Australian Society for Veterinary Pathology

VETERINARY PATHOLOGY REPORT

PUBLICATION NO. PP544059/0003

EDITOR: Cleve Main

Number 52 **APRIL 1999**

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PRESIDENT'S REPORT

This is the immediate pre-AGM issue of the newsletter and through it I urge as many members as possible to register for our conference in Melbourne where a very interesting program has been built around a group of excellent plenary speakers in the field of toxicology. I am sure there will be plenty of "meat and potatoes" for anyone who attends as well as stimulating conceptual material. I am also hoping for a batch of top quality member presentations, and am particularly looking forward to those from our younger members. I already know of a couple which will be full of interest.

On matters political and organisational, I encourage members to come to our AGM with vision and ideas. We are all well aware of the major issues – the trick is how best to deal with them. We need to stop problems from converting to dilemmas – problems should have solutions while dilemmas (or "trilemmas", quadrilemmas" etc) are endlessly circled around. It is up to us to play an active role in generating solutions.

Anyway, enough of sermonising, I look forward to the pleasure of seeing and hearing from you in Melbourne. Here's to a great conference.

Clive R. Huxtable

HON. PRESIDENT

EDITORIAL

Well, this is the last ASVP Report for the financial year. Please read it carefully as it contains valuable information on the forthcoming conference.

Thank you all for your contributions throughout the year. My apologies to those contributors in NSW and Victoria who on two occasions missed out on getting their contributions published. To add insult to injury, it was then suggested by myself that they were not trying their hardest to contribute.

The problem has been identified and hopefully rectified. On both occasions, the error was because the contributions fell in a crack in the electronic floorboards somewhere between Dave Forshaw's email system and mine.

On a more positive note, presentation of contributions by email is working well and really is the way to go.

Enclosed with this Report is an invoice for subscriptions for the next financial year. All of you will note that subs have increased in line with that adopted at last year's AGM. Some of you will note that you have been asked for overdue subs. Before you get upset, I would like to point out that we have over 150 members on our books, but only 106 financial members. This represents a $1,000 shortfall in our income this year and we need every cent to pay for publication of this report as well as the conference proceedings. Remember, we are only a small (but growing) organisation and we need your support.

Finally, in line with our efforts to keep you up to date with your Executive's activities regarding continuing education and pathology standards, I urge you to read the attached reports from Committee member, Barry Richards. Note that the topic of ACVS fellowship training has been listed as an agenda item for the AGM. So too, is proficiency testing in anatomic pathology, medical laboratories carrying out veterinary testing and the Slide of the Month series. I will also foreshadow the inclusion of the topic of holding the 2004 conference and AGM in Brisbane to coincide with the International Congress of the International Academy of Pathology. This, as the name suggests, is a world wide organisation and has approximately 15,000 members. Your Executive is keen on the idea, and looks forward to membership support.

PATHOLOGY STANDARDS AND THE APSA PROPOSAL

Progress on the Society's APSA (Animal Pathology Standards Australia) proposal has been molluscine following the Australian Animal Health Council's review of laboratory systems which was completed in November 1998. The Review had identified pathology standards and continuing education as significant issues and recommended that AAHC invite tenders for delivery of a training/competency testing package.

News from AAHC informs that the Chairman of the review team, Dr Mike Carroll, had addressed the Board on 3 February on all recommendations including this one. The Board then decided that it needed a "concept paper" to address the benefits that will accrue to members of the AAHC (Livestock Industries, Commonwealth Government, AVA etc) if it were to adopt the recommendations, and to suggest options to broker funding of the program. The AAHC Board was due to meet again in April to consider the matter.

ACVSc FELLOWSHIP TRAINING

The ASVP has expressed concern over the conditions specified for attainment of Fellowship status in Pathobiology through the Australian College of Veterinary Scientists. In the opinion of the ASVP Executive, the ACVSc has set unrealistically tough conditions for candidates to meet before being eligible to sit the Fellowship examination. Most aspiring Australian pathologists would find it extremely difficult, if not impossible, to meet the current specifications across the full range of production and companion animals. Approaches by the ASVP to have ACVSc soften its attitude have so far been unsuccessful. This is a serious issue that affects the future of veterinary pathology in Australia and our Society should join the debate with some vigour. Our objective is to encourage and foster excellence in veterinary pathology. Unfortunately, the current pathway to the highest form of specialist recognition in Australia is sadly out of reach for most. An issue for discussion at the AGM.

ANNUAL GENERAL MEETING

You are hereby notified that the 1999 Annual General Meeting of the Society will be held at 3:30pm on Saturday 15 May 1999 at the Veterinary Preclinical Centre, University of Melbourne, Parkville, VICTORIA.

AGENDA

1. Welcome and Apologies
2. Minutes of AGM 1998
3. Business arising
4. Correspondence
5. Reports of Office Bearers
   - President
   - Secretary
   - Treasurer
   - Report Editor
   - Slide of the Month Coordinator
   - Registrar - NRDAP
   - Management Committee - NRDAP

GENERAL BUSINESS

1. Medical Pathology Laboratories
2. QA Standards
3. Slide of the Month program
4. ACVS Fellowship training program
5. Other Business

ELECTION OF OFFICE BEARERS

1. President
2. Secretary
3. Treasurer
4. Committee members
5. Slide of the Month Coordinator
6. Registrar and Management Committee – NRDAP

NEXT MEETING

NOTE:

Many of you will be aware that our President, Clive Huxtable is leaving Australia for greener pastures in the USA. This if course means that unlike the rest of your Executive, he will not be available for re-election. It has been the practice of the Society to rotate the Executive through the States on a 3 year cycle and under normal circumstances, this AGM would confirm the re-election of the current Executive. To allow the continuation of Western Australian custodianship, Dr David Pass has agreed to allow his name to be put forward as a candidate for Society presidency for the year 1999/2000. This of course does not prevent any of our members for nominating for that or any other position. I will accept nominations now and there will be a further opportunity on the day of the meeting itself.

Cleve Main

HONORARY SECRETARY

ASVP Conference, Melbourne, 1999

Harold White Theatre, Arts Building, University of Melbourne, Parkville

Saturday 15 May 1999

8:00am – 9:00am	**Registration and coffee, Harold White Theatre, Arts Building**
9:00am	Polioencephalomalacia revisited: thiamine, selenium and sulphur. *Professor Merl Raisbeck, Visiting Scholar, CSIRO, Geelong*
10:00am	Biomonitoring methods in environmental toxicology *Dr Paul Wright, RMIT, Melbourne*
10:45am	**Morning tea, Graduate Centre Bistro**
11:00am	Drugs as toxins, perspectives on toxicokinetics and drug registration for veterinary pathologists *Dr Tim Dyke, National Registration Authority, Canberra*
12:00 noon	New perspective on toxic algae: Cylindrospermopsis and other cyanbacteria *Professor Alan Seawright, University of Queensland*
1:00pm	**Lunch, Graduate Centre Bistro**
2:00pm	Indospicine poisoning in dogs; a complex naturally-occurring environmental intoxication *Professor Roger Kelly, University of Queensland*
2:45pm	Federation of Australian Scientific and Technological Societies *Professor Snow Barlow, University of Melbourne*
3:00pm	**Afternoon Tea, Graduate Centre Bistro**
3:30pm	Annual General Meeting
7:00pm	Conference Dinner, Asti Restaurant, 680 Swanson St, Parkville
	The cost per person for the meal will be $30. The restaurant does not hold a liquor licence but will also allow us to bring our own dinner wines without charging a corkage fee. Alcoholic beverages will be in addition to the meal price. There will be a limited choice of menu but it is cheaper if we have a restricted range of fare.

ASVP Conference, Melbourne, 1999

Harold White Theatre, Arts Building, University of Melbourne, Parkville

Sunday 16 May 1999

8:30am	**Registration and coffee, Harold White Theatre, Arts Building**
9:00am	Research on envenomation by arachnids *Ken Winkel, Australian Venom Research Unit, University of Melbourne*
9:45am	Bracken fern syndromes *Barry Smith, AgResearch, Rualcura Research Centre, New Zealand*
10:30am	**Morning tea, Graduate Centre Bistro**
11:00am – 1:00pm	Member Presentations
1:00pm	**Lunch, Graduate Centre Bistro**
2:00pm – 4:00pm	Member presentations
Meeting close	

ACCOMMODATION GUIDE

The following information should prove helpful, but please arrange your own bookings.		Phone
Budget Accommodation		
The Hotel Y 489 Elizabeth Street	From $30.00 (shared accommodation)	03 9329 5188 1800 249 124
International House 241 Royal Parade	From $55.00 (includes meals)	03 9345 7576
Flinders Station Hotel Backpackers 35 Elizabeth Street	From $12.00 (shared accommodation)	03 9620 5100
Toad Hall 441 Elizabeth Street	From $17.00 (shared accommodation)	03 9600 9010
Mid-Priced Accommodation		
Rydges 186 Exhibition Street	Four Star Rating Corporate Rate $135.00	03 9662 0511
Elizabeth Towers 792 Elizabeth Street	Three Star Rating Corporate Rating $95.00	03 9347 9211
Victoria Hotel 215 Little Collins Street	Three Star Rating Corporate Rate $75.00	03 9653 9441
Travel Inn Grattan Street, Carlton	Four Star Rating Corporate Rate $105.00	03 9347 7922
Luxury Accommodation (many more available, but generally not close or on convenient tram stop)		
Grand Hyatt	5 Star hotels typical charge $250+ per day, often discounted for weekend deals	03 9657 1234
Hilton on the Park		03 9419 2000
Le Meridien		03 9620 9111
Sheraton Towers		03 9696 3100
Windsor		03 9633 6000

University of Melbourne

Main Campus Map

ASVP Meeting – Harold White Theatre, Arts Centre

Location:

- Corner of Grattan and Swanson St, SE Corner of campus

Entrance:

- From Swanson Street

Parking:

- Off street parking available at University gate access points. Cost is $2 per day and requires a daily ticket, able to be purchased at the gate at the time of entry.
- Street side metered parking available along Grattan St but space is limited along Swanson St near the Arts Centre until the afternoon

Public Transport:

- Tram stop is opposite the Arts Centre, and taxis are readily available
- Refer to map on page 13

Map

STATE REPORTS NORTHERN TERRITORY – ANTON JANMAAT

Melioidosis in dogs

Helen Parkes, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industries and Fisheries

Melioidosis has been very active in the Top End of the NT this wet season, with 42 cases so far in humans, and more expected. At BVL, we have isolated *Burkholderia pseudomallei* from two dogs this year; one was from an abscess on the hindleg of a young dog, the other was from the placenta and a mammary abscess of a bitch that aborted and subsequently died. Following a request from a veterinarian for melioidosis serology on a dog that presented initially with unusual skin lesions, then developed neurological signs three weeks later, we obtained very high CFT and IHA titres (IHA titre of 640, CFT titre of 256). However, we didn't know how this related to "normal dogs" so did melioidosis CFT and IHA tests on 45 stored canine sera for comparison. (Sera were collected this wet season – between November 1998 and February 1999. Some were from healthy dogs; others were samples from clinically ill dogs, sent in for biochemical testing.) Many of the sera were reactive, particularly in the CFT. This may reflect previous exposure to *B. pseudomallei* (not surprising, considering dogs' often intimate exposure to soil). Our small survey suggested that, in dogs, the IHA may be a more useful diagnostic test than the CFT or higher. Of these, two dogs had IHA titres greater than 640 and CFT titres of 128. These two were both sick dogs, one with a high fever of unknown cause, and the other with neurological signs. (I contacted the vet who had submitted these samples – both dogs have apparently recovered with antibiotic treatment, but she will keep an eye on them for recurrence of signs). We also tested serum from the dog with the *B. pseudomallei* abscess, and obtained an IHA titre of 160 and CFT titre >1024.

These results suggest that *B. pseudomallei* infections in dogs are perhaps more common in the Top End than we realise, and may be responsible not just for wound infections, but may also cause systemic disease.

Exotic mussels in Darwin waters

Helen Parkes, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industries and Fisheries

There is an ongoing battle in Darwin to clear the harbour's marina areas of an exotic marine mussel (*Congeria sallei*) that grows rapidly, causes major fouling problems and has the potential to severely compromise the pearl industry in northern waters. Just before the mussel story broke in the press (on 1 April, which resulted in many locals not believing it), we received about 100 of the little blighters to see whether they were sexually mature. The answer was a definite yes, which DPIF was happy about (sort of!), because it confirmed they had made the right decision in closing the Cullen Bay marina and taking very active measures to seek and destroy the pest.

STATE REPORTS NSW – PAUL GILL

New Head for Veterinary Pathology at Sydney

ASVP member Paul Canfield has recently been appointed Head of the Department of Veterinary Anatomy and Pathology at the Sydney veterinary school. He takes over from Michael Bryden who is soon to be retiring. Paul will continue his involvement in the department's diagnostic and teaching activities as well as pursuing research interests in wildlife pathology and immunopathologic aspects of disease in companion animals.

Norseq Veterinary Pathology Group Meeting

Twenty-two interested people, mainly veterinary pathologists, met on 13 March at VPS in Brisbane to hear Dr Richard Miller speak on "The histological diagnosis of fungal diseases". Case presentations included Aphanomycosis in Australian bass (Wendy Townsend), saprolegniasis in silver perch (Dick Callinan), *Hemoproteins* sp infection in a magpie (Roger Cook), meningothelial meningioma in a Rottweiler pup (Steve Yeomans), superficial necrolytic dermatitis in an aged terrier (Neill Sullivan), myelokathexsis-like syndrome in a persistently neutropaenic Border collie pup (John Mackie), dermatophytic mycetoma in a Persian cat (Melissa Carlisle), synovitis and foetal bronchopneumonia associated with Mycoplasma Bovis infections and Anthrax in a cow (Steve Hum), Hendra viral pneumonia in an adult thoroughbred horse (Geoff Mitchell) and Lyssa viral encephalitis in a flying fox (Graeme Fraser).

Flaky skin in a nude mouse

Malcolm France, Department of Veterinary Anatomy and Pathology, University of Sydney

A 4 month old athymic (nude) mouse had developed dry, powdery, scaly skin over most of its body and particularly on the dorsal midline; it also appeared depressed and so was euthanased. A cage male which had recently developed similar, although less extensive, skin lesions had been isolated and recovered without therapy over 7 to 10 days. Histological examination of the skin revealed moderate to marked hyperkeratosis, moderate acanthosis and a mononuclear infiltrate in the dermis.

The clinical and histological findings here are consistent with scaly skin disease of nude mice. Investigation of field cases, experimental transmission and molecular studies suggest that *Corynebacterium bovis* is important in the development of this disease. It is claimed that diagnosis can be assisted by observing small coryneform bacteria in the stratum corneum. However, in my experience, it is very difficult to be certain whether such bodies are bacteria or something else such as keratohyaline granules, and often the stratum corneum stains heavily in a non-specific manner making it difficult to see bacteria even if they are there.

Culture is necessary for confirmation although PCR for *C. bovis* is offered commercially by some rodent diagnostic labs in the USA. Subclinical infection and contaminated fomites appear to be important in the transmission of this organism.

Suspected pestiviral enteritis in a calf

John Glastonbury, Elizabeth Macarthur Agricultural Institute

Fresh and fixed tissues were submitted from a 1 month old Angus bull calf, which had been recumbent and aggressively treated with fluids for 2 days prior to death.

Significant histological findings were restricted to the jejunum and ileum. They consisted of moderate congestion, multiple small foci of acute necrosis in the lamina propria towards the tips of the villi, mild hyperplasia of the crypts of Lieberkuhn, severe acute diffuse apoptosis of mature small lymphocytes in the Peyer's patches and acute segmental necrosis of the dome epithelium. The foci of necrosis in the lamina propria appeared to be associated with necrotising microvasculitis, and the "medullas" of the patches consisted of sheets of large reticulum-like cells.

As pestivirus was high on the list of aetiological possibilities, fresh spleen was subjected to the pestivirus antigen capture ELISA (PACE). Alas, there was a negative result.

Fortunately, the owner of this herd is a veterinarian and she had been undertaking serological testing in an endeavour to identify and cull pestiviral carriers. The dam of this calf gave a reaction of 3 in the GDPT for pestivirus when tested in March 1998; the calf was born in early October 1998. My theory is that the calf was infected in utero during the first trimester of gestation and became immunotolerant to pestivirus. Superinfection with cytopathogenic pestivirus postnatally would explain the pathological findings.

In our experience, tissues from such animals are often negative in the PACE because of the high level of maternally derived antibody; lithium heparin blood is the preferred specimen.

Sulphur poisoning in cattle

John Glastonbury, Elizabeth Macarthur Agricultural Institute

A dairy farmer was ad lib feeding his herd with a 15% pelleted ration along with ammonium sulphate as a limiter. Several animals became depressed and staggery and exhibited gut pain before becoming comatose and dying. Gross post mortem findings included a high percentage of concentrate in the rumen, hepatic congestion and inflammation of the abomasum.

The kidney was the only organ, out of a range examined, to reveal histological lesions. In the medullary tubules, presumably Henle's loops, there were many pale basophilic amorphous uroliths, associated with acute segmental necrosis of the neighbouring renal tubular epithelium; the lumens of the distal convoluted tubules appeared dilated.

Fresh kidney was found to contain 1310 mg/kg of sulphur; there was <0.05 mg/kg of the commonly used sulphonamides. D-lactate estimations on material from some of the other cattle confirmed lactic acidosis as the cause of their deaths.

There would appear to have been a mixing problem with the ration, leading to a "Catch 22"! If an animal ingested a nidus of the limiter, it died from sulphur poisoning, whereas if it did not ingest the limiter, it succumbed to ruminal lactic acidosis!

***Malva parviflora*, marshmallow poisoning in sheep**

John Glastonbury, Elizabeth Macarthur Agricultural Institute

In late September, 6,000 quality fat lambs were held at Quirindi in small paddocks over the weekend, prior to transportation to Goulburn on Wednesday for sale. Despite traversing half way across the state, the agent refused to take delivery of the lambs, because in his opinion, they looked "crook". Well, following the return journey on the Friday, they really did look "crook", and about 30 died. Clinically they were staggery and incoordinate, with prominent muscular tremors; treatment with "calcigol" intravenously and subcutaneously had little effect.

Five serums submitted to the laboratory yielded: AST, 551 to 5920 IU/L; CK, 3408 to 30340 IU/L; BUN, 6 to 36 mmol/L. Mild acute segmental ischaemic renal tubular necrosis associated with myoglobin-like casts was found histologically. Unfortunately, no skeletal muscle was submitted.

Further questioning found that the small holding paddocks at Quirindi contained considerable *Malva parviflora*, which had been eaten out over the weekend. Ingestion of *M. parviflora* causes a primary degenerative myopathy but the nature of the actual toxin is unknown.

Yersiniosis in pigs

John Glastonbury, Elizabeth Macarthur Agricultural Institute

In a 15 sow side-line piggery at Gundagai, pigs were observed to have diarrhoea, lose weight and die within 2 to 3 weeks of weaning at 6 weeks of age.

Severe acute segmental erosive necrotic enteritis and colitis, associated with characteristic microabscesses containing prominent bacterial colonies, were detected histologically. Profuse growths of *Yersinia pseudotuberculosis* were recovered from all segments of the intestinal tract cultured.

Clinical disease in pigs due to *Y. pseudotuberculosis* infection is relatively rare, having been reported in the literature from Victoria, New South Wales, Great Britain and Argentina. At a laboratory in Queensland, 1.5% of routine diagnostic intestinal samples from pigs yielded *Y. pseudotuberculosis*, while in Germany, sampling of pigs at slaughter found the organism in 0.5% of 1,098 rectal samples and in 5.8% of 480 tonsils.

Cerebellar abiotrophy of fine wool Merino Sheep

Paul Gill, Regional Veterinary Laboratory, Wollongbar

Each year, 0.25% of 1,200 adult, fine wool Merino ewes on a property in the Glen Innes district of New South Wales became increasingly incoordinate and ultimately died. Histological changes in the brain of a typical case included a loss of Purkinje cells and a moderate proliferation of Berman's glia. The occasional shrunken, eosinophilic Purkinje cell was evident. There were a few eosinophilic spheroids in the cuneate nucleus.

These changes are similar to those described in fine wool Merino sheep in the Yass district of NSW by Peter Harper *et al*, 1986[1]. There the disease was colloquially known as Yass ataxia. Stud rams from the Yass district had been used in the Glen Innes flock.

Lead Intoxication in cattle grazing in a paddock containing an ore treatment plant

Barbara Moloney, RVL Orange

Fifteen ex 50+ mixed age/sex Hereford cattle died over a period of months after exhibiting signs of dullness, recumbency and tooth-grinding. The animals were in light to fair condition and no prominent neurological signs were reported. The cattle had access to some areas of an ore treatment plant which had a stockpile of ore, processed ore concentrates and tailings. The lead content of the ore may have been up to 20%.

Blood lead levels were reported by the NSW Agriculture Diagnostic and Analytical Services, Wollongbar, to be in the range of 0.16 to 0.88 mg/L for samples submitted in EDTA (mean 0.66 mg/L) and in the range of 0.48 to 2.0 mg/L for samples submitted in Lithium Heparin (mean 1.1 mg/L). Normal blood lead levels for bovines are reported as <1.2 micro mols/litre (BVA Veterinary Laboratory Data) which converts to 0.25 mg/L. Subsequent tissue analyses had results varying from 150 mg/kg in faeces to 50 and 11 mg/kg in two tissue samples.

Laryngeal Chondritis in a Texel cross ram

Barbara Moloney, RVL Orange

A 16-month-old Texel cross ram presented with recent onset of signs of respiratory obstruction, became severely distressed when caught and died en route to the vet. Two 'purebred' Texels had died with similar clinical signs in the previous 15 months.

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[1] Harper, PAW, Duncan DW, Plant JW and Smeal MG. Cerebellar abiotrophy and segmental axonopathy: two progressive ataxia of Merino sheep *Aust vet J* 1986; 63: 18-21.

STATE REPORTS SOUTH AUSTRALIA - RUTH REUTER

Chronic glomerulopathy in rats

John Finnie, Veterinary Division, IMVS, Adelaide

Five 6-8 week old male, Sprague-Dawley rats presented with unusual necropsy findings. There was marked ascites and hydrothorax, the former causing obvious abdominal distension, and the kidneys were bilaterally enlarged, soft and uniformly white on both capsular and cut surfaces. Microscopically, there was a severe, diffuse glomerulopathy involving nearly all glomeruli to varying degrees. The glomerular lesion was characterised by marked thickening of glomerular and, to a more limited extent, capsular basal laminae with occasional crescent formation. Bowman's space was frequently obliterated or less commonly the glomerular tuft was atrophic; the tuft was sometimes partially hyalinised. Numerous proteinaceous casts were found in dilated tubular lumina and the interstitium was oedematous with lymphocytic infiltration.

The cause of this glomerulopathy, which only occurred in a few rats on one occasion, was not determined, although testing for immune complexes was not performed. Chronic progressive glomerulopathy is recognised in older (>12 MO), male, Sprague-Dawley rats with proteinuria but our animals were young and it is difficult to attribute the abundant clear fluid in body cavities to hypoproteinaemia alone.

Feline oxalate urolithiasis

John Finnie, Veterinary Division, IMVS, Adelaide

Over a 12 month period, 6 DSH cats ranging in age from 3 to 18 months presented with weight loss and intermittent haematuria and clinical pathology suggesting renal disease. At necropsy, the kidneys were reported to be pale, very firm and lobular in appearance. Microscopically, there was marked dilatation of numerous tubular lumina by refractile, crystalline material resembling oxalate and fewer proteinaceous casts. The tubule epithelium was often degenerate and attenuated and sometimes mineralised. Interstitial fibrosis was patchy and inflammation scant. The widely distributed calculi were best appreciated under polarised light. No calculi were found in the bladder or on microscopic examination of urine sediment.

In cats, struvite calculi are most frequently found with oxalate, occurring as calcium oxalate, rather uncommon. However, although acid urine favours oxalate formation, the mechanism is unclear. The cause of urolithiasis in these cats was not determined, but may have a genetic basis. In man, oxalate nephrosis may be primary (inherited in an autosomal recessive manner) or acquired (ingestion of oxalate precursors such as ethylene glycol or large doses of ascorbic acid, pyridoxine deficiency or a complication of gastrointestinal disorders).

Avian encephalomyelitis in chickens

Julia Lucas - Veterinary Pathology Services, Adelaide

A commercial egg producer reported nervous signs and death in 3 week old chicks. Several birds had become weak and unable to stand. The clinical signs progressed to lateral recumbency with whole body tremor and death. Brain and heart in formalin were submitted to the laboratory.

Microscopic examination of the brain revealed a diffuse non-suppurative encephalitis affecting all areas of the brain, with the most severe lesions observed in the brainstem and cerebellum. There was widespread spongiosis and gliosis, degeneration of neurons characterised by chromatolysis and vacuolation of neuron bodies. In the neuropil there was formation of spherules, axonal swelling and myelin degeneration. Lymphoplasmacytic perivascular cuffs and lymphoplasmacytic foci were also observed in the neuropil. In the molecular layer of the cerebellum there were *flame* lesions. These were linear foci of gliosis arising from and perpendicular to the Purkinje layer. This lesion is considered characteristic of avian encephalomyelitis. In the heart, there were multiple foci of lymphocytes infiltrating the myocardium.

The lesions in the brain and heart were consistent with avian encephalomyelitis. This disease is caused by infection with picornavirus[2], [3] and primarily affects birds under 6 weeks of age[2], [3]. After 6 weeks of age, birds develop resistance to the virus and do not exhibit clinical signs[2]. Transmission of the virus may be vertical or horizontal, but outbreaks in commercial flocks are usually initiated by breakdown of vaccination regimes in the parent birds.

Careful consideration was given to the differential diagnosis of vitamin E deficiency and Newcastle disease. The lesion in this case was clearly inflammatory which excludes vitamin E deficiency. Newcastle disease may produce a range of lesions in the central nervous system including a non-suppurative encephalitis. However, Newcastle disease tends to produce severe, haemorrhagic lesions in the brain and other viscera[3]. In this case, the typical lesions of avian encephalomyelitis included focal clusters of glial cells and focal clusters of lymphocytes in the myocardium. The “flame lesions” in the molecular layer of the cerebellum were pathognomic.

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[2] Ritchie BW, Harrison GJ and Harrison LR (1994). Avian Medicine: Principles and application, Chapter 32, Viruses. Wingers, Florida.

[3] Riddel C (1987) Avian Histopathology. American Association of Avian Pathologists, Saskatchewan, Canada.

STATE REPORTS TASMANIA - ROY MASON

Uronema infection in a Spotted Handfish

David Taylor, Animal Health Laboratory, DPIWE, Mt Pleasant, Tasmania

A Spotted Handfish (Brachionichthys hirsutus) from the CSIRO breeding program was submitted with respiratory distress and sunken eyes.

Their epidermis was fragmenting and missing in many areas and was replaced by haemorrhage inflammatory infiltrate. There was considerable diffuse inflammatory response within the muscle and subcutaneous tissue consisting of both mononuclear and polymorphonuclear cells, congestion and haemorrhage, oedema, muscle fibre degeneration and necrosis. Occasional bacteria were also present in the area of the Uronema parasites.

Parasites were also present within the spinal cord, brain and interstitial tissue of the kidney. The eye appeared to be particularly heavily infected with parasites present within the choroid, retina, iris and cornea. The lamina propria and muscular serosa of the gut were also infected with some oedema and thickening. There were parasites within the liver with resultant cellular necrosis but little inflammatory response. There was congestion of the secondary lamellae, epithelial hyperplasia and degeneration, inflammatory infiltrate into the basal epithelia and Uronema attached to and within the epithelium.

Uronema ciliates are generally regarded as commensals but have been recorded to cause losses in aquaculture species, notably Southern bluefin tuna. This case was one of a number of handfish diagnosed with Uronema infections and probably relates to the management of the aquaria.

Intervertebral and spinal abscessation in chickens

David Taylor, Animal Health Laboratory, DPIWE, Mt Pleasant, Tasmania

Four chickens of five and a half weeks of age were presented with loss of mobility. There were no gross lesions internally or associated with the sciatic or brachial nerves. Histologically, there was no evidence of Mareks disease in the spinal cord or the brain, nor was there any evidence of encephalomalacia or encephalitis. Within the cervical vertebral column, one bird had an epidural abscess, spinal meningitis, spinal cord degeneration and localised spinal leukomalacia with a mixed, mainly mononuclear inflammatory infiltrate and clusters of coccoid organisms. A mononuclear perivascular response was also observed. Within another vertebral section there was intravertebral fibromyxomatous marrow with multicystic cavitation (some containing pink staining material) which was determined to be inflammatory response in the region of an airsac (thanks to Rod Reece). A second bird had an intervertebral abscess of the cervical vertebra and intense mononuclear perivascular infiltrate of the epidural vessels. Many coccoid bacteria were visible within the abscess.

Although cultures were not performed, the bacteria closely resembled staphylococci which have been recorded to cause such lesions and which have been implicated in similar cases recently at this laboratory.

Feline non-suppurative meningitis

Phil Ladds, Animal Health Laboratory, DPIWE, Mt Pleasant, Tasmania

A 10 month old castrated male Burmese had for 6 weeks exhibited signs of nervousness, head pressing and ataxia with petit mal seizures and loss of weight. Fever was never observed and haematological parameters on several occasions were normal although there was slight elevation of globulin.

No gross changes were apparent but microscopically there was extensive lymphoplasmic infiltration of the meninges of most areas of the brain with pronounced perivascular cuffs. Similar infiltrates were in the choroid plexus. A non-suppurative meningitis, possibly of viral origin, was diagnosed. Serum from the cat was negative on ELISA for Ross River Virus and Flavivirus.

Summers *et al* (1995) in Veterinary Neuropathology (page 156) note that "… examples of pure meningitis of viral cause, so-called aseptic meningitis in humans, are not recognised in animals." Absence of non-CNS lesions in this cat ruled against coronavirus infection or a lymphoproliferative cause.

STATE REPORTS VICTORIA - MALCOLM LANCASTER

Bovine dermoid cysts affect beef exports to USA

Malcolm Lancaster, VIAS Attwood

Container loads of Australian beef are periodically rejected by US authorities on the grounds of "faecal contamination". Australian authorities now suspect that at least some of this "faecal" material is actually the content of dermoid cysts.

One abattoir in Victoria that "hot bones" the neck musculature is reporting several of these cysts each week. Each cyst is 30mm to 40mm long, and filled with a wad of greenish fibrous material. The fibres are shed hairs, and the wall of the cyst contains pilosebaceous units, tubular glands and melanocytes beneath a typical stratified squamous epithelium. Because these cysts are located deep in the neck musculature, between muscle groups, routine "cold boning" does not detect them.

Have other people seen these cysts? Does anyone know their pathogenesis? One suggestion is that epidermal implantation may occur with some cervical injections. Alternatively, the cysts may be developmental anomalies.

Pilchard mortalities

Peter Hooper, CSIRO Australian Animal Health Laboratory, Geelong

I guess everybody will have heard there has been another outbreak of deaths in pilchards along the southern coastline. There are striking similarities in the pattern of the epidemic, especially its origin off Eyre Peninsula, then fanning out west and east. There are variations, such as involvement of juveniles, which were not evident in 1995. An interesting development was to see lesions in advance of the main wave. Fish were examined that were taken from near Lakes Entrance in eastern Victoria and well away from the visible front at the time, approach Port Phillip Bay from the west. One in a group of 4 that were suitable for histology had early lesions. The main epidemic then reached that area, 4 to 5 weeks later, a "visible-epidemic incubation period". This would be the time allowed for virus to firstly build up in the fish then secondly, attain a visible surplus after consumption of early mortalities by scavenging fish.

The lesions in the gills are the same as in the 1995 epidemic. Juveniles have essentially the same proliferative and infiltrative lesions. There seem to be more advanced atrophy of secondary lamellae and stunted primaries this time as well. In a way, this is consistent with a lesion in a longer-surviving fish and correlates with far fewer herpesviruses seen by Alex Hyatt, our EM man. One could postulate that there has been some natural selection.

There are numerous incidental other lesions, e.g the usual parasites, although there seem fewer amoebae in the gills. The most common other lesion, nearly as frequent as the gill virus, is the one affecting a variety of organs. The lesion is quite fascinating and needs more evaluation, so watch this space!

Preferred tissues for Newcastle disease immunodiagnosis

Peter Hooper, CSIRO Australian Animal Health Laboratory, Geelong

We have made unpublished studies of Newcastle disease that show the pathogenesis does not fit nicely with the traditional tropisms. From a pathologist's point of view, the viscerotropic virus has its principal initial action in lymphoid tissues and small blood vessels. At a later stage, if there is survival, which is rare, there is invasion to many organs. The significance of the term viscerotropic is visible necrosis and haemorrhage in caecal tonsils and other alimentary lymphocyte accumulations. The neurotropic virus is so named because the nervous disease is the killer. The actual disease is a complex starting in the conjunctivae and respiratory system, then if virulent, progressing to a number of organs, especially the heart, kidney, CNS, peritoneum, pancreas, and if sufficiently virulent, the bone marrow. Immunohistochemical reactions indicate far more virus antigen in organs other than the CNS. These results suggest preferred organs for NDV diagnosis, as follows:

If there seems to gastrointestinal involvement:

- include caecal tonsil, spleen and bursa
- but as these 'viscerotropic' viruses are so virulent and invasive, diagnosis can be effected in a variety of organs.

If neurological or respiratory:

- prefer trachea, lung, heart, kidney, pancreas (peritoneum then automatic) - but still include CNS

Whatever, if there are high mortalities:

- include bone marrow - if for immunohistochemistry, this could be a piece of fixed vertebral column to portray both spinal cord and bone marrow.

The selection of these patterns of organ tests has been proved useful in studies in the past of respiratory disease in Australian broiler chickens, and in the 1998 NSW disease outbreaks.

Remember too that the full work up of an NDV case involves serology and virus isolation.

Sera from convalescent birds is the ideal, but at least sera from 20, and preferably 30, birds in the vicinity of the sick birds. If disease in young or newly introduced birds on a farm with multiple age groups, then also include 20, and preferably 30, sera from each of the other age groups.

Virus can be isolated from the organs that show positive IPX reactions, and in the recent outbreak was isolated from kidneys and bone marrow (say a thigh bone) as well as CNS and other organs.

However, the standard sample for NDV virus isolation is swabs, both cloacal and tracheal. Preferably at least 6 birds, or even 10, should be swabbed. Swabs are usually submitted in transport media, but if none is available, still take the swabs. So long as the swab is moist the virus will quite happily survive the 1 to 2 day trip to AAHL.

Iridovirus virus infection in snakes

Mark Williamson, CSIRO Australian Animal Health Laboratory, Geelong

Ten Chondra pythons were illegally imported into Australia. Two were found dead on arrival. The remaining eight were subsequently necropsied with no abnormalities detected on gross or histopathology examination. At necropsy, the two found dead were in poor body condition with large ulcers of the buccal mucosa.

Nematode parasites were found in the nasal cavity. There was erosion and ulceration of the oral mucosa without a significant inflammatory infiltrate. There was severe diffuse periacinar degeneration of the liver with extensive areas of hepatocyte necrosis. Cytoplasmic vacuolation was present in the remaining hepatocytes. Numerous intranuclear inclusion bodies, possibly but not distinctly of viral origin, were observed in hepatocytes. There was focal peracute tubular necrosis in the kidney and increased numbers of free alveolar macrophages in the lung. Findings in the second snake were similar to the first although the liver lesion was milder with only occasional focal areas of hepatocyte necrosis. Also, there was severe necrotising inflammation of the pharyngeal submucosa with numerous macrophages, eosinophils and oedema.

Pooled tissues were prepared and inoculated onto confluent monolayers of Vero, VH-2, gecko lung, chinook salmon embryo and blue fry cells.

A cytopathic agent was identified in cell culture from the two dead snakes, but not from the other eight snakes. On electron microscopy, the agent was identified as belonging to the Iridoviridae, but was not epizootic haemopoietic necrosis virus (EHNV).

In the two snakes found dead, positive immunostaining using a rabbit polyclonal antibody against EHNV was found in the following tissues; skin, liver, heart, lung, kidney, stomach, small intestine, nasal and oral epithelium. Immunostaining was observed in a variety of cell types including vascular endothelium, fibroblasts and osteoblasts and in nasal exudate.

Vasculopathy in a Jack Russell Cross Terrier

Dr Judith S. Nimmo Wilkie, Victorian Veterinary Pathology Services

An eight-month-old Jack Russell Cross terrier was presented with a history of crusting, alopecia and pitting oedema of the ear and tail tips which had been present since she was a little pup.

Skin biopsies revealed a chronic hyperplastic dermatitis which was alternately ortho- and para-keratotic. There was subepithelial vacuolation and some vacuolar degeneration of the basal layer with focal thickenings of the basement membrane. Rare necrotic basal cells were present. The dermal lesions were striking; dermal oedema and fibrosis, very marked pigmentary incontinence and a diffuse mononuclear cell infiltrate. Vascular lesions consisted of fibrinoid changes in some superficial capillaries.

The histological changes suggested repeated episodes of basal cell damage probably due to ischaemic episodes.

The lesions were similar to those described recently in Jack Russell terriers in Canada. The condition is believed to probably be familial and possibly a condition resembling dermatomyositis of collies.

W.M. Parker and R.A. Foster. Cutaneous vasculitis in five Jack Russell Terriers: Vet. Dermatol (1996) 7, 109-115.

STATE REPORTS WESTERN AUSTRALIA – DAVID FORSHAW

Big Head in Horses

Jeremy Allen, AHL, Agriculture WA, South Perth

Tony Tully and Matt Bolam from Kununurra recently investigated a possible case of big head in horses on Springvale Station near Halls Creek.

The station manager reported that two stock horses had changes in the shape of their skulls. He suspected big head from previous experience in Queensland. A gelding and a mare in excellent condition were examined with similar, almost symmetrical bony protrusions across the face between the eyes. The protrusions were firm, but not painful. There were no other problems apparent. Samples of fresh growth from birdwood and buffel grass plants were collected from the horse paddock.

This condition is a form of chronic oxalate toxicity that may occur when horses graze on various introduced tropical grasses that contain oxalate (donkeys may also be affected). The oxalate precipitates with the calcium in the diet, reducing calcium absorption, and disturbing the absorbed Ca:P ratio. This results in a nutritional secondary hyperparathyroidism and a mobilisation of bone mineral to alleviate the hypocalcaemia (thus blood calcium concentrations are usually normal). This in turn leads to fibrous osteodystrophy and swelling of the heads of affected horses.

Hazardous pasture grasses contain >0.5% total oxalate and have a calcium:total oxalate ratio of <0.5 (DW basis). Grasses that have been associated with this condition include buffel grass (*Cenchrus ciliaris)*, green panic or guinea grass (*Panicum maximum*), setaria pangola grass (*Digitaria eriantha spp. pentzii*), signal grass (*Brachiaria sp.*) and purple pigeon grass (*Setaria incrassata*). In the current investigation, the birdwood (*Cenchrus setiger*) and buffel grasses contained 1.01% and 1.60% oxalate (DW), respectively.

Big head occurs when the offending grass makes up all, or most, of the feed on offer. The condition usually takes 6-8 months to develop, but can occur in about 2 months. The most frequent presenting signs are lameness (not present in the current case) and ill thrift (the horses presented in the current case were in good condition!!), the latter occurring even though the pasture is apparently nutritious (sheep and cattle will probably do well on it - rumen bacteria break down these small intakes of oxalate). The jaw bones become swollen in the advanced stage of the disease. Mares and foals are more susceptible because of their increased calcium requirement.

Prevention requires avoiding these oxalate-containing grasses. If they can not be avoided, all the time, then horses should graze the grasses for a maximum of 1 month at a time, with intervals of 3-4 weeks in between. If long term grazing of the grasses is unavoidable, then the horses must be given a balance calcium and phosphorous supplement.

Contact: Tony Tully and Matthew Bolam - 08 9166 4000; Jeremy Allen - 08 9368 3466

Flying Scapula of Weaner Cattle associated with Vitamin E and Selenium Deficiency

John Creeper and Martin Robertson, AHL, Agriculture WA, South Perth
Bradley McCormick, Agriculture WA, Busselton

A distinctive clinical presentation in young cattle in which the scapulae become elevated and often meet above the spine has been termed "flying scapula". The condition is the result of degeneration of the skeletal muscles of the axillary region between the scapulae and the thoracic wall. In the United Kingdom, the disorder has been recognised for over thirty years and the one published reported in housed dairy heifers describes an association with vitamin E deficiency[4]. An outbreak in Australia in which over 80% of a group of 95 Hereford weaners became affected, was attributed to selenium deficiency.[5] We have recently investigated an outbreak of flying scapula affecting weaner Murray Grey steers in which both vitamin E and selenium levels were deficient and in which hindlimb skeletal muscle degeneration was also present.

The affected group was composed of 86 mixed sex, predominantly cross bred weaners, 5 to 6 months old. The cattle were weaned and held in a yard and fed hay for 36 hours before being turned out onto a clover-ryegrass pasture. The first case of flying scapula was noticed by the owner 6 days after being turned out and a further two cases detected at closer inspection the following day. All affected weaners were pure bred Murray Grey steers. Veterinary investigation was prompted following the death of the first affected steer 21 days after weaning. The two remaining affected steers were bright and alert and able to run with their herd mates without any apparent difficulty. As they walked, their scapulae were raised above the back and it appeared all weight of the forelimbs was borne by the skin at this point. Their chests were slung low between the forelimbs and almost touched the ground and their back-line sloped down towards the head.

The worst affected steer was killed and necropsy revealed necrosis of the muscles between the scapula and the thoracic wall, and the formation in this area of a large fibrous lined sac containing 3 litres of serosanguinous fluid. The *Latissimus dorsi* and *Biceps femoris* muscles of the hindlimb appeared pale and streaky. The heart showed no gross abnormalities.

Histologically, the muscles of the axillary region had undergone coagulative necrosis giving the suggestion of an infarctive-type aetiology. The muscle attachments to the scapulae were normal for the first centimetre then showed a sharp demarcation into the necrotic muscle. Myodegeneration and necrosis of hindlimb muscles were histologically consistent with a nutritional myopathy. Selenium, Glutathione peroxidase and Vitamin E levels are recorded below:

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[4] Hannam DAR, Holden LR, Jeffrey M, Twiddy N. Flying Scapula of cattle. *Vet Record* 134: 356, 1992.

[5] Allen JD, Friend SCE. Suspected nutritional skeletal myopathy in weaned calves. *Aust Vet J.* 54:547-548; 1978/

Liver selenium	0.10 mg:/kg (normal levels 0.3-1.5 mg:/kg)
Liver Vitamin E	5.66 mg/kg (normal levels 9-44 mg/kg)
Glutathione peroxidase	12 U/g Haemoglobin (normal levels >50 U/g Hb)

Haemophilus somnus pleuritis in feedlot cattle

David Forshaw, Agriculture WA, Albany

Haemophilus somnus was isolated from two steers with severe fibrinous pleuritis from a feedlot at Manypeaks, east of Albany. More than 20 animals have died since the first diagnosis. Lesions consist of severe acute fibrinous pleuritis with extension along interlobular septae into the parenchyma of the lung.

Some animals have died very suddenly without prior signs of illness, but the farmer has treated successfully with antibiotics, animals that he considered were in the early stages of the disease. These animals were identified only by twice daily careful observation of all stock in the feedlot. There is a distinct “danger period”, 3-4 weeks after introduction into the feedlot when animals are likely to show clinical signs. Hot weather resulted in more clinical cases.

Haemophilosis in cattle has previously only once been confirmed in WA by Agriculture WA labs and this was also as a pleuritis syndrome in cattle from a feedlot at Margaret River. The main syndromes described with *H. somnus* infection are thromboembolic meningoencephalitis and myocarditis but pleuritis is also mentioned in some texts. I’d be interested to know if others are seeing a pleuritis syndrome associated with the infection.

Polioencephalomalacia in an adult ostrich

David Forshaw, Agriculture WA, Albany

Polioencephalomalacia was diagnosed in an adult ostrich on dry pasture being fed grain. The lesions consisted of acute laminar cortical necrosis in the cerebrum. There was also a focus of blood vessels medial to the necrotic lesions which showed marked perivascular accumulation of intensely eosinophilic homogeneous material, presumably plasma. The cause was not determined.

A progressive ill thrift syndrome followed by death is also being experienced in the same group of birds and necropsies of two have shown marked iron pigment accumulation in gut and liver as the only significant lesion.

Suspect urea poisoning of seagulls

Clive Huxtable, Murdoch University

Apparently, urea was being unloaded from a ship in Fremantle and was spilt on the wharf. It then rained and the urea dissolved in puddles. Shortly afterwards, 30-40 seagulls were found dead or dying. Three dead seagulls were submitted by the Department of Environmental Protection together with a sample of the puddle water which had been kept in a jar in the fridge overnight, together with the dead birds. At necropsy, all birds were in good condition and there were no significant gross abnormalities, apart from a moderate quantity of catarrhal material throughout the small intestine. A small amount of fluid was obtained from the ventriculus of two birds and was analysed for urea along with the water in the jar. The fluid contained 308 and 382 mM urea respectively, while the water contained 4M urea and its pH was 5.5. There were no significant histologic changes in a range of tissues including the brain.

It was concluded:

- That large amounts of urea had dissolved in the puddles but no significant conversion to NH3 had taken place at that stage.
- That the birds had indeed drunk the water.
- Gross and histologic evidence of urea/ammonia poisoning as seen in mammals was not present, but that the intake of urea had probably killed the birds, and that therefore 4)4M urea is an effective gullocide. Does anyone have any information on urea poisoning in avians?

Multicentric and epitheliotropic lymphoma in a dog

Clive Huxtable, Murdoch University

A 14-year-old male castrate Cocker Spaniel had a 2 month history of progressive severe skin disease and general deterioration in condition. Liver ultrasound revealed enlargement and some nodularity. In the last two weeks, it developed severe dyspnoea, and general lymphadenomegaly. Clinical DDX included hepatocutaneous syndrome and metastatic neoplasia. Euthanasia was performed. At necropsy, there were numerous crusting ulcerating lesions of the skin and oral muco-cutaneous junction. Superficial lymph nodes were enlarged, pale firm and homogenous. The spleen was enlarged, pale and “meaty”. The lungs had bilateral dorso-caudal pale “meaty” consolidation (it looked like pneumocystis pneumonia). The liver was diffusely enlarged with some mild nodular remodelling.

Histologically, the diagnosis was lymphoma with an extensive epitheliotropic muco-cutaneous pattern, and diffuse pulmonary infiltration together with more conventional hepatic, splenic and lymph node involvement.

The case is unusual:

- because systemic involvement in cutaneous lympho of this type usually occurs very late after a long skin disease
- because of the diffuse pulmonary infiltration, and
- because extension of systemic lympho to the skin form found is not reported to my knowledge. It just goes to prove the old saying “lympho can do anything”.