VETERINARY PATHOLOGY REPORT

Number 65 June 2003

President's Report

Details of the activities of your new management Committee are given in the secretary's report below. I congratulate Barry Richards on making significant progress with funding for the registry and proficiency testing programs. Let's hope this gets sorted out in the next few months. The value of the registry has recently been demonstrated by the traveling slide show hosted by Clive Huxtable. You must get tickets to this show when it comes to your state.

Your committee plans to meet regularly. I therefore encourage you to be active and to communicate regularly with us so that we can discuss issues of importance in a timely manner. E-mail is the best means of communication (see Committee report for e-mail addresses of Committee members) as it allows rapid sharing of documents. When we work out how that part of the website works, we will be harassing you until you are all logged onto the facility that will make it possible for us to send you unsolicited e-mail. In the meantime, you must actively visit the website (http://www.asvp.asn.au/) for updates and of course, the Vet Pathology Report.

Richard Miller
Honorary President

Editorial/Secretary's Report

The hiatus between this and the last issue has to do with the rather untidy transfer of the ASVP Management Committee from South Australia to Queensland. No nominations were received for membership of the committee in time for the annual general meeting in April, and to compound the problem, none were received at the meeting itself. So various Queenslanders had to be bullied into the positions: this unorthodox procedure is at variance with the ASVP constitution, which of course requires elections to be held. If there is a motion to throw out the unconstitutional committee, it can be put to a postal vote or it may be put it on the agenda for the next AGM, but, given the demonstrated apathy of the rank and file in the matter of nominations, we are not expecting much activity on that score.

So the management committee as now installed is:

President - Richard Miller (richard-miller@idexx.com)
Secretary - Roger Kelly (roger.kelly@mailbox.uq.edu.au)
Treasurer - Anita Gordon (anita.gordon@dpi.qld.gov.au)

Committee members:

Mark Carrigan (mark-carrigan@idexx.com)
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At present there is no convener for the Slide of the Month program. It may be that we will be able to run it as a component of the Quality Assurance/proficiency program should that receive financial support (see below).

The Scientific Meeting of the Society was held on April 12th & 13th at the Elizabeth MacArthur Agricultural Institute, Menangle NSW, immediately after a successful 3 day training course in gross pathology for field veterinarians organised by staff at the EMAI and the Postgraduate Foundation in Veterinary Science. Members are urged to visit the ASVP web-site for the Abstracts booklet for the ASVP meeting. Should they be interested in obtaining a copy of the CD that is to be made available to participants in the Gross Pathology course, they should be able to do so by contacting the Foundation.

The annual business meeting of the Society was held on April 12. A full set of minutes will be published before the next AGM, but issues that were raised included the venue and timing of the AGM for 2004. The Society has been invited to hold its meeting jointly with the big International Academy of Pathology meeting which is to held in the Brisbane Convention Center from October 10 - 15th. A tentative proposal is to hold our two day meeting with the first day on the 15th, which the IAP is at this stage planning to keep for veterinary contributions, and have our usual member contributions (short papers) the next day by ourselves, when we can also hold our annual general business meeting. So pencil in October 15th and 16th, 2004. If you can get to Brisbane earlier, you might want to attend some of the general IAP sessions: to that end, we will try to keep you informed of the detailed program as it coagulates, and also some idea of daily registration costs.

There is a move to hold another joint meeting the following year, with the World Association of Veterinary Laboratory Diagnosticians, should they hold it in Australia in 2005. At this stage Melbourne and Cairns are bidding for the conference. Again, we will keep you posted.

Phillip Ladds was presented with a certificate of Life Membership of the Society; a decision that was approved by acclamation.

Discussion was again had on the matter of connection with the Australian Veterinary Association. Your new executive will actively explore ways of increasing our ties with the rest of the profession. These will include examining ways of coordinating our activities with those of the Australian College of Veterinary Scientists (see President's message in this issue).

Your next annual subscription has been increased to $45 for Australian members ($55 for overseas members).

Members voted to lobby SCAHLS to lobby the Animal Health Committee for financial support for the National Registry of Animal Pathology and the Quality Assurance/Proficiency programs run by the Society and the Registry. Members are urged to read the Turner Review of NRDAP (on the ASVP website) if they have not already done so. Late breaking news has just come in from Barry Richards, and his report is given below under Continued funding….

Other matters: The ASVP website was temporarily dormant while Cleve Main was away, but one of our objectives is to have all members subscribe to it so that it can be used for instant communication, as was so effectively demonstrated with Pathmail for those of us who were able to subscribe to that notice board when it was set up by the Western Australians a few years ago. The following advice is from Cleve:

"Members should subscribe and they can do so through the web-page or by sending an empty e-mail to:
asvp_central-subscribe@asvp.asn.au

They will then receive a reply asking them to confirm their intent and, having done that, will receive an e-mail of welcome - much the same as Promed.

To send a message to all, use the address asvp_central@asvp.asn.au and it should go to everyone who has subscribed. Of course they may be a few glitches (as every one knows, Murphy is alive and well and his law still operates). Note that if you reply to an e-mail and want it to go to all members, click on "reply to

all" at the top of the screen. To reply or respond to one person only, just click on "reply". We hope you will in fact "reply to all". That way we all benefit.

In the unlikely event of you ever wanting to drop off the mailing list (unsusbscribe), send an empty e-mail to:
asvp_central-unsubscribe@asvp.asn.au

As more of our members subscribe to the mailing list, I as webmaster can issue you bulletins regarding the ASVP web page and your ASVP Executive can use it to keep you up to date with current events. We hope that it will eventually replace pathmail (for ASVP items only)".

From the Secretariat: Barbara Gill wishes to remind members that credit card payment of subscriptions is only available for overseas members (not including Tasmania!). This is not Ludditism; it saves a good deal of trouble and expense for the Secretariat and thus helps our subs go further, and it really isn't a lot of trouble to write a cheque or buy a postal money order. If you insist on using a credit card, then a surcharge will have to be levied. We will look into electronic transfer and see if that would be cost-effective. Some members ask Barbara for a for a tax invoice with the GST component on it, but we do not charge GST, so please remember that. Note that she also no longer sends receipts.

Case Reports from Members. The pathogenesis of the dreadful deficiency in this issue of State Reports from members is no doubt multifactorial. We Queenslanders can hardly carp about it, since we haven't produced anything at all. But I urge us all to exdigitate for the next one.

Continued funding for NRDAP, Q/A & Proficiency Testing

SCAHLS, through Barry Richards and Mike Nunn, have been battling for quite a while now for continuing funding for the Registry and the associated QA/Proficiency Testing programs, ever since the Animal Health Australia Review of the NRDAP was announced. The delay was caused primarily by the untimely and tragic death of Terry Nichols, who was to have conducted the review. It took some time to arrange another reviewer but the Turner Report was finally delivered late last year (see the ASVP Website). No action was subsequently taken by AHA, so SCAHLS responded to the recommendations of the review by proposing to Animal Health Committee that a new and vibrant NRDAP should rise phoenix-like from its own ashes and be funded by the States, Commonwealth, CSIRO and AHA through a Primary Industry Standing Committee (PISC) agreement. AHC met by teleconference on June 26 to discuss the proposal and (after considerable debate) approved the concept but with a modified funding equation that excludes AHA and only runs for 12 months (2003-04). The next task is to compose a new agreement for the following years with something to satisfy the AHC call for some private sector contributions. What happens now is that AHC will recommend the 12 month proposal to PISC for its next meeting in August. Barring earthquakes, that should be successful. AHC have called for a revised one year plan to be quickly put to PISC so we can get up and running for 2003-04 (essentially the budget needs to exclude a contribution from AHA who have declined to be involved). But for the years that follow, a more comprehensive plan is needed, with growth built into it and some concrete milestones (e.g. website up and running by Dec 2004, national electronic conference operating monthly by March 05, on-line training modules by June 05, etc). Mike and Barry agree that the NRDAP Management Committee should start the process of appointing a full time registrar with the proviso that it will be 12 months with a possibility (strong, we hope) of extension (4-5 years). The

proposal calls for the appointment of a full time registrar with a brief to completely revamp and modernise the registry, to make all of its resources more accessible using the internet and digital imaging, to organise the annual training sessions and to run the proficiency testing program.

Roger Kelly
Honorary Secretary

Obituary

Barry Laing Munday BVSc, MVSc, DVSc, MACVSc

Veterinary science in general, and the Australian veterinary pathology community in particular, lost a powerful force on May 10 with the death at Launceston of Barry Munday in his 71st year.

Barry's long and productive career was almost entirely served in Tasmania. After primary and secondary schooling in Hobart, he was awarded a Tasmanian Department of Agriculture Veterinary cadetship which enabled him to study at the Sydney Veterinary School, from which he graduated with honours in 1956. He worked first as a field veterinary officer in northern Tasmania, then for 3 years on King Island in the 1950s, in the days when government veterinary officers in more remote areas were called on to perform a wide range of clinical tasks in addition to their regulatory duties. There seems always to have been something of the pioneer about Barry. During his time on King, in collaboration with AK (Sandy) Sutherland, then of the Nicholas Institute in Victoria, he defined cobalt deficiency as a production-limiting problem on the island, and worked on brucellosis control in cattle and sheep.

He began work as a pathologist at the Mt Pleasant Laboratories at Launceston in 1960, learning the hard way in comparative isolation, without the benefit of formal post-graduate course-work in the discipline. However, his native ability and capacity for collaboration served him well, enabling him to benefit from distant mentors such as Bill Hartley, with whom he published a series of papers on neurological and other diseases of production animals. He was also influenced by Keith Meldrum, then Tasmania's Chief Veterinary Officer, who encouraged Barry's interest in wildlife at a time when relatively few veterinarians had such interests: the pioneer again. His establishment of the Australasian section of the Wildlife Disease Association was duly recognised by the WDA with their Distinguished Service Award at their 1985 meeting in Sweden. A Wool Research Trust Fund Post Graduate Scholarship enabled him to conclude a successful Masters research program on ovine toxoplasmosis at the Melbourne Veterinary School in 1970. Back in Tasmania he was successively Senior, then Chief Veterinary Pathologist at the Mt Pleasant Diagnostic Laboratories from 1973 to 1988. He became heavily involved in the Tasmanian salmonid industry at the time of its rapid expansion (and of the appearance of the diseases that dog new intensive animal industries), and this led to a transition to Senior Research Fellow and Reader at the National Key Centre for Teaching and Research in Aquaculture, University of Tasmania. Not only was this an exciting opportunity to transmit his accumulated wisdom to under- and postgraduate students, but (by his own

admission) it enabled him to escape the perils of a purely administrative job which he saw looming in his senior years.

In 1992 he was admitted to the DSc degree by the University of Melbourne on the strength of his published scientific papers, which by that time numbered more than 150 (there were more to come). A long list of international awards, appointments and travel grants (WHO toxoplasmosis workshop Holland 1989; fish health official appointments by US, UK and Canada; 1992 Smithsonian Bilateral study grant; 1994 French bilateral science & technology grant; 1996 guest speaker on virus diseases in marine aquaculture at Hiroshima, Japan; invited paper at the 1996 OECD-sponsored Ecotoxicology workshop in Maui) illustrates his international standing and the breadth of his recognition. And then there were the demands of thesis examination for many universities in Australasia, and memberships of the Australian Veterinary Association (several executive posts in the Tasmanian branch), the Australian Society for Parasitology, Australian Society for Veterinary Pathology, the European Association of Fish Pathologists, and the Tasmanian State Thyroid Advisory committee.

Barry was driven by a relentless energy. In 1998, three years after his coronary bypass surgery, I floundered about in the mud of a stream near Launceston helping this "retired" scientist set nets for platypuses (part of his fungal ulceration study on that species), and remember being more concerned for him than for the animals. It is sad to think that he was not given some time to rest after his strenuous career, but that seems not to have been part of his nature. His tremendous drive notwithstanding, he was very helpful and encouraging to younger colleagues and students, and it must be some comfort to his friends and family that he was able to influence so many of the next generation of scientists in such a positive way.

It is gratifying that in March 2003 the "Munday Lecture" lecture series in Human Life Sciences was named in his honour at the University of Tasmania, and the first lecture was a celebration and recognition of his contributions to science. He is survived by his wife Fay, and by his children Phillip, Paul and Louise, to whom we wish to convey our condolences.

R.K.
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Australian College of Veterinary Scientists Pathobiology Chapter Report

The Australian College of Veterinary Scientists recently held its annual 3 day conference -Science Week- at the Gold Coast. Once again, the Pathobiology Chapter was noticeably absent. In future years it may be beneficial that college members present a half day of lectures on issues that are of interest to both pathologists and clinicians. This will raise of profile of the college members of the ASVP.

In recent years the College has mainly been associated with holding examinations. Most of these have been for membership with several for fellowship. Membership examination is designed to investigate the breadth of knowledge of the candidate. The fellowship examination investigates a depth of knowledge.

Recently, NATA has indicated that there will be a requirement for the main signatory of a NATA-accredited laboratory to have membership of the Australian College as the minimal qualification. I respectfully suggest to all members of the ASVP that they should demonstrate to their peers their prowess in veterinary pathology by sitting for membership unless they have higher qualifications obtained by examination.

The next examinations for membership in pathobiology will be held in June/July 2004. Applications to sit examination must be received by December 2003. Subsequent examinations will be held every two years, i.e. 2004, 2006, 2008 etc.

For those of you who are interested, membership examinations are in two parts. The written examination in June covers general pathology and veterinary pathology with emphasis on significant endemic and exotic diseases as well as topical issues. The second part in July covers practical aspects of veterinary pathology including slide review, gross pathology and an oral examination.

For more information please contact myself,
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Roger Cook(roger.cook@agric.nsw.gov.au)

Richard Miller
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STATE REPORTS

NSW – Paul Gill

Dermal Vasculitis in slaughtered pigs

Paul Gill & John Boulton
Regional Veterinary Laboratory, Wollongbar NSW

History: One of 200 baconer Large White pigs from one herd and one of 81 porker Large White gilts from another herd had widespread blotchy cutaneous haemorrhages particularly over the hindquarters. Both pigs appeared normal at ante mortem inspection. These cutaneous haemorrhages were evident after depilation and scalding. The viscera appeared normal to both the meat inspectors and the abattoir's veterinarian.

Gross Pathology: In both pigs , the submitted skin portions were mainly uniformly red or congested dull red with a few randomly distributed ecchymoses (up to 1 cm in diameter). The redness extended into the hypodermis. Muscles, fat: appeared to be normal in colour and consistency. The dermal lymph nodes: were normal in size, shape and consistency. On section, the cortices were dull red.

Histopathology: Skin (3 portions): Much of the epidermis is missing and the upper dermis is reduced to a pink coagulum peppered with nuclear debris as a result of scalding and depilation post mortem. There is marked vasculitis in the dermis characterised by segmental to circumferential fibrinoid degeneration of the tunica media of arterioles with moderate infiltration of granulocytes into the affected segments; and perivascular accumulation of leucocytes. There are intraluminal deposits of fibrin and thrombosis frequently with partial occlusion of the lumen and recanalisation. There are randomly distributed haemorrhages into fat and connective tissue. These changes extend into the upper dermis. In one section of skin, vascular proliferation and perivascular fibrosis are evident.

Lymph node (2 nodes): In one lymph node, the subcapsular sinuses, paracortices and trabeculae contain many granulocytes (both neutrophils and eosinophils). The other lymph node contains blood and granulocytes in the subcapsular sinuses, paracortices and trabeculae.

Microbiology: There was no significant growth from both primary and enriched inocula of subcutaneous tissues cultured aerobically on 5% sheep blood agar at 35°. Virology results are not yet available. Morphological diagnoses are subacute dermal vasculitis and acute lymphadenitis. The current hypothesis is that the condition may be due to an idiosyncratic hypersensitivity reaction. Some cases of arteritis in other species are immune mediated. Comments are invited from the Veterinary Pathology Report readership.

Verminous encephalomyelitis in greyhound pups

Paul Gill
Regional Veterinary Laboratory, Wollongbar NSW

History: Five out of six eight week old greyhound pups developed hind quarter paralysis and hyperaesthesia after being placed in a newly built run. Four pups died. A rat's nest was disturbed during construction of the run and snails were observed in the run.

Histopathology: There are multiple, large, circumscribed pyogranulomatous foci randomly distributed through the white matter of the lumbar spine. The pyogranulomas comprise degenerate granulocytes centrally ringed by a thick collar of histiocytes with the occasional giant cell, this inflammation extended along short segments of adjacent blood vessels. A transverse section of nematode larvae was evident in one pyogranuloma. A few small, random haemorrhages were also evident. There are also several, circumscribed pyogranulomatous foci randomly distributed through the white matter of the thoracic spine, the cervical spine and the medulla. The morphological diagnosis is pyogranulomatous encephalomyelitis and the likely cause, the rat metastrongylid lungworm, Angiostrongylus cantonensis.

Ovine Johne's Disease: Histological examination of Material collected by Meat Inspectors at Commercial Abattoirs in NSW 2001 - 2002

Cor Lenghaus; Patrick Staples; Graham Bailey.
Regional Veterinary Laboratory, Orange Agricultural Institute, NSW 2800.

As part of the National Abattoir Surveillance Program for Ovine Johne's Disease (OJD), meat inspectors at various commercial abattoirs submitted formal-saline-fixed terminal ileum, caecum, and ileo-caecal/terminal mesenteric lymph nodes, from a maximum of three sheep from any cohort of sheep sent to slaughter. These tissues were selected on the basis of a variably thickened intestinal wall, cording of lymphatics and/or enlarged lymph nodes.

Where tissues from three sheep from a single cohort were submitted, a complete transverse section of the terminal ileum was examined histologically from each sheep. Where two sheep represented a single cohort, terminal ileum and lymph node were examined histologically from each. Caecum was also examined if tissues were only received from a single sheep out of any flock. Diagnosis of OJD was based on the Australian Standard Diagnostic Techniques, now slightly amended as the Australian and New Zealand Standard Diagnostic Procedures (2002).

Overview of Results

From November 2001 through April 2002, sheep from more than 800 different cohorts were examined, with over 70% of these cohorts positive for OJD. At this time it is not known how many different properties this represented. The most severe intestinal lesions usually, but not always, had the most acid fast bacilli (AFB), but AFB were generally very scarce in lymph node sections. Overwhelmingly, epithelioid cells rather than multinucleate giant cells were the dominant inflammatory cell in positive OJD cases. In <1% of positive cases, the diagnosis was based solely on the lesion in lymph node/s.

Lymph Node Lesions

Sporadically in the nodes submitted, there were poorly encapsulated areas of greyish coloured caseation, up to 1cm diameter, or 1-2 mm chalky, white lesions, mainly in the cortex, seen histologically as encapsulated foci of mineralisation. AFB were demonstrated in these affected lymph nodes and in the caseous material which partly occluded associated lymphatics. In summary, the caseating lymphadenitis was similar to that described for Johne's disease and tuberculosis in deer; the small, mineralised foci were similar to resolved, end-stage tubercular lesions seen in lymph nodes especially of the head, in open-range pigs.

Lymphangitis

In ~10% of submissions there was a generalised lymphangitis but without the granulomatous enteritis or lymphadenitis associated with OJD, and AFB were not

found. In its mildest form there were lymphocytic infiltrates into and around a thickened connective tissue wall of serosal lymphatics. The lymphocytic infiltrates followed through into the submucosal lymphatics. At a more severe stage, macrophages, epithelioid cells and multinucleate giant cells infiltrated and partly occluded the lumen of serosal lymphatics, and dense clusters of epithelioid cells and small numbers of other leucocytes were associated with the submucosal lymphatics.

This lymphangitis did not contribute significantly to any inflammation seen either in the intestinal mucosa or in the cortex of the lymph node. Grossly, in its more severe form, the lymphangitis caused an obvious cording of serosal lymphatics and contributed significantly to the overall thickness and turgidity of the gut wall.

In the 50 cases of lymphangitis in which it was possible to trace back to the properties of origin, 64% were found to be in the designated OJD Protected Zone, where only 16 of 14,752 properties, incorporating 61% of NSW sheep, were known to be OJD infected as at April 2002. We do not know in all cases whether the specific sheep examined were bred on the properties under review.

We have concluded that the lymphangitis as described, is a distinct lesion separate from that of typical OJD. The lesion may however be readily confused with OJD, both at necropsy and during histological examination, which could potentially lead to considerable economic loss for affected producers.

Addendum, June 2003
More recently, in Victoria and in South Australia, one of us (CL) has diagnosed OJD in unthrifty, mature-aged sheep on the basis of a focal granulomatous hepatitis, with aggregates of epithelioid cells scattered throughout the hepatic parenchyma. This lesion is well documented for Johne's Disease generally, however it may be dismissed as non-specific, particularly if, as in the present cases, gut and mesenteric lymph nodes were not submitted. AFB were usually found as singletons in some granulomas, although rarely they were present at >10 bacilli per granuloma.

NT – Anton Janmaat

No report.

QLD – Bruce Hill

No report.

SA – Stephen Yeomans

No report.

TAS – Judith Handlinger

No report

VICTORIA – Malcolm Lancaster

Different iridoviruses in redfin (Perca fluviatilis) and Murray cod (Maccullochella peelii)?
Malcolm Lancaster, Mark Williamson
VIAS Attwood

High fingerling mortality rates occurred in wild redfin and farmed Murray Cod this summer. Fish of both species contained basophilic intracytoplasmic inclusions and apoptotic cells in many tissues. Icosahedral virions morphologically consistent with an iridovirus were detected electron-microscopically. However, whilst the relatively small redfin inclusions were labelled by a polyclonal antiserum to Epizootic Haematopoietic Necrosis Virus, the consistently larger cod inclusions were not.

WA – David Forshaw

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

Ovine - Yersinia and sheep in WA(Russell Graydon and David Forshaw)

Yersiniosis is rare in sheep in Western Australia, however a recent case confirms the existence of the disease if not the clinical syndrome. Abattoir (Narrikup) specimens were collected during OJD surveillance. One animal had mucosal microabscesses in the SI, associated with colonies of gram negative coccobacilli and Yersinia pseudotuberculosis was isolated.

In another case, sheep were selected from farms (Kojonup) for a non-parasitic scour project ("allergic" enteritis) and were necropsied according to an experimental protocol. Yersinia enterocolitica and Pseudomonas aeruginosa were isolated but mostly from non-scouring animals and no lesions consistent with yersiniosis were seen.

Bovine - SBE. (Russell Graydon)

10/180 mixed sex Shorthorns cattle at Mingenew were ataxic with swollen joints. One was euthanased and necropsied. Lesions identified included non-suppurative meningoencephalitis with lymphoid cuffs and glial nodules, and in some areas acute-subacute vasculitis and focal ischaemia. Several affected animals had Chlamydia CFT titres up to 1:128, and unaffected in-contact animals also had low to medium titres. This seems an unusually high number of affected animals (we usually see only one or two affected) and vasculitis is not commonly seen in WA cases.

The species Chlamydia pecorum has been renamed Chlamydophila pecorum. C.pecorum strains are serologically and pathlogically diverse, having been isolated only from mammals: ruminants, koalas and swine. C.pecorum has been associated with abortion, conjunctivitis, encephalomyelitis, enteritis, pneumonia and polyarthritis.

Rock Rat - Renal disease. (John Creeper)

A Rock rat (*Zyzomys argurus*)form the Perth Zoo with end stage renal disease similar to that seen commonly in Stick Nest Rats at the Perth Zoo. Lesions of end stage glomerulonephritis were present - proteinaceous casts in ectatic tubules, interstitial fibrosis and sclerotic glomeruli - changes that affected more than half of the renal parenchyma. Of interest is the presence of immature appearing glomeruli and evidence of acute ongoing inflammation within surviving glomeruli. The histopathology indicated a primary glomerulopathy. Nutritional and/or toxic aetiologies were suspected although there may be genetic susceptibility. Electron micrographs of the diseased glomeruli were examined; there were no deposits on the thickened basement membranes, which suggests that the disease process was not immune mediated.

Comments from Karrie Rose at Taronga Park Zoo: *"We've searched through the Registry and while we do have one or two cases of glomerulo-nephropathy in sticknest rats on file (both from Taronga Zoo and Adelaide Zoo), but we can not find any published information. We have seen both a membranous glomerulopathy, and amyloidosis in aging native rats. We have been looking at a number of native rats from all over Australia, and haven't seen embryonic rests in the renal tissue of adult animals. It would be interesting to look at some more rats of that species to see if it was particular to that animal, or that species"*.

Bovine - Familial Neuromuscular Disease of Gelbvieh cattle (Russel Graydon)

Gelbvieh cattle (Belgian breed) at Morawa with 2/30 affected animals, this one 18 months of age. Clinically unable to stand. Histologically there is mild wallerian degeneration of the dorsal white matter columns in the cervical cord (the only segment submitted), mild degenerative skeletal myopathy, and quite striking renal glomerular lesions of haemorrhage, proliferative glomerulitis, haemoglobin (or myoglobin) nephropathy and occasional vasculitis of small arteries. This condition is recently described in 13 animals in 6 herds of Gelbvieh cattle in USA (Moisan DG et al 2002, J Vet Diagn Invest 14:140-149). It is a degenerative skeletal muscle disease with vascular, neurologic and renal lesions and a probable familial distribution in 4-20 month old purebred Gelbvieh cattle in North Carolina, Kansas, Nebraska, Missouri and Georgia. Clinically there is ataxia, weakness and terminal recumbency. Vitamin E levels were also deficient in 6/7 animals tested.

Equine - Polioencephalomyelitis.(Cleve Main, Clive Huxtable)

Two year old Thoroughbred gelding from suburban Perth with acute onset spinal ataxia. Histopathology shows a severe poliomyelitis in sections of spinal cord from cervical to lumbar segments, and a less severe polioencephalitis in parts of the brain. The lesion consists of lymphocytic cuffs with some PMNs, occasional vasculitis, gliosis, parenchymal lysis in some foci, but minimal neuronophagia. The lesions are suggestive of a viral aetiology and Clive Huxtable, who is familiar with West Nile Encephalitis in horses in USA, considers that the lesions seen here are indistinguishable from WNV. The lesions are not typical of EHV1. At AAHL, no WNV antigen was detected by ELISA and no CPE observed after inoculation into

BHK cells for 19 days. AAHL does not offer any immunohistochemical tests for flaviviruses.

Bovine - Kikuyu Poisoning or Ammonia Poisoning? (David Forshaw)

Cattle at Welstead had been grazing lush kikuyu pastures for 3 days. 8 dead, 15 sick, 38 total. History of paddocks treated with sulphate of ammonia 200kg/ha end Nov 2002. No rain until March 03 and then 4 inches over 2-3 weeks. In affected paddock, water pooling and pools "smell like urine" according to farmer. 37 cows in another herd were on similar pasture and drinking from troughs only - no illness in this mob. Sick cows showed salivation, some diarrhoea, weakness, staggering and collapse. Some were just found dead death. The farmer moved the affected stock, pumped out the pooled water and fed hay. One further animal died. He then returned the animals to the paddock after short period (3 days?) and had no further losses.

The only described lesions from a necropsy of one cow were disseminated serosal petechiae through the viscera. Histologically there was severe multifocal omasitis but no lesions were seen in the rumen (4 sections examined). The omasal change was not noted in the gross description. There were also focal areas of acute myocardial necrosis.

A diagnosis of probable kikuyu poisoning made but ammonia toxicity cannot be discounted.

Bovine - Neonatal Salmonellosis. (David Forshaw)

Herd of 200 breeders at Albany. Calves reported to be born healthy and suckle normally, then become sick, often within hours of first suckling and die within a week despite intensive treatment. Some developed diarrhoea before death and 23 died. In 3 PMs, two had acute torsion of the small intestine with associated fibrinous peritonitis, and very wet heavy congested lungs. The third also had a SI torsion with loose adhesions and severe inflammation of the lower SI and LI. Fibrino-necrotic cores of tissue were present in the lumina in affected areas. Regional lymph nodes were swollen with extensive discrete sub-capsular zones of pallor. Histologically there was severe acute enteritis of the jejunum, ileum, caecum and colon. There was also a very severe lymphangitis and lymphadenitis. Salmonella typhimurium was isolated from liver, lung, kidney, gut and brain of the first animal necropsied, from viscera of the second and from the mesenteric node of the third. Interestingly Salmonella were not isolated from the gut but this animal had received long term antibiotic treatment.

Equine - Ulcerative Stomatitis cause unknown (David Forshaw)

Nine of ten mares used for polocross competition and stabled together at Kojonup developed ulcers in the oral cavity (gums and tongue) but without fever or pain. Three animals had very severe lesions. There were no skin lesions and the horses were otherwise clinically normal. Five geldings, held separately on the same property, did not have lesions. The only common feed source was good quality hay and no other treatments or supplements had been administered recently. Some of the horses had received dental treatment 1 week previously, but not those with the severe lesions. All had competed in recent polocross events and had extensive contacts with other horses. 15 in-contact cattle on the property were also inspected and none had lesions. Tests for vesicular stomatitis virus at AAHL (SNT, virus culture) were negative. A week later, lesions were reported by

the owner to be resolving. The cause remains unknown but similarities with "Balclutha horse disease" were noted.

Caprine - Nocardial Pleuritis(David Forshaw)

A four year old goat at Denmark had shown lethargy, inappetance and a distended abdomen for two weeks before it died. At necropsy purulent exudate was present in the thoracic cavity and the pleura was greatly thickened. Histologically the pleura was replaced by a thick layer of mature granulation tissue, overlain by a mat of exudate containing filamentous bacteria which occasionally form colonies with a 'star-burst' appearance. The bacteria stained poorly with Gram but are argentophilic (Grocott). Aerobic culture produced no growth. Reculturing under anaerobic conditions resulted in a moderate growth of Nocardia sp.

Bovine - Monensin Toxicosis (John Creeper)

Seven of 40 calves at Busselton died suddenly with respiratory signs after being massively overdosed with Monensin (mixed at 5:1 rather than 1:5). The necropsy description was poor but histologically there was extensive central and paracentral necrosis in the liver and diffuse congestion and oedema in the lungs. PMNs have infiltrated the lesions in both organs suggesting a lapsed period of 12-24 hours (consistent with dosing history). No muscle or heart were submitted. Comment: Monensin at such a highly toxic dose may have a direct effect on the liver. However, one would normally expect the lesions in liver and lung to be associated with severe myocardial damage.

Fish - Dinoflagelate Branchitis (John Creeper)

Fish were collected from a large fish kill in Swan River in Perth. The deaths were reported to be due to anoxia caused by algal bloom, in turn caused by a flush of fertilizers after heavy rain. Histologically, sections of gills fixed immediately after death show severe proliferative branchitis with the presence of dinoflagelates (probably Gyrodinium sp) in the lesion. These organisms are said to leach oxygen from the water. The liver also showed dissociation of hepatocytes and some necrotic cells but the significance of this change is unknown.