Newsletter June 2014

QML Pathology | Vetnostics

Dr Jeanine Sandy

WELCOME DR JEANINE SANDY, VETERINARY PATHOLOGIST

Dr Jeanine Sandy BVSc (Hons), PhD, MACVSc, DACVP

Dr Sandy graduated from the University of Queensland with her Bachelor of Veterinary Science in 1991 with first class honours. Having completed her PhD at the University of NSW in 1999, Dr Sandy lectured in Veterinary Pathology at the University of Melbourne from 2000, before moving to the USA in 2009 to take up a clinical lecturing/instructor role at NCSU. Dr Sandy successfully attained her specialist qualification in Veterinary Anatomic Pathology from ACVP (American College of Veterinary Pathologists) in 2010.

Dr Jeanine Sandy joins us with extensive experience having held a number of veterinary and university lecturing positions both here in Australia and the USA.

NEW PRICE LIST - COMING SOON!

Soon we will be sending out copies of our new 2014 price list. We have redesigned the format to include codes of test panels. These codes will assist you when speaking with our staff about tests, and can be used as an alternative to the test name when filling in request forms.

Our extensive list is always growing and this year has been expanded to include more analytes and more of the tests we are regularly asked about.

All prices have been reviewed, with some of the tests that are sent to external laboratories having a larger price increase reflective of the costs of transport. QML Pathology Vetnostics still remains extremely cost competitive compared to other laboratories.

As a policy, we try to keep the price of our send away tests as low as possible (compared with our competitors) to allow Veterinarians to investigate their cases as comprehensively as possible.

If you have any further queries, please contact Vetnostics on email vetnostics@qml.com.au or phone 1300 838 765 (1300 VET QML) – option 6.

Dr Sue Foster
Veterinary Medical Consultant

ADRENALS: What you won't find in a textbook

Dr Sue Foster BVSc, MVetClinStud, FACVSc
Vetnostics Small Animal Medical Consultant

PART 3: ROUTINE CLINICAL PATHOLOGY

Hyperadrenocorticism (hyperA)

HAEMATOLOGY

1. Lymphopenia and absolute eosinopenia are the most frequently cited haematologic abnormalities (approximately 80% of dogs according to Feldman and Nelson 2004). However, they are not always present. Lymphocyte counts in hyperA dogs at Vetnostics quite often seem to be normal which would be consistent with the finding in one study that only 14% dogs with hyperA had lymphopenia (Peterson, 1984). Eosinopenia seems far more common than lymphopenia (consistent with the figure of 84% by Peterson, 1984). However, eosinophils can be normal to increased if there is a concurrent eosinophilic process (uncommon but occasionally seen).
2. Nucleated red cells are seen quite commonly (usually in low numbers, 1-3/100 WBC). Whilst this number would be classed as normal, it is more common in dogs with hyperA than in other dogs of similar age with no reported history or signs typical of hyperA.

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3. High normal or increased platelet counts are often apparent. The cause and significance of this is unknown.

BIOCHEMISTRY

1. Increased alkaline phosphatase (ALP) is widely cited as the most common routine laboratory abnormality but it is not always increased. A normal ALP does not rule out hyperA.
2. Increased ALP is largely due to induction of a specific ALP isoenzyme by glucocorticoids. Although this isoenzyme can be evaluated, it has been shown that an increase can be caused by a variety of disorders and is not specific for hyperadrenocorticism (Solter et al 1993). The steroid-induced isoenzyme cannot be used to distinguish spontaneous or iatrogenic hyperA from liver disease or diabetes mellitus for example. It is often stated that 70-100% of the increase in hyperA dogs will be due to this isoenzyme but this is certainly not always the case with either iatrogenic or spontaneous hyperA (Feldman and Nelson 2004).
3. Lipaemia is very common in hyperA dogs. Most of the old studies that reported biochemistry findings did not report the frequency of hypertriglyceridaemia in hyperA dogs as veterinary laboratories have not typically run triglyceride concentrations. The triglyceride increases in hyperA dogs are often quite marked and the serum/plasma consequently often has a strawberry milkshake appearance, even on a fasted sample. HyperA should always be on the DDx list for fasting hypertriglyceridaemia in an otherwise well dog (or cat!). This also holds for breeds such as Miniature Schnauzers, not of all which will have familial hypertriglyceridaemia; Miniature Schnauzers do get hyperA.
4. Mild hyperglycaemia is reported as occurring in 45-60% of hyperA dogs (Peterson 1984, Feldman and Nelson 2004) but this would not be the case in my own patients or Vetnostics' cases. Whilst hyperglycaemia can occur, it would seem to occur at a much lower rate in our patients.
5. Alanine aminotransferase (ALT) is commonly increased but again, not necessarily. It is not usually increased to the same extent as ALP.
6. Urea concentration may be decreased due to polydipsia.
7. Hypokalaemia and hypernatraemia may occasionally be seen and are probably more common in dogs with adrenal tumours as the cause of their hyperA (presumably excessive mineralocorticoid secretion).
8. Bile acids test results may be increased in dogs with hyperA (Center et al 1985).
9. Serum lipase may be increased by exogenous corticosteroids (dexamethasone) thus possibly by endogenous glucocorticoids also. Although it would not be routinely measured in hyperA cases, this must be borne in mind when analysing lipase in potential pancreatitis cases. The effect of glucocorticoid excess on cPLI is currently unknown.

URINE

1. Urine in dogs with hyperA is usually isosthenuric or hyposthenuric and in one old study (Meijer 1980), 80-85% of dogs had a USG <1.013. However, as we often pick up hyperA much earlier (i.e., before they become textbook classics) that figure is probably an overestimate in modern medicine. Not all hyperA dogs have polydipsia/polyuria as presenting signs; only 82% of 300 hyperA dogs had PU/PD in one report (Peterson 1984). In addition, many dogs can concentrate their urine reasonably after being in a hospital so the urine concentration measured at any one moment, could be hyposthenuric, isosthenuric or concentrated.
2. Urinary tract infection (UTI) reportedly occurs in 40-50% of hyperA dogs (Feldman and Nelson 2004). Again, I think it would be interesting to review that figure. Frequency of UTI has probably

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Roth L, Tyler RD. Evaluation of low sodium: potassium ratios in dogs. J Vet Diagn Invest 1999;11:60-64

Seth M, Drobatz KJ, Church DB, Hess RS. White blood cell count and the sodium to potassium ratio to screen for hypoadrenocorticism in dogs. J Vet Intern Med 2011;25:1351-1356

Solter PF, Hoffmann WE, Hungerford LL et al. Assessment of corticosteroid-induced alkaline phosphatase isoenzyme as a screening test for hyperadrenocorticism in dogs. J Am Vet Med Assoc 1993:203:534-538

decreased with earlier detection of the disease but as urine culture is not routine, this is impossible to assess.

3. It is important to remember that a) hyperA dogs with UTIs may not have any pyuria or haematuria (presumably because of the anti-inflammatory effect of excess glucocorticoids) and b) routine sediment examination on a wet preparation may fail to detect white cells and bacteria in dilute or weakly concentrated urine. A stained, air-dried smear will increase detection of both white cells and bacteria but culture is usually required to detect UTIs in dogs with hyperA. UTIs may well be undiagnosed in hyperA dogs.

Hypoadrenocorticism (hypoA)

HAEMATOLOGY

1. Lack of a stress leucogram in a sick dog can be an indication of hypoA and may be the only clinicopathologic abnormality in dogs with glucocorticoid deficient (atypical) hypoA. When I ask vets about the leucogram in suspected hypoA cases, the common response is “Everything is normal”. Remember, a normal leucogram can be quite abnormal for a collapsed dog and each count should be assessed with respect to the dog.
2. Lymphocytosis is not always present.
3. Lymphopenia is a good ‘rule-out’ for hypoA. I have never seen a hypoA case with lymphopenia, however, a recent study on lymphocyte counts in dogs with hypoA (Seth et al 2011) did identify a few low lymphocyte counts. In this study, 100% of hypoA dogs had a lymphocyte count >0.75x109/L and 92% had lymphocyte counts >1.00x109/L.
4. Eosinophilia is also not always present.
5. I have never yet seen a hypoA dog with an eosinophil count of 0, thus an eosinophil count of 0 would be a good ‘rule-out’ for hypoA. Let me know if you have a hypoA dog with an eosinophil count of 0!

BIOCHEMISTRY

1. A Na:K ratio of <27 is NOT diagnostic for hypoA. In one study only 24% of dogs with Na:K ratio <24 had hypoA and 41% of dogs had renal disease (Roth and Tyler 1999). It is worth noting that all dogs in that study with Na:K ratios <15 had hypoA. Other diseases causing low Na:K ratios include whipworm (and other gastrointestinal diseases), renal disease, pancreatitis, diabetes mellitus, pyometra and body cavity effusions. Another larger, more recent study showed that whilst hypoA was the most common cause of a Na:K ratio <27, only 16.7% of dogs with Na:K ratio <27 had hypoA (and that was after the cases with suspected EDTA contamination had been removed from the sample population).

GENERAL

1. Combining the Na:K ratio with the lymphocyte count is diagnostically superior as a screening test than either alone (Seth et al 2011).
2. A faecal flotation test should be performed in all dogs with a low Na:K ratio. Occasionally dogs have both whipworm and hypoA!!
3. A manual differential white cell count (of at least 100 cells) is mandatory in order to detect hypoA leucocyte ‘patterns’ with any accuracy. Most of the in-house analysers would not perform with high enough accuracy (Papasouliotis et al 1999, Bienzle et al 2000, Papasouliotis et al 2006) to detect the subtle ‘patterns’ of hypoA.

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Figure 1

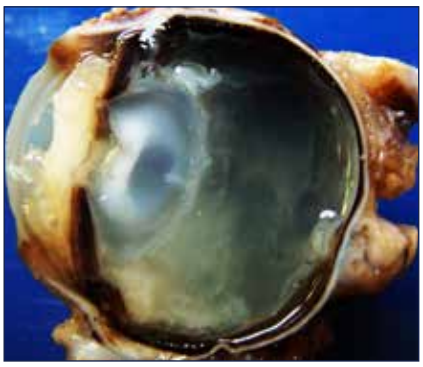

Figure 2

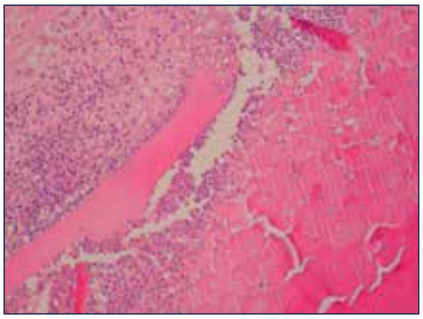

Figure 3

FOCUS-EYEPATHLAB CASE PRESENTATION 1: Investigation of a case of acute onset glaucoma in an English Springer Spaniel

Dr Karen Dunn, Consultant Veterinary Ocular Pathologist

The eye from a five-year-old entire male English Springer Spaniel was submitted to FOCUS-EyePathLab from Ireland. The dog had a one week history of a cloudy, red left eye, and on referral he had an opaque cornea (Fig. 1), with possible hypopyon in the anterior chamber; the eye was glaucomatous and buphthalmic, with an IOP of 76 mmHg. The iridocorneal angle in the fellow eye was narrow on gonioscopy, but appeared sufficient.

The eye was enucleated, and vertical sagittal sectioning of the globe at gross examination revealed a distorted, cataractous lens, with an apparent anterior capsular deficit, along with exudative material (coagulated by fixative) in all ocular chambers (Fig. 2).

Histologically, there was a severe, chronic suppurative to granulomatous and focally necrotizing anterior uveitis, with fibrinosuppurative exudate in all chambers, including the vitreous (vitritis); there was also retinitis and pre-iridal fibrovascular membrane formation. The anterior lens capsule was ruptured, with fraying of the edges, and there was advanced cataractous change and 'phacitis' (inflammation within the lens cortex) (see Fig. 3). Small numbers of Gram-positive bacteria were found within the lens cortex, and a penetrating injury to the eye was considered likely, with intraocular bacterial localisation causing anterior uveitis, progressing to endophthalmitis; lens capsule rupture would also have resulted in a phacolytic component to the uveitis. The dog recovered well without any post-operative complications.

This is an example of secondary glaucoma associated with a penetrating injury and bacterial endophthalmitis; histopathological examination was able to rule out other differential causes of glaucoma in this case, such as goniodysgenesis and intraocular neoplasia, thereby assisting in establishing the prognosis for the fellow eye.

Fig 1. The affected left eye showing marked corneal oedema and peripheral neovascularisation. Clinical image kindly supplied by Natasha Mitchell MVB DVOphthal MRCVS of Eye Vet, Ireland.

Fig 2. Macro photograph after vertical sectioning of the globe showing a distorted, cataractous lens with an apparent deficit at the centre of the anterior capsule (the suspected lens penetration site), and showing turbid, exudative material in all chambers; the exudative material forms a plug across the pupillary aperture, and floccular exudative material is present in the ventral posterior and vitreous chambers.

Fig 3. Photomicrograph demonstrating discontinuity and fraying of the anterior lens capsule at the site of perforation, surrounded by degenerating neutrophils and macrophages (exudate), with globular, degenerate cataractous lens material, H&E stain, 100x magnification.

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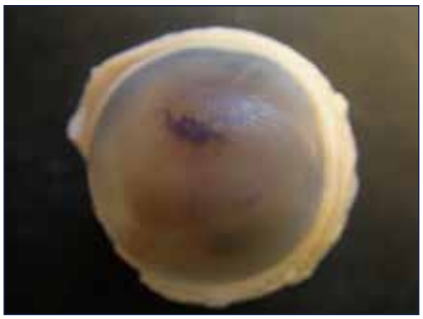


Front and caudal views of eye correctly submitted. Trimming away excess tissue allows optimal fixation.

SUBMISSION ADVICE FOR OCULAR PATHOLOGY SPECIMENS

Dr Karen Dunn, Consultant Veterinary Ocular Pathologist, FOCUS-EyePathLab

SUBMISSION FORM – please provide as much detail as possible.

- Indicate the eye affected, and if the condition is uni- or bilateral.
- Use diagrams provided to mark areas/lesions of interest, or site of biopsy, if applicable.
- **Detailed clinical history** is vital to interpretation of histological findings.
- Please indicate if an intraocular lens implant (IOL) is present in the globe, and if it is a hard or soft (foldable) implant.

TISSUE PREPARATION

- **Whole globes – trim away excess tissues** for optimal fixation, unless involved in disease process (the nictitans/third eyelid will not interfere with fixation).
- **Please leave enucleated globes intact** (incision causes collapse and distortion, hampering gross evaluation, and neutral buffered formalin diffuses readily into intact globes trimmed of excess tissues).
- Globe orientation is still possible without lids; however, location of **lesions of interest may be marked with a suture** if desired.

GENERAL SUBMISSION ADVICE

- **Handle tissues gently** to avoid artifacts (be aware that cautery will cause artifactual changes).
- Place tissue **into fixative rapidly** to avoid desiccation.
- Always use a **suitable volume of fixative**, ideally a 1:10 ratio of tissue sample to neutral buffered formalin (in the case of a large equine eye, you can prefix for 24 hours, and then reduce the volume of fixative for mailing).
- Use a **suitable size** of leak-proof submission container (please DO NOT cram tissue into a narrow-necked container, as the container may need to be broken to retrieve the biopsy once it is fixed and no longer flexible).
- **Label the submission container** with the animal and owner's name, and site of biopsy if applicable.

FOCUS-EyePathLab (run by Dr Karen Dunn), is now available as an ocular pathology consultancy service for Vetnostics in both Queensland and NSW. Karen is a Queensland veterinary graduate who worked in the UK for many years before returning to Brisbane just over 12 months ago. FOCUS-EyePathLab was set up by Karen in 2006, and is a dedicated ocular pathology service for specialist veterinary ophthalmologists and clinicians with a special interest in ophthalmology; FOCUS-EyePathLab continues to receive samples from veterinary ophthalmologists in the UK and Europe, and is now also receiving samples from specialists in Australia.

Karen, along with the Vetnostics pathology team in Brisbane, conducts monthly eye pathology rounds for veterinary ophthalmologists at QML Pathology at the central lab in Murarrie, and has been invited to speak at the combined Ophthalmology and Pathology chapter day at Science Week in July this year.

Samples can be submitted to FOCUS-EyePathLab using the routine courier service for QML Vetnostics; the cost of the service is $160 + GST*. Further information on the ocular pathology service offered by FOCUS-EyePathLab through QML Vetnostics can be found at www.FocusEyePathLab.com, or by contacting Vetnostics Veterinary Pathology.

FOCUS
EYE PATH LAB

Submission Form

Karen Dunn BVSc (Hons)

Referring Veterinary Surgeon

Vetnostics Doctor Code: _____

Email: _____

Patient

☐ Dog ☐ Cat ☐ Horse ☐ Other: _____

Breed (or likely cross if mixed) : _____

Age: _____ ☐ Male ☐ Female ☐ Spayed/Neutered

Owner: _____ Your Reference: _____

Animal Name / ID: _____

Please email the pathologist on karen.dunn@FocusEyePathLab.com when you submit a sample.

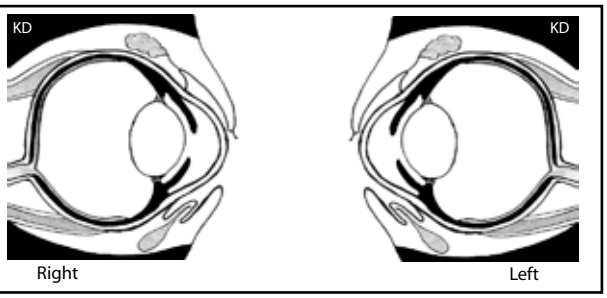
Submitted Material

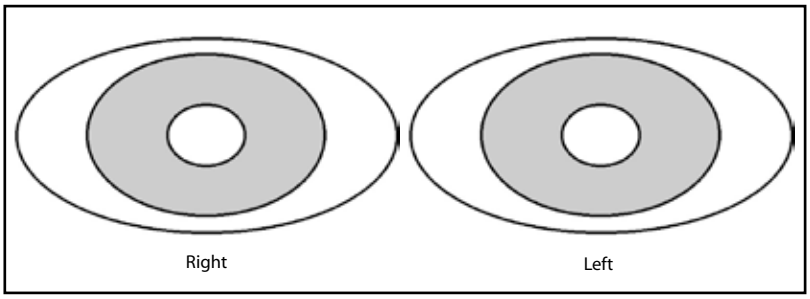
Whole eye ☐ Left ☐ Right

Biopsy site ☐ Wedge ☐ Excision Sites: _____ Number of pieces: _____

Other: _____

Date Sampled _____ **Previous Submission** Lab No. / Date _____





Clinical Differential Diagnoses

History

Additional information (please complete as appropriate)

Glaucoma: Yes / No IOP: _____mmHg Duration: _____ | IOL Implant Type: Hard / Soft (foldable) | Blue Eye: Yes / No Haircoat Colour: _____

Treatment given and response

Submit samples to:
FOCUS-EyePathLab
C/- Vetnostics Veterinary Pathology
in your state

LABORATORY USE ONLY

Collection Date:	Histo Samples Received:
Request Date:	Vetnostics DE Code: HVE, VKH (QML) VOH (LAV)

Samples submitted to FOCUS-EyePathLab may be used for anonymous disease surveillance and research purposes unless otherwise requested at the time of submission.
Submission of samples constitutes acceptance of our terms & conditions which can be found at ***www.FocusEyePathLab.com***

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Professional development for Australia's veterinarians

AVA APPROVED
AVA members automatically accumulate Vet Ed points when they provide their AVA number.

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The program enables veterinarians to systematically assess their diagnostic accuracy in the clinical setting, with the ultimate goal of improving both their pathology skills and the quality of patient care.

EARN CPD POINTS

Earn **one** structured CPD point for every **four** specimens submitted, where a clinical workup has been sufficiently documented and clinical diagnoses are accurate.

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Participants simply need to submit samples together with a completed **QML Vetnostics Pathology CPD Program request form** to participate and automatically receive program reports and CPD points.

RECEIVE REPORTS ANNUALLY

A CPD points certificate, and report will be provided to participants annually, detailing:

- total number of examined specimens
- prevalence of lesion types observed
- diagnostic accuracy for each lesion type
- margin clearance (Histo only).

To order QML Vetnostics Pathology CPD request forms, please email vetnostics@qml.com.au or call 1300 VET QML (1300 838 765) and select option 6 (All other enquiries).

Available Now

Vet diagnostic test kits

QML Pathology is a leading supplier of vaccines to the medical and veterinary community.

In conjunction with veterinary vaccines and consumables, QML Pathology has expanded its vaccinations catalogue to include the Witness range of in-house diagnostic kits available to our veterinary customers.

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NCS INTERNATIONAL CERTIFIED QUALITY MANAGEMENT SYSTEM ISO 9001

QML Pathology Veterinary Vaccines

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- **phone: (07) 3121 4523**

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- **Next day delivery** is available (excluding weekends and public holidays) for surgeries located within metropolitan and regional centres throughout QLD and Northern NSW.
- Commonly requested **veterinary consumables** are also available for purchase to bundle with in-house kits and vaccines.

QML Pathology Vetnostics Service

For further information about QML Pathology's Vetnostics Service

Phone: 1300 VET QML

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