymph node. The sample was divided with one section in formalin for histopathology and the other for culture and speciation if mycobacterial infection was confirmed. The chest radiographs revealed an interstitial/military pattern, which is apparently typical for cats with tuberculosis (TB).

Not surprisingly the histopathology was consistent with mycobacterial granuloma, so treatment for TB was instigated. TB treatment ideally consists of an initial phase of rifampicin-fluoroquinolone-clarithromycin/azithromycin, followed by a continuation phase of rifampicin and either fluoroquinolone or clarithromycin/azithromycin. C.J. was therefore put onto rifampacin (Rifadin) 35mg sid, marbofloxacin (Marbocyl) 5mg sid and azithromycin (Zithromax) 24mg sid for an initial 2 months.

Clinically the ocular lesions were beginning to regress and as she was a very bright, appetite positive cat throughout, they were the only indicators to her clinical progress. After 2-3 months (when the bottle ran out!) the azithromycin was removed from the regime and a dual drug regime was continued for another 4 months by which time active ocular lesions had completely resolved and survey chest radiographs appeared clear.

About 3 months into the treatment regime the species was confirmed as *Mycobacterium microti*.

Discussion: Professor C Formston wrote an article, posthumously published (JSAP 1994 **35,** 5-8) *which although of limited clinical significance, could be of value to veterinary clinical history.* His article on retinal detachment and bovine tuberculosis in cats pointed out that the introduction of pasteurisation meant that the incidence of TB in cats had dramatically reduced.

A classical dissertation by Hancock and Coates (1911) described tubercle of the choroid in six separate cats. Kirk (1925) then diagnosed tuberculosis in a cat with bilateral vitreal haemorrhage and retinal detachment, on the basis of the Hancock and Coates (1911) findings. Although it is not mentioned in the article (Kirk) Professor Formston records that on post mortem no evidence was found of the disease.

Certainly in the early decades of the 20th century retinal detachments and haemorrhage were felt to be highly associated with tuberculosis, and probably often was due to the ingestion of unpasteurised milk.

Feline tuberculosis is currently infrequently recognised. When diagnosed, it is usually caused by either the cattle form, *M. bovis* or the vole form *M. microti*. The current thinking is that most infections in cats result from "a last laugh" by predated species as moles and rats can carry *M. bovis* and wild mice and voles often carry *M. microti*. Most commonly TB in cats manifests itself initially with non-healing skin lesions on the face and legs and this is in fact the way that C.J. did initially present.

Acknowledgements: I would like to thank Chris Belford, David Gould and Danielle Gunn-Moore for their help in the diagnosis and treatment of C.J.

References

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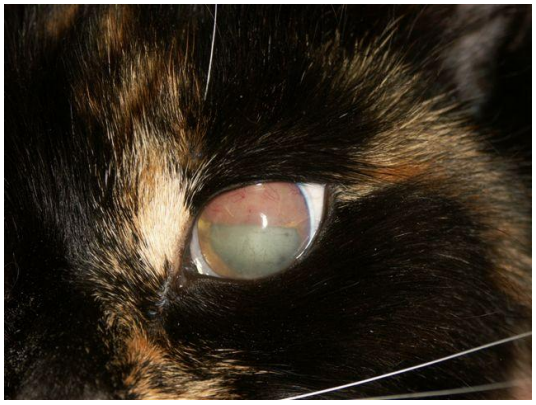


Figure 1. Left eye at presentation

Figure 2. Right eye viewed through a Volk 2.2 Pan-retinal lens

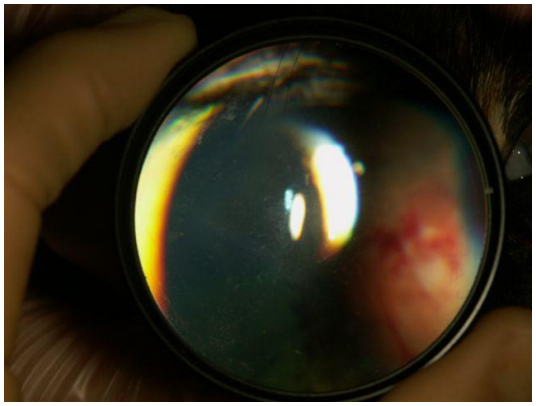


Figure 3. Right eye viewed through a Volk 2.2 Pan-retinal lens

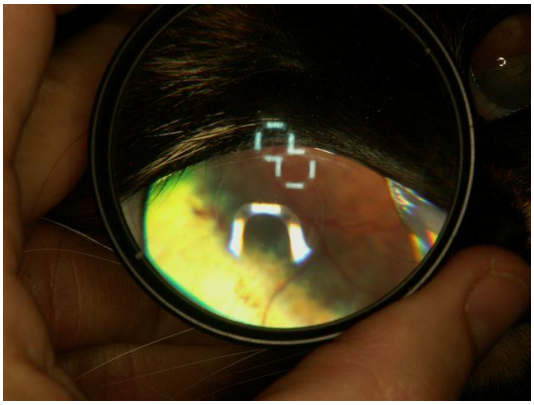


Figure 4. Right eye viewed through a Volk 2.2 Pan-retinal lens

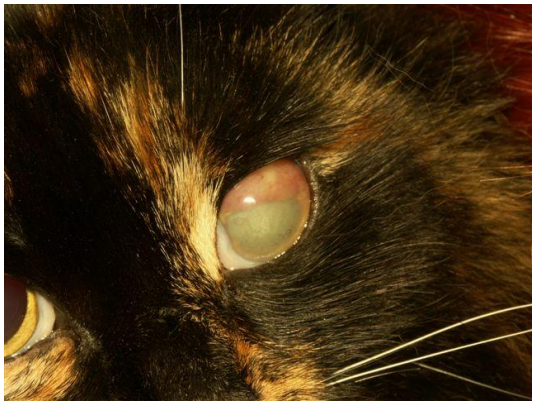


Figure 5. Left eye 2 weeks post presentation at the start of treatment

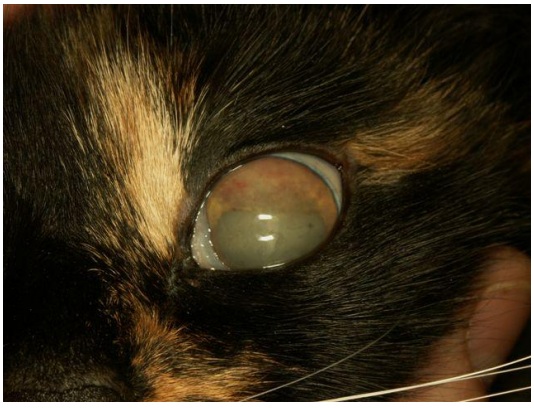


Figure 6. Left eye 2 months after the start of treatment

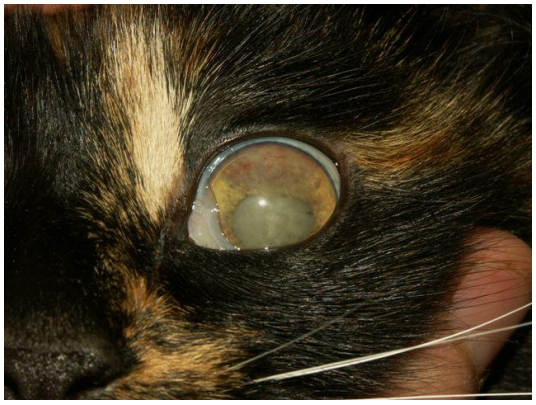


Figure 7. Left eye 4 months after the start of treatment

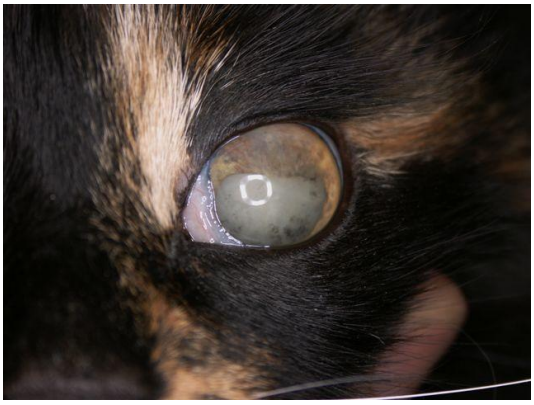


Figure 8. Left eye 6 months after the start of treatment

Figure 9.
Chest x-ray before treatment.

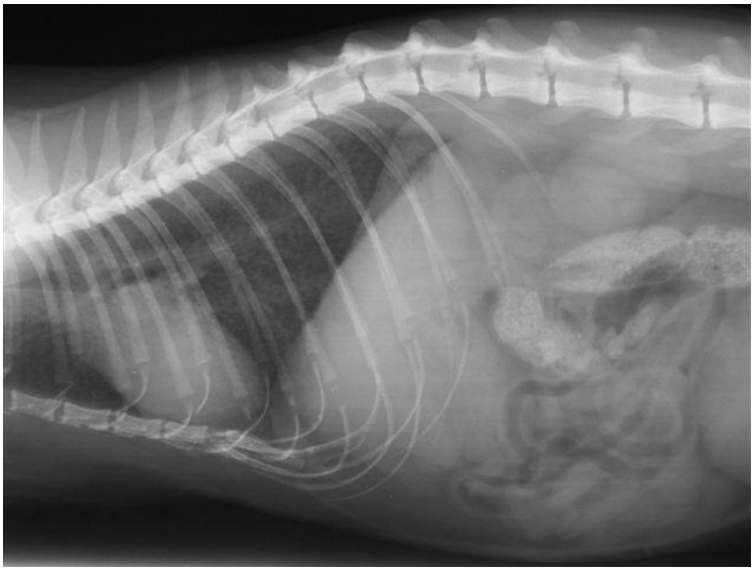
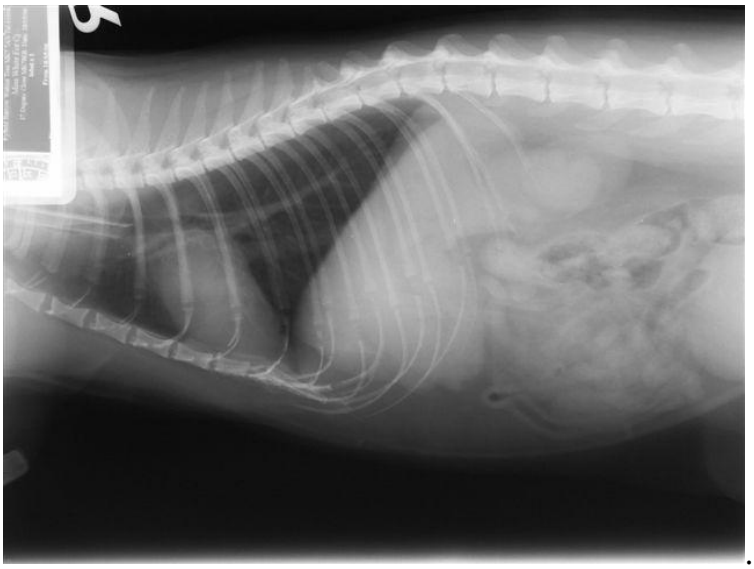


Figure 10.
Chest x-ray at the end of treatment.



Feline Central Retinal Degeneration

J.D. Wills

Severn Veterinary Centre, Tybridge House
St. John's, Worcester WR2 5BA

A twelve year old, female neutered domestic short-hair cat presented with inappropriate urination and defecation, dark loose faeces, vomiting and polydipsia of several months' duration. Her diet consisted solely of Hi-life tuna flakes (Town and Country Petfoods, Melton Mowbray), a complementary cat food. Previous treatment consisted of anthelmintics and robotics, both unsuccessful.

General physical examination revealed 10% weight loss since last seen 3 years previously, cardiac arrhythmia, enlarged kidneys and gassy/liquid intestinal contents. A syncopal episode occurred when stressed. Ophthalmic examination revealed bilateral, horizontal oval-shaped areas of tapetal hyper-reflectivity dorsolateral to the optic discs, with sharply defined borders. No other ocular abnormalities were noted. Complete haematology and routine biochemistry were carried out, together with urine specific gravity (USG) and urine dipstick analysis. Mild azotaemia, mild hepatic enzyme elevation, neutrophilia and inappropriately low USG (1.015) were present. Subsequent urine culture yielded a heavy growth of *E. coli*. Faecal analysis was unremarkable. Electrocardiogram (fig. 1) showed normal sinus rhythm at 170/min, interrupted by regular supraventricular premature complexes (SVPCs) averaging 50/min. Echocardiography (fig. 2) showed evidence for dilated cardiomyopathy (DCM) (fractional shortening 37.7%, normal 40-50%). The diet manufacturer's data showed adequate taurine: 0.25% dry matter (recommended minimum 0.2%).

Feline Central Retinal Degeneration (FCRD) was diagnosed, considered pathognomonic for taurine deficiency and causing central retinal degeneration and DCM. The DCM further supports this. Here the retinal lesions are considered secondary to poor taurine absorption. Azotaemia with concurrent low USG, bacteruria and renomegaly suggest pyelonephritis. The other clinical pathology results are mild and non-specific. SVPCs are usually associated with atrial stretch or dilation (e.g. hypertrophic, restrictive or dilated cardiomyopathy) or may be associated with cardiac neoplasia or secondary to systemic disease (e.g. hyperthyroidism, hypertension secondary to renal disease). Echocardiography showed evidence of DCM. Vomiting and diarrhoea with melaena may be due to many different causes. The immediate response to change of diet and unremarkable faecal analysis support dietary sensitivity/hypersensitivity, although other causes may be involved.

Treatment included:

- Benazepril (Fortekor®, Novartis) 2.5mg SID for 2 months (DCM)
- Enrofloxacin (Baytril®, Bayer) 15mg SID for 1 month (based on urine culture)
- Diet changed to Hills Feline Oral Care
- Taurine 250mg SID (Taurine, Solgar) indefinitely

The diarrhoea and vomiting resolved immediately following the change of diet. The inappropriate urination and polydipsia were not noted while hospitalised, nor reported later; subsequent urinalysis was not performed. Four weeks after initiating treatment, the heart rate was reduced to 140/min, with a more regular rhythm developing. The retinal lesions remained static indicating that taurine levels were adequate. No visual deficits were clinically evident at any point. Repeat clinical pathology, ultrasound and ECG were not possible due to financial circumstances.

The prognosis is good. DCM should resolve with adequate taurine levels [1]. There will be permanent blindness and tapetal hyper-reflectivity in the area centralis, since the changes are irreversible [2]. The rest of the retina should function normally and visual deficits are unlikely to be clinically apparent [3], provided that taurine deficiency does not recur. The diarrhoea, vomiting and urinary tract infection should not return.

Taurine deficiency is the only known cause of FCRD. Taurine deficiency is usually seen with low dietary intake [3] and the condition is today considered rare since commercial diets now contain high levels. This case was interesting because the diet contained adequate levels of taurine. Taurine is secreted in bile and absorbed by the enterohepatic circulation [4]. The diarrhoea seen in this case may have increased faecal losses by reducing transit time and thus re-absorption of taurine. Bacterial overgrowth may also degrade taurine in the lower bowel [4]. With a history consistent with dietary sensitivity/hypersensitivity (the immediate cessation of diarrhoea following diet change), it is also possible that intestinal malabsorption may have caused the taurine deficiency; intestinal biopsies would be required to support this potential aetiology, but the animal's poor cardiovascular status precluded general anaesthesia. Other reasons for taurine deficiency despite dietary taurine levels higher than recommended have been reported in the literature, but no definitive cause has been proved. Pion and Kittleson [1] hypothesised that the recommendations were too low or that the taurine was not adequately absorbed. Morris *et al* [4] reported that "diets containing similar concentrations of taurine...support vastly different plasma levels of taurine". They postulated that canning produces substances that inhibit taurine absorption or increase taurine degradation by enteric microbes. Changing to a non-canned diet may have been the important factor, rather than taurine supplementation.

Aguirre [3] classified the retinal changes seen in taurine deficiency with a 5-stage scheme. Lesions begin at the area centralis (fig. 3) and eventually form

a horizontal band-shaped lesion superior to the optic disc, which can spread to involve the entire retina. More recent work [5] showed the periphery of the retina to be affected also, forming a circumferential annulus, confluent with the horizontal band. This cat had bilateral stage 2 lesions, i.e. solitary areas of tapetal hyper reflectivity dorsolateral to the optic disc. The hyper-reflectivity in these lesions is due to retinal degeneration and atrophy (Bellhorn and Fischer, 1970). The factors for the distribution of lesions remain unclear: it cannot be accounted for by a preferential effect on cone photoreceptors, or by light damage (Leon *et al*, 1995). These authors also showed pathological changes in photoreceptors over an area larger than that evident macroscopically.

Unfortunately plasma taurine levels were not assayed in this case due to cost so the diagnosis of taurine deficiency must be tentative. Since the retinal damage is permanent, and since taurine levels were unknown at presentation, it is possible that this cat may have had a low taurine episode that had resolved long before presentation leaving the hyper-reflective areas in the retinas. The concurrent DCM, however, implies that the plasma levels *were* low at presentation: DCM is almost exclusively caused by taurine deficiency, the prognosis being grave without dietary correction. With normal plasma taurine it resolves [1] within a few weeks. Benazepril was used to support cardiac function in this case as it is licensed for use in cats with renal insufficiency. Further discussion of DCM treatment falls outside the scope of this report.

Taurine deficiency is not associated with renal pathology in the literature, but is associated with immune dysfunction [1,4]. This may have caused the pyelonephritis in this case, or it may have been coincidental. Certainly breaching of the intestinal mucosa (as evidenced by the melaena) will predispose to translocation and circulation of *E. coli*, and hence haematogenous urinary tract infection. Conversely, increased renal taurine loss may have contributed to the deficiency. This case underlines the importance of an ophthalmic examination in apparently non-ocular cases, as many such cases show ocular manifestations of systemic disease.

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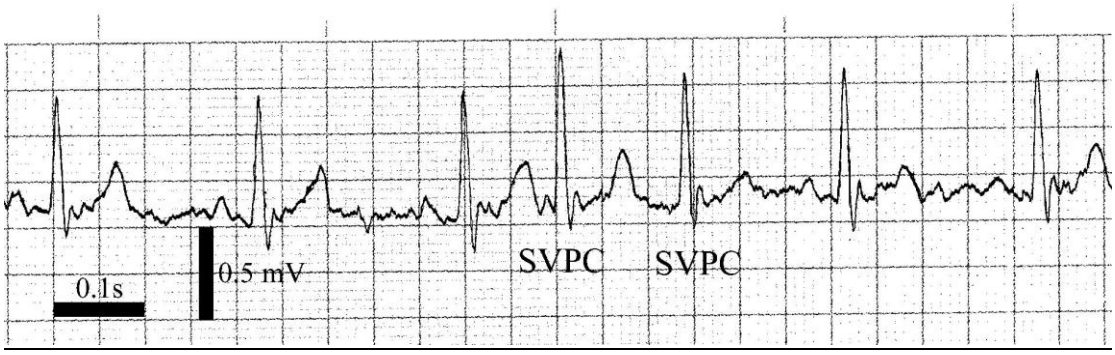

Figure 1 Electrocardiogram

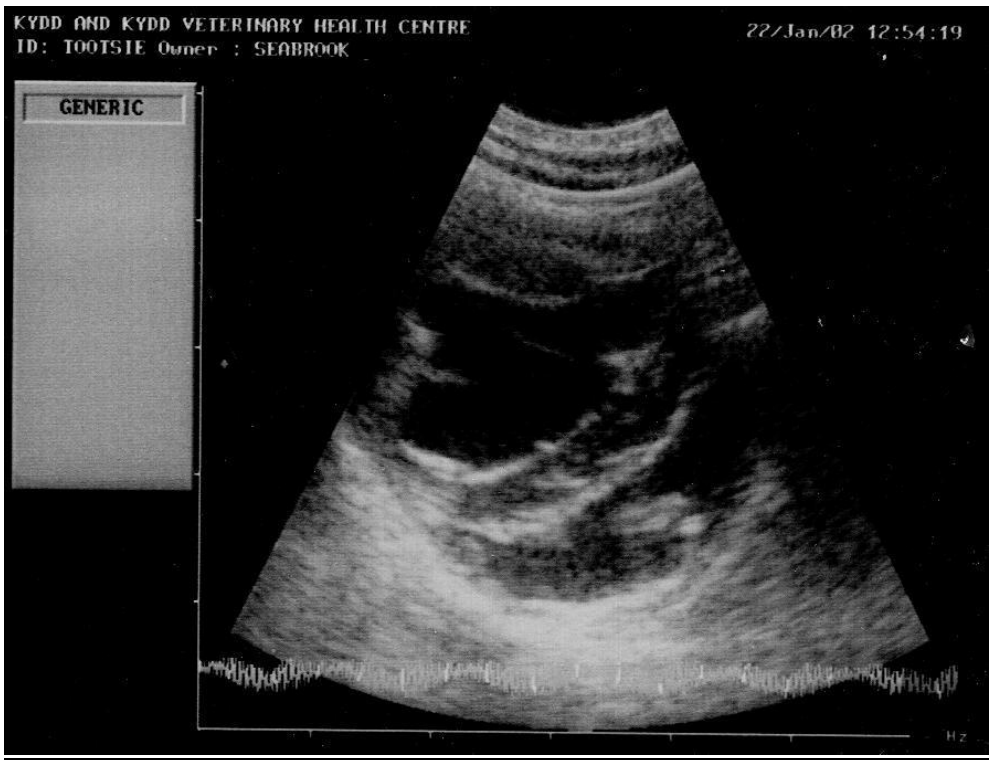

Figure 2 Cardiac ultrasonogram

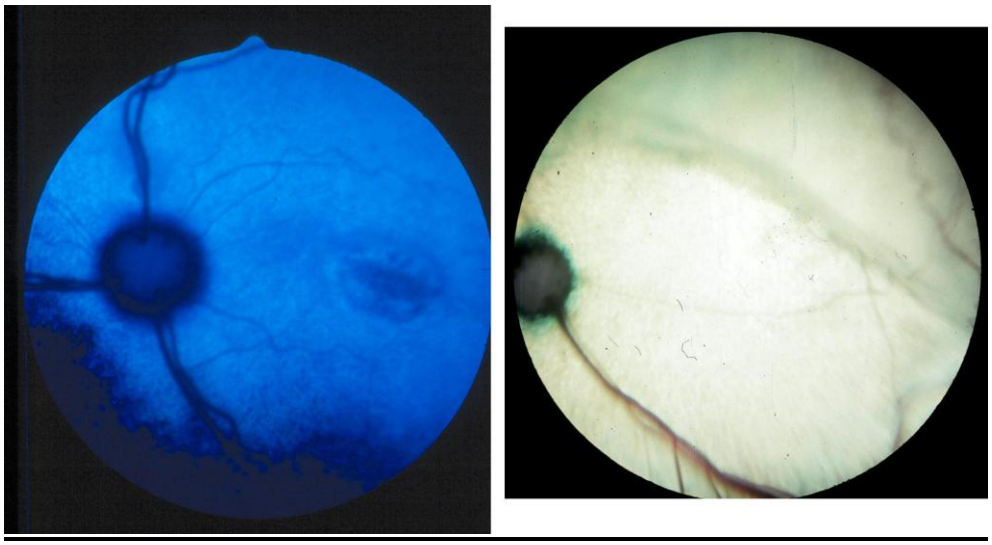

Figure 3 Retinal lesions (courtesy of Martyn King)