AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

QUARTERLY REPORT

EDITOR: JOHN FINNIE
Production: Barbara Gill

Print Post Approved Publication No: PP44059/0003

Number 60 February 2001

PAGE CONTENTS

SECRETARIAT

Barbara Gill
PO Box 114 Walkerville SA 5081
Phone: 08 8344 6337 Fax: 08 8344 9227
avasa@senet.com.au

ASVP EXECUTIVE

President — Ruth Reuter
IDEXX-VPS
PO Box 445
GLENSIDE SA 5065
ruth-reuter@idexx.com 08 8372 3700

Secretary — John Finnie
Institute of Medical & Veterinary Science (IMVS)
Veterinary Division
101 Blacks Rd
GILLES PLAINS SA 5086
john.finnie@imvs.sa.gov.au 08 8261 1033

Treasurer — Rob Rahaley
IDEXX-VPS
PO Box 445
GLENSIDE SA 5065
rob-rahaley@idexx.com 08 8372 3700

COMMITTEE MEMBERS

Julia Lucas
IDEXX-VPS
PO Box 445
GLENSIDE SA 5065
julia-lucas@idexx.com 08 8372 3700

Martin Copland
IDEXX-VPS
PO Box 445
GLENSIDE SA 5065
martin-copland@idexx.com 08 8372 3700

Peter Phillips
IDEXX-VPS
PO Box 445
GLENSIDE SA 5065
peter-phillips@idexx.com 08 8372 3700

STATE REPRESENTATIVE

NSW — Paul Gill
RVL
WOLLONGBAR NSW 2480 02 6626 1261

NT — Anton Janumaat
PO Box 990
DARWIN NT 0801
anton.janmaat@nt.gov.au 08 8999 2240

QLD — Jim Taylor
Toowoomba Vet Lab QDPO
PO Box 102
TOOWOOMBA QLD 4350
TaylorJ@prose.dpi.qld.gov.au 07 4688 1351

SA — Ruth Reuter
"As Above

TAS — Philip Ladds
DPJW&E
PO Box 46
KINGS MEADOWS TAS 7249
Philip-ladds@dpiwe.tas.gov.au 03 6336 5216

VIC — Malcolm Lancaster
VIAS
475 Mickleham Road
ATTWOOD VIC 3049 03 9217 4200
Malcolm.Lancaster@nre.vic.gov.au

WA — David Forshaw
AHL, Agriculture WA
ALBANY WA 6330
dforshaw@agric.wa.gov.au 08 982 8444

EDITORIAL

In addition to having the best wine, arts festival and nuclear waste in Australia, South Australia will be the home of the ASVP Executive for the next few years.

The Executive met in November and January with the following issues being addressed:

* it was resolved in Perth this year to actively pursue Proficiency Testing for veterinary ` pathologists. Since this appears inevitable (and desirable), it is better that it is initiated by the ASVP rather than having it thrust upon us.

  Tony Ross is to be congratulated for assembling a small group of pathologists to commence Proficiency Testing. The concept of using the "Slide-of-the-Month" (sans diagnosis) will be trialed and details are provided by Tony in this Report.

* the following dates (weekend before AVA Conference) and venues are proposed for the next 2 ASVP Annual Conferences:

May 12-13	2001	Melbourne
May 4-5	2002	Adelaide

The theme for meeting 2001 will be: "Advances in Diagnostic Microbiology". Thanks to Ron Slocombe for securing the venue.

* It was decided to investigate the feasibility of the ASVP having its own Web-site. This would not only permit members (and external organisations/individuals) to access information, but publication of the Vet Path Report on this site would result in considerable savings to the Society. A hard copy of the Report could also obviously be obtained in this manner if desired. A means of ensuring confidentiality when appropriate would also have to be put in place.

  Such a site could also serve as a vehicle to publish a register of veterinary laboratories in Australia (and NZ). The current mix of public and private (and recent changes to the latter) now makes this register more pertinent. We may be losing, for example, potential contract pathology opportunities by not being visible enough. A listing in each capital city telephone book under "Veterinary Laboratories" could be trialed with the Web-site indicated, however, this could be a subject for discussion/approval at the next annual meeting.

* In accordance with the Perth resolution, financially recalcitrant ASVP members 2 years or more in arrears have been contacted (twice) and those not responding by reaching for their cheque books have been suspended. A list of financial members is printed in this Report and it would be appreciated if you could supply telephone numbers and e-mail addresses so these can be published in the next Report. Any aggrieved members not included should also contact me. Membership forms have been inserted to facilitate the inclusion of new pathologists.

Many thanks to the WA Executive for the excellent Fremantle Conference and for facilitating the transfer of responsibility to SA. We look forward to a robust 2001 for the ASVP.

John Finnie
Honorary Secretary.

PRESIDENT'S REPORT

Your executive has been looking into a number of issues over the past few months. The most immediate one is arrangement of the upcoming Annual General Meeting, which is being held in Melbourne on the weekend of May 12-13th, preceding the AVA conference. Conference venue is the "Harold White Theatre", Arts Building, University of Melbourne and conference accommodation has been booked at the "Downtowner on Lygon" in Carlton with a block of rooms being held for conference attendees. The theme for the Conference is "Advances in Diagnostic Microbiology". When "microbiology" is mentioned, many people think primarily of culture and antibiotic sensitivity testing. While this is an important area, particularly in view of some of the current discussion on antibiotic resistance, there are many other aspects which are involved and recent discoveries which are of significance to our profession. All of us would encounter diagnostic cases in which microbiological questions surface. Case reports on these will add significantly to the discussions at our conference.

Hopefully all of you have now received a questionnaire from Tony Ross regarding the Trial Histopathology QA program. This is a very important part of diagnostic pathology. Accreditation of laboratory systems, procedures and technical expertise is accepted as the Gold Standard for customer service, and the public is becoming more aware of its right to a quality service. It is important for a similar standard to be applied to the more difficult area of competence in pathology. If we do not do this ourselves, there will be pressure for a system to be put in place for us. Please return your questionnaires to Tony if you have not already done so.

After serious deliberation, we have made the difficult decision to remove all unfinancial members from the mailing list of the Veterinary Pathology Report. If you have not received your last newsletter or know of colleagues who have not, please contact John Finnie. He will check the list and let you know your financial status. Although the ASVP is a relatively small organisation, it represents your interests. If you are not a member, your voice cannot be heard on some of the important issues which we are facing.

I look forward to seeing you all at the conference in May. Please bring as many cases with you (microbiological or not) as you wish, to help stimulate a lively discussion.

Ruth Reuter.

NEW SOUTH WALES - Paul Gill

ILLTHRIFT IN WEANER PIGS

John Glastonbury, Regional Veterinary Laboratory Camden, Elizabeth Macarthur Agricultural Institute, NSW Agriculture. Allan Kessell, Veterinary Clinical Centre, University of Sydney, Camden. Graham Wilcox, Division of Veterinary and Biomedical Sciences, Murdoch University.

For over a year a large intensive piggery on the southwest slopes of New South Wales has been experiencing problems with a wasting syndrome in weaner pigs. During November 2000 it was decided to investigate the condition fully. Pigs appear healthy at weaning, but 10-14 days later, at about 5 weeks of age, 7% become illthrifty, cough, develop a long hair coat and approximately 25% of affected animals die. Post mortem examinations carried on the farm found poor body condition, variable degrees of pneumonia and well formed faeces.

Material, including ileum, mesenteric lymph node, lung, liver, spleen, kidney and brain (1 animal) fixed in neutral buffered formalin, fresh spleen and serum (4 animals), from 11 pigs has been submitted to the Regional Veterinary Laboratory Camden. Mild to moderate chronic diffuse interstitial pneumonia and mild chronic enteritis have been observed in most cases. The former was characterised by increased numbers of mononuclear leucocytes, mainly lymphocytes, in alveolar walls, while villous atrophy and hyperplasia of the crypts of Lieberkuhn signified the latter. Four pigs contained isolated syncytial giant cells in their Peter's patches. Moderate chronic non-suppurative interstitial nephritis, very mild chronic meningitis, suppurative bronchopneumonia and bronchointerstitial pneumonia, suggestive of mycoplasmal pneumonia, were detected in one animal each.

The fresh spleens were forwarded to the Murdoch University. They were subjected to a multiplex polymerase chain reaction (PCR) to detect porcine circoviruses (PVC) types 1 and 2. Of five spleens tested to date, one was positive for PCV type 2, two were positive for PCV type 1 and two were negative. In the near future, in-situ hybridization will be performed on unstained histological sections in an endeavour to demonstrate the presence of PCV in association with pathological changes.

PCV type 2 infection has been incriminated in a variety of pig diseases, including post weaning multisystem wasting syndrome (PMWS), porcine dermatitis and nephropathy syndrome, interstitial pneumonia and reproductive failure, in many countries. PMWS is the most prevalent and economically important. Whether the condition described is PMWS is most prevalent and economically important. Whether the condition described is PMWS is problematical. Differences with reports from overseas are jaundice was not found clinically and histologically there was no portal hepatitis or lymphadenopathy, except for the presence of a small number of syncytial cells. PCRs on the remaining spleens and in-situ hybridizations are awaited with considerable interest.

The group at Murdoch has received funding from the Pig Research and Development Corporation for this work. They would appreciate receiving material from all states in the country from normal weaners, weaners in poor body condition, neonates with congenital tremor and suspect cases of dermatitis nephropathy syndrome. Fresh spleen, lymphoid organs, lung, liver and kidney as well as serum are required. Please help, and if you can, contact Graham Wilcox at wilcox@numbat.murdoch.edu.au.

Equine foetal granulomatous pneumonia

John Glastonbury, Regional Veterinary Laboratory Camden, Elizabeth Macarthur Agricultural Institute, NSW Agriculture.

During the second quarter of 2000, we investigated an outbreak of abortion on a Thoroughbred stud on the north coast of New South Wales. By the end of July when this particular case was examined, eight of 12 mares had aborted.

An aborted female Thoroughbred foetus was submitted to the laboratory. It had a crown-rump length of 60cm, minus its lower limbs it weighed 8.7kg and hair was developing about the eyelids. Autolysis was moderately advanced, subcutaneous oedema was present in the inguinal and ventral abdominal regions and the pleural and pericardial cavities contained excessive volumes of blood stained fluid. A rupture on the diaphragmatic surface of the right middle liver lobe had led to appreciable haemoperitoneum.

A full range of tissues was fixed in neutral buffered formalin. Significant histological findings were restricted to the lungs. They revealed diffuse primary non-aeration, diffuse congestion, occasional fibrin emboli in interstitial blood vessels, mild cuffing of bronchioles by small numbers of lymphocytes and plasma cells and marked exudation of neutrophilic polymorphonuclear leucocytes into the lumens of bronchioles and alveoli. Alveolar lumens also contained alveolar macrophages and large numbers of multinucleate giant cells of variable morphology, including Langhan's foreign body and bizarre types. Gram and periodic acid Schiff stained sections failed to reveal the presence of any significant bacteria or fungi, respectively.

Pericardial fluid was found to contain 1250 µg of IgG/ml by RID at the Scone Diagnostic Veterinary Laboratory. Routine bacterial culture of liver, lung, spleen and foetal stomach contents failed to isolate any likely pathogens. Lungs were submitted for specific culture for *Taylorella equigenitalis* and mycoplasmas, again with negative results. No equine herpesvirus, equine viral arteritis virus or arbovirus was isolated from liver, lung, kidney or spleen.

Largely out of ignorance, we were quite excited by the granulomatous foetal pneumonia. Morphologically, it appeared to be bacterial or fungal in origin. Our failure to demonstrate or isolate any possible pathogens was attributed to the fact that the mare had been treated with antibiotics due to a suspicion that the abortion storm was associated with *Leptospira Bratislava* infection. You are never too old to learn, as Bill Hartley has pointed out to me that during a survey he conducted in New South Wales in 1978 and 1979 he found a similar pneumonia in 32% of 142 aborted foals examined. From affected lungs, he recovered a variety of bacteria, including streptococci, staphylococci and diptheroids. However, it would seem to be unusual if such sporadic causes of equine abortion were responsible for this storm or cluster.

Suspect Sporidesmin toxicity in an Easter Grey Kangaroo - Steven Hum

In November last year a 2 year old, 32kg male eastern grey kangaroo started to drink excessively and become depressed. It was raised on the 3.5 acre property, which was completely fenced off with short mixed pasture. The diet was supplemented by commercial kangaroo pellet. On clinical examination no overt signs were detected but clinical pathology revealed elevated liver enzymes, AST 291 U/L (30-60 U/L), GGT 130 U/L (5-20 U/L), TBIL 38 umol/L (<10 umol/L). Its condition continued to decline and died 3 weeks later weighing 22kg.

Gross post mortem examination revealed small pale liver but there were no abnormalities detected in other organs in the body. Fixed liver collected and submitted to the laboratory revealed a severe diffuse chronic cholangiohepatitis characterised by diffuse vascular congestion, severe diffuse bile duct proliferation with advanced periportal fibrosis, sometimes infiltrated by moderate numbers of mononuclear leucocytes. Bile ducts proliferated randomly, individual ducts were often 2 or 3 cells thick and frequently without lumen. In other areas there were extensive interstitial scarring and nodular hepatocellular hyperplasia with frequent individual hepatocellular necrosis.

Findings would be consistent with sporidesmin toxicity. At the same time of this incident sporidesmin toxicity has been reported in cattle in the neighbouring properties indicating that conditions for the growth of Pithomyces chartarum were favourable around the district. There was no evidence of other hepatotoxins involved.

Sporidesmin toxicity is suspected to be quite rare in free living or entirely hand fed native animals probably because of insufficient amounts of spore uptake but the restricted grazing opportunities and heavy fungal pasture contamination may have precipitated the condition in this case.

NORTHERN TERRITORY - Anton Janmaat

Meliodosis in a Puma (Felis concolor)

Anton Janmaat, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

Nothing unusual about Burkholderia pseudomallei infection in the Top End but a first for its occurrence in a puma (to add to the first in an alpaca - ASVP Report 47, December, 1997). The puma's handlers suspected snakebite as the cause of death. They received the necropsy and histo reports below.

Necropsy

The animal had its skin and head removed prior to post-mortem examination. There was some bruising over the right shoulder. Opening of the cadaver, revealed a stomach distended by gas, an enlarged spleen and voluminous dark lungs with numerous firm pale nodules up to 15mm in size (dull on cut surface). There were a considerable number of roundworms in the stomach and intestines. There was advanced autolysis and apart from the apparent lung lesions, all changes were considered to be autolytic i.e. autolysis was presumed to have masked any ante-mortem changes.

Histopathology

As anticipated from the post-mortem examination, there was extensive autolytic change in the eight sections prepared from the tissues stored in formalin. Nevertheless, multiple foci of necro-purulent inflammation surrounded by extensive haemorrhagic areas were still recognizable in the lungs. There were multiple small abscesses in the spleen, explaining the small pale foci seen grossly (remembered in retrospect) and there was one such lesion in a section of kidney. These findings, together with the isolation of *Burkholderia pseudomallei* make for a convincing diagnosis of melioidosis. The widespread and acute lung involvement explains the sudden demise of this animal. As a matter of interest, in our experience, melioidosis in domestic cats is rare (one case of natural infection in the last 10 years).

Reference:
Low Chou J., Mayo M., Janmaat A, Currie B.J. (2000). Animal melioidosis in Australia. Acta Tropica 74, 153-158.

Acute hepatotoxicity in cattle - Helen Parkes, Berrimah Veterinary Laboratories.

In September, field veterinary staff investigated the deaths of approximately 70 cattle (cows and heifers) out of about 800 in a paddock, over about a two week period. Only one paddock on the northwest NT property was involved. Animals were either found dead (sometimes in groups of two or three), or were recumbent and unable to rise but relatively alert. No struggling was evident. Unaffected animals appeared normal (i.e. no sick ones were found) and in good condition. Post-mortem examinations showed enlarged, mottled livers and pale kidneys. Carcases were jaundiced. Serum biochemistry showed markedly elevated hepatocellular and biliary enzymes and elevated urea. Histological examination showed severe hepatic necrosis, ranging from periacinar to massive necrosis, with periacinar haemorrhage. There was prominent cytosegresome formation in some animals and variable degrees of dissociation of hepatocytes (sometimes quite marked). The kidneys showed focal, acute tubular necrosis, affecting mainly proximal tubules. There was focal, quite severe, subendocardial haemorrhage, with scattered myocardial degeneration, but no regenerative response.

An acute, severe hepatotoxin was suspected. Unfortunately, no specific agent was identified. Blue-green algae was a strong suspect. The paddock has numerous watercourses with many small billabongs, ponds and soaks. At that time of year it was very dry and getting hot, so the waterholes were getting smaller and hadn't flowed for a while. No evidence of an algal bloom was found, but apparently there are about 70 separate waterholes in the paddock and some were not accessible.

Part of the paddock had been burnt about 2 weeks before, and there was some regrowth. Pyrrolizidine alkaloidosis was considered, especially as crotalaria is plentiful in the paddock, however, the severity and acute nature of the lesions would have been unusual. Samples of rumen content and fresh tissues were sent to AAHL for pyrrolizidine alkaloid detection, and were negative.

The cattle were moved out of the paddock and the deaths stopped, further supporting the case for a toxic cause. An examination of the paddock about 3 weeks after the cattle had been moved out did not detect any known hepatotoxic plants.

Not melioidosis this time! - Helen Parkes, Berrimah Veterinary Laboratories.

On the basis that common things occur commonly, this should have been a straightforward case of melioidosis. A two week old male goat (one of triplets) was presented thin and weak, with increased respiratory effort. The previous kid born to the same dam had been euthanased at four weeks old, due to hind leg paralysis of one week's duration. At post-mortem examination, it was found to have spinal abscesses, as well as abscesses in the spleen and submandibular lymph nodes, all of which grew *Burkholderia pseudomallei*. The dam and sire both had high titres to *B. pseudomallei.* These goats were "pets" and euthanasia wasn't considered an option, but the owner was warned not use the milk and not to breed again from either of them. However, the female "got pregnant" again (as goats do).

The kid was euthanased. Post-mortem examination showed numerous, firm, creamy-white nodules up to 10mm in diameter, from which a small amount of caseous material could be expressed, in all lobes of the lungs. In fact the lungs looked rather like a large bunch of fleshy white grapes. As well, there was a ventricular septal defect about 5mm in diameter, dilated right ventricular wall and excess pericardial fluid.

Histological examination of the lungs showed purulent exudate in bronchioles and in alveoli around the edges of nodules. In some areas the exudate was also fibrinous. The nodules consisted of consolidated pulmonary tissues and granulomatous inflammation forming bands around remnants of bronchioles and ruptured alveoli filled with purulent exudate. In some nodules the inflammation was necrotising, leading to central cavitation and abscess formation. Gram positive, filamentous organisms were easily found in the purulent debris.

Culture of the lesions resulted in a moderate growth of a *Nocardia* species. Direct and enrichment culture for *B. pseudomallei* were negative. The kid had high CFT and IHA titres to *B. pseudomallei* (presumably maternal antibody).

A diagnosis of pulmonary nocardiosis was made.

Cerebellar abiotrophy in a kitten - Helen Parkes, Berrimah Veterinary Laboratories.

A four month old male Burmese kitten was euthanased with only about one week's history of progressively worsening cerebellar disease. The kitten was initially ataxic but bright and alert, with incoordination and intention tremors, progressing to being dull and depressed with inability to stand. On post-mortem examination the cerebellum was noted to be slightly small and firm. Also, the left kidney was less than 1cm in diameter with a few small cystic spaces, but no recognisable cortex and medulla.

Histological examination shows extensive loss of Purkinje cells in the cerebellum with a markedly reduced granule cell layer. The remaining Purkinje cells tend to be vacuolated or shrunken and pyknotic. Increased numbers of large astroglial cells are associated with the degenerate Purkinje cells, or mark the spaces where Purkinje cells are missing.

The history and histological picture are suggestive of cerebellar abiotrophy, i.e. premature or accelerated degeneration of a cerebellum that was normal at birth. This is recorded in several species and is often hereditary, but is considered "exceptionally uncommon in cats" (according to Summers, Cummings and de Lahunta, Veterinary Neuropathology, 1995). This kitten was locally bred, and as far as the breeder knew, none of the others in the litter were showing similar signs. This was the third litter from the dam - the first litter was apparently normal, but the second litter all died soon after birth with "flu-like symptoms". It was the first and only litter by this sire (now desexed).

QUEENSLAND - Bruce Hill

Caprine subcutaneous mycotic granuloma (presumed Curvularia lunata). Ross McKenzie, Yeerongpilly Veterinary Laboratory, Queensland Department of Primary industries.

A focal mycotic granuloma was seen in the subcutis of the ear of a Boer goat from Bargara via Bundaberg in early August 2000. The submitting veterinarian described circular crusty and sometimes pustular raised lesions on the ears of 3 of 20 goats. The lesion submitted contained numerous large chlamydospore-like bodies engulfed by macrophages and giant cells. Gomori's methenamine silver stain revealed mostly singular chlamydospore-like bodies with some sporting short thick hyphae and some in short chains. Fungal culture yielded *Curvularia lunata*, an *Alternaria* sp., *Aspergillus fumigatus* and *Trichosporon cutaneum*. Given the morphology of the fungal elements in the lesion, the Curvularia lunata was considered the agent most likely to be responsible for the lesion. Curvularia spp. have been associated with black-grained eumycotic mycetomas in dogs in Australia. This caprine lesion contained no grains.

Streptococcus dysgalactia polyarthritis in goat kids - Ross McKenzie.

Streptococcus dysgalactiae was isolated from 2 synovial fluid samples from neonatal goat kids with polyarthritis in herd of Anglo-Nubians at Teviotville via Kalbar in late August 2000. Approximately 15 kids had been affected in the two weeks before specimen submission. Kids were born normal and became affected within 48 hours with severe joint swelling. Laboratory investigations for the Mycoplasma and caprine retrovirus infections were negative.

Vitamin E deficiency in chickens - Ross McKenzie.

Vitamin E deficiency was diagnosed as the cause of deaths in 2-week-old Transylvanian naked-neck chickens from a flock at Mt. Crosby. The owner reported recumbency and death within 24 hrs. Initial examination of dead chickens revealed no lesions. Subsequent examination of live affected chickens revealed the birds to be recumbent, apparently unconscious and undergoing clonic spasms. Histology of three of the birds submitted alive revealed mild degenerative changes in the cerebellar white matter and small blood vessels of the brain. Liver Vitamin E concentrations from these birds were <0.1, 0.4 and 0.4 mg/kg (normal 15-40 mg/kg). Liver Vitamin A concentrations were also deficient at 7, 11 and 11 mg/kg (normal 60-300 mg/kg).

Acute bovine interstitial pneumonia - Ross McKenzie.

Acute interstitial pneumonia was seen in specimens from necropsy of two 12 month-old Santa Gertrudis-cross steers from a herd at Monto in early November 2000. Nine steers had died and 30 were ill in a herd of 170 cattle introduced to the property in early June 2000. No significant bacteria were isolated from either animal. No animal suitable for viral culture was available. A drought ration including copra and urea was being fed.

Hypocalcaemia in dairy cattle on lush buffel grass - Ross McKenzie.

Hypocalcaemia attributed to relatively large concentrations of oxalate in lush buffel grass (Cenchrus ciliaris) was seen in a dairy herd at Mundubbera in mid November 2000. Due to wet pasture conditions, cows were put onto a lush buffel grass pasture instead of the usual rye grass and lucerne. Thirty cows became recumbent and were treated with subcutaneous Ca and Mg, which reversed the condition in all but 7. A further 13 became recumbent overnight. Serum calcium concentrations in 4 cows sampled were 1.37, 1.59, 1.70 and 2.05 mol/L (normal 2.128 mmol/L). No evidence of kidney dysfunction was indicated by the clinical chemistry results.

Analysis of a lush buffel grass sample revealed (all on a dry matter basis) calcium 0.34%, magnesium 0.23%, total oxalate 6.50%, soluble oxalate 5.00%. These are the highest concentrations yet recorded for oxalate in buffel grass.

Aspergillosis in broiler chicks - Anita Gordon, Yeerongpilly Veterinary Laboratory, Queensland Department of Primary Industries.

Mortalities of 2.3% (expected rate = 0.5%) occurred in a group of 4,000 three-day-old broiler chicks introduced as day-old chicks to a research facility. Twelve of thirteen dead chicks submitted for necropsy exhibited very uneven growth rates, uni or bilateral pneumonia/airsacculitis and many also had evidence of yolk sac infection. Affected lungs were purple and consolidated, and beset with miliary white foci up to 1mm in diameter. Similar white foci were present in affected air sacs, which were thickened and cloudy. Histologically there was severe, subacute, multifocal to coalescing, necrotising, mycotic pneumonia, as well as mycotic infection of yolk sac. *Aspergillus fumigatus* was isolated from lung swabs of two chicks. No significant bacteria or viruses were isolated.

Nitrate poisoning in cattle - Anita Gordon.

Seventeen of 26 Murray Grey cows were found dead the morning after being fed lush forage sorghum hay. Samples of aqueous humour yielded 100ppm nitrate and 10ppm nitrite, and three samples of the hay yielded nitrate concentrations of 3.2% 9.7% and 9.7% DM, respectively. Hay samples were negative for cyanide. The property, a poultry farm, had a history of previous episodes of nitrate poisoning. The hay had been produced on the farm, from paddocks fertilized with poultry manure.

Suspected tick (*Ixodes*) in Rusa deer - Anita Gordon.

Fifteen of 120 Rusa deer died unobserved over a two-week period in September. One fawn presented for necropsy was bright and alert, but had flaccid paralysis, more pronounced in the hindquarters, and laboured respiration. A total of eight engorged and semi-engorged female *Ixodes holocyclus* were removed from the submandibular region, as well as three males. Significant gross and histological lesions were absent, negative results were obtained for the botulism **ELISA,** and a presumptive diagnosis of tick paralysis was made.

Colibacillosis in a guinea pig - Anita Gordon

A guinea pig, which had received an intraperitoneal injection of botulism toxoid one week previously, was found dead with faecal staining around the cloaca. Gross and histopathology revealed severe, acute, haemorrhagic typhlitis, associated with masses of Gram-negative bacilli deep within the mucosa. Intense focal infiltrates of polymorphs were occasionally visible in the lamina propria and submucosa, and an occasional mucosal vessel appeared thrombosed. *E.coli* was the only significant pathogen cultured from the caecum. Colibacillosis was thought to be unrelated to the botulism vaccine.

Ovine Campylobacteriosis - John Gibson, Queensland Department of Primary Industries, Toowoomba Veterinary Laboratory.

An outbreak of abortion, stillbirth and neonatal deaths was diagnosed as Ovine Campylobacteriosis. Some 25 lambs died in a flock of 22o Border Leicester ewes. Three sets of twins were submitted for laboratory examination. Five lambs were born alive and one was still born. No gross abnormalities were detected in viscera, but histologically all lambs had a suppurative bronchopneumonia. Silver stained sections of foetal lung revealed a serpentine bacterium consistent *Campylobacter sp* in all lungs. A *Campylobacter sp* was isolated from lungs and stomach contents and has been referred for definitive identification.

Lead poisoning, still a common cause of neurological signs in cattle - Jim Taylor, Queensland Department of Primary Industries, Toowoomba Veterinary Laboratory.

Four separate cases of lead poisoning were confirmed in cattle during September. Three cases involved yearly steers and the fourth involved adult Angus cows.

Mortality rates varied from 2-10% with all cases involving access to lead batteries or old dumps. All cases consistently had neurological signs with one case also having gastrointestinal signs. Toxic lead levels were detected in blood, liver, kidney, rumen contents or faeces. Lead metal fragments were found in rumen contents from two cases. A brain was submitted from one of the adult cows with a moderate diffuse subacute polioencephalomalacia consistent with lead poisoning.

SOUTH AUSTRALIA - Julia Lucas

Panepidermal pemphigus in an Akita Dog

Julia Lucas - Indexx - Veterinary pathology Services.

A four year old mail neuter, Akita presented with severe scabbing hyperkeratosis along dorsal nasal area and a smaller amount on ear pinnae. The footpads were erythematous and had hyperkeratosis. Similar lesions were beginning to develop around the eyes and tarsals. The dog had received a trial course of cephalosporins but lesions progressed whilst receiving antibiotics.

All sections of skin examined had a similar lesion characterised by intraepidermal pustules that varied from supra basilar to mid epidermal pustules. There was also marked orthokeratotic and some focal areas of parakeratotic hyperkeratosis and marked hyperplasia of the epidermis. Within the intraepithelial pustules there were free acantholytic epithelial cells.

All sections of skin examined had a similar lesion characterised by intraepidermal pustules that varied from suprabasilar to mid epidermal pustules. There was also marked orthokeratotic and some focal areas of parakeratotic hyperkeratosis and marked hyperplasia of the epidermis. Within the intraepithelial pustules there were free acantholytic epithelial cells.

In the superficial dermis there was evidence of pigmentary incontinence in the sections from the feet, with accumulation of melanin granules in macrophages. There was a superficial dermal, perivascular inflammatory infiltrate composed of a mixed population of neutrophils, lymphocytes and plasma cells.

The characteristic epidermal changes including intraepidermal pustules throughout the epidermis, containing free acanthocytes and hyperplastic changes suggest the diagnosis of panepidermal pemphigus. This is a rare autoimmune disease that has been described in the dog, especially Chow Chow and Akita[2]. The specific target of the auto antibody has not been determined in panepidermal pemphigus as in other pemphigus disorders of the dog.

Akitas in Adelaide account for a large number of the pemphigus group diagnoses made by IVPS yet make up a fairly small proportion of the dog population. Akitas are reported to be predisposed to other autoimmune diseases such as pemphigus foliaceous and pemphigus erythematosus.

Differential diagnoses include Pemphigus vegetans, Pemphigus foliaceous and Pemphigus erythematosus. Pemphigus vegetans is considered a benign variant of pemphigus vulgarism [2]. The cases previously reported in the literature as Pemphigus vegetans have since been reclassified as panepidermal Pemphigus [1]. Pemphigus vegetans disease is characterised by suprabasilar pustules and papillomatous hyperplasia of the epidermis [2].

Pemphigus foliaceous is the most commonly diagnosed of the pemphigus group and is characterised by sub-corneal pustules. Pemphigus erythematosus is characterised by subcorneal pustules and interface dermatitis with basal cell degeneration. This lesion was classified as panepidermal pemphigus because the pustules occur in all layers of the epidermis and are not confined to a single layer. Tests are not readily available in Australia for specific auto-antibodies to further define this condition and diagnosis usually depends of typical histology, signalment and response to therapy.

1. Mason Ken, 2000, personal communication.
2. White Stephen, 2000, *Diagnosis of Autoimmune skin disease*, Fourth World Congress of Veterinary Dermatology Conference Proceedings, San Francisco, California.

John Finnie, Institute of Medical & Veterinary Science, Adelaide.

Chronic respiratory disease in ferrets

Numerous ferrets purchased for experimental purposes arrived with an intermittent cough that persisted until they were necropsied. Macroscopically, there were many soft, white nodules in the lungs. Microscopically, these nodules corresponded to a severe, chronic bronchitis characterised by epithelial hyperplasia/dysplasia, marked lymphocytic infiltration of lamina propria (with a few plasma cells) and robust peribronchial lymphoid proliferation.

The aetiology was not determined, but was possibly viral.

Rat urolithiasis

Many, predominantly male, DA (and occasionally SD) rats presented hunched and ruffled with weight loss. At necropsy, the bladder was invariably greatly distended with heavily blood-stained urine, seminal vesicles were very dilated with accumulated secretion, and there was mild hydronephrosis. Uroliths of varying size were occasionally found in the bladder. Microscopically, there was a severe, focally ulcerative, haemorrhagic cystitis with extensive haemorrhage in the bladder wall. Mild nephrocalcinosis was evident at the renal corticomedullary junction. Culture of urine and seminal vesicles was negative.

The incidence of spontaneous urolithiasis in laboratory rats is generally low and, although the chemical composition of calculi in our cases was not ascertained, they are usually struvite in rats. Attempts to define the cause of urolithiasis in rats are usually unrewarding, but our feed source has been changed to see whether the incidence declines.

Botulism in waterfowl - Ruth Reuter, IDEXX/VPS, Adelaide

20 ducks and a swan were found dead over several days, on a lake in the Botanical Gardens in Adelaide. The man-made lake is fed by water coming off the parklands in the area and flushed through the storm water drains. The weather had been consistently hot and sunny, and an algal bloom had been recorded in the area. No spraying had taken place for some months. Several other ducks were apparently lethargic, having difficulty flying or walking and lying with necks outstretched. Two days later several birds, including a black swan, were found dead in the river Torrens near the outlet of the drainage from the Botanical gardens.

Post mortems were performed on a wood duck and a black swan. The birds were in good body condition. Gizzards and proventriculi were empty apart from small collections of sand and grit. No other gross abnormalities were evident. Intestinal contents were sent to the Animal Research Institute in Queensland for analysis for botulinum toxin. Antigen ELISA test on the sample from the wood duck was negative for *Cl. botulinum* toxin. However, the sample from the swan was strongly positive for *Cl. botulinum* toxin type C or D.

Waterfowl appear to be particularly susceptible to clostridium toxins found in decaying organic matter. Fly larvae can be a source of intoxication for species which eat maggots. Typical clinical signs, history and analysis of intestinal contents can help confirm a diagnosis of botulism. However, false negatives can be recorded on antigen ELISA tests. Differential diagnosis would include exposure to toxic algal blooms. Water contaminated with bacteria such as *Aeromonas hydrophila* has been reported to cause large numbers of deaths in waterfowl in summer.

TASMANIA - Roy Mason

Department of Primary Industries Water and Environment, Mount Pleasant Laboratory Report - Philip Ladds

Cases of general interest.

In addition to the syndromes specifically reported below, the following cases were of interest:

Bovine - Deaths of calves due to *Salmonella typhimurium* infection.

Ovine - Listeric meningoencephalitis, necrobacillosis in neonatal lambs, yersiniosis in lambs, deaths of a number of lambs post-marking due to *Clostridium chauvoei* infection, erysipelas causing suppurative bronchopneumonia and deaths in lambs, Campylobacteriosis causing abortion in a number of flocks, and severe gastro-intestinal parasitism - with total worm counts in excess of 35,000

Caprine - Aural squamous cell carcinoma with lymph node metastases.
Porcine - Cases of haemorrhagic enteropathy.
Canine - Parvovirus infection considered the result of vaccine breakdown, with deaths of a number of pups in a litter and concurrent infection with *Isospora* sp.
Avian - *Erysipelothrix rhusiopathiae* septicaemia causing death of a hen.
Lapine - Cases of severe hepatic coccidiosis and calcivirus infection.
Wildlife - Salmonellosis with intussusception in a kangaroo, deaths of large numbers of migrating Mutton birds (Puffinus tenuirostris) apparently due to exhaustion/inanition - perhaps with concurrent parasitism contributing, ringworm in a wallaby, and severe fibrinopurulent (presumed bacterial) meningitis in a koala that was found dead at the base of a tree in a wildlife park.

Myopathy and scoliosis in juvenile Atlantic salmon - DP Taylor, Fish Health Unit, DPIWE, Tasmania.

A syndrome of juvenile fish developing pallor from the dorsal fin caudally, variably present with paralysis and vertebral deviation was detected on a number of marine farms. In all cases the affected fish had been transported from the hatchery within the previous 10 days.

Necropsy of a number of these fish revealed haemorrhagic lesions in the dorsal musculature directly beneath the dorsal fin associated with fractures in the vertebral column and tearing of muscular attachments. Whirling disease digests of the vertebral structures and heads were negative. Microscopically, located either side of the spinal cord but not distributed symmetrically, were clusters of small, angular, dark atrophic myofibres containing usually one or a few nuclei. There was a modest sarcolemmal response and patchy fibrosis between fibres. The atrophic fibres were mostly noted to radiate along fascial planes, but not all fibres within a fascial bundle were affected. Occasionally within an affected bundle there were a number of large fibres infiltrated by macrophages undergoing floccular degeneration.

A visit was made to the hatchery supplying these fish. Although no fish were found to have grossly detectable vertebral fractures, caudal body pallor and paralysis were noted from fish collected from the bottom of the tank. Histologically, one fish was found to have a prolapse of the notochord through a fracture on the ventral aspect of a vertebral body. All of the fish examined had muscle lesions similar to that described above. The farm manager indicated that there had been a problem with intermittent electrical leakage into some tanks.

Evidence to support deficient mineralisation of bone or defective cartilage was inconclusive. Lightning strikes induce fracturing in the region of the dorsal fin because this area is considered to be the “fulcrum” and is subjected to much movement. Sublethal exposure to organophosphates causes hypercontraction of muscle through cholinesterase inhibition. Vitamin C deficiency is well described in catfish and salmon as causing broken backs by affecting collagen synthesis and bone deposition. Scoliosis is also recognised in salmon with tryptophan and in Vitamin A toxicity by causing differential maturation of the growth plates of the vertebrae.

Reference:

Ferguson H. (1989) Systemic pathology of fish. Iowa State University Press, Ames.

Monensin toxicity in 112 dairy heifers - L.J. Gabor, C. Dwyer and G. Downing.

AHL was initially contacted concerning the sudden death of thirteen 4-6 week old dairy heifers on a farm in Northern Tasmania. According to the owner, the only clinical signs were slight lethargy, failure to feed, then sudden death (often before the owners’ eyes). The local practitioner sent rectal swabs and colostrum samples for microbiological examination and small amounts of intestine, liver and spleen for histology. Apparently, formalin at 0.1% was no longer being added to the colostrum in storage, and there were suspicions that this had somehow affected the feed. Following the death of 8 more calves, a government

field veterinarian visited the farm to carry out further necropsies. The only gross findings in samples submitted from 2 freshly dead calves were pale regions in the myocardium. Histologically in one calf there was severe, extensive myofibrillar vacuolation with little cellular infiltration; extensive aggregates of mineralized muscle fibres were also present. In the second calf, the changes were milder and focal haemorrhages were observed in the deeper aspects of the ventricular wall.

At this point a diagnosis of acute severe myocardial necrosis was made, with a tentative aetiological diagnosis of monensin toxicity. Other possible differentials were nutritional deficiencies (Se, thiamine, K, Cu) or other exogenous toxins such as gossypol, vetch or Acacia.

To rule out monensin toxicity, a sample of the pre-prepared calf rearing mix was sent to a major pharmaceutical company's quality assurance laboratory where four samples were found to have actual concentrations of monensin between 10, 179-10,631 PPM. The stated concentration on the packaging, however, claimed a theoretical value of 1000 PPM. Liver copper, cobalt, selenium were all normal.

Further deaths of calves from the same farm were investigated and in all cases the only histological findings were severe cardiac necrosis. In all cases, the animals had received the calf formula from a specific batch. Retrospectively, a case of sudden death in a dairy calf from a different farm approximately 2 weeks prior to the major outbreak was re-examined. No significant findings had been recorded in this moderately autolysed case, although following serial re-sectioning of the heart, regions of severe vacuolation and focal necrosis were found, so a tentative diagnosis of monensin toxicity was reached. It was found that calves on this farm had also been fed a calf rearing mix from the same batch.

The deaths continued for almost 14 days and involved an additional farm. In total, of 258 calves fed calf rearing mix of the same batch number, 112 died. In all cases the clinical signs were few - slight lethargy followed by death. In all cases analysis confirmed monensin concentrations in excess of 10,000 PPM. The laboratory notified the manufacturer, and feed with the appropriate batch numbers was recalled.

Monensin is a commonly used carboxylic polyether ionophore produced by some strains of *Streptomyces* spp. Other ionophores include compounds such as lasalocid, salinomycin and narasin. Monensin was originally used as a coccidiostat in poultry, but has since become commonly utilized in ruminant nutrition to improve feed conversion efficiency by regulating rumenal fermentation end products. Monensin achieves this in cattle by increasing the proportion of proprionic acid produced in the rumen, increasing rumenal protein bypass, and decreasing the incidence of feedlot disorders by its antibiotic effect on major lactate-producing micro-organisms in the rumen.

The mode of action of ionophores such as Monensin is complex. In summary, they combine with metal ions and serve as a carrier for these products to cross the cell membrane. Monensin largely mediates Na^+ and H^+ exchange and causes cellular damage to coccidia with a massive influx of Na^+ into the protozoa.

As with most widely used pharmaceutical products, monensin toxicity has been reported on numerous occasions in many species including ruminant cattle, horses, sheep, pigs, dogs, poultry chickens, turkeys, quail, guinea fowl, ostriches, camels and laboratory animals. Published LD50 dosages vary greatly depending on the species, in cattle, however, between 12.5-80mg/Kg has been reported. In most instances, reported cases of toxicity have been due to handler mixing error which has led to inadvertent pockets of feed in which concentration of the drug are high.

Signs of toxicity in cattle have been published in detail and include near complete anorexia, depression, diarrhoea, tachypnoea, ataxia and death. The pathological and clinical pathological findings in adult cattle reflected rumenitis and severe cardiac and skeletal muscle damage. In experimental studies, the extent of pathology has largely been dose-dependant. Interestingly, there appears to be no report of toxicity in immature (pre-ruminant) cattle, as in the present case. Following withdrawal of the feed, the deaths ceased and there have been no recurrences. To this point, no corporate entity has admitted liability.

Dicoumarol toxicity in dairy calves - L. Gabor, C. Dwyer and G. Downing.

A farmer from a dairy in north-west Tasmania with seasonal (August) calving reported the deaths of six calves within the first three days of birth. The calves had appeared healthy at birth having stood up and suckled. In the following hours to days, however, these calves had become listless, with swollen umbilical regions and they died soon after with haemorrhage from the mouth, nose, anus, vulva and/or umbilicus.

A private practitioner carried out a field post-mortem on a recently deceased calf and noted extensive free blood throughout the abdomen, the umbilical region and the medial ligament of the bladder. There was also extensive haemorrhage in the thorax with petechial and ecchymotic haemorrhages on the epicardium. The plain blood sample was noted for not clotting, even after several hours. Haematology was unremarkable apart from a PCV of 0.07, lowered RBCC and Hb.

The following day three calves were presented to the clinic, including a calf empirically treated with injectable Vitamin K on the previous day and two calves which were born overnight. All three calves were recumbent, listless, had increased heart rates, were pale and had subnormal temperatures. The calves were placed in a warm environment and administered with intravenous fluids (Hartman's fluid) and IV trimethoprim/sulfadiazine, IM Vitamin K intramuscularly at 1.5mg/kg, and Richtafort (phosphorous and Vitamin B12) intramuscularly.

The AHL was contacted and a further field post mortem carried out. The three treated calves had died up to 7 hours earlier. Despite this, frank blood was observed in all body cavities, and all organs oozed blood when incised. Clotted blood was rarely observed. The visible connective tissue was swollen and often contained large amounts of blood.

Histologically the tissues exhibited frequent parenchymal haemorrhages. The livers displayed consistent periacinar fatty vacuolation. At this point in the examination, an exogenous anticoagulant which was somehow affecting the entire herd was suspected. The average prothrombin time from cows in the affected herd was over 80s, compared to 45 for cows from an unaffected neighbouring property.

Careful questioning of the owner revealed that the only change in husbandry worth noting was a change in the source of hay, which was still moist when cut and baled. Physically, the hay submitted for examination appeared to have a black core and was moist. It was composed of rye grass and sweet vernal grass. Results of hay analysis revealed dicoumarol concentrations of 27 and 39ppm, and serum dicoumarol levels in an affected calf and its dam of 3.1 and 14mg/L respectively.

A diagnosis of clotting deficiency due to dicoumarol toxicity was reached. In total, 18 calves died during the period of investigation.

Dicoumarol toxicity is a haemorrhagic disease of animals caused by the feeding of spoiled hay or silage. Coumarin, ferulenol and melitotin are contained as normal constituents in plants of the *Melilotus* spp (sweet clovers) and *Anthoxanthum odoratum* (sweet vernal grass). These chemicals are converted to dicoumarol by moulds including the *Aspergillus* spp during the making and storage of hay or silage containing these plants. Dicoumarol competitively inhibits the synthesis of Vitamin K dependent coagulation factors VII, IX, X and prothrombin. This results in impaired fibrin stabilization of platelet plugs causing internal and external haemorrhage and anaemia. The disease can occur in all species but is seen most commonly in cattle, less so in sheep and deer and is very rare in horses. There is, to our knowledge, only one report in the literature of dicoumarol poisoning in Australia. The report is of poisoning in South Australia which occurred in 1961 and affected adult cattle and sheet fed *Melilotus* species based hay (Wignall WN).

Reference:

Banks A.W., Hackett M.A., Irving E.A. (1961) Dicoumarol Poisoning of Cattle and Sheep in South Australia. *Aust Vet J* **37**:456-459.

Diet-related ulcerative rumenitis in cattle - Phillip Lads, Les Gabor, David Taylor, Caroline Ash & George Downing.

For two consecutive years during spring and early summer, specimens of rumenal lesions were received from adult cattle originating from several properties, and slaughtered at an abattoir in north-west Tasmania. In no case was clinical illness observed at ante-mortem inspection. Affected cattle had been grazed on lush green pastures and were in good to excellent body condition at time of slaughter. Both steers and cows were affected. Because a number of animals in each draft of cattle had lesions that resulted in condemnation of the rumens for tripe, the lesions represented an important source of financial loss.

Lesions appeared to be confined to the rumen. Typically they were multiple, irregular in shape, up to ~10mm in size and often confluent, covering an area of up to ~10cm in size. Colour was red-brown, progressing to pale scarring in healing lesions. Beneath erosions and ulcers the rumen wall was thickened by oedema to ~10mm.

Microscopically there was coagulation necrosis of epithelium with associated luminal protozoa and bacteria. Some ballooning degeneration of superficial viable epithelium was also apparent. Immediately beneath necrotic epithelium or erosions with attenuated epithelium, there was fibrin exudation and mixed inflammatory cell infiltration, sometimes with micro-abscessation. In the submucosa there was focal haemorrhage, marked oedema and fibrin exudation, prominent capillaries with swollen epithelium and a moderate diffuse infiltration of mixed inflammatory cells, especially polymorphonuclear neutrophils and eosinophils. Occasional multinucleate giant cells were also present, sometimes associated with or present within (lymph or blood) vessels. Shapely defined spaces, presumed to be gas bubbles, were also present in these areas in some animals. A diagnosis of ulcerative pyogranulomatous rumenitis, was made.

Other than superficial protozoa and bacteria, no organisms likely to be causal, were identified in sections stained routinely or by special stains. Likewise, direct microscopy of material from lesions, or culture, revealed no likely pathogen. Serum from one animal was tested and found negative for pestivirus infection.

A dietary/metabolic cause was considered probable. The deeper layers of the rumenal epithelium metabolise the short-chain volatile fatty acids, particularly butyric, acetic and propionic, the chief products of fermentation, so presumably diet-related, marked but localised concentrations of these acids could induce focal epithelial necrosis. Then penetration of resident micro-organisms or their products would induce inflammation. Possibly other dietary elements that might compromise rumen motility could presumably exacerbate the problem by preventing adequate mixing of ingesta. The granulomatous response seemed associated with the gas bubbles and has parallels in interstitial pulmonary emphysema in cattle, and intestinal emphysems in pigs.

VICTORIA - Malcolm Lancaster

Scrapie and Bovine Spongiform Encephalopathy Exclusions - Peter Hooper, AAHL

The Australian surveillance program to prove freedom from scrapie and bovine spongiform encephalopathy is now well underway. The program consists essentially of histological examinations of brains of those aged sheep and cattle that have neurological signs.

You will all know that the present AAHL tests of suspicious cases of transmissible spongiform encephalopathies (TSE's) are immunohistochemical (IHC) tests for prion protein on the fixed tissues and electron microscopic examination on a small piece of frozen unfixed upper cervical spinal cord for scrapie associated fibrils (SAF). A number of laboratories have been submitting the fresh frozen tissue to AAHL rather than paraffin blocks for immunohistochemistry as a first stage of exclusion. AAHL's preference is to do the IHC test before the SAF test.

This means a laboratory should first submit the paraffin blocks of the brains, especially the blocks with the suspicious areas, as well as the key areas for the two species, medulla for sheep and midbrain and obex for cattle. The frozen piece should be retained at the submitting laboratory and only submitted if the case still remains suspicious after IHC examination. In the event of a negative test, there will be no problem if a laboratory wants return of the paraffin blocks or wants them securely retained at AAHL.

Pathologists are reminded that they should consult Cook *et al*, "Transmissible spongiform encephalopathies" (Australian and New Zealand Standard Diagnostic Protocols), which gives guidance on histological exclusions of the TSE's.

WESTERN AUSTRALIA - David Forshaw

WESTERN AUSTRALIA

Non-suppurative encephalomyelitis in a horse - Phil Nicholls, Murdoch University, WA 6150.

A 17y M equine Thoroughbred with a several month history of hind limb ataxia and weakness with possible narrowing of C3-4 and C5-6 radiographically, was euthanased. No compressive lesions of the spinal cord or vertebral articular lesions were found during post-mortem examination, but histopathological examinations revealed a non-suppurative encephalomyelitis. The predominantly lymphocytic lesions were primarily asymmetrical and random in both grey and white matter of brain and spinal cord, including a mild leptomeningeal infiltrate. Wallerian degenerations randomly affected all funiculi. PCR for EHV-1 proved negative. Paraffin blocks were sent to Dr. Peter Hooper at the Australian Animal Health Laboratory, Geelong, Victoria for immunohistochemistry. Results of immunohistochemistry to date have proven negative for Hendra virus, Nipah virus and Japanese encephalitis. There will be further tests for a variety of orbiviruses and flaviviruses, as part of an on-going opportunist project on determining the causes of sporadic encephalitides in horses and wildlife.

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

ANNUAL CONFERENCE 2001

May 12-13 MELBOURNE

Conference venue: Harold White Theatre, Arts Building, University of Melbourne, off Swanston Street.

Conference accommodation: "Downtowner on Lygon", 66 Lygon Street, Carlton.

Theme: "Advances in Diagnostic Microbiology"

In order to facilitate conference planning, members are encouraged to:

(1) **send abstracts of papers to be presented to John Finnie (E-mail: john.finnie@imvs.sa.gov.au) particularly (but not confined to) infectious diseases cases.**

(2) **indicate ASAP to John Finnie (not the hotel) whether they will need accommodation and the type of room desired:**

Conference rates:
single/double/twin $135/room/night
single $149/room/night inc. full hot buffet breakfast
twin $149/room/night inc. 2 full hot buffet breakfasts