VETERINARY PATHOLOGY REPORT

Number 62 December 2001

President's Report

Since the AGM in Melbourne in May, several issues have received the attention of the Executive. The Histopathology Quality Assurance Programme organised by Tony Ross is well underway, with the second of the periodic submissions having been circulated and returned for examination. This is a valuable exercise, which is becoming more important with the increasing emphasis on competence in the professions.

Problems facing the community with the general ageing of rural veterinarians and pathologists and lack of younger replacements, has received publicity in the AVJ and has reached Government level, even becoming a topic for discussion during the last Federal election. This topic needs to be addressed by our membership.

Roger Kelly presented his Continuing Education course on a range of emerging diseases, with particular emphasis on viral and other neurological conditions. This was a valuable update for us and Roger is to be congratulated on his hard work in organising such a diverse group as we are for such attendance.

A vacancy on the Management Committee of the National Registry of Domestic Animal Pathology was filled with Dr Karrie Rose, of the Taronga Zoo. Karrie is well qualified for this, having been involved with a similar registry at the Zoo.

Communication among members is a critical part of any organisation. In today's world, the 'Net is rapidly replacing "snail mail" and other forms of communication. Your Executive is working on a Website, which we hope will lead to improvement in this area. The possibility of an electronic version of the Veterinary Pathology Report, which will markedly decrease labour and postal costs, is being examined.

Another issue which will be brought to the membership for the next AGM, is a proposal to consider a bid for the International Symposium of Veterinary Laboratory Diagnosticians conference to be held in Melbourne in 2005. This is an exciting development and one which, we feel, should be carefully considered by the membership.

Work is well underway on the organisation of the 2002 conference, to be held in Adelaide just prior to the AVA conference. The theme will be "Forensic Pathology", a topic increasingly important to everyone involved in diagnostic pathology in these days of increasing litigation. We are fortunate to have a strong Forensic Science group here in Adelaide, with two top persons accepting the role of keynote speakers at the sessions.

These, and many other issues, appear likely to keep us all busy in 2002. Best wishes to all for the Christmas season and the New Year. I look forward to seeing you in Adelaide in May.

Ruth Reuter
President, ASVP
December 2001.

Editorial/Secretary's Report

Veterinary Pathology Training in Australia

There has recently been some discussion about appropriate levels of training for veterinary pathologists in Australia as part of a wider AVA push to increase the commitment of government to veterinary education. This issue is pertinent for several compelling reasons: (1) the current age profile of veterinary pathologists; (2) the funding squeeze on veterinary schools and demise of many government veterinary laboratories, both traditional training mechanisms; and (3) the lure of higher salaries and greater opportunities in the United States.

It is incumbent upon the many of us who have enjoyed satisfying careers in veterinary pathology to ensure that similar opportunities are available to new graduates. The well-being of the livestock industries in this country, in particular, also demands that sufficient experienced veterinary pathologists are available.

As the professional body established to promote the practice of veterinary pathology in Australia, the ASVP should adopt a stance on pathology training after discussion at the next conference in Adelaide and, if deemed appropriate by members, be prepared to lobby for the required changes.

Mandy O'Hara, Dip ACVP

The ASVP congratulates Dr Mandy O'Hara, Pathology Registrar, Murdoch University on her success at the ACVP board examinations in September. When asked what she intended to do with her life post-boards, Mandy replied: "I intend to spend it drinking champagne!"

John Finnie
Honorary Secretary

The Australian Society for Veterinary Pathology

2002 Annual Conference

Robson Theatre, Institute of Medical and Veterinary Science
Frome Road, Adelaide SA 5001

May 4-5, 2002

Conference Theme: Forensic pathology Agenda items: Members wishing to bring any business before this meeting should e-mail the Secretary at john.finnie@imvs.sa.gov.au

ASVP executive: South Australia's term expires next year so start thinking about the succession.

Tony Ross
Convenor tony.ross@agric.nsw.gov.au

A Nasty New Worm

Tony Ross at the Regional Veterinary Laboratory at Menangle NSW believes a nasty new worm may have been imported in alpacas and llamas from South America. Lamanema chavezi is a pathogenic helminth of llamas and alpacas in Peru and Chile. It has recently been reported for the first time in Argentina (Cafrune et al 2001).

It is an nusual trichostrongyloid nematode as its 3rd and 4th larval stages migrate from the gut to the liver (and sometimes elsewhere). Affected livers show haemorrhagic and necrotic migration tracts which can result in small abscesses throughout the liver. In Peru 12-19% of livers in alpacas and llamas are condemned at abattoirs due to this parasite.

More importantly it can cause liver malfunction, anaemia, anorexia, emaciation, prostration and death.

Tony asks veterinary practitioners and owners to keep an eye out for lesions in the liver (and occasionally the heart and other organs) which may be caused by L. chavezi. The abscesses and tracts can be quite florid. In endemic flocks they mainly infect immature weaned animals. However, in naive populations, weaned animals of all ages could become clinically ill.

Lesions should be submitted to state government veterinary laboratories as a suspect exotic disease.

Investigation strategies could include: formalin fixed lesions, faecal egg counts (the eggs are large and embryonated) and total worm counts of C3 and the small intestine.

A 17 month old alpaca recently died in NSW with abscesses in the liver and heart. Formalin fixed samples were submitted for histopathology. Its mother which was imported from South America died with similar pathology - but no laboratory work-up occurred. Liver abscesses in the younger animal contained viable helminths - which were not liver fluke or tapeworms or common ascarids. A presumptive diagnosis of L.. chavezi infection was made. Further investigations are underway. For more information contact Tony Ross phone 02 4640 6312, fax 02 4640 6400 or email tony.ross@agric.nsw.gov.au.

Reference

Cafrune, M.M et al (2001) *First report of Lamanema chavezi in llamas from Argentina*. Veterinary Parasitology 97 (2): 165-168.

STATE REPORTS

NSW – Paul Gill

GASTRIC ULCERS IN ALPACA

Steven Hum, RVL Camden

A 7-year-old alpaca became lethargic and depressed after shearing and died a few days later. On post mortem examination the body and internal organs appeared to be pale and the blood was thin. There were ulcerations in the true stomach (C3) and the intestines contained blood stained ingesta. Histological examination of the true stomach revealed severe, diffuse vascular congestion with areas of mucosal haemorrhages in the cardiac region and infiltration of lamina propria by mixed inflammatory cells. Unfortunately tissues from ulcerations seen grossly were not included in the section. The renal medulla revealed multifocal, interstitial accumulations of haematopoietic cells and there were large numbers of megakaryocytes in the spleen. Sections of liver revealed a severe, periacinar, acute, coagulative hepatocellular necrosis and there were large numbers of haematopoietic cells throughout.

The pathogenesis is consistent with chronic gastric ulcerations, periodic haemorrhages with subsequent chronic anaemia. The chronicity of anaemia is evidenced by extramedullary haematopoiesis in the spleen, kidney, liver and the severity is indicated by the extent of hepatocellular necrosis centred on the central vein.

Gastric ulceration is a common disorder in lamoids.

Reference: ME Fowler: *Medicine and Surgery of the South American Camelids*, 1989, Iowa State University Press.

THIAMINE DEFICIENCY IN A CAT

Alan Kessel, University of Sydney, Camden
Email : akessell@camden.usyd.edu.au

Animal: feline, DSH, male neutered, 9 years, "Ned"

Relevant History: The animal is an indoor cat and has a history of 1 week of weakness before referral to specialist clinic. The animal had had elevated bilirubin, ALP and amylase and had failed to respond to treatment for pancreatitis. The animal developed central vestibular/cerebellar signs and deteriorated neurologically to become very depressed despite treatment. The cat was euthanasia and forwarded to the UVCC for post mortem examination.

Please Note
Toxoplasma titre >2560
Cryptococcus titre on CSF = negative
CSF normal
Serum cholinesterase level normal

Post Mortem Findings:
The animal is in good body condition and had been chilled and thus there is only mild post mortem autolysis.
Abdominal Cavity: the liver is markedly jaundiced, as are many body tissues including kidney etc. The bile duct is patent. The stomach and GIT are empty.
Thoracic Cavity: the left side and the caudal half of the right side of the lungs are moderately mottled and congested.
Brain: there are scattered areas of superficial congestion over the cerebral cortices.

Histological findings: CNS: listed below are changes noted at various levels of the CNS, starting at the obex (medulla):
1) Obex: there is moderate margination of neutrophils inside meningeal vessels and a small number of extravascular neutrophils in the meninges over the cerebellum and brain stem. In the brain stem itself there are a small number of degenerate neurones in grey matter around the central canal as well as scattered small haemorrhages.
2) Cerebellum: there are large numbers of necrotic hypereosinophilic Purkinje cells in the nodulus and vermis, with lesser numbers elsewhere in the cerebellum. Often the Purkinje cells are totally absent and have left small holes. As well there is a hypercellularity associated with the Purkinje cell layer that consists of macrophages and glial cells; small numbers of mitotic figures are seen. The severity of change is greatest at the base of cerebellum and attenuated dorsally. There are also small numbers of necrotic hypereosinophilic neurones in the molecular layer. Accompanying these changes are mild but diffuse spongy change in the white matter tracts of the cerebellum as well as a more focal area of spongy change in the lateral part of the caudal cerebellar peduncle.
3) Brainstem level of vestibular nuclei: there are multifocal areas of haemorrhage and spongy change in the white matter tracts. However the major change is centred bilaterally on some of the nuclei found at this level primarily the vestibular and rostral olivary nuclei. These changes consist of a hypervascularity with hypertrophy and hyperplasia of the endothelial lining of capillaries, mild to moderate numbers of spheroids, a patchy necrosis of neuropil, scattered necrotic neurones, scattered but prominent haemorrhages, and an increase in glial cells especially macrophages. The changes are bilateral but do not appear to affect the motor nerve of the trigeminal nerve.
4) Caudal Colliculi: in the three large nuclei here - the central oculomotor

nucleus and the ventrolateral red nuclei - there are similar changes as those described in the vestibular nuclei.
5) Thalamus: there are similar changes in the thalamic nuclei, but perhaps they are more severe and widespread. There are small deposits of what ap pear to be haemosiderin inside macrophages as well as fresh haemorrhage and an increased number of spheroids over more caudal nuclei. As well there are small perivascular cuffs, 1-2 cell thick, around scattered vessels here that consist of slightly enlarged lymphocytes.
6) Cerebrum: the changes are widespread and are in all 4 sections examined. They consist of segmental hypervascularity in areas where there is moderate necrosis of individual neurones at various levels in the cerebral cortex.. As well there are small scattered haemorrhages and perhaps an increase in sponginess of neuropil here.

Thyroid: there are several variably sized cystic follicles scattered throughout the parenchyma of the thyroid. Also in the fascia near one thyroid is an approximately 2 mm multiloculated cavity full of eosinophilic fluid and discontinuously lined by ciliated columnar cells that have basal nuclei and are in single layer (this represents an embryonic remnant).
Pancreas: mild fat infiltration
Kidney: mild autolysis
Liver: most hepatocytes are markedly distended with a single vacuole of fat, although this change is less severe in hepatocytes around the central veins where hepatocytes do however contain small to moderate amounts of bile intra-cytoplasmically.
Heart, GIT, bladder, spleen: NAD

In summary, the relevant findings at post mortem examination were:

The CNS changes are pathognomonic for thiamine deficiency in the cat. The changes are most prominent in the periventricular grey matter and also neurones in the cerebellum and cerebrum. The types of changes - hypervascularity (which is really later event in response to earlier necrosis), neuronal necrosis and scattered haemorrhages along with spheroid formation- are classically described. Thiamine deficiency has been studied experimentally in dogs and can develop as quickly as 30-40 days, but these were dogs on thiamine sufficient diets before the study. The reasons we may have thiamine deficiency here are:

1) The cat was on a thiamine deficient diet before the illness eg fresh fish, cooked food, meat preserved with sulphur dioxide (very common in the pet food industry)
2) The cat was hovering on the edge of thiamine deficiency, became anorexic, and hepatic lipidosis intervened to make the cat completely anorexic
3) The cat actually had hepatic lipidosis and became anorexic for a period of time.

The factors worth examining here are the diet of the cat, the length of time the cat was off its food, and the possibility that the cat was suffering from hepatic lipidosis. The liver was certainly consistent with hepatic lipidosis, and the anorexia this causes can certainly predispose to thiamine deficiency. Thiamine is a water soluble vitamin and is an essential element in the diet of carnivores - normally it would not be deficient in a normal carnivore diet but under certain circumstances eg consumption of thiaminases with raw fish, sulphur containing foods that destroy thiamine, prolonged anorexia- thiamine status can be affected. Thiamine is an essential co-factor in the TCA cycle as well as the pentose pathway and as the brain is largely reliant on glucose for energy requirements thiamine deficiency will result in an energy lack necrosis in those cells most sensitive to this ie large

neurones. The distribution of change varies between species eg ruminants are more likely to have changes in the cerebral cortices, carnivores will have change around the central ventricular areas.

NT – Anton Janmaat

Did *Actinomyces canis* cause nocardial-like peritonitis in a dog.

Anton Janmaat and Suresh Benedict (bacteriologist), Berrimah Veterinary Laboratories (BVL), NT Department of Business, Industry and Resource Development

A male Boxer was presented for post-mortem examination after dying unexpectedly.

Opening of the abdominal cavity revealed excess (about 2L) dirty brown fluid with numerous small white granules. The parietal peritoneum and mesentery were reddened/haemorrhagic. The omentum was a reddened/haemorrhagic "knobbly" mass, due to adhesions, with some greyish pus-filled spaces. The gastro-intestinal tract was empty and seemed intact.

Histological examination showed a layer of granulomatous inflammation on all peritoneal surfaces examined. The omentum showed pyogranulomatous inflammation, often with microcolonies surrounded by neutrophils as in nocardial peritonitis (see JK&P, fig 4.8B, p438, vol II), focal necrosis and haemorrhages usually surrounded by palisading macrophages. The extent of fibroplasia suggested that the inflammatory process had been going on for several days. The white granules consisted of one or more radiating filamentous arrays which stained poorly in the Gram stain but looked like filamentous bacteria. Scattered colonies of tangled, branching, filamentous bacteria which at times stained Gram positive, were always present and appeared to increase in number towards the periphery of the granules.

Bacteriological culture of an omental swab yielded a heavy growth of Fusobacterium sp, a moderate growth of Actinomyces canis and a light growth of E. coli. The Actinomyces canis was identified in the biolog system. It is a new species first described by Hoyles et al (2000). So far we have only had access to the (online) extract of the paper.

Reference: Hoyles L, Falsen E, Foster G, Pascual C, Greko C and Collins MD. *Actinomyces canis* sp nov., isolated from dogs. *International Journal of Systematic and Evolutionary Microbiology 2000;50:1547-1551*.

Feather follicle mites in a red-collared lorikeet

Anton Janmaat and Lois Small (parasitologist), Berrimah Veterinary Laboratories (BVL), NT Department of Business, Industry and Resource Development

A feather specimen from a red-collared lorikeet (*Trichoglossus haematodus rubritorquis*) from the Territory Wildlife Park was submitted as a feather cyst with a request for virological culture. This was more expensive than expected and the submission was discontinued except that the specimen receptionist convinced me to look at the little bulge at the bottom of the feathers for "our own interest".

Histological examination revealed distended feather follicles filled with numerous parasites, presumably various stages of the life cycle of a mite. KOH digestion, in parasitology, of the formalin fixed sample freed large numbers of adult and larval mites. These were identified as the prostigmatid mite, *Harpirhyncus rosellanicus*. Harpyrhynchid mites have been reported in a scaly-breasted lorikeet (*Trichoglossus chlorolepidotus*) and the same group has been reported in feather follicles and as causing skin tumours.

QLD – Bruce Hill

Mycoplasmosis in goats

Anita Gordon and Greg Storie
Yeerongpilly Veterinary Laboratory

Two properties in SE Qld reported sickness and mortalities in Anglo-Nubian goat kids during September 2001. Clinical signs included transient fever, harsh respiratory sounds, joint swelling, paresis and recumbency. Two dead kids were presented for necropsy. One had severe, subacute, diffuse interstitial pneumonia, and mild non-suppurative meningoencephalitis. The second had severe, suppurative polyarthritis, together with mild to moderate, acute, patchy interstitial pneumonia, and mild subacute meningitis. *Mycoplasma capricolum* was isolated from the lung of kid 1 and the carpus and hock of kid 2. No other bacterial pathogens were isolated. Kid 2 had been purchased at one week of age from kid 1's property. This property had problems with mycoplasmosis last year as well.

Orbivirus infection in a Tammar wallaby

Anita Gordon
Yeerongpilly Veterinary Laboratory

A two-year-old female Tammar wallaby from the Animal Research Institute colony as found dead. Apart from mild generalised congestion, and a few scattered subcutaneous ecchymoses, the necropsy picture was unremarkable. Histological changes in tissues comprised congestion and haemorrhage, most severe in the liver, where there was acute necrosis of occasional acini. Tissues forwarded to EMAI for viral culture yielded orbivirus, similar to previous isolates from cases of "Tammar Wallaby Sudden Death Syndrome".

Neonatal listeriosis in a goat

Anita Gordon
Yeerongpilly Veterinary Laboratory

A four-day-old Boer cross goat kid died in July 2001, after a short illness. Necropsy revealed severe fibrinous peritonitis. The liver was at least twice normal size, swollen and beset with miliary pale foci. Histological examination revealed multifocal necrosis and inflammation in liver, hepatic lymph node, heart, kidney, spleen and thymus, and masses of small Gram-positive bacilli were present within microabscesses in these tissues. Mild to moderate, patchy, non-suppurative meningoencephalitis was also present, most severe in the midbrain. *Listeria monocytogenes* was isolated in pure culture from the liver, heart and peritoneum.

Gossypolpoisoning in feedlot lambs

John Gibson
Toowoomba Veterinary Laboratory

Gossypol poisoning was diagnosed as causing the death of 60 ram lambs on a property at Blackall. The lambs were from a group of 420 drafted into a feedlot pen from a consignment of 1000 ram lambs. The 1000 ram lambs were 10 to 14 weeks-of-age and recently weaned when they arrived on the property. The feedlot lambs were fed lucerne hay until 9 days-on-feed (DOF) when cottonseed meal (CSM) was introduced at 140 grams/head/day. At 22 DOF whole cottonseed (WCS) was included in a ration. The ration containing 35% WCS, 13% CSM and 52% hay was fed at 730 grams/head/day. The ration was not premixed; components were placed separately into troughs, allowing some lambs to selectively feed on the cottonseed components. This was more noticeable after barley hay was substituted for lucerne hay. The first deaths occurred at 38 DOF and by 57 DOF when the cottonseed components were withdrawn from the ration, 27 lambs had died and by 64 DOF 60 lambs had died.

Initially, few clinical signs were seen, apparently healthy lambs were found dead in the pen. This was often after a period of excitement such as feeding. Affected lambs that were seen were recumbent and panting rapidly. They invariably died soon after. Lambs dying after the cottonseed components were withdrawn had clinical signs of lethargy, anorexia, poor exercise tolerance, dyspnoea and submandibular oedema.

Gross pathological changes included severe serosanguinous ascites and hydrothorax, haemoglobinuria and mottled livers. Consistent histological changes included severe periacinar necrosis of the liver and pulmonary congestion and oedema. Less frequently there was myocardial oedema, congestion of the spleen and occasional haemoglobin casts in renal tubules.

The CSM and WCS contained 840 ppm and 7650ppm free gossypol respectively. This equates to a free gossypol concentration in the ration of 2972 mg/kg. Selective consumption of the cottonseed components, especially the WCS, would greatly vary the dose of free gossypol per lamb. Levels of free gossypol from 100 to 220 mg/kg in the diet have been reported toxic for preruminant calves (1) and 8 week-old feeder lambs dosed with gossypol at 409mg/day all died within a 30 day trial period (2).

Ref:

(1) AVJ (1995) 72: 10 pp 394-398
(2) Am J Vet Res (1988) 49: 4 pp 493-499.

Botulism in commercial broiler poultry

Ross McKenzie
Yeerongpilly Veterinary Laboratory

Intoxication with botulinum toxin type C was diagnosed in a commercial broiler poultry flock at Capalaba, Brisbane, in mid-October 2001. Deaths were largely confined to one shed with 2 others being marginally involved. The total number of deaths was 14,000 birds over a 20-day period. Sheds initially contained 24,000 birds each. Affected birds became recumbent and died. Typical clinical signs of ascending flaccid paralysis were not seen in live birds submitted for laboratory examination. These had "leg weakness", but continued to hold their heads erect. They were dehydrated, and during the initial investigation, had diarrhoea. No

significant abnormalities were seen at necropsy. Alimentary tract contents were scanty in most birds. Histopathology revealed no significant lesion, thus profoundly reducing the likelihood of significant infectious diseases and a number of intoxications as likely diagnoses. Botulinum toxin was detected in serum and alimentary tract contents by mouse inoculation, and in alimentary tract contents and litter from the affected shed by ELISA for types C&D. Tests for botulinum toxin and *Clostridium botulinum* organisms on feed and feed components were consistently negative. Initially, only serum was submitted for testing by ELISA for botulinum toxin. These results were negative. Alimentary tract contents were first submitted for testing 10 days later and were positive in the ELISA. At or about this time, mouse inoculation tests on serum were positive for botulinum toxin and mouse protection tests confirmed type C botulism. The large death toll prompted quarantine and elimination of infectious diseases (Newcastle disease, avian influenza, and severe bursal disease) as an urgent early response. Laboratory testing at AAHL, Geelong, ruled out exotic virus infections. Histopathology did not support infections as a credible cause of the incident. Testing for ionophore coccidiostat overdose in feed, insecticide residues in organs and cholinesterase activity in sera did not support these possible agents as credible alternative or additional diagnoses.

Erysipelas in free range layers
John Gibson
Toowoomba Veterinary Laboratory

Avian influenza was excluded as the cause of increased mortalities, depression and facial swelling in a flock of 6000 free range layers. Mortalities had increased to 40 birds per day. Eight birds were examined. Grossly five of the eight had oedematous swelling of the face and combs, four had a suppurative peritonitis and one had splenomegaly and hepatomegaly with miliary foci in the liver. Microscopically, the most consistent lesion was an acute cellulitis characterised by oedema, heterophil infiltration and perivascular lymphocyte cuffing. Peritonitis and acute multifocal hepatitis were also present. Gram stained sections demonstrated large numbers of Gram-positive bacilli in the liver, heart, skin and peritoneum with fewer in the spleen. *Eyrsipelothrix rhusiopathiae* was recovered in pure growth from birds cultured. Samples sent to AAHl Geelong were negative for Avian Influenza virus and Erysipelas was diagnosed.

SA – Ruth Reuter

Microsporidiosis in Princess Parrots (Polytelis alexandrae)
Peter Phillips, IDEXX Laboratories, 33 Flemington Street
GLENSIDE SA 5065

Separate submissions of fixed tissues from young adult princess parrots were received from a local zoo. The submitting veterinarian's history of the case was that the birds were emaciated and one had been treated for a gastric yeast infection and died of pulmonary oedema, while the other had megabacteria in a faecal smear, although this was suspected to be secondary to another underlying disease. The second bird had been treated for chlamydiosis two months previously and suspected to have circovirus.

Histopathology revealed a huge microsporidian infection in the lamina propria of all sections of small intestine from both birds. In one bird there were also multiple

granulomas associated with microsporidian infection throughout the liver. It also had a heavy megabacterial infection of the surface of the cuticular layer of the gizzard with no associated inflammation.

Microsporidiosis with concurrent megabacteriosis has been described in psittacine birds including peach-faced, masked and Fischer's lovebirds, budgerigars, an Amazon parrot and eclectus parrots. Ultrastructurally the microsporidia in two of the cases at least was identified as being consistent with *Encephalitozoon* species. PCR and Southern blot analysis identified the organism in those cases as *Encephalitozoon hellem*, which causes keratoconjunctivitis and respiratory infection in humans with acquired immunodeficiency syndrome.

Reference: Black SS, Steinohrt LA, Bertucci DC, Rogers LB & Didier ES. 1997. *Encephalitozoon hellem in budgerigars (Melopsittacus undulatus),* Vet Path 34(3):189-198

Pulparampil N, Graham D, Phalen D & Snowden K. 1998. *Encephalitozoon hellem in two Eclectus parrots (Eclectus roratus): Identification from archival tissues.* J Eukaryotic Microbiol 45(6):651-655

Coccidiosis in Black Eared Miners (*Manorina melanotis*).

Peter Phillips, IDEXX Laboratories, 33 Flemington Street
GLENSIDE SA 5065

Tissues from juvenile black-eared miners from a local zoo were submitted on separate occasions. This species has been difficult to breed and maintain in captivity. Gross pathology mentioned by the submitting veterinarian included a mildly enlarged spleen, inflammation of the duodenum, petechial haemorrhage of the proventriculus mucosa and reddish-brown intestinal contents.

Histopathology revealed widespread coccidian organisms within the mucosal epithelial layer of the small intestines, with local foci of necrosis and distension of small intestinal crypts and glands containing cellular debris, sometimes associated with large numbers of coliform bacteria.

Faeces were negative for chlamydial antigen. Culture of the spleen produced a light growth of *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*.

Previous findings have included rickets, aspergillosis and coccidiosis. It is speculated that captivity stress may reduce immuno-competency in these birds.

Rat respiratory disease

John Finnie, Veterinary Services Division, IMVS

A number of outbred Sprague-Dawley rats from a research facility died after 2 hours under anaesthesia; some showed minor stertor prior to use.

At necropsy, the lungs were firm and uniformly greyish in colour. Fresh and formalin-fixed lung was submitted. Microscopically, there was a diffuse interstitial pneumonia characterised by mononuclear cell (lymphocytes, plasma cells, macrophages) infiltration of alveolar interstitium, with a few polymorphonuclear leucocytes present in some areas; inflammatory cells were uncommon in alveolar spaces and airways. There was prominent perivascular lymphocytic aggregation, with fewer plasma cells, and peribronchiolar lymphoid hyperplasia.

Routine bacteriology on submitted lung was negative. PCR performed by the IMVS Murine Virus Monitoring Service (MVMS) for mycoplasmas (which detects all, not just rat, mycoplasmas) and Sendai virus was also negative. Serology for Sendai virus and *Mycoplasma pulmonis* was negative, but positive for pneumonia virus of mice (PVM). The latter was probably the aetiological agent, although these other pathogens produce similar morphological changes in rat lungs.

TAS – Philip Ladds

David Taylor, who has been at this laboratory for about four years, recently resigned from the DPIWE to take up a Residency position in Veterinary Pathology at the University of Florida. David expects to be overseas for about two years. David's position has been filled temporarily by Christina Mc Cowan. Christina has taken a several month break from her PhD studies at the University of Melbourne to help out at Mt. Pleasant.

Interesting cases were as follows:

Bovine.

Over a period of one week, Polioencephalmalacia occurred in a mob of 150 calves ~4 months-of-age at pasture. Two died and 6 were ill. Signs included stiffness, depression, opisthotonus, diarrhoea, dehydration, and a subsequent comatose appearance with apparent blindness then lateral recumbency. Treatment with thiamine and tetracyclines gave some improvement but not complete recovery. One recumbent calf was submitted alive for euthanasia and necropsy. Rectal temperature was 37oC; the calf was comatose and unable/unwilling to rise.

At necropsy there was an apparent excess of (clear) CSF noticed when the head was removed. The urinary bladder was very distended with dark clear urine, and multiple petechiae and ecchymoses were present in one area of the transitional epithelium.

No significant changes were seen histologically in sections of liver, kidney, spleen, lung, heart, adrenal, pituitary, lymph node or gut at various levels. Sub-epithelial haemorrhage in the urinary bladder, unassociated with inflammatory infiltration, was confirmed. In the brain, no significant changes were seen in the brain stem but in the cerebral cortex there was a marked vacuolated appearance involving the cortical lamina with neuronal degeneration, oedema and prominent infiltration beneath the leptomeninges and around some blood vessels of macrophages. Sections of cerebrum fluoresced under UV light. Polioencephalomalacia, moderate, multi-focal, was diagnosed.

Complete recovery of calves with this degree of brain damage would not be expected, regardless of treatment administered.

Two cases of arthrogryposis in calves born to cows at Smithton were attributed to an hereditary defect arising from use of semen from a bull in Victoria. Previous offspring from this bull were arthrogrypotic so tissues from a Smithton calf were collected and sent to VIAS for continued collaborative investigation.

Other interesting single cases included Globus amorphous, and mycotic abortion with severe foetal meningo-encephalitis.

Ovine. Hypocalcaemia was considered the cause of death of death of 8 near-term ewes in a mob of ~1000 that were in excellent body condition and on good feed. Calcium level in aqueous humor was 0.5 mmol/L (normal 1.3-1.73 mmol/L).
Several outbreaks of abortion caused by *Campylobacter jejuni* were diagnosed; suggestive hepatic gross lesions were seen in only several of the 10 lambs necropsied.
Single cases of toxoplasma abortion, and pituitary abscess associated with meningo-encephalitis, were seen in sheep from other flocks.

Caprine. *Yersinia enterocolitica* infection in a doe with septicaemia leading to foetal infection and abortion was diagnosed; there was a history of diarrhoea and abortion in other does.
Necropsy of one of two aborted kids from another herd revealed marked hyperplastic goitre, the precise cause of which, was not ascertained.

Avian. Ionophore toxicity, apparently due to inappropriate mixing, was the cause of death of a number of 18 week-old turkeys. Extensive rhabdomyolysis was suspected grossly and confirmed histologically.
In another flock of pullets with a problem of soft-shelled eggs and ceasing to lay, there was focal ulcerative enteritis, and *Erysipelothrix rhusiopathiae* was isolated.

Wildlife. Diffuse serositis in a magpie (*Gymnorrhina tibicen*) was associated with abdominal filariasis, and concurrent infection with *Brevibacterium sp*.

Staphylococcus sp. was isolated from a crow (*Corvus sp*.) with pyogranulomatous encephalitis.

Culture of tissues of one of four Eastern Rosellas (*Platycerus exemius*) that died, indicated *Yersinia pseudotuberculosis* as the cause.

Enormous numbers of presumed *Toxoplasma gondii* were associated with severe pneumonia and encephalitis in an Eastern Barred bandicoot (*Perameles gunnii*) submitted from the wild.

VICTORIA – Malcolm Lancaster

NO REPORT

WA – David Forshaw

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

Porcine Erysipelas.

An SPF herd reported deaths in four; eight-month-old-gilts with clinical signs 3-4 days prior to death of pyrexia, lethargy and anorexia. There were no skin lesions, respiratory symptoms or scours. A necropsy performed on a 3 month pregnant

gilt that had died showed red-purple discoloration of the lungs and excessive blood-tinged pleural and pericardial fluid. Histologically the lung showed acute haemorrhagic pneumonia with myriad colonies of gram positive rods. *Erysipelothrix rhusiopathiae* was isolated in pure culture.

In another outbreak on another piggery, there were 20 deaths in a group of 80, six-week-old weaners with clinical history of post weaning illthrift, respiratory distress and sudden deaths. Two affected animals were euthanased; both had infarcted spleens and cranio-ventral pneumonia and one was also jaundiced. Histologically there was a severe suppurative broncho-pneumonia and numerous sub-acute splenic infarcts of some duration (there is surrounding fibroplasia). There were not as many organisms in sections as in the previous case, *E. rhusiopathiae* was isolated in pure culture.

Photosensitivity associated with grazing Biserrula legume pasture

The spring season this year has seen widespread reports of photosensitization in sheep, particularly in those flocks grazing the legume Biserrula, but also on sub-clovers, ryegrass and capeweed dominant pastures. In one group of 800 weaners introduced to a Biserrula pasture, clinical signs of photosensitization developed within 24 hours. Blood samples from affected animals showed elevated unconjugated bilirubin and no conjugated bilirubin. There was no elevation of GGT or GLDH. Histologically, skin sections show superficial epidermal and full thickness necrosis typical of photosensitization.

Plant researchers postulate the increased prevalence of photosensitization is due to seasonal factors that have "stressed" pasture species, particularly the legumes. At the time of the outbreaks, Biserrula was intensely green, having very high concentrations of chlorophyll. phylloerythrin overload is the likely cause of the photosensitization.

Hypocalcaemia associated with oxalate containing *Chenopodium sp*.

Tetanic-like convulsions with collapse and death of 40 ewes in a flock of 300 ewes was associated with grazing *Chenopodium sp* dominant pastures. There was pulmonary oedema seen at necropsy and clinical chemistry revealed marked hypocalcaemia. Histologically there were small numbers of oxalate crystals within renal tubular lumens. Tubular epithelial cells appeared unremarkable.

Acute oxalate toxicity is uncommon and usually associated with hungry sheep being given access to oxalate containing pastures. In Western Australia, chronic oxalate nephrosis is more typical.

Bovine Intestinal fibrinoid vasculitis.

Histological examination of necropsy specimens collected on-board ship from cattle (ex Victoria and South Australia) during export to the Middle East, showed 8/105 animals affected by a circumferential fibrinoid change in the media of medium sized submucosal vessels of the jejunum and ileum. In some there is adjacent oedema but in all the mucosa is normal. Some advanced lesions have obliterated the lumen and there is proliferative change in the media and adventitia. Similar lesions are seen in oedema disease of swine and reproduced recently by experimental inoculation with shiga toxin 2e into pigs (AFIP Wed Slide Conf, 11, 2000-01). Stx2e binds to endothelium and media of arterioles in susceptible organs. This is likely to be an incidental lesion in these cattle.

Ovine - Coccidiosis and *E.coli*-Associated Watery Mouth Syndrome

An outbreak of disease in three-month-old lambs with 5 % mortalities and a further 10% of the flock of 300 sick was investigated on a property in the eastern wheatbelt. Sick lambs show "watery" mouths but no scouring. Histologically there are severe lesions of coccidiosis in the colon and SI with patchy secondary bacterial (Clostridial or Fusobacterial) necrosis of the superficial mucosa. There are also some submucosal vessels in the colon showing early fibrinoid vasculitis with fibrinoid change and swollen endothelial cells. A pure growth of *E coli* was obtained from a regional enteric LN. "Watery Mouth" syndrome (hypersalivation) in very young lambs in the UK is thought to be caused by endotoxinaemia. Not described in Australia to our knowledge.

Yabby Virus infection.

Yabby virus was responsible for severe necrosis in the cuticular epithelium. Large viral inclusions were observed in the nuclei of many cells. These changes are diagnostic for infection with a yabby virus that is novel and hitherto uncharacterized.
Although this virus has been seen previously in WA associated with mortalities, it has not been reported elsewhere in Australia or in the world. The virus infection appears to be a secondary problem, in that many yabby populations appear to harbour a subclinical infection but disease only occurs following a period of prolonged stress. Toxicological analysis of the water indicated that zinc levels were normal, which excludes zinc toxicity from being the cause of the problem. Variation in water temperature of more than a few degrees per day may be responsible for precipitating clinical disease and subsequent mortalities in this case. Results of a survey of WA properties for the yabby virus are to be found in:Jones, J.B., Lawrence, C.S. (2001). Diseases of yabbies (*Cherax albidus*) in Western Australia. Aquaculture 194: 221-232

Prawns - Baculovirus infection.

Histological examination of post larval prawns found a high prevalence of infection with a baculovirus (occlusion bodies) in the epithelial cells of the hepatopancreas. It is highly likely that the baculovirus infection was at least partly responsible for mortalities seen in this group. Given that the mortalities have now almost ceased, it is probable that the virus has the potential to cause some losses up to about PL5, but is of little consequence thereafter. A baculovirus histologically identical to this one has been found in virtually 100% of endeavour prawns from Exmouth Gulf that were examined during an FRDC-funded disease survey of wild prawns. The same virus was also present at low prevalence (about 4%) in brown tiger prawns and western king prawns.

**Note: ***Baculoviruses are large dsDNA viruses of arthropods (eg. Nuclear Polyhedrosis Viruses of insects) which produce occlusion bodies in the nuclei of infected cells. Occlusion bodies resemble inclusion bodies histologically and contain large amounts of virus, however they differ from inclusion bodies by being much more durable in the environment. Crystallization of polyhedrin, a non-structural viral protein that sets into a durable resin-like substance in which viral particles are embedded, allows the occlusion body to remain intact until it is broken down in the intestinal tract of the host arthropod. The genus formerly included some non-occluding members (eg. White Spot Virus of prawns), however these have now been moved to a new genus ("Nudiviruses"). All baculoviruses are now occluding viruses, by definition.