

# VETERINARY PATHOLOGY REPORT

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Number 66

December 2003

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## **President's Report**

Committee meetings are held monthly and by teleconference. These seem to be working well although individuals are sometimes hard to track down at 1.00pm. The minutes of the committee meetings are now published on the web site.

It was great to see all the discussion on the NATA issues associated with signatures on reports. As a member of the NATA advisory committee, I will take your comments to our next meeting for discussion.

Congratulations and thanks to Clive Huxtable for his efforts in taking the neuropathology training course around the states. Everyone who attended has been rewarded with greater knowledge of these diseases.

It is that time of the year when renewal of state registration occurs. Several of the states are now requiring proof of individual CE effort. Reading the Vet Path Report and attendance at ASVP conferences and registry training sessions are valuable ways at gaining CE points. I encourage you to plan for attendance at these training programs next year.

Seasons greeting to all. I would specially like to thank all members of the committee for their participation this year and I look forward to further planning and discussions in 2004.

PS. With the approaching public holidays during the next few months, it must be time for an outbreak of exotic disease.

Richard Miller

**Honorary President**

## Editorial/Secretary's Report

The management committee has held monthly meetings since the transfer of executive in June this year. The minutes of these meetings have been posted on the ASVP website after each meeting, so only a brief summary of management activities need be given here.

**NATA Definitions:** A fair bit of comment has been generated by NATA's Georgina Kanizaj-Clark's proposed definitions of veterinary pathologists for NATA purposes. Below I reproduce Georgina's response (5/12/03) to the responses:

"Many thanks for the correspondence. It makes for very interesting reading.

"I do however feel that of the 35 odd responses I have received (this includes individuals outside ASVP) only a handful of responders have commented on the purpose of these definitions.

"I think I will need to re-emphasize to everyone that the purpose of these definitions is stated as follows:

*NATA proposes that the following standardized terms be used on NATA-endorsed test reports to purvey a person's qualifications and their role as a NATA approved signatory*

"This is in accordance with clause 5.10.2.j of ISO/IEC 17025 which states that:

*Test reports ..... shall include .... the name(s), function(s) and signature(s) or equivalent*

"I don't believe the intention was that the terms would be mandatory. They were designed to be a guide for individuals/submitters who receive veterinary testing reports.

"As such, it is proposed that the standardized terms be used by individuals who are:

1. veterinarians
2. NATA approved signatories and
3. issuing diagnostic comments on NATA endorsed test reports.

"I take note of the comments/issues/objections/support raised and will discuss these with the Veterinary Testing Accreditation Advisory Committee.

**Members Mailing List:** 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) or ph.(07) 3371 4463

**Tasmanian Correspondent:** Stephen Pyecroft is to be commended on volunteering for this position even as his application for ASVP membership was being processed. His reassuring voice was heard in a radio interview on the Tasmanian Devil lymphoma epidemic, aired on Radio National's Earthbeat on Sat. Dec 6<sup>th</sup>, so his career in veterinary journalism is off to a flying start.

**2004 AGM:** It was decided at the December 2<sup>nd</sup> management committee meeting that the ASVP should hold its usual two-day week-end meeting on October 16-17th immediately after the International Academy of Pathology Congress in Brisbane, during the last day of which (Friday 15<sup>th</sup>) there has been allocated a veterinary pathology symposium. ASVP members will be able to attend any of the Academy sessions during this congress (for a discounted registration). For details of the IAP program, stay in touch with the IAP website <http://www.iap04.im.com.au>. One of the themes for the ASVP session will be exotic disease; it has not yet been decided whether to run this on the Friday or the week-end sessions.

**Registry and Slide-of-the-Month Scheme:** The Slide-of-the-Month scheme is not operating at present because there is no-one in a position to be able to act as Co-ordinator. Ideally, there should be a full-time Registrar of the National Registry of Domestic Animal Pathology who would be Co-ordinator of this scheme *ex officio*. In the last Vet Path Report there was heartening news of imminent Registry re-funding, but unfortunately this has stalled at a higher level. Barry Richards and Tony Ross *et al* continue to strive, however, and have produced a draft business plan for the Registry which is a requirement necessary for funding, and continue to strive for it.

So, in the interim, if members want a Slide-of-the-Month scheme to continue, someone needs to volunteer to be co-ordinator. It is not a terribly onerous task. Details of what is involved are available from the Secretary.

**Vet Path Report:** It is very gratifying to see this issue of the Report in much better nutritional status, compared to recent issues. The State correspondents are to be congratulated for expressing such a good response from contributors, who of course are thanked, too.

**Addendum to Minutes of the April 2003 AGM:** A regrettable omission from the Minutes of this meeting was record of this year's Young Presenter's award, which was divided between Selina Ossedryver of ARI, Yeerongpilly ("Acute oxalate poisoning from buffel grass (*Cenchrus ciliaris*) in cattle"), and Richmond Loh of Animal Health Lab, Kings Meadows ("Tasmania albatross diseases").

**News of 5<sup>th</sup> edition of Jubb, Kennedy, Palmer & (?):** Robert Foster in an e-mail from Guelph informs us that the almost despaired-of 5<sup>th</sup> edition of this text is still in preparation, and that some chapters have been sent to the publisher. He undertook to send author/chapter lists soon.

Roger Kelly  
**Honorary Secretary**

## STATE REPORTS

### NSW – Paul Gill

#### **Trichomoniasis in Large Beef Herd** *Tony Ross, RVL Menangle*

Scans revealed poor pregnancy rates, mummified fetuses and pyometra in a group of beef cows in the tablelands of northern NSW. Trichomoniasis was confirmed in 13 of 110 bulls by culture of preputial washings. Only one group of cows had obviously low pregnancy rates but breeding cows are moved between 4 properties. Therefore bulls on all 4 properties were tested. Twelve positives in a group of 32 bulls on one property were found to be infected with a further positive detected during a second round of testing. Quarantine and a management plan based on movement restrictions, testing and culling have been implemented on one property. Another property in the group was diagnosed with trichomoniasis five years ago, managed and released from quarantine one year ago.

#### **Paramyxovirus Infection in Snakes in NSW** *Tony Ross*

Background: Paramyxoviral infection of snakes (Ophidian paramyxovirus – OPMV) was first recognised in 1976 as a cause of respiratory disease in captive snakes in Switzerland. It has since been identified as cause of significant epizootics of respiratory and nervous disease in snakes in a variety of private and zoological animal collections/parks/zoos throughout the United States of America and Europe. OPMV has also been reported from Brazil, Canary Islands, and the UK, but is considered exotic to Australia.

This disease affects some species (including rattle snakes) more than others but can cause 'die-offs' in viperids, elapids, boids and colubrids. All of Australia's venomous snakes are elapids.

Most snakes develop a respiratory syndrome, stop eating, waste and die over 2-8 weeks. Grossly changes range from diffuse haemorrhage and pulmonary oedema to accumulations of caseous necrotic debris in the lumen of the lung and sometimes the trachea.

Histological changes in the lung reveal hyperplastic alveolar cells, septal congestion and oedema and sometimes interstitial and exudative pneumonia. Some snakes develop a demyelinating encephalopathy and waste away.

The disease must be distinguished from inclusion body disease of boid snakes that causes regurgitation, variable CNS signs and deaths in boids (pythons and boa constrictors). Histologically the presence of eosinophilic intracytoplasmic inclusion bodies in H & E sections of multiple organs including kidney, pancreas, liver and brain differentiate it from paramyxoviral infection.

Paramyxovirus infection produces both intracytoplasmic and intranuclear inclusion bodies particularly in lung and brain. Phylogenetic analysis suggests that a new genus in the Paramyxoviridae should be created for reptilian paramyxoviruses to distinguish them from avian paramyxoviruses.

In overseas reports, a diagnosis of OPMV is usually made on gross and histological findings. In some studies paramyxoviruses have been isolated and identified. Immunohistochemical detection of the virus has been achieved in some

studies. Haemagglutination inhibition assays to measure antibodies against OPMV infection have been developed at Weybridge in the UK and at the University of Florida in the USA.

Situation: In late 2002 NSW Agriculture was notified of suspected OPMV in a large captive snake collection in NSW. The diagnosis was based on "outbreaks" of respiratory and neurological disease and histological confirmation by Dr Neill Sullivan, a senior pathologist with IDEXX Brisbane. Investigations concluded:

1. Dr Sullivan had made diagnoses of OPMV in several collections in Qld and NSW on histological grounds. The Qld cases were brought to the attention of CVO Qld.
2. Other pathologists warned of the dangers of confirming OPMV on clinical and histological grounds alone due to the existence of other inclusion body forming diseases including Boid Inclusion Body Disease.
3. NSW cases were reviewed by Dr Tony Ross (NSW Agriculture EMAI) and Dr Karrie Rose (Taronga Zoo, Sydney) who concluded that some but not all cases were suggestive of OPMV on clinical, gross and histopathological grounds. Additional materials were also submitted to both EMAI and Taronga.
4. Attempts at EMAI and AAHL to isolate OPMV from 3 live cases were unsuccessful and OPMV like viruses were not seen on electron microscopy of selected tissues from affected snakes.
5. A panel of 25 sera from snakes in various collections in NSW was assembled and sent to Ms Ruth Manvell of the Avian Virology section of VLA Weybridge UK. Ms Manvell has worked with OPMV. She examined the sera using 2 OPMV isolates in a haemagglutination inhibition test (see attached results).
6. The results showed that 9 of 25 snakes were positive for antibodies to OPMV-1 and 7 of 25 to OPMV-7. Positive snakes originated from 2 separate collections in NSW where there have been clinical and pathological findings consistent with OPMV. The significance of individual titres and differences between species is not yet clear.

Conclusion: The presumptive diagnosis of OPMV made on clinical and gross and histopathological grounds is supported by the serological results. This appears to be the first confirmation of paramyxoviral infection in snakes – a disease previously thought to be exotic to Australia.

Recommendations:

1. CCEAD liaise with interested parties in the public and private sectors to determine the significance of this finding to the wild snake populations of Australia. In particular, further serological surveillance is warranted. This should include an adequate number of samples from representative species in wild populations.
2. NSW Agriculture, Qld DPI and AAHL continue attempts to isolate the virus of OPMV and develop antibody and antigen testing capabilities in Australia.
3. CCEAD liaise with the representatives of the captive snake industry to develop a risk analysis and response options to the likely presence of OPMV in Australia.

## References:

Suedmeyer K Paramyxovirus and inclusion body disease of snakes. Exotic Pet Practice 5 #1 1-2 2000

Franke J et al Identification and molecular characterisation of 18 paramyxoviruses isolated from snakes. Virus Research 80 #1-2 67-74

### **Videoconferencing at NSW Agriculture Laboratories: Tony Ross**

Broadband 386 k videoconferencing equipment has been installed in the microscope rooms of the 3 regional veterinary laboratories of NSW Agriculture at Menangle, Orange and Wollongbar. This allows real time videoconferencing between labs including audio discussion, video images of the pathologists and high quality histological images. Powerpoint presentations and stored digital images can also be broadcast and discussed. High or low resolution still images can be taken during the conference for later use.

The laboratories are able to participate in interactive continuing education programs such as the "Bugs Breakfast" series provided by NSW Health.

Other laboratories with broadband equipment and an ISD number should be able to conduct live conferences with pathologists at NSW laboratories. This will ultimately allow remote access to selected images in the National Registry of Domestic Animal Pathology.

### **NT – Anton Janmaat**

Spinal pachymeningitis in a pig-tailed macaque (*Macaca nemestrina*) Cathy Shilton Berrimah Veterinary Laboratories

A twelve-year-old, female pig-tailed macaque (*Macaca nemestrina*) became acutely ill and was completely unable to use the left front limb. The monkey appeared otherwise normal and was bright and alert. There were no remarkable findings on general physical examination and careful examination of the left front limb under anaesthesia. Radiographs of the affected limb were also unremarkable. A full blood count revealed moderate neutrophilia and serum biochemistry showed a moderate elevation in alkaline phosphatase. The macaque was treated with injectable tetracycline and recovered from anaesthesia. Next day the macaque was weak and unable to rise, but still fairly bright. Two days later, despite still being bright and with normal cranial nerve reactions (as far as could be determined from a distance), it appeared to be quadriplegic and was killed humanely.

At gross necropsy, there were no significant findings in the left front limb. Following meticulous removal and dissection of the entire vertebral column and spinal cord, gross findings were limited to a 1x2x4mm poorly-defined focus of yellowish-white material firmly adherent to the dorsal dura mater in the region of the first cervical vertebra. The adjacent spinal cord and vertebra were grossly normal, as were the cranium and brain. The only other significant gross findings were scant creamy white vulvar discharge and mild enlargement of the uterus, which exhibited patchy mural congestion and a small amount of intraluminal

creamy white exudate, haemorrhage and friable yellow material loosely adherent to the endometrium.

Histopathological examination of the spinal cord revealed marked regional thickening of the dura mater due to fibroplasia and intense infiltration by neutrophils and aggregates of lymphocytes, with subdural focal aggregates of degenerate neutrophils intermingled with fibrin. These lesions were most severe in the cranial cervical region, where the enlargement of the dura was noted grossly, however, they continued in a diminished form to the upper lumbar level. There was particularly marked unilateral dural inflammatory infiltration surrounding brachial nerve roots, consistent with the initial presenting sign of severe unilateral forelimb lameness. The spinal cord parenchyma appeared normal in all sections except in the most cranial cervical section where there was a large region of white matter vacuolation containing scattered swollen axons, gliosis and infiltration by phagocytic cells. This indicates that the enlargement in the dura mater in this region was likely exerting a compressive effect, and perhaps explains the development of quadriparesis. Various sections of the brain were normal except for mild infiltration of the meninges of the brainstem with neutrophils, macrophages and lymphocytes.

Histopathological examination of the uterus revealed marked congestion of the myometrial vasculature, and marked endometrial lymphoplasmacytic infiltration. Towards the luminal surface, there was also focal endometrial oedema, haemorrhage and moderate infiltration by neutrophils. There was haemorrhage and fibrin intermingled with degenerate neutrophils in the lumen. In the liver, kidney and heart, there were a few random foci of necrosis with associated fibroblastic response and infiltration by neutrophils and lymphocytes.

The suspected aetiological agent for the spinal meningeal inflammation was *Burkholderia pseudomallei*. This bacterium is an environmental saprophyte that is widespread in soil and untreated ground water in Northern Australia. Infection with *B. pseudomallei* is common in many animals, including humans, and is particularly frequent in non-human primates from the facility where this macaque lived. The typical manifestations of infection in primates are bacteraemia with widespread focal suppuration in internal organs, pneumonia, pyometra, osteomyelitis and skin and soft tissue infections, with neurological melioidosis being relatively uncommon.

Unfortunately, in this case, the animal had been treated with antibiotics prior to euthanasia, probably explaining the negative general and *B. pseudomallei* enrichment culture results for the spleen and cerebrospinal fluid. Culture of the uterine lumen, however, yielded *B. pseudomallei*, indicating that the chronic suppurative endometritis may have been a focus for bacteraemia and seeding of the spinal meninges, resulting in the severe chronic suppurative pachymeningitis seen in this monkey.

## References

Choy JL, Mayo M, Janmaat A, Currie BJ. Animal melioidosis in Australia. *Acta Trop* 2000; 74: 153-158.

Currie BJ, Fisher DA, Howard DM, Burrow JN. Neurological melioidosis. *Acta Trop* 2000; 74: 145-151.

## **Canine cutaneous-subcutaneous pythiosis**

*Anton Janmaat Berrimah Veterinary Laboratories.*

*Case 1:* A one year old female German Shepherd dog presented with a swollen right hind paw and was treated with clavulox. Three weeks later (to cut a long clinical story short) there was marked tissue thickening of the right caudal upper tibia up to the mid-femur. The right popliteal and inguinal lymph nodes were enlarged. Fine needle aspirates were obtained from the swelling between the 2<sup>nd</sup> and 3<sup>rd</sup> toes, the mid-femur swelling and the popliteal node and an excision biopsy of the upper tibia was submitted for culture and histopathological examination.

Cytological examination of all three FNA smears shows a background of erythrocytes with prominent tissue macrophages and increased numbers of eosinophils. These findings were considered indicative of a chronic inflammatory process. Two sections prepared from the biopsy show granulomatous inflammation with macrophages, often in sheets, predominant. The centres of the sections are mainly necrotic with necrotic foci scattered throughout. Thrombosed vessels, haemorrhage and oedema and fungal elements are recognizable in the necrotic areas. Eosinophils are plentiful, often necrotic, and plasma cells are present as is the occasional giant cell. The PAS stain shows fungal elements throughout although more numerous in the necrotic areas.

There was no growth on bacterial culture after 48 hours incubation. Fungal culture showed a possible *Pythium* sp after four days incubation. The reference laboratory identified the isolate as a fungus resembling a water mould (presumably because zoospore formation could not be induced?).

*Case 2:* A two year-old male German Shepherd dog presented with thickened and erythematous right metatarsal skin. The dog was treated with clavulox tablets. A fortnight later the owner reported that the skin lesion extended beyond the original area. The dog appeared clinically normal but had enlargement of the right popliteal lymph node. Skin biopsy samples were submitted fresh and in formalin.

The biopsy shows a deep dermal and subcutaneous inflammatory process with oedema, eosinophils, macrophages and other round cells. Necrotic foci within the inflamed area contain increased numbers of inflammatory cells as well as fungal elements. The PAS stain confirms the presence of fungal elements.

Bacterial culture showed no growth after 48 hours incubation. Fungal culture resulted in the isolation of a probable *Pythium* sp. after 24 hours. The reference laboratory confirmed the diagnosis of a probable *Pythium* sp.: definitive identification was not possible as zoospore formation could not be induced.

The dog was treated with fluconazole 200mg bid. A skin lesion submitted three weeks later shows fungal elements in inflamed areas.

## **Comment**

Dykstra *et al* found that 6/15 of their cases occurred in German Shepherd dogs. They gave the condition a poor prognosis because of difficulty/delay in definitive identification of the fungus and lack of effective therapy. Antifungals that interfere with ergosterol synthesis are ineffective because *Pythium* is not a true fungus and does not utilise ergosterol as the main sterol in cellular membranes. In their study, the only dog to survive underwent amputation of the affected limb.



## Reference

Dykstra MJ, Sharp NJ, Olivry T, Hillier A, Murphy KM, Kaufman L, Kunkle GA and Pucheu-Haston C. A description of cutaneous-subcutaneous pythiosis in fifteen dogs. *Medical Mycology* 1999; 37:427-433.

### **Polyomavirus infection in Gouldian finches (*Erythrura gouldiae*)**

*Helen Parkes and Cathy Shilton, Berrimah Veterinary Laboratories*

A large finch aviary was experiencing increased mortality in juvenile Gouldian finches (*Erythrura gouldiae*). Laboratory submissions of birds found dead over several weeks were inconclusive due to autolysis. The only significant finding was the presence of tracheal mites, likely *Sternostoma* spp., described previously in Gouldian finches from the Northern Territory. Finally, a juvenile bird was observed sick and was removed from the aviary to a separate cage, where it died within hours and was submitted immediately for post-mortem examination.

The major gross finding was sanguineous intestinal content, smears of which contained blood without inflammatory cells. Faecal flotation for parasites was negative. There were no significant isolates on bacterial culture of the intestine or liver. Histologically, there was random multifocal to coalescing hepatic necrosis with associated haemorrhage but minimal inflammatory cell infiltration. Many cells associated with the necrotic foci had enlarged nuclei with marginated chromatin and a pale basophilic glassy appearance. In the spleen there was marked lymphoid depletion and necrosis and many cells with enlarged pale glassy nuclei. Scattered cells in renal tubules were necrotic, and there were cells with enlarged pale glassy nuclei both within tubular epithelium and glomeruli. Finally, in the small intestine, most notably associated with areas containing lymphoid tissue in the duodenum, there were necrotic cells and typical enlarged glassy nuclei. A tentative diagnosis of polyomavirus infection was made. Transmission electron microscopic examination of the liver was consistent with this diagnosis, with regular, 40-50 um virus-like particles present in arrays within both nuclei and cytoplasm.

The first case in an adult Gouldian finch occurred two weeks after the diagnosis of polyomavirus infection was made. The bird appeared fine in the morning, but was found dead by early afternoon, and submitted immediately to BVL. Post-mortem examination revealed a bird in good body condition, bright yellow discolouration of the skin and adipose tissue, and a light orange liver with red mottling. These latter two findings were suggestive of hyperbilirubinaemia, an unusual finding in a bird.

Histopathologically, there were severe random coalescing foci of necrosis, haemorrhage and cholangiolar proliferation with associated moderate infiltration by lymphocytes, plasma cells and heterophils. Throughout the liver, there were numerous hepatocytes with enlarged nuclei with marginated chromatin and a pale basophilic glassy appearance centrally. There was also diffuse moderate hepatic lipidosis. There were no significant findings in other tissues.

Polyomavirus infection is well described in psittacines, with various manifestations of disease depending on the age and species of the bird. The disease is less well described in passerines, although it has been reported in Gouldian finch aviaries in Australia. Sudden death or death following a 24-48 hour illness is typical in nestlings, juveniles and occasionally young adults, with hepatic necrosis a consistent finding, and lesions in other tissues more variable. The virus appears to have a tropism for the macrophage/mononuclear cell system, including Kupffer

cells, splenic histiocytes and glomerular mesangial cells. The histopathological presence of enlarged nuclei with marginated chromatin and a pale glassy basophilic appearance centrally is considered pathognomonic for polyomavirus infection in birds. Infection is typically subclinical, with only a few birds manifesting the disease. Faecal shedding of the virus by clinically and subclinically infected birds may continue for several days to weeks, with some birds apparently remaining subclinical carriers and intermittent shedders.

Control of the infection within an aviary is based on waiting for the major mortality to subside, halting breeding to remove the most susceptible portion of the population, testing the remaining birds on multiple occasions for the presence of the virus in blood or faeces and removal of birds that repeatedly test positive. In Australia, a PCR for polyomavirus is available to enable this testing, which is being considered in the aviary in this case.

## References

Forshaw D, Wylie SL, Pass DA. Infection with a virus resembling papovavirus in Gouldian finches (*Erythrura gouldiae*). *Aust Vet J* 1988;65:26-28.

Phalen DN. Viruses. In: Altman RB, Clubb SL, Dorrestein GM, Quesenberry K editors. *Avian Medicine and Surgery*. W.B. Saunders Company, Philadelphia, 1997:281-322.

## QLD – Bruce Hill

**Pompe's disease** *Anita Gordon and Selina Ossedryver Yeerongpilly Veterinary Laboratory*

Two cases of generalised glycogenosis Type II (Pompe's disease) were diagnosed recently in young (6-9 months old), unrelated cattle on two different properties in the Longreach district. The first case was a Brahman heifer, which was recumbent and had evidence of mucosal disease at necropsy (upper alimentary ulceration). However this animal tested negative in the bovine pestivirus PCR. The second case was a Brahman-cross steer with paresis. Necropsy of this animal revealed unilateral "nephritis", pale liver and white striations of the heart muscle.

Histological examination of tissues from both animals revealed spectacular vacuolation of skeletal and cardiac muscle (including Purkinje fibres), and widespread vacuolation of neurons, glial cells and neuropil throughout the brain, most obvious in some brainstem nuclei. PAS-positive, diastase-sensitive material was demonstrated within vacuoles of a range of tissues, but not myocardial fibres. Other findings included mild vacuolation of enteric smooth muscle in both animals, and severe, multifocal nephrosis of unknown cause in the steer.

**Bovine papular stomatitis** *Anita Gordon*

Five of 25 Friesian calves from the Atherton Tablelands were reported to have discrete to coalescing ulcers of the dental pad, gingiva, lips, tongue and hard palate. Digital images sent by the practitioner in August revealed "target" (coin-shaped) lesions on the dental pad and lips. Four of the five calves were also scouring, and one was febrile. No lameness or foot lesions were reported. Biopsy of the gingival lesions from one calf revealed mild, focal epithelial thickening, with

ballooning degeneration mostly confined to the intermediate layers of the epithelium. Some eosinophilic intracytoplasmic inclusions were observed in the ballooned cells. EM of negatively stained fresh tissue revealed characteristic parapoxvirus particles, resembling balls of yarn. Diarrhoea was attributed to either or both of *Yersinia pseudotuberculosis* and *Eimeria zuernii*, which were recovered from faeces of one calf.

**Acute ptaquiloside poisoning in cattle** *Grant Campbell, John Gibson and Jim Taylor; Toowoomba Veterinary Laboratory*

Acute ptaquiloside poisoning associated with bracken or mulga fern ingestion was diagnosed or suspected as the cause of death of cattle on 5 properties during April and May. Animals were of mixed ages and clinical signs consisted of fever, swelling of the lower head and neck, mucoid nasal and oral discharge sometimes tinged with blood, diarrhoea and straining (again sometimes bloody) and some mucosal haemorrhage. Haematology from most animals showed a severe leucopaenia and thrombocytopaenia, occasionally pancytopenia. On histology, bone marrow (where supplied) was hypoplastic and, in addition to haemorrhagic foci, bacterial infarcts were detected in sections. The cause was confirmed by identification of plant material in the rumen, or suspected by history of access to offending plants.

**Poisoning with *Swainsonia* sp. in sheep and cattle.** *Jim Taylor*

Darling pea (*Swainsona* sp.) poisoning was diagnosed as the cause of progressive neurological signs in sheep and cattle from which samples were submitted for transmissible spongiform encephalopathy (TSE) exclusion. Both groups of animals were from the same property and goats were also reported affected. All animals had been grazing areas with heavy Darling pea growth. The sheep were more severely affected clinically than the cattle. Clinical signs included incoordination, hypermetria, hyperaesthesia and, in the case of the sheep, coma and death. Lesions were typical in brains from both sheep and cattle, with foamy distension of neurons of the central nervous system. In the sheep, hepatocytes and renal proximal convoluted tubular epithelium were also affected.

**Bacterial meningoencephalitis in a Sacred Baboon** *John Gibson*

A 20-year old male Sacred Baboon (*Papio hamadryas*) was submitted for necropsy from a local zoological collection. The animal was undersized and had a history of chronic weight loss, but had suddenly become lethargic and died. The large intestines had a heavy infestation of whipworms that are apparently common in baboons, but the degree of infestation was the most likely cause of the weight loss. Cause of death was severe, acute, multifocal thromboembolic meningoencephalitis with numerous short-chained streptococci in vessels. The organism was most likely *Streptococcus pneumoniae*, a cause of acute meningoencephalitis in non-human primates.

**Tyzzler's disease in foals** *Jim Taylor*

Two Thoroughbred foals, 10 and 20 days of age, died on the same stud within the same week. The first was found dead with slight jaundice and multifocal coalescing miliary pale foci throughout the liver. The second foal was found comatose and failed to respond to supportive treatment. The only gross change noted at necropsy was again slight jaundice.

Both foals had marked/moderate multifocal necrotizing hepatitis. Steiner-stained sections of liver revealed numerous slender bacilli at the periphery of the necrotic foci. Organisms were not readily evident in H&E sections and did not stain with Gram stain. According to the 9th edition of Veterinary Medicine by Radostits, Gay, Blood and Hinchcliff, the causative agent is now known as *Clostridium piliformis* (formerly *Bacillus piliformis*).

### **Pyogranulomas in lymph nodes associated with *Mycoplasma bovis*. Jim Taylor**

Recently a number of pyogranulomas (submitted under the National Granuloma Submission Program) had a distinct gross and histologic appearance. Unlike other routinely seen pyogranulomas, Gram positive cocci and club colonies (Splendore-Hoeppli phenomenon) were not detected in these lesions. Lesions were restricted to mainly the retropharyngeal and occasionally parotid lymph nodes. They were well-encapsulated, focal, sometimes multifocal, and no bigger than a centimetre in diameter and contained thick green pus.

Histologically there was a central caseous core sometimes with scattered nuclear material that stained quite intensely with eosin. A few neutrophils, macrophages, lymphocytes and a fibrous capsule surrounded this. Giant cells were not observed in any of these lesions. Routine cultures and stains were negative. Six nodes were cultured anaerobically and a very small, Gram-negative coccobacillus was isolated. The organism and cultural characteristics were suggestive of *Mycoplasma* sp. The isolate was referred to Oonoonba Veterinary Laboratory where it was identified as *Mycoplasma bovis*. Investigations are continuing.

## **SA – Stephen Yeomans**

### **Streptococcal septicaemia in Barramundi**

*Peter Phillips, IDEXX Laboratories, Glenside, SA.*

Eight moribund barramundi fingerlings and one 30cm specimen were submitted with a limited history of increasing mortalities. A couple were noted to have cloudy eyes and a little redness around the base of pectoral fins.

Histopathology of fingerlings and the larger fish revealed numerous gram-positive bacilli free in the blood vessel lumens. Culture of viscera produced an alpha-haemolytic *Streptococcus*, which was sensitive to oxytetracycline.

### **Lymphosarcoma in a bilby** *Peter Phillips*

A five year-old female Bilby died in a local zoo after chronic weight loss. She had temporarily responded to antibiotics. On post-mortem a large piece of colon was found to be thickened by infiltrative tissue extending into the mesentery. The mesenteric lymph node was enlarged and contained a small caseous lump.

Histopathology revealed effacement of the architecture of both the colon and the lymph nodes by round cells which displayed anisocytosis, pleiomorphism, anisokaryosis, nuclear pleiomorphism and variable chromaticity with regionally variable mitotic rate. A diagnosis of lymphosarcoma was made.

### ***Chlamydophila* in captive pigeons & doves** Peter Phillips

About 20 white-headed pigeons, Wonga pigeons and brown cuckoo doves in a large aviary in a local zoo were found dead in rapid succession. They were aged from 6 months to 6 years, and ranged in condition from good to poor.

Necropsies revealed polyserositis, which was particularly marked on the liver and heart. Birds also had splenomegaly and hepatomegaly. Histopathology revealed marked splenitis and hepatitis and fibrino-granulomatous polyserositis in which there were numerous macrophages containing small basophilic organisms within their cytoplasm.

There was no bacterial growth from the affected organs of one pigeon necropsied at the lab, and mild growths of non-haemolytic *E. coli* and *Serratia* sp. were cultured from lung and heart of a dove. Direct smears from the spleen and liver from the pigeon and a dry swab of the dove's liver surface gave rise to positive *Chlamydophila* (*Chlamydia*) PCR.

As the aviary concerned was a walk-through type, this outbreak had public health implications, and the aviary was closed pending diagnosis and resolution of the problem following administration of tetracyclines in the drinking water.

The outbreak occurred after 4 fruit doves were introduced to the aviary, but noticed to be unwell, and removed. It is postulated that they may have introduced the disease. Alternatively wild psittacines overfly and land on the aviary roof and could have been implicated.

### **TAS – Stephen Pyecroft**

No report

### **VICTORIA – Malcolm Lancaster**

***Aspergillus clavatus* toxicity in cattle** Malcolm Lancaster, Mark Williamson, Charles El Hage DPI Victoria

A farmer continued to feed sprouted barley to about 100 weaner cattle, despite the presence of a blue-green fungal growth around the base of the shoots. Several cattle developed hind-limb ataxia and went down never to rise again.

Microscopic examination of the fungus revealed large clavate vesicles characteristic of *Aspergillus clavatus*. Histologically, large neurons in the red nucleus and ventral horns of the spinal cord were chromatolytic, hypereosinophilic and sometimes vacuolated, with nuclear degeneration. A total of eighteen calves had to be killed over the next 3 weeks, with fifty seven of the remaining herd displaying varying degrees of hind-limb ataxia.

## **WA – David Forshaw**

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

### **Avian – Suspect (low virulence) Infectious Bursal Disease.** *John Creeper*

Metro Perth: Rapid increase in mortality rate in a group of 6,500 thirty day old layer replacements. Mortality increased to 350-400/week. Lesions included severely atrophic and inflamed bursa, necrotising hepatitis (probably bacterial), and focal (probably fungal) nephritis. Immunodiffusion test for IBD were positive.

Comment: IBD virus (not the hypervirulent strain) is present in Australia and capable of causing mild disease. Deaths stopped after the first lab submission and adjustment of shed heating and correction of water and feed supply. Further submissions of live birds: bursal and other lymphoid tissue including thymus populated with lymphocytes.

Conclusion - previous deaths were due to multiple factors including chilling and possibly water deprivation. The factors precipitated stress-induced bursal lymphoid depletion. No viruses were isolated by allantoic viral culture of bursa and kidney. Hypervirulent IBDV is less likely, based on the flock mortality rates. Virus neutralisation testing to distinguish strains is not performed in Australia.

### **Bovine Peripheral myelinopathy.** *David Forshaw*

Albany. One of three 6 month-old Santa Gertrudis calves bought at local sale yard. One individual developed progressive ataxia of both fore and hind limbs. Histologically, the only lesions are in all peripheral nerves examined which have segmental thinning and ballooning of myelin sheaths producing a moth-eaten appearance. Segmental reduction in myelin staining is readily apparent in sections stained with luxol-fast blue/silver. Axons are wider than normal but presumably filling up dilated cylinders. The cause of this condition in a single calf is not clear.

### **Porcine serosal hyperplasia?** *Cleve Main*

Intestine submitted from abattoir, age of pig unknown. The serosal surface was covered with irregular nodules of pale firm tissue, projecting up to 1 cm from the surface, and coalescing in places. A similar change also involved the mesentery to a lesser degree. Histologically, the serosal 'growth' consists of connective tissue, within which are many lymphatic channels and deep invaginations from the surface mesothelium. The rest of the gut was essentially normal although there was increased submucosal fibrosis in the ileum. There were no inflammatory cells to indicate that this is a post-inflammatory sequel.

Comment: Several possibilities were considered including mesothelioma, lymphangiectasia, chronic lymphangitis, serosal and mesenteric lymphatic dysplasia. We were reminded that a similar lesion had been described in koalas by Canfield et al (AVJ 67:342-3, 1990) where the conclusion was 'probably mesothelioma'. Images of this case was circulated via the ASVP mailing list.

**Rabbits: Vitamin A deficiency.** *John Creeper*

Rabbit submitted from university breeding unit. Three month history of neonatal deaths. Young survivors often had keratoconjunctivitis and tetraparesis. Four necropsies all revealed the same lesions: severe internal hydrocephalus and wallerian degeneration of the dorsolateral white matter tracts of the spinal cord. Follow up history revealed the feed contained 4,000 IU/kg vitamin A (10,000 IU/kg is recommended). In addition, all feed is autoclaved before feeding (common practice) which would do little for preservation of vitamin A activity.

**Ovine Hepatic Listeriosis.** *Cleve Main*

Kojonup. Three Merino rams died within a few months after a period of ill-thrift. Gross lesions of multiple 4mm caseous lesions were found throughout the liver from one of these animals. Histologically there were several large, irregular necrotic lesions bordered by inflammatory cells and gram negative rods. Additionally there were many small focal necrotic areas containing gram positive coccobacilli. Both *Listeria monocytogenes* and a haemolytic *E.coli* were cultured from fresh tissue. While hepatic listeriosis is described, to our knowledge, it has not been previously observed in WA in adult sheep.

**Rock Rat – Encephalomyocarditis Virus Infection?** *Russell Graydon*

Zoo animal. Common rock rat found dead. Grossly there were pale foci throughout the liver and approximately one third of the heart was occupied by a pale lesion centred on the apex of the ventricles. Histologically, the liver has numerous random foci of hepatocyte loss and fibrous replacement (old Tyzzer's lesions?). The heart lesion consists of massive, predominantly mononuclear inflammatory cell infiltration which has resulted in severe loss of myocardiocytes. The brain was normal (one section). The differential diagnosis included EMC virus infection and nutritional myopathy but the predominant inflammatory change raises the possibility of a viral myocarditis.

**Murray Cod – Cystic necrosis of the kidney.** *John Creeper*

Murray Cod grown commercially in tanks. A few have died each day recently out of 7,000 at risk. The kidneys have multifocal areas of parenchymal lysis which has progressed through to liquefaction and cyst formation in several areas. Some of the foci are quite large (1 mm). There are no organisms associated with the lesions and surrounding tissue looks normal. Strangely, there is no inflammatory reaction associated with this change. Nothing like this is recorded in the literature.

**Barramundi nodavirus (viral encephalopathy and retinopathy).** *Brian Jones*

Barramundi raised in tanks in Broome displayed signs of CNS disease and problems with swim bladder control. Distinctive 'holes' are present in the retina, spinal cord and brain. Each vacuole is approximately 20 microns wide and could represent swollen cells rather than empty spaces. A large number of cell types are affected by the virus, including neurons, astrocytes, oligodendrocytes and microglia. This is the first diagnosis of nodavirus infection in WA (notifiable disease). Source of virus is thought to be an introduced wild-caught (WA) male. This facility and two trace-forward facilities have been depopulated in an attempt to eradicate the virus which is known to cause heavy mortalities. Samples will be sent to the University of Queensland for comparison with Queensland and

Northern Territory strains. An experimental PCR is available in both the Northern Territory and Queensland.

### **Poorly Differentiated Glial Cell Tumour in a Bovine.** *Cleve Main*

The subject was a 7 year old female Aberdeen Angus that developed shuffling, stumbling gait, tended to circle and chewed compulsively. It was yarded a week after developing clinical signs but died 3 days later after an episode of bellowing and recumbency.

Following necropsy a unilateral lesion was found in the enlarged right cerebrum. A cross-section at the level of the optic chiasm revealed a 3-4 cm diameter area of softening that bordered on liquefaction. Histopathological examination showed this area was populated by mononuclear cells with ill-defined eosinophilic cytoplasm. In many areas there were festoon-like pseudo-palisades of these cells that bordered areas of necrotic neuropil. Multi-nucleated cells were present in some places and in others, especially close to lateral ventricles, there was glomeruloid vascular proliferation.

The tumour is consistent with glioblastoma as described in: Summers BA, Cummings JF and de Lahunta A *Veterinary Neuropathology* 1994:63.

### **Pan-encephalomyelitis in a Boer goat** *Cleve Main*

A seven week old Boer kid with a 2 day old history of scoliosis of the cervical vertebrae was presented for examination. It was otherwise bright, alert, and able to eat, drink and walk normally although it had been reported to be slightly off colour two weeks previously.

Euthanasia and subsequent necropsy yielded little in the way of additional information except that the scoliosis did not relax until the nuchal ligament was cut. Histopathological examination of the brain and cervical spinal cord on the other hand revealed moderate to severe bilateral inflammation of both grey and white matter of the midbrain, medulla and ventral cervical cord. Typically there was marked perivascular cuffing of blood vessels with histiocytes, lymphocytes and lesser numbers of plasma cells and neutrophils. In many areas there were glial aggregates frequently accompanied by infiltrates of neutrophils. In places, the proportion of neutrophils was such that a description of micro-abscessation was appropriate. In some areas of cervical cord the inflammatory process was severe and extended well into white matter and ventral nerve roots. In addition to these lesions there was also a mild lymphocytic meningitis.

Additional lesions were present in the liver and were characterised by random dense focal aggregates of plasma/lymphocytes with occasional mitotic figures. The diagnosis is unclear. CAE is high on the list of differential diagnoses but the presence of so many neutrophils is not typical. Their presence and micro-abscessation is more suggestive of listeriosis; however, gram-stained sections failed to demonstrate gram positive rods. The role or significance of the lesions in the liver is unclear and one could speculate that the kid was suffering from more than one disease. It was interesting to note that its twin and mother were serologically positive for *Neospora sp.* Investigations are continuing.



## **Goldfish – Severe muscle atrophy.** *Fran Stevens*

Imported goldfish from a metro pet shop were used in a teaching demonstration. They had apparently been raised in large ponds, then moved to the pet shop and placed in small tanks.

Histologically there is severe muscle atrophy with few thin surviving fibres and a lot of empty sarcolemmal tubes. The intervening space is filled with fluid, rather than fat. It is possible that well-exercised and well-developed fish have subsequently suffered from disuse atrophy. Nerve ganglia also appeared abnormally large, raising the possibility of innervation deficits. Splenic necrosis raised the possibility of viral involvement, but the fish had all been sold before further investigation carried out.

## **Canine Leishmaniasis** *Peter Irwin*

*(Peter is senior lecturer in small animal medicine at the Murdoch University School of Vet Science, and has been kind enough to provide this summary of cases that had been reported in the mass media)*

"During 2003, leishmaniasis was reported in some kangaroos at a wildlife park in the Northern Territory and in an imported dog in Perth. The cases in the kangaroos resulted in a flurry of sensational reporting in The Bulletin on 15/9/03, referring to the "hideous flesh-eating disease" which was previously thought not to occur in Australia.

"The dog was (is) a 5 year old miniature Poodle that had been imported from Portugal with two other dogs, about three years ago, and all 3 went through quarantine. All three had been fine until one developed severe weight loss and was referred for a work-up. Its main presenting signs were emaciation, splenomegaly and scaly skin. It had pancytopenia, hypoalbuminaemia, hyperglobulinaemia and a severe proteinuria consistent with severe glomerulonephritis. This was associated with laboratory evidence of chronic renal failure. A diagnosis of leishmaniasis was made by splenic aspirate cytology and we have since had the species confirmed to be *L. infantum* by molecular genotyping in Switzerland.

"With the assistance of Merial, we were able to import some meglumine antimonate, which we have used in conjunction with allopurinol to treat the parasite infection, and we've managed its GN with prednisolone at first, and then by a combination of enalapril and aspirin.

"The infection was notified to the State CVO as it is exotic, and considered opinion here in WA and in general, is that the sand fly vectors (*Phlebotomine* spp.) that are required for transmission, do not exist here, thus there was no obvious danger to the owners or the other dogs (neither of the other dogs were sero-positive, whereas the affected dog of course was.) The issue of vector is concerning the people involved with the kangaroos, though.

"The case reminds us about the importance of travel history, and that our quarantine system is not perfect. Leishmaniasis is well known to have very long incubation periods in some individuals - up to 7 years, so cases such as this are inevitable from time to time."