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VETERINARY PATHOLOGY REPORT

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Management Committee Report

Welcome to the second Veterinary Pathology Report for 2005.

The Cairns AGM: Preparation for the 2005 Cairns AGM is well advanced. It seems that the idea of holding it as a curtain-raiser to the World Wildlife Disease Association Congress has borne fruit, in that not only has a healthy number of our members indicated they will attend, but we will have the stimulus of a considerable number of visitors who want to warm up at our meeting before tackling the splendid fare on offer at the WDA Congress immediately following.

The WDA program is finalised and can be visited *via*
http://www.rainforest-crc.jcu.edu.au/events/WDAConf.htm

The program for our ASVP meeting is by no means finalised, although acceptances for abstracts closed on April 30 and we have a healthy number of interesting abstracts to hand.

Now is the time to let the honorary secretary Roger.Kelly@uq.edu.au know of any items you wish to have placed on the AGM agenda. This, with the conference program, will be circulated *via* mailing list very soon.

Conference registration forms have been distributed by email, together with some advice about accommodation in Cairns. If you haven't received these mailings, please contact the secretary.

Please note that there will be no credit-card facilities at the registration desk: it shouldn't be difficult to raid a local ATM for the $100 registration fee.

The Australian Animal Pathology Standards Program (AAPSP)
The continuing professional development/proficiency testing trial on CD evoked disappointingly few responses but those that were received were very useful and the respondents are thanked. The analysis of the results has been circulated by mailing list. Any one interested in this exercise who did not receive this report can ask the Secretary for a copy.

Contract Proficiency testing *via* glass slide circulations has been commenced by the QDPIF lab at Toowoomba. Jim Taylor Jim.Taylor@dpi.qld.gov.au should be contacted for details of the program.

The AAPSP has received funding from Animal Health Australia to (a) convert all archival copies of the ASVP Veterinary Pathology Report and Proceedings of annual meetings to searchable digital text files, and (b) to support ongoing maintenance of the histology and transparency collections and begin the process of digitally archiving them, so that they will be more widely accessible to clients. The text archiving is already well under way. When it is completed, members will be able to either search the files online for articles, authors, diseases, etc. Or they can download the files for storage on their own systems so they can be searched off-line. Recent issues of the Report and the Proceedings can of course be added as soon as they are published.

More details of AAPSP activity will be presented by Tony Ross in his Scientific Coordinator's report which will appear in the AGM Proceedings.

Roger Kelly (honorary secretary)
for the **Management Committee** May 16, 2005

NOTICE OF ASVP ANNUAL GENERAL MEETING, 2005

Agenda of the annual business meeting of the Society to be held at 4.00pm, Saturday June 25th, 2005.

Venue:
The RSL Club, The Esplanade, Cairns

AGENDA

1. **Welcome; apologies**

2. **Minutes of AGM 2004**

3. **Business arising**
   National rationalisation of veterinary registration
   Proposed association of veterinary laboratory diagnosticians

4. **Correspondence**

5. **Reports of office-bearers**:
   Presidential
   Secretary/VPR editor
   Treasurer
   Scientific Coordinator, AAPSP
   Webmaster

6. **General Business**
   The ASVP Website: utility and costs of upgrading
   Location of next executive

7. **Election of Office-bearers**
   The incumbents of these positions offer themselves for election or re-election:

President:	Richard Miller
Secretary/VPR Editor:	Roger Kelly
Treasurer:	Anita Gordon
Committee Members:	Mark Carrigan, Dick Sutton, Richard Ploeg

   Other nominations (seconded) for these positions should be in the hands of the Honorary Secretary in time for the opening of the meeting

   The following positions are un-elected and the members named have kindly undertaken to continue in office:

Scientific Coordinator, AAPSP:	Tony Ross
Webmaster:	Cleve main

   State Correspondents (un-elected): no changes foreshadowed at this date.

8. **Date & Venue of the next AGM:**

Members who wish to place additional notices of motion and additional items on the agenda are asked to submit these to the honorary secretary within 13 days of the annual general meeting.

Roger Kelly
Hon. Secretary, ASVP

STATE REPORTS

NEW SOUTH WALES

State correspondent: Paul Gill

Ingested *Eucalyptus viminalis* implicated in oxalate nephropathy of marmoset monkeys

B.A. Vanselow[1], M. Pines[2], J.J. Bruhl[3] and L. Rogers[2] *[1] NSW Agriculture Beef Industry Centre, [2] Centre for Neuroscience and Animal Behaviour, [3] School of Environmental Sciences and Natural Resources Management, Botany; University of New England Armidale 2351*
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Seven marmosets from a laboratory colony of 17 died between February and August 2002. Death was attributed to kidney failure from oxalate-induced nephropathy. The source of oxalate was identified as *Eucalyptus viminalis.* Eucalypt branches, both dried and freshly cut from various sources had always been provided for climbing. Just prior to January 2002, the branches used were *E. radiata*. Branches from a new source, a recently pruned *E. viminalis,* had been put in the marmoset cages in early January 2002 and the marmosets were observed to chew on leaves and bark. The deaths commenced in February and the branches were removed in March. Urinalysis indicated that all the surviving marmosets had chronic renal damage and deaths continued until August 2002.

The colony had been founded at the University of New England in 1992. Prior to this episode only 5 deaths had occurred in the colony. At the beginning of 2002 the colony comprised 6 males and 11 females. They were housed indoors but had access to outdoor cages. They were fed a selection of foods once daily *ad libitum*: orange, beans, pear, peach, banana, sultanas, peanuts, yoghurt, cheese, boiled egg, bread, dog-biscuits, mealworms, Nutri-Grain®, Pentavite®, banana cake and meatloaf. Water was also available *ad libitum.* No dietary changes had been made prior to the deaths: the only management change was the introduction of the new eucalypt branches.

Post-mortem examination revealed enlarged pale kidneys. Histopathologically the kidneys were undergoing a chronic degeneration with tubular and glomerular atrophy, chronic inflammatory cell infiltrates and fibroplasia. Numerous crystals were observed within dilated tubules and within macrophages and epithelial cells. These were confirmed as calcium oxalate by special stains (Von Kossa's and Pizzolato's) as well as by the use of crossed polarised filters. Many crystals were observed in kidneys of the 3 monkeys that died before the suspect branches were removed; fewer were present in kidneys of those that died later. Further, a kidney from a marmoset that died in May 2001, prior to the outbreak, had no oxalate crystals.

Crystals were observed in high concentrations in the suspect leaves and bark. They were confirmed as calcium oxalate: birefringent under polarised filters, soluble in hydrochloric acid (50%) and insoluble in acetic acid (45%).

Urine samples were collected from the surviving marmosets 80 and 122 days after contact with the suspect branches. Urinary protein levels were elevated (mean 500mg/dl), and ketone levels were elevated in some individuals. Three new marmosets were introduced into the colony in 2003. Analysis of their urine showed low protein (Mean 10mg/dl) and no ketones.

Insoluble calcium oxalate crystals can precipitate in the kidneys of animals and man. The resulting nephrosis can result in death, either acutely or from chronic renal failure. The source of soluble oxalate can be either endogenous or exogenous. The epidemiology of this outbreak

suggests an exogenous source and there is strong evidence it was the bark and leaves of *Eucalyptus viminalis*.

WESTERN AUSTRALIA

State correspondent: David Forshaw

Bovine chronic placentitis with live calf

David Forshaw *Animal Health Laboratories, Albany*

A Mount Barker cow with dystocia.
A veterinary practitioner successfully delivered a live calf but noted a thickened placenta with tortuous blood vessels, particularly in the non-cotyledonary area. Microscopically there was localised congestion, haemorrhage and fibrosis with a surface layer of granulation tissue infiltrated by neutrophils. Small vessels often contained fibrin thrombi. Within cotyledons there were multifocal areas of acute necrosis and intense mixed inflammatory cell infiltration including many neutrophils. Bacterial culture yielded a mixed growth that included *Streptococcus bovis* but with Gram staining, lesions contained only mixed Gram-negative rods.

Spirochaetosis (*Borrelia anserina*) in ducks

Samantha Wong *Animal Health Laboratories, South Perth*

Seven-month-old mixed-sex Muscovy ducks from Popanyinning had profuse green-tinged diarrhoea, and leg weakness that progressed to paralysis. Within 3 to 4 days 12/500 birds had died with another 36 sick. Necropsy of three ducks revealed copious green-tinged fluid intestinal content and multiple pale 2 - 3 mm foci throughout livers and spleens. Histologically both liver and spleen showed medium to large randomly distributed focal acute to sub-acute coagulative necrosis. Numerous filamentous serpentine organisms consistent with spirochaetes were evident in silver-stained sections. In a muscle section of one bird there was a focal extensive area of haemorrhage and oedema with myocyte necrosis. Additionally there was fibrinoid necrosis of many blood vessels. Borelliosis caused by *Borrelia anserina* is an acute septicaemic disease of birds characterised by greenish diarrhoea, weight loss, elevated body temperature, inactivity, anorexia, paresis and/or paralysis. The organism does not survive outside the host and fowl ticks classically serve as the reservoir, however it can be transmitted via blood, excreta or tissues. Biting insects including mosquitoes and fowl mites can also transmit the disease.

Ovine oesophageal myopathy

Cleve Main and Shane Besier *Animal Health Laboratories, South Perth*

Regurgitation, wasting and deaths in a flock of 150 Merino ewe weaners at Yallingup with 15 dead and 15 affected. Three sheep were necropsied initially and common to all was extensive myopathy of the oesophageal lamina muscularis. This was characterised by swelling and pallor of muscle fibres so that they manifested only delicately thin myofibrils with little if any trace of the characteristic striations (yes; the ruminant oesophageal lamina muscularis is entirely striated). Mild acute myopathic lesions were also present in sections of skeletal muscle. Later samples showed more chronic lesions with early attempts at repair. Given reports of a similar condition in sheep called Vermeersiekte (vomiting sickness) due to *Geigeria spp* in South Africa and spewing disease attributed to other plants such as *Helenium spp* and *Hymenoxys spp* in the USA, the

property was searched without success for similar plants that may have been responsible. The acute nature of some of the lesions present in the earlier cases indicates that the toxin was still available in mid- to late February. Failure to find the suspect plant three weeks later, combined with histological evidence of muscle repair, suggests that the plant is no longer present and that the property will need to be visited again during spring/early summer. Note that South African researchers have found that dried milled *Geigeria ornativa* retains its toxic properties for at least four years when stored at room temperature (van Heerden J, van der Lugt JJ, and Durante E.J. S Afr Vet Assoc. 1993 Jun; 64(2): 76. Experimental vermeersiekte (*Geigeria ornativa* O. Hoffm. poisoning) in sheep. The active principles are known to be α,β-unsaturated-8-sesquiterpene lactones.

Multiple subcutaneous osteomas? in a bobtail lizard
Cleve Main *Animal Health Laboratories, South Perth*

Sample from "papillomatous" growths near the external auditory meatus of a bobtail lizard at a wildlife park in Perth. The mass was approximately 1cm x 0.5 cm and was very hard, requiring decalcification for sectioning. Histologically there were plaques of mature bone beneath the dermis below which there was a core of loose adipose connective tissue. Most plaques contained osteocytes and lacunae and lamella formation was evident in many areas. No osteoclastic activity was evident suggesting that no remodeling was taking places. There were perivascular infiltrates of lymphoid cells in the connective tissue immediately below the bony proliferations. Discussion revolved around whether bony plaques could be a normal component of lizard anatomy but they are plainly abnormal in this case. The cause of these bizarre lesions is unclear but it is not a malignant change.

Nephropathy in heifers
John Creeper and Mark Kabay *Animal Health Laboratories, South Perth*

Chronic wasting with three-week history of weight loss, weakness, increased thirst and frequency of urination with scours in 3-year-old heifers at Esperance.
Six /150 dead and 10 more affected. One animal was killed for necropsy. Emphysema of lungs was noted as well as "degenerated changes in kidney and scarring of liver". Histologically the lung showed multifocal bullous emphysema with some thickening of surrounding connective tissue. The most significant changes were in the kidney where there was acute necrosis of renal tubular epithelium with accumulation of degenerating neutrophils within lumina. There was also some regeneration and attenuation of tubular epithelium, diffuse interstitial fibrosis and mild multifocal interstitial lymphocytic infiltrates. The aetiology of this moderate, polyphasic, suppurative tubular nephrosis and interstitial nephritis is unclear but a nephrotoxin should be sought.

***Pseudomonas* pneumonia in feedlot cattle**
Cleve Main *Animal Health Laboratories, South Perth*

Problem with severe pneumonia in a Busselton feedlot containing 12-month-old mixed sex and breed beef cattle.
Ten/1000 cattle had died and another 50 or so had respiratory problems. Necropsy of one animal revealed advanced pneumonic lesions characterised by large irregular areas of coagulative necrosis bordered by zones of bacteria and inflammatory debris. Additionally there was marked

thickening of the pleura by fibrous tissue overlayed by proteinaceous exudate. The interlobular septa were dilated by sero-fibrinous exudate, fibrous tissue and dilated lymphatics that contained fibrinous clots. Alveolar septa were thickened, in some areas by infiltrating mononuclear cells, but in many other places, by proteinaceous exudate and capillary thrombi. Surprisingly, culture of lung yielded pure and heavy growths of *Pseudomonas aeruginosa* rather than the expected *Pasteurella, Mannheimia* or *Haemophilus* spp. Similar focal lesions were evident in the liver but also present were septate branching fungal hyphae consistent with *Aspergillus* sp., suggesting that the hepatic lesions may have spread from rumenitis that went undetected at necropsy.

Bovine neonatal multifocal encephalomalacia

Cleve Main *Animal Health Laboratories, South Perth*

Ill-thrift and mortalities in Brahman calves on a Northampton farm. Four/40 died in a similar manner within the first week after birth. Some calves appeared not to be able suckle the udder but attempted to suckle other parts of the cow. They lost condition, become dehydrated and died after 3 - 4 days. No significant lesions were described at necropsy. In one calf, there were spectacular histological changes in the brain, with multiple foci of acute rarefaction that contained ghosts of swollen axons, necrotic neurons and glial cell nuclear debris in the thalamus, collicular and cerebellar white matter. Proteinaceous lakes were present around several capillaries. Overall, blood vessels appeared prominent and many had reactive endothelial cells. In short this was multifocal acute leucoencephalomalacia with vascular leakage suggesting endothelial damage as the basis of the condition. There was apparent discrepancy between duration of the clinical description (4 days) and the age of the lesions (hours). A blood sample from another affected calf showed elevated GGT, urea and creatinine, and elevated potassium and magnesium but samples from the calf that was necropsied and another affected calf did not show these abnormalities. Multifocal encephalomalacia is an unusual lesion in a neonatal bovine and if the opportunity arises, further necropsies should be done to see if there is a consistent pattern of lesions.

Bovine thrombotic meningoencephalitis

John Creeper *Animal Health Laboratories, South Perth*

A 10 month-old Waroona heifer presented recumbent, pyrexic and dull with rough lung sounds and nasal exudate. At necropsy, purulent exudate was noted in cerebral ventricles and basilar meninges. There was some reddening of the lungs, particularly on dependent side. In the brain, there were bacterial thrombi within arterioles of both the meninges and neuropil accompanied by an intense neutrophilic infiltrate. Several vessels showed fibrinoid necrosis of the media. A morphologic diagnosis of thrombotic encephalitis was made but the likely candidate, *Haemophilus somni* (previously *Haemophilus somnus*) was not able to be isolated from brain or lung.

Sporadic Bovine Encephalitis

John Creeper *Animal Health Laboratories, South Perth*

Yearling cattle at Gingin were grazing lush pasture but were performing poorly and exhibiting severe wasting with weakness and incoordination that progressed to lateral recumbency. Necropsy of an affected animal revealed fibrinous pericarditis, swollen liver and purulent meninges. Histopathological examination showed severe inflammation in the meninges, brainstem and cerebrum with intense cuffing of meningeal and cerebral vasculature by histiocytes, lymphocytes and plasma cells. Changes were most intense in the caudal areas of the

cerebrum and brainstem and characterised by intense gliosis, leucocytic vasculitis with thrombosis and focally extensive areas of ischaemic malacia. The process extended into the leptomeninges with similar severity and into the spinal cord. The heifer was serologically positive to *Chlamydia* with a CFT titre of 1:64.

Renal dysplasia in a potoroo AA-05-0120

David Forshaw *Animal Health Laboratories, Albany*

This captive elderly female potoroo from Albany had been under observation for some months. It has previously lost condition but then recovered only to deteriorate again over the past month. It was found moribund and was euthanased. Microscopically, there were segmental areas of dilation of Bowmans capsules and renal tubules extending from the medulla to the outer cortex. Tubules often contained amorphous eosinophilic material. Over the renal papilla in particular, the pelvic epithelium was obviously proliferative and there were inclusion cysts within the papillary proliferations. The renal lesions were unlike any previously seen in potoroos at Albany and the consensus was that this was likely a long standing developmental defect, presumably congenital. The presence of numerous *Toxoplasma*-like zoitocysts in the skeletal muscle drew comment. They were unassociated with any inflammatory change and were not the cause of the animal's illness. The cysts were stained strongly using immunohistochemistry with a polyclonal *Toxoplasma* antibody but interestingly sections were negative in a PCR for *Toxoplasma* conducted at a local hospital.

Enteropathy in Kangaroos

Shane Besier *Animal Health Laboratories, South Perth*

Samples from 2 moribund kangaroos necropsied near Yalgoo were submitted for laboratory testing. The submission formed part of an on going investigation into mortalities of kangaroos across a large area of pastoral country. In the Yalgoo area alone, shooters estimated that over 3000 of an estimated 10,000 (at least) died in recent weeks. 150 - 300 animals have been reported sick but clinical signs had not been described. Similar reports from other areas, including Pindathuna, Laverton, Meekatharra and Cue had been filtering in from a variety of sources for several weeks. The condition of the dead kangaroos has ranged from good to extremely poor. Some have had lumpy jaw (oral necrobacillosis). Necropsy of these 2 animals revealed emaciation and worms were present in the stomach and bile duct. A necrotic focus was present on the tongue of one.

Histopathological examination provided much more information. In both animals there were severe sub-acute villus atrophy and loss of normal crypt architecture of the small intestine. There was a strong suggestion of loss of crypts but no evidence of active crypt epithelial necrosis. The remaining crypt epithelium was very disorganised, with markedly enlarged and vesicular nuclei, while the cells lining the stunted villi and luminal surface ranged from squamous to cuboidal to reactive columnar. In many places a thick diphtheritic exudate covered the attenuated mucosal surface. There was severe sub-mucosal oedema, an absence of neutrophils, a paucity of plasma cells and lymphocytes and macrophages in the lamina propria. There was also apparent transformation of the lamina propria of stunted villi into tight whorls of fibroblasts. Suggested causes included virus infection and exposure to a plant toxin. Salmonella were isolated from the gut and organs of one animal but not the other. Further necropsies failed to show similar lesions and following widespread rain in the area, the kangaroos dispersed and we have been notified of no further sick animals.

Ovine sarcocystosis
David Forshaw *Animal Health Laboratories, Albany*

Thirty/680 weak hogget wethers at Tambellup on a 100ha 'eaten out' paddock.
Clinically affected sheep were 'doughy', lethargic and weak. 15-20 lay down on walk back to shed. Vitamin E deficiency was suspected. Examination of tissues from 2 lambs revealed myonecrosis and numerous sarcocysts in the myocardium and skeletal muscle. Both thin-walled *(S. arteiticanis*) and thick walled (*S. tenella*) sarcocysts were present. Sarcocysts are common in skeletal muscle in sheep but in this case there was a neutrophil-dominant inflammatory cell infiltration of the cysts, which extended into the surrounding interstitium. Most but not all sarcocysts were affected. The cause of the clinical syndrome is unclear. There was insufficient evidence of nutritional myopathy, but the animals were on poor feed, mobilising fat reserves and a worm egg count indicated that intestinal parasitism might have been a contributing factor. Sarcocystosis is described in the literature but we have no cases on file in the AHL.

Bovine tubular nephropathy
Cleve Main *Animal Health Laboratories, South Perth*

Unexplained deaths in a small mob of Murray Gray heifers at Beverley.
Two/15 dead and a third died later. Common clinical signs were weight loss, then recumbency with rumen stasis. Microscopically there was severe pulmonary oedema and fibrinoid necrosis of intestinal sub-mucosal arteries. However the most interesting changes were found in the kidney and were characterised by diffuse interstitial oedema and widespread acute to sub-acute tubular epithelial necrosis involving both cortical and medullary tubules. Affected tubules were commonly dilated and their epithelium attenuated. They frequently contained low to moderate numbers of neutrophils or proteinaceous casts or both. Glomeruli were unaffected.

Ovine verminous hepatitis AA-04-0879
David Forshaw *Animal Health Laboratories, Albany*

Abattoir samples from a line of 265 ewes from Williams.
About 60 livers contained scattered focal 1-4mm nodules throughout the parenchyma. There was also widespread distribution of smaller gritty nodules in lungs. Additional nodules were also present on small intestinal and omental fat. Histopathological examination of lung, liver and intestine revealed multiple nodules with thick fibrous capsules surrounding central zones of degenerating eosinophils attended by numerous multi-nucleated giant cells and lymphocytes. In rare instances nematode fragments were evident in the central zone. *Oesophagostomum columbianum*, or “nodule worm” was thought the most likely cause of these nodules but *O. columbianum* has not been identified before in WA. Infestation by *Strongyloides sp.* was also suggested as a possibility. A faecal sample was taken from the only surviving ewe on the property as well as from several lambs belonging to the previously slaughtered ewes. The worm egg count of the ewe was 200 epg and those of the lambs ranged from 0 – 450 epg. A Baermann test on a pooled faecal sample proved positive *to Muellerius sp*. Infestation with *Muellerius sp* might explain the lung lesions but no references describing involvement of other organs has yet been located. An interesting observation by the farmer was that a mob of emus was consistently present on the property. There was some speculation that the sheep may have picked up a heavy burden of nematodes originating from the birds but a subsequent check of emu dung failed to find any nematode eggs.

Mortalities in Weedy Sea Dragons
John Creeper *Animal Health Laboratories, South Perth*

Death of a second weedy sea dragon from a Perth aquarium
Grossly there were haemorrhages over the surface of the ovaries and swim bladder with multiple coalescing depressed areas of pallor throughout the liver. Histologically there were focally extensive areas of acute coagulative hepatic necrosis with intra-lesional Gram-positive rod shaped bacteria. Similar bacteria were present throughout the coelomic cavity and extending through the ovarian and renal parenchyma. Bacteriological findings were not consistent with the histological evidence and no Gram-positive bacteria were isolated. A previous submission 4 weeks earlier was characterised by Gram-positive rod-shaped bacteria associated with acute tissue necrosis and eosinophilic granular cell inflammation within multiple organ systems. Despite intense bacteriological work-up the bacterium was not cultured.
The major cause of mortalities in Weedy Sea Dragons, Leafy Sea Dragons and Sea Horses in Western Australia is the marine scuticociliate *Uronema nigricans*, which penetrates the mucosa of the intestinal tract and multiplies throughout the peritoneal cavity, often invading splanchnic organs and tracking through to the exterior. The ciliate also is responsible for deaths in tuna in cage culture at Port Lincoln and is associated with meningoencephalitis. The pathogenetic mechanism is believed to involve initial penetration of the olfactory rosettes.

NORTHERN TERRITORY
State Correspondent: Anton Janmaat

Infectious Canine Hepatitis
Anton Janmaat *Berrimah Veterinary Laboratories*

A 12-week-old male Bull Mastiff was submitted for necropsy in January this year. The dog had been lethargic and inappetant for two days and had vomited twice overnight prior to presentation at a veterinary clinic. Shortly after admission the dog developed acute respiratory distress, went into cardiac arrest and, despite intensive attempts at resuscitation, died. The pup had been vaccinated three days earlier and a parvovirus antigen test was negative.

Post-mortem examination revealed pale mucous membranes. There was generalised lymph node enlargement; the nodes appeared haemorrhagic and the thymus was large and oedematous. Excess bloodstained fluid was present in the abdominal and thoracic cavities. There was reddening of the gallbladder, urinary bladder and part of the left diaphragmatic lung lobe, and red spots were present on the intestinal serosa. The mesenteric lymph nodes were enlarged and haemorrhagic. Both spleen and liver were slightly enlarged and the liver showed a periacinar pale pattern. The left endocardium was haemorrhagic, and from stomach to colon there was bloodstained essentially fluid content, with more mucus in the jejunum and ileum, while the colon content was partially clotted.

When asked by a colleague veterinary pathologist about the possible cause of this essentially haemorrhagic syndrome, the response was: "If you'd asked me 30 years ago, I would have said infectious canine hepatitis but does it still occur in urban areas of Australia?" Apparently it does. Although the submitting veterinarian did not request histopathological follow-up, a full range of tissues was sectioned. The liver section was diagnostic and showed

periacinar necrosis and haemorrhage with typical intranuclear inclusion bodies in Kupffer cells and hepatocytes.

The submitting veterinarian was informed of this diagnosis. He was later told that the owner had taken the pup on several camping trips in the bush near Dundee Beach. Perhaps the virus is still circulating in wild dogs.

Streptococcosis in Sea-Caged Barramundi *Lates calcarifer* in the Northern Territory

John Humphrey *Berrimah Veterinary Laboratories*

The pathological, histopathological and bacteriological findings are described in an outbreak of streptococcosis caused by *Streptococcus iniae* in farmed, sea-caged barramundi in the Northern Territory. The disease occurred following stress, trauma and environmental disruption caused by cyclone Ingrid in March 2005 and affected all age groups of fish across the farm.

Gross examination showed multiple irregular focal haemorrhages in the integument. Diffuse areas of severe congestion and haemorrhage were prominent in and around the base of the fins, especially the dorsal fin. Severe unilateral or bilateral exophthalmos was present, with intra-ocular haemorrhage in most cases. Gills showed marked congestion and were excessively mucoid. The skeletal musculature was diffusely reddened with fine haemorrhages throughout. Multiple focal haemorrhages to approximately 8 mm were present in the abdominal fat, on the intestinal serosa and the peritoneal surface. The pericardial sac contained bloody fluid. Intestinal contents were yellow, fluid and flocculent. Spleens were small.

Histopathological changes were referable to fulminant Gram-positive bacterial septicaemia, with localisation of the organism in tissues including skeletal muscle, liver, kidney, gill, eye, spleen, intestinal tract and fins. Vascular hyperaemia, haemorrhage and numerous colonies of Gram-positive cocci in the microvasculature and occasional septic micro-thrombi were accompanying changes.

Other changes included severe, generalised granular necrosis of skeletal muscle fibres, marked interstitial oedema and mild focal necrosis of interstitial cells in the kidney, and loss of splenic architecture, with generalised depletion of white cells, fibrin deposition, haemorrhage and necrosis. Massive colonisation by Gram-positive cocci occurred in kidney, spleen, epicardium and in the vascular choroid of the eye.

The causative agent, *S. iniae*, was seen in high numbers in Gram-stained smears of pericardial fluid, liver, kidney, brain and spleen and was readily isolated from these tissues. Identification of the organism was based on morphological and biochemical examination in comparison with a reference strain and the diagnosis was confirmed by PCR assay by Oonoonba Veterinary Laboratories, Townsville.

This is the first confirmed occurrence of *S. iniae* in the Northern Territory. The pathogen was previously recognised as a major threat to the successful farming of fish with quarantine measures imposed to prevent its introduction. Following its occurrence, measures to limit further spread have been implemented. Although there is little data on the epidemiology of *S. iniae* in fish, it appears probable that infection occurred from the local environment subsequent to the stress, trauma and environmental disruption caused by the cyclone and does not represent an incursion from external sources.

Stillbirths and Weak Neonates in a Piggery

Cathy Shilton *Berrimah Veterinary Laboratories*

A small 150-sow piggery in rural Darwin experienced problems with stillbirths and weak neonatal piglets during Sept-Nov. 2004. Of 40 sows farrowing during the period, 30 litters were affected. Sows appeared to farrow normally, however most piglets in affected litters were either born dead or very weak, trembling and dying within hours. Piglets that survived a few hours went on to become apparently healthy. No deformed piglets were noted, and mummies were rare. Sows were fat, kept outside on dirt until farrowing and fed exclusively on locally baked white bread. Sows and growing pigs appeared clinically healthy.

Gross post-mortem findings in eight piglets revealed near or at-term, at least partially haired piglets. There was moderate oedema of subcutaneous and connective tissues primarily in the head, neck, shoulders and forelimbs in four. Two piglets had focal swellings with associated haemorrhage in the subcutaneous tissue of the forelimbs. Most piglets, particularly the more autolysed ones, had a scant to moderate amount of blood-tinged clear fluid in the abdomen or thorax. One piglet had a few epicardial petechial haemorrhages. Thyroid glands were prominent, being approximately the same diameter as the trachea. Histological examination of various tissues from several piglets was largely unremarkable, with no evidence of tissue necrosis or inflammation. Thyroid gland follicles appeared small, generally ranging from 30-75 μm in diameter, with occasional follicles up to 200 μm in diameter. Follicles were lined by tall cuboidal, occasionally jumbled epithelium and appeared either devoid of colloid or contained very pale eosinophilic, usually foamy material. However, comparison with images of the histological appearance of thyroid glands from normal neonatal pigs, kindly provided by an eminent colleague with abundant pig pathology experience, revealed only equivocal differences.

Bacterial culture of abdominal fluid and filtering organs from affected piglets was either unproductive, or yielded primarily *E. coli*, with occasional isolates of *Streptococcus* spp. or other miscellaneous bacteria. Virus isolation was unsuccessful from spleen and stomach content of three piglets using baby hamster kidney and pig testes cell lines. Virus neutralisation tests for Japanese encephalitis were negative on abdominal/thoracic fluid from three affected piglets and two sows that had recent stillborn litters. These two sows were also negative in a MAT for antibodies to *Leptospira interrogans* serovars *hardjo*, *pomona* and *tarassov*i. Referral testing at AAHL included porcine parvovirus ELISA and HI testing on abdominal/thoracic fluid from four affected piglets, and Aujesky's disease latex agglutination, porcine reproductive and respiratory syndrome indirect ELISA, classical swine fever NPLA and Menangle VNT on sera from four sows, three of which had recently farrowed stillborn/weak litters. All of these tests were negative. Porcine circovirus immunoperoxidase testing for antibodies was also conducted and was positive. However, this was judged to be insignificant given that the features of this disease problem were not typical of porcine multisystemic wasting syndrome, and seropositivity to these viruses is high in Australia.

Our advice was that this was not likely an infectious disease problem. Based on the clues of a diet limited to white bread, the disease apparently manifesting exclusively as stillborn/weak piglets, some with substantial oedema of the anterior subcutaneous tissues and prominent thyroid glands, a tentative diagnosis of congenital hypothyroidism was made. Australia is generally considered iodine deficient, and soil in coastal areas in particular may be low in iodine due to leaching from heavy monsoon rains. The farmer was advised to add a multivitamin/mineral supplement to the sow diet. Instead, being unjustifiably confident of our diagnosis, he added only iodised salt. There have been no more stillbirths or weak piglets since. So impressed was the farmer of the effectiveness of this remedy that he also administered it to his wife and reports that she is feeling much better and is far less crabby.

QUEENSLAND

State correspondent: Brett Stone

Fatal nephrosis in a horse due to suspected brown snake envenomation.

Selina Ossedryver *Yeerongpilly Veterinary Laboratory, DPI&F*.

A 3 year old Australian Stockhorse gelding from Monto in the Burnett district was found sweating and in pain and was treated for severe colic and laminitis with anti-inflammatories and sedation. Initial treatment was relatively successful but the horse was reluctant to stand, with muscle tremor of the hind limbs when standing, and preferred to lie down. The animal died approximately 36 hours after clinical signs were first noted. No significant gross changes were noted at necropsy. The submitting veterinarian had noted a slight positive result on the CSL Venom Detection Kit and suspected brown snakebite.

Histopathology revealed widespread severe nephrosis. A significant proportion of renal cortical tubules were necrotic (figure 1) and there was massive protein accumulation in glomeruli and in dilated collecting tubules and ducts. Necrotic tubular debris was also seen in some dilated collecting ducts, and the kidney was generally congested. Significant rhabdomyolysis was not noted grossly or microscopically, although there was degeneration of scattered individual muscle fibres with infiltration by phagocytic macrophages in some skeletal muscle sections. I concluded that the severe nephrosis was the likely cause of death and was probably of toxic aetiology.

Renal cortex - necrotic cortical tubules and protein accumulation.

Envenomation by the common brown snake (*Pseudonaja textilis*) has been recorded as producing acute tubular necrosis and renal failure in humans in the absence of rhabdomyolysis, myoglobinuria or significant coagulopathy, thus implying a possible direct nephrotoxic effect of the venom (Acott, 1988). Other potential nephrotoxins include fluorine compounds (eg. superphosphate fertilizer), mercury, NSAIDs and nephrotoxic antimicrobials.

References

Acott, CJ. (1988) Acute renal failure after envenomation by the common brown snake; *Medical Journal of Australia*, 149: 709-710.

Pigeon Circovirus with possible Polyomavirus co-infection

Roger Kelly *IDEXX Labs, Brisbane*.

Fixed tissues were submitted from a juvenile pigeon, one of a succession of mortalities in a loft in Sydney. The bursa of Fabricius was very atrophic and follicle centres contained apoptotic debris and numerous fused, degenerate lymphocytes with dense basophilic intranuclear inclusions of the so-called “morula” type. These changes are quite consistent with those induced in lymphoid tissue by circovirus-2 in pigs with porcine multisystemic wasting syndrome, and have been recorded in outbreaks of pigeon circovirus infection. In the same bird, however, the liver sections showed widespread ballooning degeneration of hepatocyte nuclei, many of which contained faint eosinophilic inclusions (see figure).

Pigeon liver H&E: polyomavirus-type intranuclear inclusions

The spleen showed no convincing inclusions,

but its lymphoid elements were depleted. Many crypts in the small intestine were distended by small protozoal forms consistent with *Hexamita* sp., but there was little evidence of associated enteritis. There was mild peritonitis. Kidney was not submitted.

The changes in liver are more suggestive of those produced in this tissue by polyomavirus in psittacines and some other bird groups. Polyomavirus infection has not as yet been reported in pigeons, but if it were to occur in a pigeon, then one affected by circovirus would seem to be a good candidate, since immunosuppression seems to play an important role in virulent circovirus infections (including in pigeons).

Anthrax exclusion/*Myoporum sp*. poisoning in Hereford-cross steers

Jim Taylor *QDPIF Regional Veterinary Laboratory, Toowoomba*

Four 12 month-old steers died suddenly and another presented with hind limb ataxia, blood in the rectum and a temperature of 40.4 C. Necropsy of one of the dead steers revealed mottled liver; pale kidneys, and haemorrhages over the surfaces of the abomasum and omentum, endocardium and throughout the pleural cavity. Jaundice was also noted, along with inflamed, haemorrhagic intestines. Biochemistry on one animal revealed azotaemia, liver damage and elevated bilirubin and magnesium. Haematology revealed slight anaemia. The most significant histopathological changes were seen in the livers submitted from two animals: both showed changes consistent with Ellangowan poison-bush poisoning; *ie* moderate periportal hepatocellular necrosis and haemorrhage with perivascular haemorrhage around portal veins. Mild bile duct hyperplasia and moderate hyperplasia of the bile duct epithelium were also noted.

Fragments of *Poaceae*, possibly Family *Myoporaceae*, were found in the rumen contents. This was consistent with the animals having exposure to Ellangowan poison-bush (*Myoporum deserti*) overnight. Anthrax was ruled out following examination of a polychrome methylene blue-stained blood smear (Jim Taylor, TVL).

Leukaemia in a horse

Jim Taylor *QDPIF Regional Veterinary Laboratory, Toowoomba*

An Australian Stock Horse presented with chronic weight loss, ill thrift and mild pyrexia. Haematological examination revealed anaemia (Hb 9.1 g/dl, PCV 26%, RCC 4.98 x 10^{12}/l *(normal values Hb 11-19 g/dl; PCV 32-52 %; RCC 6.5-12 x 10^{12}/l)* and white cell count of 79.8 x 109/l *(normal 5.5-12 x 10^{9}/l)*. The leucocytosis was due to proliferation of a monomorphic population of medium to large lymphocytes, which comprised 94% of the differential count.

Congenital diaphragmatic hernia in a foal

Wendy Townsend *QDPIF Regional Veterinary Laboratory, Oonoomba, Townsville*

A newborn foal appeared lethargic and had very rapid respiration. Attempts to place the animal in sternal recumbency were unsuccessful and it was found dead a few hours later. Necropsy revealed a defect in the left side of the diaphragm that had allowed the abdominal viscera into the thoracic cavity. The defect appeared to be developmental as the left lung lobes were markedly smaller than normal.

Salmonellosis in layer chicks

Jim Taylor *QDPIF Regional Veterinary Laboratory, Toowoomba*

Nine hundred 12-day-old layer birds were found dead on a property holding 39,000 birds. Sixteen thousand layers were considered to be at risk. The submitter mentioned that "some chilling" might have occurred the week before and that that the birds had had access to sawdust when they were a day old. Thirteen chicks were submitted for post mortem examination. Ten of the twelve birds examined had little or no crop or gizzard contents. Five of the twelve birds had evidence of pericarditis or perihepatitis with occasional necrotic foci in the livers, and slight to moderate splenomegaly. Histological examination of heart, liver and spleen revealed diffuse subacute fibrinous pericarditis, mild multifocal necrotising hepatitis and moderate multifocal necrotising splenitis, with hyalinisation of periarteriolar lymphoid sheaths. Gram-negative bacterial emboli were present throughout the sections. A pure, heavy growth of *Salmonella typhimurium* was recovered from primary non-selective culture in two of the birds.

Suspect ergotism due to *Claviceps paspali*

John Gibson *QDPIF Regional Veterinary Laboratory, Toowoomba*

Ergotism due to *Claviceps paspali* was considered the likely cause of neurological signs in a mixed breed group of 110 weaners on a property on the NSW/QLD border. Ten to 15 weaners had clinical signs. The weaners were grazing paspalum infected with *Claviceps paspali*. The fungus was identified by DPI & F plant pathologist, Dr M Ryley. Biochemical and haematological results on 6 animals were unremarkable.

Cytological Diagnosis of Malignant Histiocytosis in a Rottweiler

Brett Stone *Veterinary Pathology, The University of Queensland*

An abdominal ultrasound performed on an 8 year-old spayed Rottweiler revealed the presence of multiple splenic masses and enlarged sublumbar lymph nodes. Ultrasound-guided fine needle aspirate biopsies were obtained from the splenic masses and from enlarged lymph nodes and high-quality smears were submitted for cytology. The smears from aspirates of both sites were of high cellularity. The nucleated cell population consisted predominantly of small mature lymphocytes; however, numerous very large round to ovoid cells with histiocytic appearance were scattered throughout the smears (see figure). These cells had distinct cell borders and a moderate to large amount of light blue (Diff-Quick), occasionally vacuolated cytoplasm. They had very large ovoid, frequently reniform, eccentrically-located purple nuclei with a homogenous to reticular chromatin pattern and prominent, frequently multiple nucleoli. Binucleate cells and mitoses were often present, with up to 3 mitoses per 400X field observed. These neoplastic cells demonstrated marked anisocytosis (ranging in size from approximately 4 to 20 times the size of a neutrophil), anisokaryosis and anisonucleoliosis. Bizarre nuclei, nucleoli and mitotic figures were also frequently present. As these histiocytes demonstrated multiple cytological criteria of malignancy[(1)] and were present within both the spleen and abdominal lymph nodes, a diagnosis of malignant histiocytosis was made.

Diff-Quik-stained smear from splenic mass in a Rottweiler (oil immersion)

Literature on the spectrum of histiocytic neoplasia can be confusing and differentiating these diseases may be difficult. The histiocytic proliferative disorders recognized in dogs include canine cutaneous histiocytoma, cutaneous histiocytosis, systemic histiocytosis, histiocytic sarcoma and malignant histiocytosis (MH). There is an excellent overview of these diseases on the UC Davis website (see link below). Histiocytic sarcoma and malignant histiocytosis follow a

rapid clinical progression despite therapeutic intervention and occur with high incidence in Bernese mountain dogs, Rottweilers, Flat Coat Retrievers and Golden Retrievers[(2)]. Histiocytic sarcomas occur as localized growths in spleen, lymph nodes, lung, bone marrow, skin and subcutis, brain, and periarticular tissue of large appendicular joints[(2)]. Histiocytic sarcomas can also occur as multiple foci in single organs (especially spleen), and rapidly disseminate to involve multiple organs. Disseminated histiocytic sarcoma is often difficult to distinguish from MH (multicentric origin), since disease progression is often advanced at the time of clinical signs/diagnosis. MH is a multi-system, rapidly progressive disease in which there is simultaneous involvement of multiple organs such as spleen, lymph nodes, lung, bone marrow, skin and subcutis.[(2)] The subclinical period is unknown when it occurs in cryptic sites such as spleen, lung or bone marrow.

References:

(1) Brown, D.E. *et al*., Cytology of Canine Malignant Histiocytosis. Vet Clin Pathol (1994).
(2) www.histiocytosis.ucdavis.edu

VICTORIA

State correspondent: Russell Graydon

Lupinosis

Julie Wayne *Gippsland Veterinary Pathology Service*

Samples were submitted from a sheep property located in western Victoria. A mob of 250 sheep which had been grazing lupin stubble had developed signs of photosensitisation and jaundice. A large proportion of the flock was affected and 8 animals had died. Blood was submitted for analysis and revealed a neutrophilia (15.0 x 10^9/L) and cholangiohepatopathy (GLDH 113 U/L, GGT 194 U/L, bilirubin 52 µmol/L). The submitting vet reported a jaundiced carcass and swollen orange liver at necropsy. Histopathology of the liver revealed diffuse marked anisokaryosis with chromatin clumping and prominent nucleoli, and there was moderate fatty change. Single cell necrosis of hepatocytes was seen as well as increased numbers of mitotic figures. Inflammation was mild, patchy and neutrophilic. There was mild biliary hyperplasia with occasional peribiliary fibrosis. Histological findings were compatible with lupinosis, which is a mycotoxic hepatopathy subsequent to ingestion of phomopsin. This toxin is produced by the fungus *Phomopsis leptostromiformis* which parasitises Lupinus plants. Phompsins A and B may be produced after the host plant dies and the stubble becomes moistened. An assay for phomopsin was not performed in this case due to financial considerations. The flock was removed from the lupin stubble and placed into a shaded area. Two further deaths were recorded.

Copper Deficiency and Chronic Arthritis in Red Deer

Mike Forsyth *PIRVic Attwood*

A practitioner submitted two 3 to 4 month-old red deer for examination. The herd had 30% of the fawn drop affected by chronic arthritis and poor coat condition. The arthritis was apparent soon after birth and had not resolved with treatment. Necropsy revealed the carpi and tarsi to have greatly thickened joint capsules. The joints contained straw-coloured fluid in one case and blood stained fluid in the second. The articular surfaces were pitted. Other joints examined did not appear to be affected. The hair coat was unkempt and appeared faded in colour. Bacteriology on joint samples did not reveal any bacteria including mycoplasma. Histopathology of the joint capsule revealed thick fibrous tissue and the synovial membrane showed hypertophy and

proliferation of the epithelium. What was surprising was the relative lack of acute or chronic inflammatory cells to suggest that an infection had been involved.

In the history of the case it was mentioned that the normal annual copper supplementation had not been given to the herd. Liver copper analyses from the calves showed them to be markedly deficient. Liver vitamin B_{12} and glutathione peroxidase levels were normal. Copper is important in the development of the matrix in which the bone is laid down and it is suggested that this joint defect occurred because of a congenital deficiency of copper.

Acceleration of *Pasteurella*-associated mortalities in free range chickens
Mike Forsyth and Banydhuro Oyay *PIRVic Attwood*

The farmer had noticed a nagging level of mortality in the previous laying season. Mortalities accelerated with the onset of hot weather when some birds were on the point of lay for the new season. In two days, he lost 50 birds in one flock and 4 birds in a second, out of seven multi-aged flocks of about 500 birds each.

Post mortem examination of six birds revealed mucoid exudates in the respiratory passages, white and yellow spots in congested livers, enlarged spleens and some abdominal haemorrhages. Three out of 3 birds had heavy growth of *Pasteurella multocida* from livers and spleen. *Mycoplasma* sp were also cultured. Acute focal hepatitis and interstitial pneumonia were observed histologically.

The owner was advised to make sure he diligently picked up carcasses that would have been a source of infection and to seek advice about treatment. Two months later and still with a mortality problem, he submitted more birds from which *Pasteurella multocida* was again cultured.

His mortality has dropped after 3 months but he has lost about 1800 birds this season. The value of this in terms of lost production could be as high as $100,000 (Greg Parkinson).

Cerebellar abiotrophy in rabbits
Russell Graydon *Pirvic Attwood*

A syndrome of progressive ataxia leading to complete loss of coordination and a requirement for euthanasia was investigated in a colony of laboratory rabbits. T en of a group of 160 were affected. Two rabbits were necropsied and no gross lesions were seen. On histopathology there was necrosis and loss of Purkinje cells and swelling of the proximal axons in the cerebellum. Marked wallerian degeneration of white matter tracts was a feature in the spinal cord. Based on the histopathology and history, a diagnosis of cerebellar cortical abiotrophy was made. This is thought to have a genetic basis in other species and is probably the case here as this rabbit colony had been closed for more than 80 years.

SOUTH AUSTRALIA
State Correspondent: Stephen Yeomans

(no report)

TASMANIA
State Correspondent Stephen Pyecroft

(no report)

U.S.A.

Correspondent: Paco Uzal

(no report)