

# VETERINARY PATHOLOGY REPORT

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## **Management Committee's Report**

Welcome to the second Veterinary Pathology Report for 2004.

There are details in this issue (below) about the up-coming AGM to be held on October 16-17th as a satellite of the International Academy of Pathology Congress which runs in Brisbane over the preceding 5 days. Please note dates and details and ensure that your abstracts are submitted on time.

The ASVP website [www.asvp.asn.au](http://www.asvp.asn.au) is still up and visible most of the time, despite your ham-fisted secretary's fumbblings in the field of website maintenance. Fortunately, Cleve Main has been standing in slips as website OIC and has been a great support. One problem with the site is that it is being sadly under-utilised by you the membership, from whose subscriptions the web hosting fee and domain charges are deducted. Please do not hesitate to point out errors you find on the site, and please send Cleve or me any items of news or information that you think should be placed on the site for the benefit of all our members. Right now most search engines do not pick up the site, and your management committee has decided that it should be more visible. However, before we do that, it is hoped that the site can have more material placed on it that will be of greater interest to the casual browser, not to mention any senior policy maker. In other words, the site could let the wider public know more about the Society, its strengths and its contributions to the community, in addition to serving as a resource for its members. Please give some thought to this because suggestions and inclusions will be actively sought from now on.

The mailing list seems to be functioning as it should after the odd glitch earlier in the year, but again, it is being used precious little by members. We pay the same regardless of how many or few messages are posted, so why not master your shyness and post items of interest? You can post images as well as text; the only proviso being that file sizes should be kept below about 250KB so as not to enrage subscribers who are on dial-up connections rather than broadband.

If, despite your membership being paid up to date and your having tried to subscribe to the ASVP Mailing List, you have not received any mailings, please contact the Secretary, Roger Kelly [Roger.Kelly@uq.edu.au](mailto:Roger.Kelly@uq.edu.au) ph.(07) 3371 4463

## **ASVP Annual General Meeting, Oct 16-17th, 2004**

Invitations for papers for the weekend AGM have been sent out and have been on the website for some time now. The chosen themes this year are exotic disease and clinical pathology. A distinguished panel of speakers has been drafted for the exotics section, including Gardner Murray, Rod Campbell, Hume Field and Francisco Uzal. In addition, Bruce Williams (AFIP) and John Cullen (North Carolina

University) are coming to the IAP and will stay on for our meeting. There have as yet been no offers of presentations on clinical pathology, so please give that some consideration. Members wishing to present papers on either theme or on any other topic are invited, nay urged, to submit abstracts as soon as possible to avoid congestion or even missing the cut. The deadline for submission of abstracts is September 1st.

Abstracts for member presentations (non-themed) will be limited to one A4 page of Times Roman 12 font text: larger abstracts may be brutally edited to size. Abstracts from invited speakers and other themed abstracts may be longer, depending on the mood of the Proceedings editor on the day.

**Agenda Items for the AGM:** these will include a proposal to broaden our membership base by admitting non-pathologist laboratory-based veterinary diagnosticians to the Society, since there has been a suggestion to form an Australian branch of the World Association of Veterinary Laboratory Diagnosticians. Joining forces with such a group might produce a more effective body of laboratory-based veterinarians. Details of this and other agenda items will be sent out closer to the meeting. Members are urged to contact the honorary secretary ([Roger.Kelly@ug.edu.au](mailto:Roger.Kelly@ug.edu.au)) as soon as possible with items they wish to have included on the agenda of the business meeting to be held on the afternoon of Saturday, Oct. 16th, 2004.

**Venue for the AGM:** The AGM will be held in Room 37 on level 6 of the Webb Centre, Queensland College of Art (Griffith University, Southbank Campus, Merivale St, South Brisbane). This venue is quite close to the IAP Congress venue (see below); this is for the convenience of those who want to attend both meetings.

**Accommodation** Bookings in the South Brisbane area are likely to be tight, especially during the 5 days of the IAP Congress. Below is a list of local hostels with a range of tariffs. Delegates should use the contact details provided to make their own bookings, and give some thought to booking early to avoid disappointment.

**Mater Hill Motel**, 1 Allen St, South Brisbane 07 3846 3188 \$79 - 89  
single/double/ twin/share

**Southbank Mater Motel** cnr. Graham St & Raymond Tce 07 3844 9133 \$80 - 90  
- 95 single/double/ twin/share

**Hillcrest Central Apartments**, 31 Vulture St 1800 678 659 \$100 - \$124  
single/twin

**Diana Plaza Hotel**, 12 Annerley Rd 'Gabba 07 3896 1692 \$135/n

**Riverside Hotel**, 20 Montague St, S. Brisbane 1800 301 101 \$140 twin/share

**Sapphire Resort Motel**, 55 Boundary St, S. Brisbane 07 3217 2588  
[www.sapphireresort.com.au](http://www.sapphireresort.com.au) \$85 - \$99

**Edmonstone Motel**, 24 Edmonstone St S. Brisbane 07 32550777  
[www.edmotel@bigpond.net.au](mailto:www.edmotel@bigpond.net.au) \$85 - \$95/n

**Parkview Motel**, cnr. Russel & Edmonstone Sts, S. Brisbane 07 3846 2900  
www.parkviewmotel.ozemail.com.au (n/a)

**The International Academy of Pathology Congress (Oct.11-15th)** will be held in the Brisbane Convention Centre, South Brisbane. The program for this big meeting is extremely varied and exciting as can be seen by visiting the Congress website at [www.iap04.im.com.au](http://www.iap04.im.com.au) where the full program is now displayed. Congress organizers have set aside a session on Friday afternoon for veterinary pathology, and it has been decided to make regional emerging zoonoses (SARS, Nipah, influenza, Hendra, etc) the theme for this session, since these topics are likely to be of particular interest to the medicos. ASVP members who wish to attend one or more days of the IAP Congress can do so on payment of the daily registration of \$150, which is the rate set for other non-medical Congress delegates, and includes refreshment breaks and lunch.

**Slide-of-the-Month Scheme:**Your management committee, as expected, has been underwhelmed by offers from people prepared to resuscitate the Slide of the Month scheme. But there has been good news on funding of the Registry and associated activities which may enable a suitable alternative (see Tony Ross's report below: Australian Animal Pathology Standards Program).

**Digital archiving** of Veterinary Pathology reports and Registry case records may be undertaken with the aim of providing all members with online access to searchable text files of all Reports and Registry cases. A small trial has shown that even the tattiest copy of an old Report can be scanned with optical character recognition software to yield useful digital text (admittedly with plenty of mistakes) that could be edited into very valuable files that could be searched by author, keyword, date, geographical location, etc. Members will be kept informed of developments.

Roger Kelly  
**Honorary Secretary**  
**for the Management Committee**

## **Australian Animal Pathology Standards Program**

The Australian Society for Veterinary Pathology is delighted to announce a major new initiative. After several years without funding, the activities of the National Registry of Domestic Animal Pathology (NRDAP) have been expanded, re-badged and funded for 5 years from 1 July 2004.

After gaining support along the way from SCAHLS and AHC (Subcommittee on Animal Health Laboratory Standards and Animal Health Committee) the Primary industries Standing Committee approved an ASVP proposal for funding for 5 years. The budget for 2004/5 is \$104 460. A further 4 years of funding is agreed subject to higher levels of cost recovery from public and private users of the program as well as a review of the formula for attributing governments' share of the funding.

NRDAP has been renamed the Australian Animal Pathology Standards Program (AAPSP) and its business plan places greater emphasis on proficiency testing and diagnostic preparedness for emergency animal diseases. It continues the strong commitment to continuing education and adds development of teaching modules, which will be made available to members everywhere via the Internet as well as CD/DVD.

ASVP has entered into a partnership with Animal Health Australia to manage the program. AHA will provide ongoing administrative and financial support. ASVP will provide the scientific leadership and technical coordination. SCAHLS will monitor the program. Tony Ross has been appointed by the ASVP executive as Scientific Coordinator of the program.

Progress to date includes:

1. Formation of a management committee chaired by Peter Morcombe of Animal Health Australia with representatives of ASVP (Tony Ross), SCAHLS (Malcolm Lancaster) and the current host institution NSW Agriculture (Keith Walker).
2. Calling for expressions of interest from relevant organisations to become the host institution for the next 5 years. See notice in this Vet Path Report.
3. Proposal for a revised funding formula that is based on production animal populations in each state, as used in other AHA programs.
4. Invitation to all veterinary pathologists in Australia to join the program.
5. Confirmation that access to all program activities will be restricted to pathologists and pathology groups who are financial stakeholders in the program.
6. Determination to present the first continuing education module to members by Christmas 2004 on CD/DVD.
7. Following up the offer by AHA to use its website to access continuing education modules.
8. Discussion of the proposed veterinary pathology proficiency-testing program with existing and potential providers.
9. Performance of a stock-take of NRDAP assets.

10. Draft an ongoing program of continuing education.

The management committee and the ASVP executive would like to particularly thank Dr Barry Richards for his skilled preparation and persistent advocacy of this initiative.

All members are encouraged to contact Tony Ross and other members of the management committee with feedback about any aspect of the program.:

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[Peter.morcombe@aahc.com.au](mailto:Peter.morcombe@aahc.com.au)

[Malcolm.lancaster@dpi.vic.gov.au](mailto:Malcolm.lancaster@dpi.vic.gov.au)

[Keith.walker@agric.nsw.gov.au](mailto:Keith.walker@agric.nsw.gov.au)

### **EXPRESSIONS OF INTEREST**

Animal Health Australia and the Australian Society for Veterinary Pathology invite expressions of interest from institutions and companies to host and mentor the Australian Animal Pathology Standards Program.

The AAPSP was formerly known as the National Registry of Domestic Animal Pathology (NRDAP). It is currently hosted by the NSW Dept. Primary Industries at the Elizabeth Macarthur Agricultural Institute, Menangle, NSW.

The AAPSP requires office and laboratory support for its Registrar and visitors and collection of histological slides, kodachromes, digital images, case reports, reprints and books.

The host institution is asked to provide access to good quality microscope, digital imaging and computer equipment. AHA will provide financial administration and the ASVP will provide scientific direction.

The term of the hosting period is 5 years beginning in late 2004. Expressions of interest close on Friday 13 August 2004. For further information contact scientific coordinator Dr Tony Ross ph 0246 406312 [tony.ross@agric.nsw.gov.au](mailto:tony.ross@agric.nsw.gov.au) or Dr Peter Morcombe, Technical Services Manager AHA ph 0262 033944 [peter.morcombe@ahc.com.au](mailto:peter.morcombe@ahc.com.au)

Send expressions of interest to:  
The Hon. Secretary, ASVP  
48 Twelfth Ave,  
St Lucia Qld 4067  
([Roger.Kelly@uq.edu.au](mailto:Roger.Kelly@uq.edu.au))

by Wed 11 August 2004

**Tony Ross**  
**EMAI Menangle, NSW Agriculture**      [tony.ross@agric.nsw.gov.au](mailto:tony.ross@agric.nsw.gov.au)

## **The Australian Wildlife Health Network**

ASVP members who are also part of the Australian Wildlife Health Network will have received mailings recently about leatherjacket mortalities. I have also forwarded one or two of these (with the blessing of the coordinator of the Network) to ASVP mailing list subscribers, in case some might not be aware of the network. It seems to be a mailing list much like our own: I quote from its charter:

"The Australian Wildlife Health Network (AWHN) is a National initiative of the Commonwealth Government and is managed under the Wildlife Exotic Disease Preparedness Program (Australian Department of Agriculture, Fisheries and Forestry). Its mission is to promote and facilitate collaborative links in the investigation and management of wildlife health in support of human and animal health, biodiversity and trade".

Members who wish to be included in the Network can approach its coordinator, Dr Rupert Woods, [rwoods@zoo.nsw.gov.au](mailto:rwoods@zoo.nsw.gov.au)

## **NRDAP CE Course**

Solutions to the Case Studies in Clive Huxtable's 2003 NRDAP CE course.

Clive has kindly provided a set of notes which will be complementary to the cases he provided on CD to the participants in the Continuing Education (Neuropathology) road-show in 2003. These notes have now been posted to the mailing list, so subscribers to that will have received the notes by the time they read this.

# STATE REPORTS

## NSW – Paul Gill

### **Australian Bat Lyssavirus (ABLV) positive flying fox in Metropolitan Sydney**

Steven Hum\*, Tony Ross\* and Karrie Rose±

\*NSW Agriculture EMAI Menangle and ±Taronga Zoo

A wild adult male grey-headed flying fox was found behaving abnormally inside the outer layer of a two-layer wire mesh cage of captive flying foxes in the Lane Cove National Park, Sydney. The captive flying foxes were used for on-site and off-site education purposes. Experienced vaccinated bat handlers captured the wild bat and put it in a cage without incident. In the cage it was very aggressive, attacking objects, vocalising, convulsing and biting its genitalia. It had severe bilateral corneal ulceration and conjunctivitis and an ulcerated tongue. Its condition worsened overnight and it was taken the next day to Taronga Zoo veterinary quarantine station for euthanasia. Concerns were raised about ABLV and the possibility of exposure of the captive bats.

The body was sent to NSW Agriculture, EMAI, for necropsy examination. It appeared to be in good body condition (0.85kg) and had numerous small punch marks on the wing skin but no major trauma. Both corneas were dark green, the gastrointestinal tract was empty but no gross abnormalities were found internally.

Histologically there was occasional neuronal necrosis, and mild multifocal gliosis in the brainstem and cervical spinal cord. There was moderate diffuse lymphocyte infiltration of a dorsal root ganglion associated with acute neuronal necrosis. The cornea was diffusely infiltrated by neutrophils and had areas of ulceration.

The fluorescent antibody test performed on brain and parotid gland at AAHL was positive for the presence of lyssavirus antigen. Similarly, PCR and immunoperoxidase staining of the brain was positive, with large quantities of intra-neuronal and neuropil lyssavirus antigen dispersed throughout all regions of the brain. Lyssavirus was isolated from brain tissue homogenate.

ABLV in metropolitan Sydney was first confirmed in 2002 in a distressed 12 month-old female flying fox found in a tree. The histopathological changes in that case were similar and included very mild segmental non-suppurative meningitis and vasculitis with occasional neuronal necrosis and mild focal gliosis in the brainstem. Spinal cord was not examined histologically in that bat. Convincing Negri-type inclusion bodies were not detected in either of these cases which is consistent with a previous histopathological study<sup>1</sup> where typical Negri bodies were seen only 9 of the 21 bats examined. Meningoencephalomyelitis, neuronal necrosis, focal gliosis and lymphocytic perivascular infiltrates are not specific and can be very mild in this disease so histopathology alone is clearly not a sensitive test for ABLV infection.

1. Hooper et al, Aust Vet J, No 9, 1999.

## **PORCINE MYOCARDITIS SYNDROME**

Tony Ross\*, Rod Reece\*, Keith Walker\*, Deborah Finlaison\*, Peter Kirkland\*, Rob Walker\*, Sarah Robson\*, Steven McOrist<sup>2</sup>, Eric Thornton<sup>2</sup>, Robyn Smith<sup>2</sup>, Alex Hyatt<sup>+</sup> and Chris Morrissy<sup>+</sup>.

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NSW Agriculture\*, QAF Meat Industries<sup>2</sup> and CSIRO AAHL<sup>+</sup>

An apparently new infectious disease was detected in late term and neonatal piglets in 2 large piggeries in New South Wales. The syndrome appears to be caused by an as yet unidentified virus. Clinically the disease spread through each of the breeding units on the farms. There was a marked increase in stillbirths and a smaller increase in mummified foetuses. Abortions were rare. Sows of all parities were affected. Sudden deaths in unweaned piglets 1 to 20 days old rose sharply. Losses continued for approximately 3 months in each breeding unit. Most stillborn piglets showed no obvious gross lesions but a minority had mild to moderate anasarca. These cases had excessive clear to blood-tinged fluid in serous cavities. Some had minor amounts of fibrin strands strung across serosal surfaces of a variety of organs. Hearts were often dilated on both the left and right sides. The epicardium occasionally showed irregular areas of pallor and/or hyperaemia and ecchymotic haemorrhage. Lungs were often markedly oedematous. Livers were sometimes swollen and with rounded edges. Body fluids from about half the stillborn piglets examined showed raised IgG levels consistent with an in utero infection. Bacteriological cultures of a range of tissues were negative for known pathogens. Unweaned piglets were occasionally seen in respiratory distress but were usually found dead. Lesions were indicative of circulatory failure. Hearts were dilated and lungs were congested and oedematous with well-demarcated interlobular septa. Excessive watery fluid was present in most body cavities. The key histological lesion was an often mild, multifocal, non-suppurative interstitial myocarditis. Clusters of mononuclear cells (sometimes with the occasional neutrophil) were seen in the myocardium and sometimes in the sub-endocardium or adjacent to medium-sized blood vessels. Most cases showed very little myofibre damage, in contrast to the typical lesions of nutritional cardiomyopathies. However a minority of cases did show areas of early myocardial fibrosis. Marked periacinar hepatic congestion was a consistent finding, and there were occasional instances of non-suppurative interstitial pneumonitis, encephalitis, hepatitis and lymphadenitis. Serological and virological examinations at EMAI and AAHL have so far failed to identify the causal agent. Sow sera were negative for encephalomyocarditis virus (EMC), Menangle virus, Hendravirus, Nipahvirus, Porcine Reproductive and Respiratory Syndrome virus, Foot and Mouth Disease, Aujeszky's Disease, Swine Vesicular Disease and Classical Swine Fever virus. Serological examination of gilts before mating indicated consistent levels of antibodies to Porcine Parvovirus and Porcine Circovirus type 2 (PCV2). Some were positive to Porcine Circovirus type 1 (PCV1). Pericardial fluids with raised IgG levels were seronegative for a similar range of pathogens including toxoplasma and a range of leptospire. Piglet tissues were negative in PCR assays for EMC, PCV2 and PCV1. Electron microscopic examinations at EMAI and AAHL of heart tissue revealed crystalline arrays of approximately 20nm diameter virus particles within vascular endothelial cells in some samples. A range of examinations and experimental studies is in progress.



## **NT – Anton Janmaat**

### **Unusual Findings in Crocodile Mortality**

Cathy Shilton, Berrimah Veterinary Laboratories, NT Department of Business, Industry and Resource Development

For the past several months, there has been 5% mortality in the yearling crocodiles at a Northern Territory crocodile farm. Affected animals become anorexic, develop brown spots on the head, have a tendency to stay out of water, lose body condition and eventually die. Blood samples from four affected crocs were taken prior to euthanasia and post-mortem examination.

Serum biochemical analyses were unremarkable except for mild to moderate hypochloraemia and hyponatraemia in all four crocs, and mild hyperglobulinaemia and marked hypoglycaemia in three crocs. Full blood counts were unremarkable.

Post-mortem examination revealed wasted limbs, sunken abdomens and dark skin coloration. There were multifocal irregular 2-5mm pale crusts overlying moist eroded skin on the head, and the tips of the tails appeared necrotic. The only notable internal findings in cases #1-3 were pale consolidated lungs. In the fourth case there were pin-point to 2mm pale foci throughout the lungs and liver.

Histologically, the most remarkable finding involved the lungs of cases #1-3 in which there was severe non-suppurative interstitial pneumonia characterised by moderate to marked interstitial lymphocyte and macrophage infiltration and moderate hyperplasia and lymphocyte infiltration of respiratory epithelia. In the two most severely affected animals, airways contained proteinaceous fluid and low numbers of macrophages. Special stains, including Gram's, Ziehl-Neelsen and Gomori's methenamine silver, did not reveal organisms in the lung. The lung of case #4 was different, in that it contained multifocal small abscesses containing Gram-negative bacteria. Another unusual finding was ureteritis characterised by moderate lymphocytic infiltration of the epithelium and surrounding interstitium of the ureters and collecting tubules in all cases, and in the most severe case, associated patchy necrosis and sloughing of the renal tubular epithelium. In all animals there were increased numbers of lymphocytes aggregated in several organs, including the spleen, portal areas of the liver and pancreatic interstitium. The endocardium, epicardium and vascular endothelium throughout the tissues were hypertrophied and had adherent aggregates of lymphocytes and macrophages. Skin lesions varied regionally from severe ulcerative pyogranulomatous dermatitis, with large numbers of superficial mixed bacteria and a few fungal hyphae, to moderate epidermal hyperplasia with marked basal epidermal and dermal perivascular lymphoid infiltration.

Bacterial culture of the lung from cases #1-3 yielded no growth. Several samples, including the lung and liver from case #4, the liver of #3 and the skin of #2 and #3 yielded *Streptococcus dysgalactiae* subsp. *equisimilis*. This is an unusual isolate from Northern Territory farmed crocodiles and its significance is questionable since Gram positive bacteria were not demonstrated in the tissues. The liver of case #4 also yielded *Salmonella arizonae*, likely the cause of the abscessation in this animal. Fungal culture of two skin samples yielded *Fusarium* sp.

Because of the lack of significant bacterial isolates from the lung of cases #1-3, and the unusual nature of the histological lesions (compared to the usual lesions of bacterial septicaemia seen in crocs of this age), further testing was pursued. Testing for chlamydiosis using the Clearview antigen ELISA was negative on liver

and lung from cases #2 and #3. Generic Mycoplasma PCR testing was pursued on lung from cases #2 and #3, and was positive in case #3.

Mycoplasma infection is an attractive possible aetiology for some of the lesions in this case. In various animal species, Mycoplasma spp. are often associated with pulmonary infection, typically resulting in pulmonary lymphoid infiltration and epithelial hyperplasia. In the past decade, two new Mycoplasma spp. have been reported to be responsible for disease in farmed Nile crocodiles in Zimbabwe and in captive American alligators in Florida. Interstitial pneumonia, polyarthritis and lymphoid infiltration of various tissues were prominent features of the disease in those cases. In order to determine the significance of Mycoplasma spp. in this group of crocs, further investigations are planned, including postmortem examination of more crocs (with detailed examination of joints), attempted culture of Mycoplasma spp., and further Mycoplasma PCR testing on tissues with suspicious lesions.

## **QLD – Bruce Hill**

### **Pyogranulomatous panniculitis in a cat caused by Rhodococcus equi**

Mark Carrigan, IDEXX Brisbane

A 6 year-old neutered female domestic shorthaired cat had a long-term subcutaneous lesion of right flank. Initial swab cultured Corynebacterium sp. which failed to respond to antibiotic treatment. An irregular piece of soft to resilient pale mottled tan tissue measuring 13 x 9 x 6 mm was submitted. Sections were of subcutaneous adipose tissue and a small portion of pannicular muscle infiltrated by extensive pyogranulomatous inflammation which was characterised by dense aggregates of macrophages and neutrophils with low numbers of lymphocytes and plasma cells.

Moderate numbers of rather irregular medium-sized Gram-positive bacilli were detected in macrophages within the pyogranulomatous infiltrate. No organisms were detected in PAS or Ziehl-Neelsen stained sections. A light pure growth of Rhodococcus equi was recovered from a swab taken from the lesion.

The inflammatory response bore many similarities to that produced by the atypical mycobacteria, except that in this case neutrophil aggregation about fat droplets was less than that expected in the mycobacterial infections. Rhodococcus equi has been isolated from the subcutaneous abscesses/panniculitis in cats. Internal organ involvement can occur if the cat is immunocompromised (Small Animal Dermatology, 6th Edition).

### **Two wheezing Scots, twenty-two coughing Pomeranians and a lot of smouldering Cubans.**

Richard Ploeg, IDEXX Brisbane

A 12 year-old female de-sexed Pomeranian was presented for post mortem examination, having died unexpectedly during the previous night. The animal lived in a small cottage with 21 other Pomeranians, and had a persistent cough and nasal discharge for the last 6 months. The owners (of Scottish descent) enjoyed smoking cigars (Cubans). Many of their dogs coughed intermittently; this was attributed by the owners to poor tolerance of the cigar smoke.

Gross abnormalities were reported to be limited to the lungs and included patchy consolidation and congestion with numerous pale foci on the pleural surface. Histological sections revealed extensive severe chronic granulomatous interstitial pneumonia and numerous profiles of nematodes which frequently contained intrauterine embryonated eggs. Numerous larvae were also present and on occasion could be seen in blood vessel lumens as well as in bronchioles and small bronchi. Alveolar septa were thickened by mixed inflammatory infiltrates, predominantly eosinophils and macrophages (including giant cells) as well as scattered lymphocytes, plasma cells and neutrophils.

The appearance and location of the parasites, along with the presence of embryonated eggs and larvae, was consistent with Metastrongylids. The parasites were subsequently identified from fixed lung tissue as *Filaroides hirthi*. The examining parasitologist also detected eggs characteristic of *Capillaria* spp. however no adults were apparent. *F. hirthi* was first described by Georgi et al (1975) in colonies of Beagles in the United States and is believed to have spread internationally with the movement of experimental animals. This might have been the case for Australia, where it was first described in colonies of research animals (Beveridge et al 1983). Infection is typically subclinical, however severe and on one occasion fatal (August et al 1980) infections have been reported. Clinical disease is typically associated with immunosuppression and is believed to be the result of auto-infection. Chronic stress (August et al 1980), glucocorticoid therapy (Genta et al., 1984), distemper virus (Carrasco et al 1997), and a functional adrenocortical carcinoma (Valentine et al 1987) have been reported to predispose to hyper-infection. Immunity is believed to have its basis in the host's response to first stage larvae. Clinical diagnosis can be difficult with both TTW/BAL and faecal flotation being of low sensitivity. Only a small percentage of infected dogs have larvae demonstrated in faeces by the Baermann technique (Georgi et al., 1975).

In the current case faeces from five other affected animals did not appear to contain any larvae. Empirical therapy has been initiated in the other animals. The origin of the immunosuppression in this case was not apparent although the Cubans may have played a role.

August, J.R. et al (1980) JAVMA, 176: 331-334.  
Beveridge, I. et al (1983) AVJ, 60: 59.  
Carrasco, L. et al (1997) Vet. Rec., 140: 72-73.  
Genta, R.M. et al (1984) Vet. Pathol., 21: 349-354.  
Georgi, J.R. et al (1975) J. Parasitology, 61: 337-339.  
Valentine, B.A. et al (1987) J. Comp. Pathol., 97: 221-225.

## **SA – Stephen Yeomans**

### **Pyrolizidine alkaloidosis in a miniature horse.**

Peter Phillips, Gribbles Veterinary Pathology, Glenside SA 5065

A 4 year-old miniature horse stallion was presented to the clinician with a history of 2 days of inappetence and abdominal pain. The horse was in poor condition, very dull and depressed. Blood biochemistry showed a GGT of 450 U/L (normal 0-40). It was unresponsive to treatment and was killed humanely and liver submitted for histopathology.

The liver exhibited marked biliary hyperplasia, desmoplasia and megalocytosis consistent with pyrrolizidine alkaloidosis. On further enquiry it was found that the property was badly infested with *Echium plantagineum* (Salvation Jane/Petterson's Curse) and that there had been previous history of pyrrolizidine alkaloidosis in horses.

### **Fungal folliculitis in chickens.**

Peter Phillips

Small sections of skin were submitted from 4 week-old chickens which were found to have white plaque-like lesions associated with feather follicles on the surface of the skins while being processed for the gourmet restaurant industry.

Histopathology showed that the plaques were sub-corneal pustules containing multiple branching, septate fungal hyphae of moderately variable diameter. Unfortunately no fresh material was available for culture, but the fungus is thought likely to be an *Aspergillus*.

### **TAS – Judith Handlinger**

No report

### **VICTORIA – Malcolm Lancaster**

#### **Clinically normal ducks may transmit chlamydiosis.**

Malcolm Lancaster, PIRVic, Attwood

A spate of confirmed cases of chlamydiosis in workers at a duck-processing plant prompted the submission of clinically normal ducks for examination. Organisms morphologically consistent with chlamydial elementary bodies were detected in the yolk sac of embryonated eggs inoculated with material from three of 14 ducks. The elementary bodies were visualised by Gimenez staining and by immunohistochemistry (Dako kit). Swabs from two other ducks with mild conjunctivitis gave a very faint reaction in an antigen detection test (Clearview kit). Other ducks from this property have had rhinitis, air-sacculitis, pericarditis and perihepatitis, but *Riemerella anatipestifer* is usually isolated and held responsible.

The take-home message is that ducks as well as pigeons and parrots should be necropsied with caution. The literature indicates that workers in duck-processing plants are often affected with chlamydiosis, with seroconversion even more frequent.

#### **Miliary Hepatic Necrosis ("Summer Hepatitis") in Caged Layer Chickens in Victoria**

Mike Forsyth, PIRVic, Attwood

This report concerns a flock of 8000 caged layers aged between 26 to 34 weeks at the end of March. They had been in an old shed (three-tier elevated cages with fogging system curtains) for 17 weeks after being reared on earth floor with

shavings. The mortality rate began to climb from week 26 when 22 died in one row of cages. The total mortality was around 300. The owner reported that the birds were usually found dead but if they had been seen sick they died in half a day. Older birds in the same shed were unaffected. Both age groups were getting the same ration.

The three live birds presented had little white spots throughout the liver, with petechiae on the surface of the spleens, abdominal fat and pericardiums. The birds were in lay.

No organisms were recovered on routine aerobic culture from liver and spleen samples.

Histopathology: the three birds all had multiple foci of acute coagulative necrosis with associated granulocytes. There appeared to be lymphocyte depletion in the spleens. Gram, PAS and Warthin-Starry stains all failed to reveal organisms in sections.

Treatment with a tetracycline was followed by resolution of the problem.

This condition has been recognised in Victoria for about 15 years and is known also as "summer hepatitis". It is usually seen in the warmer months and mortalities increase to severe levels unless treated. Most outbreaks commence during the first 10 weeks of lay and affected birds are always in lay and in good condition. The condition has traditionally been associated with free range and barn flocks but is occasionally diagnosed in caged flock as in this instance. The aetiology remains unknown.

## **WA – David Forshaw**

***Western Australian Department of Agriculture  
Animal Health Laboratories; South Perth and Albany.***

### **Calf ventriculitis - *Streptococcus suis* type I?**

David Forshaw, WA Agriculture Animal Health Lab, Albany

One of 12 six week old red Angus calves, found dead, but seen 3 days before with no clinical signs. At necropsy the most significant changes were seen in the brain which was asymmetrical, the right half clearly larger than the left and the meninges diffusely congested and thickened. The right lateral ventricle contained clots of purulent material; the left contained no pus but was also markedly distended. Histologically, fibrino-suppurative exudate filled the lateral ventricle and mesencephalic aqueduct. In numerous places the ependyma was ulcerated and there was fibrillar astrocytosis, gliosis and perivascular cuffing in the adjacent parenchyma. Within the exudate, large numbers of Gram-positive cocci were present in chains, sometimes embedded in a densely eosinophilic matrix. There was also diffuse mild to moderate lympho-plasmacytic meningitis. Aerobic culture of a ventricular swab produced heavy pure growth identified as *Streptococcus suis* type I by biochemical reactions. This farm runs free-range pigs but not on same paddocks as the cattle; however, the farmer has frequent contact with both herds of animals. He has seen no signs of meningitis in sucker piglets.

### **Canine Nocardia/Actinomycete periorchitis** David Forshaw

A diagnosis of orchitis was made in a mature dog with severe bilateral testicular swelling and fever, and the dog was castrated. Within the tunica vaginalis and extending into the cremaster muscle and overlying soft tissues (but sparing the vas deferens and the testis itself) there was a heavy mixed inflammatory cell infiltrate consisting of macrophages and plasma cells with focal haemorrhage and large aggregates of PMNs. Occasional large serpentine colonies of Gram-positive filamentous organisms were evident amongst the inflammatory debris. At the edges of the reaction there was advanced granulation tissue response. Seminiferous tubules were lined almost exclusively by Sertoli cells with aggregates of necrotic cells within tubule lumens. The changes were confined to the tunica vaginalis and may well have been an extension of an undiagnosed peritonitis. Alternatively, the infection could have arisen in the tunica vaginalis itself and remained relatively confined. The isolate has not been definitively identified. It is sensitive to penicillin and Nocardia is not normally sensitive to penicillin.

### **Fibrous osteodystrophy in egrets** Mark Kabay & Shane Besier, WA Agriculture Animal Health Lab, South Perth

Leg deformities were seen in three sub-adult Great Egrets at Perth zoo. The bird examined had thickening and angular deformity of the proximal tarso-metatarsals, with apparent poor mineralisation of other bones and beak. Histopathological examination of the long bones revealed that in diaphysial and proximal metaphyseal areas the cortical bone was thin or absent and had been replaced by diffuse, mature fibrous tissue. There was a generalised increase in bone remodelling activity, evident as active osteoblasts, osteoclasts and a few heterophils lining the trabecular bone surfaces. Cartilaginous cores at the physis appear to be enlarged, with poor mineralisation as they approached the mature bone layers. The problem was thought to be associated with an improper diet provided by the carer who raised the birds.

### **Parasitic vasculitis in a bandicoot.**

Cleve Main, WA Agriculture Animal Health Lab, South Perth

A female bandicoot from Perth Zoo had poor appetite for three days and was found dead one morning. Marked intestinal haemorrhage was seen at necropsy. Histologically there was extensive destruction of the small intestinal mucosa that extended through the muscularis mucosae into the underlying lamina muscularis and serosa. Remnant villi were stunted, haemorrhagic and in at least one area, covered by fibrinous exudate. The lamina muscularis was oedematous and numerous aggregates of protein were present between the muscle fibres. Blood vessels within the muscle layer and the underlying proliferative serosa were necrotic or undergoing dissecting arteritis. Protozoa-like organisms were present within the vessel walls of some arteries or were closely associated with them. In other areas where the changes are less severe, numerous protozoa and some nematode larvae were found within the mucosa. There are also several focal aggregates of eosinophils associated with Splendore-Hoeppli material in the deeper mucosa. These probably represent inflammatory responses to parasites, as at least one of them contained a degenerate protozoan. Assoc Prof Peter O'Donoghue University of Queensland suggested that the protozoa seen in the tissues and blood vessels in the gut may be Hepatozoon sp, a parasite allied with Haemoproteus and Leucocytozoon. This parasite forms gamonts which arise from asexual schizogenous (merogenous) stages located in vascular endothelial cells; the gamonts then infect circulating blood cells.

### **Bovine intra-hepatic porto-systemic shunt.**

John Creeper, WA Agriculture Animal Health Lab, South Perth

Two 6 week-old calves from the Pilbara were found dead; they had appeared normal when born. In one animal there was extensive status spongiosus of brain stem tracts, mostly in mixed grey-white areas. In the liver, triads contained clusters of arterioles with often no identifiable portal venule, consistent with intra-hepatic porto-systemic shunt. Clive Huxtable reminded us that there are not actually more vessels but tortuosity of the hepatic arteriole.

### **Chicken Inclusion Body Hepatitis**

Russel Graydon, WA Agriculture Animal Health Lab, South Perth

Two week-old male breeder chickens experienced daily mortalities of fifty and thirty of an unspecified number. Birds were heat-stressed as day-olds. Fixed tissues only were received. Histologically, each of the pieces of liver were essentially the same with severe multi-focal to locally extensive hepatocellular necrosis with disruption of the normal architecture and vacuolation of the remaining hepatocytes. There were large numbers of large basophilic intranuclear inclusions and smaller numbers of eosinophilic intranuclear inclusions. In the spleens there was focal necrosis of some lymphoid follicles. Inclusion body (adenovirus) hepatitis is generally confined to chickens under 3 w of age, with mortalities up to 30% but has been seen in birds up to 20w of age. Mortalities peak at 3-4 d then drop and usually stop on day 5, but occasionally continue for 2-3 w. Immunosuppression caused by infectious bursal disease virus is a common precipitating factor but was not involved here. The birds were held up in transport and perhaps heat stress played a role?

### **Ovine Nares Necrosis**

David Forshaw

30/900 six month old wethers affected with extensive destruction of the lateral wall of the nasal cavity with exudation of bloody material onto the eroded surface. The nasal conchae were exposed in the worst affected animals. Weaners were condition score 2-3 and the only clinical description "a bit dull". Affected animals were grazing short green pasture paddock with clover/grasses in it. Two snouts with unilateral lesions were submitted. Histologically there was severe necrotising inflammation with destruction of the tissues towards the rostral end of the snout. In less severely affected areas there was focal PMN infiltration into the deep dermis centred on sweat glands with extension into the surrounding soft tissue. Adjacent large vessel walls were also infiltrated by PMNs. Eosinophilic inclusions in the adjacent epithelium were not typical of poxvirus inclusions and may have been kerato-hyaline granules. Direct EM examination failed to reveal any virus particles. The aetiology of these dramatic changes is not clear. The nature of the mildest lesions, which may have been precursors of the more dramatic destructive changes, suggests that the lesion may begin as a furunculosis or adenitis of the sweat glands. There was extensive haemorrhage and focal vasculitis suggestive of fungal involvement but no organisms were seen in affected vessels with H&E, Gram or silver staining.

### **Mycobacteriosis in passerines associated with refractile debris in macrophages**

Russel Graydon

It is not unusual to find aggregates of macrophages packed with miscellaneous debris in the liver and lungs of zoo or indeed many urban animals. Normally ZN

stains are not applied to routine sections of zoo birds, but following the diagnosis of severe mycobacteriosis in birds from one particular enclosure, additional measures were requested and acid-fast bacteria were found in such lesions in the liver and lung of a painted finch. Similar foci have now been found in several other zoo birds, but they are unlikely they have been responsible for their deaths or illness. The bacteria do not provoke any additional inflammatory reaction. Necrotic foci and giant cells, often seen in chickens, are not features of mycobacterial infection in passerine and psittacine birds. It is also worth remembering that, usually, mycobacteriosis in birds is primarily a disease of the alimentary tract, with secondary involvement of other organs.

### **Back-line necrosis in sheep.**

Cleve Main/John Creeper

We have seen a number of cases of this intriguing condition over the summer (see below for details of this year's cases but, most interestingly, note the reports of sheep with severe back-line necrosis with no history of back-lining with pesticides). Current speculation is that these are cases of severe sunburn, as there appears to be a correlation with extreme temperatures. Enoch Bergman, a private practitioner from Esperance is keen to collect details of similar cases, so if you have any, please send them to him at

Enoch Bergman [[cowvet2001@yahoo.com](mailto:cowvet2001@yahoo.com)] Swans Veterinary Services, Box 1514, Esperance WA 6450, Clinic- (08) 9071 5777 , Fax- (08) 9071 5057, Mobile- 0427 716907.

A mob of 3500 4-tooth Awassi cross ewes at Esperance was treated with Clout S following shearing. About half of the mob became affected, 124 severely so and another 120 dead. Histologically, the lesion is a sharply demarcated full-depth necrotic dermatitis. In most areas a layer of necrotic debris has replaced the epidermis with an associated moderate to severe infiltrate of neutrophils. In some places there was an overlay of haemorrhage and protein-rich exudate. No hair follicles or glandular structures remain and in many places there has been a fibrotic replacement of underlying connective tissue. There were focally extensive areas with severely dilated tubular glands that often contained fan-like sheaths of crystal-like substance, and these are associated with a granulomatous and vigorous foreign body giant cell inflammatory response. History provided by the investigating practitioner suggests that, although severe lesions were present in the purple tag mob that had been treated with Clout S, milder lesions were seen in a red tag mob that had not been back-lined.

120/3400 sheep at Esperance developed post-shearing burns following back-line treatment with Magnum. The initial lesion was severe necrotic dermatitis and 6 weeks later the zone between normal and affected skin remained well demarcated. The affected area involved both the dermis and epidermis. The epidermis was hyperplastic and hyperkeratotic while the dermis showed total replacement of normal dermal connective tissue and adnexae by dense collagenous fibrous tissue that surrounded clusters of foreign body giant cells and foamy macrophages containing acicular clefts. There were irregular lakes of a faintly basophilic stippled material that appeared to have replaced sebaceous glands and hair follicles, and this material was surrounded by the most intense granulomatous response.

Full mouth Merinos at Karlgarin were shorn in early February. Skin disease was evident when the flock was mustered for dipping 2 weeks later. The sheep had been grazing medic followed by wheat stubble prior to shearing. There were



about 7 severely affected sheep and about 200/645 sheep with minor skin lesions. Ewes grazing sub clover were not affected. Histological examination revealed a zone of severe focally extensive full depth epidermal necrosis overlain by a thick protein-rich crust of exudate and neutrophils with superficial bacterial colonies, separated from the normal epidermis by a transitional zone. There, the epidermis was markedly hyperplastic with extensive and deep epidermal peg formation and mild to moderate hyperkeratosis. Below this area and the zone of full depth necrosis was a moderate to severe dermal infiltrate of plasma/lymphocytes and neutrophils. This is an interesting addition to our data. For a start, no backline treatment was used and the problem occurred prior to dipping. Secondly there was an almost 3 month interval and yet no repair processes were evident in the samples examined. Histological examination of the liver and results of a liver enzyme panel did not indicate that the problem might have been secondary photosensitivity.

An interesting comment has been provided by Bob Nickels who sent photos of similar lesions in sheep involved in a Pindone feeding trial at Chapman Research Station in 1975.

### **Cryptobia-like flagellate infestation in fish.**

Fran Stevens, Department of Fisheries, Animal Health Lab, South Perth

Many clients with cichlids in Perth have been reporting no response in sick fish to treatment with metronidazole (Clout and Octozin). Two affected fish were presented for necropsy. A few protozoa were seen in intestinal contents, the livers were brown and small nodules were seen in liver squashes. Both fish were anaemic. Histologically there were multiple granulomas in the liver and stomachs of both fish. The stomach wall was thickened by inflammatory tissue containing lymphocytes and macrophages and a few flagellates. Stains for acid-fast bacteria, amyloid and fungi were all negative. Haemosiderin was present in the liver and spleen. *Aeromonas hydrophila* was cultured from the liver of one fish and zones of hepatocyte degeneration were typical of those often associated with this bacterium. The range of changes seen in these fish is consistent with *Cryptobia* sp infestation (Shane Raidal, Murdoch University pers. comm) and secondary infection with *Aeromonas hydrophila*

### **Microsporidiosis in pygmy perch**

Fran Stevens Pygmy perch in the most productive of one of four farm dams developed black lumps on their sides. The problem had been apparent for several months but did not appear to be associated with ill thrift. The fish continued to breed. Histopathological examination revealed several sacs filled with small microsporidian spores in the abdomen and muscle wall on the right side of the abdomen. There was a melanin layer surrounding each of the sacs of spores. There was no host reaction. Preparation of lesions for EM examination of these lesions presents difficulty as Microsporidia have very hard capsules.