VETERINARY PATHOLOGY REPORT

Number 64 December 2002

President's Report

The Army teaches us to keep our mouths shut, our bowels open, and never volunteer for anything. Unfortunately perhaps, I was never in the Army. With Ruth Reuter's departure to England, the ASVP needed a stand-in president until the executive could be handed over at our next Annual General Meeting. Having previously volunteered to take over as caretaker treasurer when Rob Rahaley also left for England, I was perhaps a prime target to provide a stopgap presidency. If I thought that this short period would be free of politics, I was to be seriously mistaken. But before venturing into any discussion on matters political and I would first like to pay tribute to Ruth for her previous 2+ years' presidency. Ruth, in her workaholic mode, dealt with the ASVP and many other non-core functions in her "copious free time".

Review of Rural Veterinary Services

Tony Ross recently spent some time with the reviewer and has suggested that our Association should respond to the discussion paper to be produced following the end of primary submissions. Tony will probably be enlarging on the situation in his article in this edition.

The AVA and the ASVP

Those of you with access to the Australian Veterinary Journal may have read a letter from Dr John Plant in which he criticises veterinary pathologists for our decision in 1979 to disenfranchise ourselves from the Australian Veterinary Association. I will not respond to John's letter at this stage for diplomatic reasons, but would hasten to add that the veterinary pathology profession was operating in a vastly different culture in 1979 than now. The letter comes at a time when our association, the ASVP, has been invited to send an observer to the AVA Policy Council. Stephen Yeomans of Gribbles Veterinary Pathology in Adelaide has been nominated as this observer, but was unable to attend the last council meeting, which Keith Walker attended on our behalf. Keith was warmly received and invited to become involved in discussions rather than merely observe. The AVA are expected to make overtures to our Association to return to the AVA fold. Apart from flag the issue, I will not discuss it until such time as we have some proposal before us. Then, with the help of the executive, I will prepare a discussion paper for publication in the pre-conference edition of the Vet Path Report, and possibly formulate a motion to be put to the next AGM.

2003 AGM & Annual Conference

The 2003 AGM & Conference looms closer. It may sound a long way off but with the advent of the holiday season it will come around all too soon and catch many

of us off guard. The venue is to be the Elizabeth Macarthur Agricultural Institute at Menangle in NSW on April 12 & 13, following the University of Sydney Postgraduate Foundation course on gross pathology at the EMAI from 9 to 11 April. I invite readers to refer to an article by Tony Ross about the proposed theme/s of the conference depending on availability of keynote presenters. In any case there will be space, as always, in the open section to present cases removed from the theme, so please start thinking about and preparing papers. Again students' presentations are welcome (and they have been some of our better papers in recent years), presenting an opportunity for some NSW post-graduate students particularly to strut their stuff.

2005 AGM & Conference

I have been approached by the Cairns Convention Centre in regard to the year 2005, during which the Association of Veterinary Laboratory Diagnosticians are proposing to hold their world conference in Australia. They have apparently targeted Cairns as the venue and I was approached to see if our Association wanted to be involved. I have tentatively said "yes", adding that our next executive (from Qld) will be in the chair, and it will be very much up to them and the membership as to what role we might play. I think that such an event would be too much of an opportunity to miss, and we should use it as our annual conference for that year and hold our AGM in conjunction with it.

Season's greetings

Finally, on behalf of the executive and our hard-working secretariat (Barbara Gill), I would take this opportunity to wish you all the compliments of the season and look forward to a new year, hopefully devoid of drought and terrorist activities. To any of you who may have been personally affected by the Bali bombing, may I extend our moral support.

Peter Phillips
Acting President

Life Membership for Philip Ladds

The Executive of the Society has decided to award Life Membership to Phil Ladds in recognition of his outstanding contribution to veterinary pathology over a long period of time.

Phil will soon retire as Principal Veterinary Officer at the Tasmanian Government laboratory in Launceston and will be relocating to the Lismore area in northern NSW. He has been awarded a 3 year grant to document in book form the pathology of Australian wildlife species and will also have an adjunct appointment at Southern Cross University in Lismore.

Phil has previously worked in pathology at James Cook University and Ross University in St. Kitts, in practice in the UK, and as a field veterinary officer with the Queensland Department of Primary Industries. He graduated from the University of Queensland in 1961 and subsequently obtained his MVSc degree from that university for work on contagious bovine pleuropneumonia. He then received a scholarship to undertake studies at Kansas State University, where he received his PhD for research on listeric abortion in sheep. Phil is a Diplomate of the American College of Veterinary Pathologists and a Fellow of the Australian College of Veterinary Scientists and the Royal College of Pathologists. He has had extensive involvement with activities of the Australian College and was Chief Examiner in 1993-4.

Phil's major research interests have included male reproductive pathology, pathology of the lymphoreticular system in cattle, and diseases of farmed crocodiles. His major responsibility for many years at James Cook University was teaching diagnostic pathology and the supervision of higher degree students in pathology. Phil has more than 120 publications as monographs, chapters in text books, or as papers in journals of international stature.

For his enthusiastic contribution to veterinary pathology as diagnostician, investigator, teacher and administrator, Phil is a worthy recipient of Life Membership of this Society.

Peter Phillips
Acting President

Editorial/Secretary's Report

As you will be aware, we have recently lost the services of Ruth Reuter (President) and Julia Lucas (Committee member) due to their move offshore and Peter Phillips has kindly agreed to act as President and Treasurer until the executive transfer to Queensland in Sydney next April. I would like to thank Ruth and Julia for their substantial contribution to the ASVP during their tenure.

The date and location of the **2003 ASVP Annual Conference** has been decided:

Elizabeth Macarthur Agricultural Institute (EMAI), Menangle, NSW

Saturday, April 12 and Sunday, April 13

Theme: Toxicology

Many thanks to Tony Ross and his team for hosting the conference and organising invited speakers. The meeting will follow the University of Sydney's Postgraduate Foundation Gross Pathology Workshop at EMAI from April 9-11.

Cleve Main continues to perform admirably maintaining the ASVP website and has effected some valuable new additions since he took the reins. It would be useful if members could give some thought about the content and usage of this site before the next AGM so we can reach some consensus on future directions.

With regard to overtures by the AVA to return the recalcitrant ASVP to the fold of the national body, we will have to wait for their detailed offer. There may be merit in having the political muscle of the AVA promoting veterinary laboratory services in this country, providing this is more than shallow rhetoric, but there may also be a number of disadvantages. It is likely that this issue will be debated and resolved at the 2003 AGM.

Best wishes for a very happy Christmas and robust New Year.

John Finnie
Honorary Secretary

Review of NRDAP

A consultancy on the National Registry for Domestic Animal Pathology (NRDAP) has recently been provided to Animal Health Australia (AHA). The report, by Andrew Turner, former Chief Veterinary Officer for Victoria, concludes that NRDAP is a valuable resource for the continuing professional education and training of veterinary pathologists. It identifies a core program involving a half-time Registrar and strengthening the NRDAP's role in continuing education and training. The report recommends that this core program be funded 80% by government and 20% by industry, which in this case would be private veterinary laboratories.

The report proposes that, although the NRDAP has been operated by ASVP for the past 13 years, consideration needs to be given to AHA and ASVP being jointly responsible and accountable for the core functions of NRDAP and the programs that it operates. This approach is seen as important for ensuring access to Primary Industries Standing Committee (PISC) and its subcommittees for reporting and operations.

The report recommends that the annual continuing education courses aim to attract presenters who are veterinary pathologists of international renown and that they present courses at only one or possibly two locations or sessions. This change from the course being presented in each State and the Northern Territory is seen as important for obtaining suitably experienced presenters.

The report stresses that quality assurance and proficiency testing provide the reference points for Australia's veterinary pathology services operating to international standards (e.g. ISO 17025) in line with Australia's commitment to the Office International des Epizooties (OIE), the World Trade Organization (WTO) and the Agreement on Sanitary and Phytosanitary Measures (the SPS Agreement). It argues that to ensure there is a continuing commitment to quality-assured systems, governments in Australia and New Zealand should contract and purchase services only from laboratories that are accredited to international standard ISO 17025.

Although proficiency testing has only just commenced in anatomical veterinary pathology, it is a key cornerstone of a quality-assured system, which should be professional, independent and not reliant on one or two people for delivery. The report notes that the Royal College of pathologists of Australia (RCPA) can provide such a service covering both histological techniques and histopathological reporting. It concludes that the RCPA can provide value for money and a proposed veterinary pathology module has been prepared for examination by ASVP members.

AHA will provide the report to Animal Health Committee out-of-session for its consideration and endorsement, most particularly to provide cost-shared funding support. It is also expected that AHA provide the report to ASVP shortly.

Annual ASVP Conference 2003

The 2003 annual conference will be held on Saturday 12 and Sunday 13 April 2003 in the conference room of the Elizabeth Macarthur Agricultural Institute, NSW Agriculture, Woodbridge Road Menangle - on the south-western edge of Sydney. It will immediately follow the 3 day "Gross Pathology of Ruminants" course at EMAI on 9 - 11 April.

Veterinary toxicological pathology is the major theme. Keynote speakers include Professor Wanda Haschek-Hock and Dr Ross McKenzie - both international experts in veterinary toxicology. Members case presentations are welcome on any subject but particularly on aspects of veterinary toxicological pathology.

A range of nearby accommodation from 4.5 star motel to student accommodation is available. Menangle is 50 km SW of Sydney. There is a direct electric rail link between Sydney airport and nearby Campbelltown.

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

STATE REPORTS

NSW – Paul Gill

Erythromycin induced hyperthermia in a thoroughbred foal.
Alan.Kessell, Rural Veterinary Centre, University of Sydney

History: An 8 week old, female, thoroughbred under treatment for suspect *Rhodococcus equi* infection was noticed lying down at approximately 4pm and found dead at 5.30 pm. A post-mortem examination was performed at UVCC within 2 hours.

Post Mortem Findings: The foal was in good condition, with mild to moderate post-mortem autolysis. All muscles had a "cooked meat" appearance in that they were pale and soft with a stringy consistency. The body was very warm to touch, with an intra-abdominal visceral temperature exceeding 45ºC.

Abdominal cavity: There was marked vasodilatation of the subcutaneous vessels of the thorax and abdomen. The spleen was moderately congested and contained moderate numbers of sub-capsular petechial haemorrhages. The liver and kidneys were moderately to markedly congested. There was approximately 10ml of opaque pink to orange coloured urine in the bladder. The serosal surface of the intestines was blanched and there was a mottled appearance to much of the mucosal surface. The stomach contained a small milk clot and a moderate amount of green fodder.

Thoracic cavity: The lungs were markedly, bilaterally, diffusely congested, and on section, copious amounts of blood freely ran. The small bronchioles and bronchi exuded yellow foam on section. There were many scattered 1-2cm firm, well circumscribed, partially encapsulated nodules with blanched haemorrhagic

centres throughout the pulmonary parenchyma with a tendency to congregate around the hilus. The heart was flabby and had a "cooked meat" appearance. There was a moderate amount of white foam in the trachea.

Limbs: There was a small, 0.5cm diameter puncture over the lateral aspect of the right carpus, with dried blood extending down the limb to the foot.

Other Tests:

Microbiology: Profuse pure cultures of *Rhodococcus equi* were obtained from various nodular lesions in the lung

Histopathology

- Lung: there are many variably sized multifocal irregular areas of necrosis and dense degenerate neutrophil infiltration. These areas are bordered by less affected areas where the degree of necrosis is variable and there are significant numbers of macrophages and also some lymphoid cells as part of the alveolar infiltrate. There is significant patchy haemorrhage and oedema associated with these purulent centres.
- Liver: frequent small clusters of degenerate neutrophils near portal areas and within sinusoids.
- Adrenal : mild congestion and oedema
- Spleen : moderate development of white matter areas
- Duodenum: there are a small number of macrogametes of *Eimeria leukarti* in the mucosal layer here.
- Heart, kidney and skeletal muscle: there is mild autolysis

Morphological Diagnosis:

1. Severe multifocal pulmonary abscessation secondary to *Rhodococcus equi* infection
2. Mild coccidial infection, small intestine

Aetiological Diagnosis: Hyperthermic reaction secondary to erythromycin therapy

Summary: this is a well recognised entity in equine medicine severe hyperthermia in foals associated with use of erythromycin antibiotic (either alone or in combination) . This animal basically died of heat stroke. Erythromycin is a very useful drug in treating *Rhodococcus equi* bacterial infections as it has good activity against the organism and can penetrate cell membranes (the organism often resides inside macrophages). The literature records a low number of these adverse hyperthermic reactions. The mechanism is not known but is thought to involve a direct or indirect effect of the drug on the temperature regulatory centre in the brain.

Atypical Ependymoma in an Angus heifer.

Roger Cook, Regional Veterinary Laboratory Wollongbar NSW, 2477

History

The brain from a 30-month-old Angus heifer with a tendency to circle and fall to the left was submitted for examination under the National TSE Surveillance

Program (NTSESP). On the left side of the medulla oblongata at the level of the caudal cerebellar peduncles immediately below the root of the vestibulocochlear (acoustic) nerve was a circumscribed , mottled, yellow to off- white area 5 to 8 mm diameter in transverse section and, and extending about 5 mm rostro-caudally along the medulla.

Histopathological Examination

Medulla (caudal cerebellar peduncles) : On the left side immediately below the root of the vestibulocochlear (acoustic) nerve was a circumscribed malacic area 5 to 8 mm in diameter, and in the adjacent neuropil there were anastomosing cords of loose fibrovascular stroma bordered by variable-sized polygonal cells with basophilic cytoplasm. Occasionally these cells formed a low columnar array with a basal nucleus and a ciliary or microvillar-like surface profile. Some of these cells were in nests. Characteristics of malignancy shown by scattered cells included marked enlargement and hyperchromasia of nuclei. Within the malacic area, the medium sized vessels had expanded oedematous adventitia and there was often a peri-adventitial border of polygonal neoplastic cells. Within the affected area there was granular mineralisation of neuronal cytoplasm and scattered spheroids and extensive infiltration by macrophages (gitter cells).

Morphological diagnosis

Atypical ependymoma, unilateral (left side), dorsolateral medulla

A small number of tumours of this type and in this location (unilateral, caudal medulla) have been reported in the UK in BSE suspects that proved to be BSE-negative. Circling and falling to the left (damaging the left eye) in this animal were consistent with the brainstem neoplasm being on the left side of the medulla. The animal was also aggressive, but there were no forebrain lesions detected to account for this.

References:

Jeffrey M (1992) A neuropathological survey of brains submitted under BSE orders in Scotland. Vet Rec 131:332-337.

McGill IS, Wells GA (1993) Neuropathological findings in cattle with clinically suspect, but histologically unconfirmed BSE. J Comp Path 108:241-260.

NT – Anton Janmaat

Hepatic Fibrosis in a Puppy

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

A 10-month-old miniature schnauzer presented with stunted growth and a two months history of vomiting, inappetence and jaundice. Serum biochemical analysis revealed marked increases of ALT, AST, GGT, ALP and fasting ammonia. A complete blood count did not suggest the presence of a primarily infectious or inflammatory process. A tentative diagnosis of congenital portosystemic shunt was made. During exploratory laparotomy, the veterinarian noted microhepatica and a very small portal vein but was unable to locate an extrahepatic portosystemic shunt. A liver biopsy was submitted for histopathological evaluation.

There was marked periportal bridging fibrosis and cholangiolar proliferation. Bile ducts were uniformly small, and did not contain bile. There was portal proliferation of arterioles. Branches of the portal vein were not readily discernible in portal areas. Most central veins were surrounded by several extra small vessels devoid of blood, presumably lymphatics. Portal tissue was mildly infiltrated by lymphocytes and occasional neutrophils. There were lymphoid aggregates and mild macrophage infiltration associated with occasional central veins. These macrophages, and scattered Kupffer cells contained hemosiderin. Hepatocytes were generally atrophic, but apart from occasional randomly scattered necrotic, binucleate or mitotic hepatocytes, and moderate sinusoidal congestion, the parenchyma remaining between portal areas and central veins was relatively normal.

A diagnosis of idiopathic periportal hepatic fibrosis was made. The presence of bridging periportal fibrosis with biliary and arteriolar proliferation typifies this poorly understood condition. Also, as in this case, branches of the portal vein are often not apparent, leading to the suggestion that the liver lesions are secondary to primary portal vein hypoplasia. The disease typically results in stunted growth, with clinical signs and serum biochemistry values consistent with portosystemic shunting appearing by two years of age.

Encephalopathy and Retinopathy in Hatchery-Reared Juvenile Barramundi

John Humphrey, Berrimah Veterinary Laboratories, NT Department of Business, Industry and Resource Development

History and Clinical Signs

Vacuolating encephalopathy and retinopathy is manifest as an intermittent, low-grade disease in juvenile hatchery-reared barramundi, typically affecting fishes 3-8 weeks of age. Clinical signs include dark colouration, failure to feed, solitary swimming, neuromuscular incoordination, rapid spiralling swimming movements, a head down attitude in the water column and death. In some populations, clinical signs and lesions are not apparent before 7-8 weeks of age. Typically, fish develop signs following transfer from larval rearing tanks with live food (*Artemia* sp.) to flow-through raceways and weaning onto solid foods.

Gross Pathology

Dark colouration and hyperaemia of meningeal vessel is evident in some fish.

Histopathology

Histopathological changes primarily relate to vacuolation in the grey matter of the brain and spinal cord and in the nuclear layers of the retina. In the brain, vacuolation is conspicuous in the neurones and neuroglia of the grey matter of the olfactory and optic lobes and in the cerebellum, with sparing of the myelinated fibres. Necrosis of neurones and neuroglia may be evident. Vacuolation may be low grade, with solitary or occasional vacuoles present, through to severe with extensive regional vacuolation of grey matter. Similarly, retinal vacuolation varies from low-grade, with single or occasional vacuoles in the neuronal layers, through to generalised, with profound vacuolar change throughout all layers of the retina, including the inner ganglion layer. Retinal necrosis may be a feature in such cases. In older fish and/or in resolving lesions, focal or multifocal histiocytic accumulations may be seen in the retina.

In the spinal cord, vacuolar change in neurones and neuroglia may occur in clinically normal fish with no other histological lesions. Vacuolation of the grey matter of the cord is also seen, however, in fish with typical nodaviral disease, usually but not always accompanied by histopathological changes in the brain and retina. This is observed more frequently in older fish (7-8 weeks).

Typical disease also occurs in the absence of discernible histological lesions in the brain, retina or cord.

PCR Assays

Positive PCR assays are invariably obtained from fish showing histological lesions. In some cases, strong positive reactions are obtained from populations of fish exhibiting clinical signs but no histological lesions. On occasions, positive PCR assays are obtained on larval and juvenile fish, in the absence of histopathological changes. This may or may not be followed by the emergence of clinical disease.

Comments

Nodaviral disease in juvenile barramundi can be presumptively diagnosed on clinical signs and histopathological examination. The detection of latent carriers cannot be effected with histopathology. The use of histopathology in evaluating the carrier status of a population is not seen as an appropriate approach, especially at three weeks of age, as nodaviral disease may not emerge as a clinical or pathological entity until 7-8 weeks of age. The vacuolation in the spinal cord is enigmatic. Possibly, a histopathological response to infection in older fish is characterised by vacuolation in the cord, with sparing of the brain and retina.

QLD – Bruce Hill

No Report

SA – Stephen Yeomans

Acanthocephalan parasite in a Crested Bellbird
Peter Phillips, IDEXX Laboratories, 33 Flemington St, GLENSIDE SA 5065
Katie Reid, Alice Springs Veterinary Clinic, 75 Bath St, ALICE SPRINGS NT 0870

A wild caught crested bellbird was being cared for at a local wildlife park. It had been treated with Panacur following discovery of unidentified faecal oocysts. The bird had a history of progressive respiratory distress with white lesions in the oral cavity. It was treated with Baytril on presumption of caseous abscessation. The bird died, and necropsy examination revealed large numbers ("100's") of white parasites present in the subcutaneous tissue, pectoral muscle and oral cavity.

Heart, liver, kidney, lung, testis and muscle were examined histologically. The lung was markedly congested but otherwise normal. The testicular tubules contained necrotic cellular debris with few spermatozoa. The skeletal muscle was normal, with an adjacent parasitic body that was eliciting no inflammatory response.

A parasite from the fixed tissue was referred to SARDI parasitologist Dr Ian Carmichael, who identified it as an intermediate stage of the acanthocephalan parasite *Oncicola pomatostomi.* Adults of this parasite occur in the intestine of feral cats (65% of 188 examined from the NT, 1991-1993) and the dingo. Intermediate stages occur under the skin of a variety of bird paratenic hosts.

TAS – Philip Ladds

Mount Pleasant Laboratory report
Philip Ladds

Some interesting recent cases submitted to the laboratory during recent months were as follows:

Bovine: Extreme spongy encephalopathy in two neonatal Hereford/Hereford-cross calves led to a diagnosis of Maple Syrup Urine Disease; history was typical and diagnosis was confirmed by DNA examination of hair samples. Interestingly, in one of the two calves there was severe, diffuse eosinophilic meningitis, presumed due to concurrent fluid deprivation?

Severe meningitis, presenting clinically as head-pressing and fever, affected ~30 dairy calves several weeks of age in a mob of 200. Response to antimicrobial treatment was good but about 6 calves died. *Pasteurella* sp. was isolated.

Campylobacter jejuni infection was confirmed as the cause of late abortion of 12 lambs in a flock of 4000 ewes. Lesions were more conspicuous in lung and brain

than in liver. In another flock, examination of two (very) autolytic foetuses (of 10 that were aborted) confirmed *Listeria ivanovii* infection as the probable cause.

Ketosis/pregnancy toxaemia was diagnosed as a cause of mortality in five flocks. Typically, heavily pregnant ewes were involved; there were elevated ketones in urine and blood, and extreme hepatic lipidosis in ewes that were necropsied. Concurrent disease included *Yersinia enterocolitica* infection in one flock, and broncho-pneumonia caused by *Corynebacterium ovis* in an affected ewe from another flock.

Other domestic species: Suppurative placentitis in an aborted equine conceptus was associated with isolation of *Streptococcus bovis* and a Group C Streptococcus (possibly *Strep. zooepidemicus*), suggesting opportunistic bacterial infection as the cause.

In another case involving death of a 5-day-old Arab foal, findings were consistent with combined immunodeficiency. Inflammation was apparent in many tissues and *Actinobacillus equuli* was isolated.

Large numbers of air sac mites (not normally considered severe pathogens) were found in 6-10 month-old commercial chickens which had a history of "coughing and sneezing" and which histologically had a severe chronic broncho-pneumonia.

Wildlife: Chlamydiosis was diagnosed in a wild Eastern Rosella (*Platycercus eximius*) that was found dead in a backyard where wild birds were fed. The case emphasised the zoonotic risk of this practice. There was splenomegaly and apparent hepatomegaly.

Severe leukoencephalopathy (similar to that described by Holz & Little, 1995, J Wld Dis 31, 509-513) was diagnosed in a Tasmanian Devil (*Sarcophilus harrisii*) with a history of "unsteadiness in hindquarters" progressing to paralysis. Material from this case sent to AAHL was negative for prion protein on immunohistochemisty.

A further case of multi-focal cutaneous sarcoma in a Tasmanian Devil (see last Vet Path Report) was also examined.

VICTORIA – Malcolm Lancaster

No Report

WA – David Forshaw

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

Laryngeal Dermatophilosis in a ram.
Cleve Main

A mob of 65 rising 6 tooth Poll Dorset/Merino cross rams experienced severe respiratory distress when driven a very short distance to the yards. Post mortem examination of a ram that became very cyanotic and remained in a dyspnoeic state revealed oedema and haemorrhage of the dorsal surface of the lung and an ulcerated larynx.

Histopathological examination confirmed widespread but patchy alveolar oedema and haemorrhage with blood present in some of the larger bronchioles. Associated with the shallow laryngeal ulcer were mild infiltrates of neutrophils and organisms resembling Dermatophilus congelensis. Neither the lung lesion or laryngeal lesions were sufficiently severe to fully explain the clinical signs. Oral dermatophilosis has been described in cats and presumably can occur in sheep although we have never seen it previously.

"Egg Drop" associated with Avian Encephalitis Virus infection
Cleve Main, Russell Graydon and Sarah Plant

A well-managed layer farm experienced a transitory drop of 5 - 18% in production, in terms of egg size and daily production. No white shelled, thin shelled or shell-less eggs were seen. The manager employed an extensive vaccination program against a range of diseases that included AE at 7 weeks and EDS at 15 weeks. Several layers in the 42 and 55 week old age group were submitted for necropsy. Evidence of avian leucosis or Marek's Disease was seen in one bird, egg peritonitis in another and possible ovarian regression in a third. No remarkable lesions were detected in another two birds.

Histopathological examination did little to clarify the situation. A small focus of gliosis was found near the decussation of the cerebellar peduncles in one bird. Numerous small focal infiltrates of lymphoid cells suggestive of Marek's disease were seen in the kidney of the same bird.

15 blood samples were collected from other birds on the farm and tested by HI for evidence of EDS 76 infection producing titres of 2048, 1024 and 512. Because the birds had been vaccinated against EDS, there was a strong argument that although the birds had been challenged, infection with EDS 76 may not have been responsible for the loss of production. The sera were consequently tested for antibody to AE virus producing positive ELISA titres, several of which were in the range of 939 - 4600.

A second layer farm located well away for this farm suffered similar losses in production. Serum samples taken from birds on the second were negative for antibody to EDS 76 but strongly positive to antibody to AE virus.

EDS vaccine is a killed oil adjuvant vaccine that is almost 100% effective. There is little scope for any loss in potency and providing it is given "intra-bird" gives excellent cover. AE vaccine, on the other hand, is a fragile live viral vaccine and it

is difficult to effectively vaccinate birds that are raised on slatted floors. It is concluded that AE vaccination failure is the most likely reason for loss of production in both cases.

AE virus infection is commonly thought of in terms of neurological signs in young chicks. It is well worth remembering that infection in mature birds may produce a temporary drop in egg production. Production levels on the layer farm discussed here returned to normal in about three weeks. Newcastle disease is an important differential diagnosis for both EDS and AE.

Photosensitisation in sheep grazing Biserrula cv Casbah
Jeremy Allen

Casbah is a new legume pasture released in WA in the late 1990's. A report of photosensitisation in sheep grazing this pasture were investigated in 1999 and another four cases were investigated in 2001. In early September this year, a further two cases occurred, both in the same week. One involved a mob of 300 mixed aged Merino ewes and lambs that had been on the pasture for 2 weeks (50% affected), and the other a mob of 200 mixed age Merino crossbred ewes and lambs that had been on the pasture for 5 weeks (25-30% affected). There have been no deaths. Preliminary investigations via clinical chemistry indicate that this is likely to be a primary photosensitisation. Epidemiological evidence suggests that the problem is most likely to occur when Casbah is the predominant pasture species, a high stocking rate is being used, the plant is rapidly growing, is about to flower or is already in flower.

Congenital tremors in piglets
David Forshaw

Breeder unit with epidemic of congenital tremors over a few months. Stillbirths were seen in badly affected litters and survivors had mild tremor syndrome that persisted to market weight. All piglets necropsied were good size at birth and had lesions confined to the cerebellar white matter. There was equivocal attenuation of the white matter with very mild coarse vacuolation (myelin splitting?) with immature glia and rare pyknotic nuclei. LFB/Ag stains showed normal myelination. Pestivirus antigen (CSF) negative, HEV negative, Aujeszky's negative by culture. Pooled lymphatic tissues positive by porcine circovirus 2 PCR test.

Congenital tremors in piglets can be either hereditary (multiple breeds described), toxic (trichlorfon toxicity), associated with in utero viral infection, notably classical swine fever virus, or due to cause unknown. Dysmyelination is seen in swine fever infection and the inherited causes. PCV-2 infection has been ascribed as the cause of congenital tremor type AII in piglets in the USA (Stevenson et al (2001) J Vet Diag Invest 13:57-62). No gross or microscopic lesions were described. Immunostaining of frozen sections showed PCV-2 infection in affected compared to unaffected animals of a greater number of neurones in the brain and spinal cord. PCV2 positivity (to the PCR test) is common in Australia (Graham Wilcox, pers comm) but has not been associated with disease. The role of PCV 2 in this outbreak is unclear.

Post script: Boars from this producer were supplied to another herd that is now experiencing similar problems.

Listeria ivanovii encephalitis - NOT - correction to report in VPR 63

David Forshaw

In the previous issue, an outbreak of encephalitis attributed to L. ivanovii in sheep was reported. Subsequent typing (API Listeria) has shown the isolates from this case to be L. monocytogenes. The original identification was based on a positive CAMP test with *Rhodococcus equii*.

Fibropapilloma in a Green Turtle

John Creeper

A green turtle in a wildlife rehabilitation centre developed several 1cm diameter raised lesions on the hind flippers. Histological examination of one such lesion revealed a focally extensive area of epidermal spongiosis with numerous eosinophilic intra-nuclear inclusions within keratinocytes. The adjacent epidermis showed hyperkeratotic hyperplasia with numerous long rete pegs ramifying into the dermis that was expanded by proliferating irregularly arranged fibrous tissue.

There have been several cases of Green Turtle Fibropapilloma virus infections seen by the wildlife carer, notably affecting the conjunctiva, flippers and penetrating within the carapace in severe cases.

Note: Readers are referred to the paper submitted by A Gordon and C J Limpus on fibropapillomatosis in marine turtles presented at the last ASVP annual conference.

Nephrocalcinosis in trout

John Creeper

Farmed trout in the wheatbelt. In the kidney there was marked tubular calcinosis diffusely throughout the renal tubules, accompanied by cellular casts. Von Kossa stains confirmed the presence of calcium salts.

Comments: The two major predisposing factors in the development of calcium within the tubules of the kidney are high water carbon dioxide levels and feeding dry pellets. High water carbon dioxide levels leads to acidosis of the fish that in turn accelerates the precipitation of dissolved calcium salts within the kidney. Obstruction of the kidney tubules or ureters causes renal failure with poor weight gains, or death.

Digenean trematode infestation in oysters

John Creeper

Denham. 10,000/20,000 black lipped oysters dead. Oysters with numerous digenean flukes in digestive gland. Tubules were atrophic with amorphous debris in lumina. Probable starvation possibly due to cold water.

Murdoch University

Multiple alimentary parasites in wild pelican

Shane Raidal

Submission: Juvenile Pelican
Status: Post natural death.

Post mortem interval: <24 hours.
Post mortem decomposition: Minimal.
Submitted history: Found dead on Swan River. DDx: Suspected toxicosis

Visible lesions:

Significant internal findings:
1. The proventriculus contained a heavy burden of contracaecum nematodes and a thick black fluid consistent with digested blood.
2. The intestines contained similar fluid and a heavy burden of worms including cestodes, trematodes and nematode species that were collected for species identification.
3. The bursa of fabricius was well developed indicating that the bird was a juvenile.
4. The adrenal glands were markedly hyperplastic.

Ancillary tests:
Wrights stained cytology of liver and spleen impression smears were normal.

Histopathological findings:
Adrenal cortical tissue hyperplasia. Many different worm species were present in sections of the gut associated with varying degrees of chronic heterophilic and eosinophilic inflammation. Large numbers of unidentified schistosome eggs were present throughout segments of the mucosa.

Parasite identification:
Contracaecum sp. (Nematoda: Anisakidae)
Armadoskrjabinia murrayensis (Cestoda: Hymenolepididae)
Echinochasmus pelecani (Trematoda: Echinostomatidae) sp. (Trematoda: Diplostomatidae)
Unidentified heterophyid fluke (Trematoda: Heterophyidae)

Final Diagnosis:
Severe alimentary parasitosis.