AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

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VETERINARY PATHOLOGY REPORT
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Newsletter Editor	Cleve Main
Working Party on Proficiency Testing	Barry Richards (convenor) John Mackie Roger Kelly Judith Nimmo-Wilkie
Conference 2000 Proceedings	David Forshaw

CONVENOR - SLIDE OF MONTH

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President's Report

I would like to start this off by saying Happy New Year but the year is no longer new and we have suddenly realised that there is only 17 more weeks to go before the highlight of 2000, the ASVP Conference on Saturday and Sunday the 25th and 26th June. We have actually been galvanised into action by Mandy O'Hara who has become the driving force behind the program for the conference. This year the theme is Dermatopathology. The speakers for the plenary session are Dr Mandy Burrows and Dr Mandy O'Hara and both are excellent speakers. Mandy Burrows is a specialist Veterinary Dermatologist and has great experience in the discipline including histopathology. Mandy O'Hara is also an experienced dermatopathologist and has worked closely with Mandy Burrows and Clive Huxtable. I guarantee that you will find their approach to the subject stimulating and informative and their presentations entertaining. We do need, however, member contributions on dermatopathology to complement the plenary session and of course, contributions on any area of Veterinary Pathology for the Sunday sessions. Details on the conference and the dates for submission of titles and abstracts are given elsewhere in this Newsletter. Please plan now to come to the conference.

The executive of the ASVP must change this year and the current Executive would like to express their sincere thanks for the offer of the South Australians to take it on.

David Pass

ASVP CONFERENCE 2000
ESPLANADE HOTEL
FREMANTLE
fortunately no longer home to the America's cup.
Last conference before GST!!!
24-25 JUNE 2000

The theme this year is **Dermatopathology** but, as usual, interesting case reports will be eagerly accepted.

Prospective contributors
TITLES required by March 31
ABSTRACTS by 19 May
preferred submission would be electronic in MS Word 6 or better.

Please send your electronic replies to

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TENTATIVE PROGRAMME

Theme: Dermatopathology

Saturday 24th June 2000

Plenary Sessions

Adhesion molecules as Targets of Disease: Drs Mandy Burrows and Mandy O 'Hara

9.00-10.30 The Dermoepidermal Junction & Disease

This session will discuss the structure and function of the dermoepidermal junction and how that relates to the clinical and histopathological presentation of diseases such as: Bullous pemphigoid, Herpes gestationis, Epidermolysis bullosa, Cicatricial pemphigoid & Linear IgA bullous dermatoses in small and large animals.

10.30-11.00 Morning Tea

11.00-12.30 The Keratinocyte & Disease

This session will discuss the structure and function of the keratinocyte and desmosome and how acquired and inherited disorders of these components of the epidermis lead to the clinical and histopathological presentation of diseases in the Pemphigus group.

12.30-1.30 Lunch

Member Presentations

1.30-3.00

3.00-3.30 Afternoon Tea

Plenary Sessions

Avian Skin: Interpretation of the feather follicle in health and disease: Dr David Pass

3.30-4.30 This session will discuss the functional anatomy of avian skin with particular emphasis on interpretation of the feather follicle in health and disease.

4.30- AGM

7.00pm Dinner

Sunday 25th June 2000

Member Presentations

New South Wales - Paul Gill

Congenital Blindness in White Shorthorn Cattle - John Glastonbury, Regional Veterinary Laboratory Camden, Elizabeth Macarthur Agricultural Institute, NSW Agriculture.

Recently I had the good fortune to examine eyes from five White Shorthorn calves, which had been blind from birth. Following weaning at 6 to 7 months of age, the calves were sent for slaughter, and the Veterinary Officer at the abattoir collected their eyes into Benin's fluid; two from two calves and one each from the remaining three. At this stage of the investigation the history is scant, being restricted to the blindness and the origin of the calves from a stud on the south west slopes of New South Wales.

Severe but variable microphthalmia was the most obvious feature. The greatest dimensions of the globes varied from 20 x 18mm to 35 x 28mm; in fact these two extremes were measured in the eyes of one animal. In two of the eyes, lens tissue was not readily discernible, and in a further two, there was a strand of tissue, about 2mm wide and 15mm long extending from the optic disc to the caudal surface of the lens.

Histological observations were dominated by truly bizarre dysplasia of the retinas. Although still attached at the ora ciliaris and optic disc, most of the retinas were detached from the underlying pigmented epithelium. The lenses which were not apparent grossly, were very small and ruptured along the caudal surface, leading to the incorporation of swollen lens fibres into the dysplastic retina. The visible strands of tissue were composed of dysplastic retina, which was associated with rupture of the lens capsule and herniation of lens fibres at the point of attachment to the lens. Dysplastic retinas were a conglomeration of retinal rosettes, nerve fibres, neurones, pigmented epithelium, swollen lens fibres, mostly with small spherical retained nuclei, and occasional blood vessels. Rosettes were one, two or three layered, with the first two being lined by undifferentiated neuroblasts. The choroids in two of the eyes were greatly thickened and contained cavernous vascular spaces.

The literature on this condition is not voluminous. It has been reported to be associated with internal hydrocephalus, the presence of which was not possible to assess in these cases. Experimental matings have incriminated either a simple autosomal recessive or an incompletely penetrant dominant mode of inheritance. Allan Kessell is going to enjoy learning more about ocular pathology and genetics in studying the condition further.

Equine Abortions due to Equine Herpes virus (EHV) Type 1 Infection - John Glastonbury and Rod Reece, Regional Veterinary Laboratory Camden, Elizabeth Macarthur Agricultural Institute, NSW Agriculture.

From July to September 1999 we received material in three submissions from six aborted equine foetuses from a Thoroughbred stud in the south west of New South Wales. The aborting mares ranged in age from a maiden to 16 years, and abortions occurred following 7.5 to 10 months of gestation. The first five aborting mares had been taken for service by "outside" stallions in 1998, and upon returning to the stud, were placed in a mob in the one paddock, with no introductions, since February 1999.

Histopathological findings varied between the three submissions. Material from three foetuses was examined in the first submission, with significant observations being severe acute segmental necrotising bronchiolitis, mild to moderate acute multifocal hepatic necrosis and marked necrosis of splenic white pulp. Eosinophilic intranuclear inclusion bodies were most frequent in the bronchiolar epithelium neighbouring the areas of necrosis. The second submission comprised material from two foetuses. In these, there was severe acute multifocal hepatic necrosis, with intranuclear inclusion bodies being evident in neighbouring hepatocytes, and moderate, subacute multifocal non-suppurative cholangiohepatitis. Findings in the lungs were restricted to diffuse primary non-aeration, with aspiration of squames and meconium, and acute multifocal interstitial and alveolar haemorrhages. A single foetus contributed to the final submission. It had

moderate subacute non-suppurative cholangiohepatitis and suppurative foetal bronchopneumonia, neither of which was associated with foci of necrosis.

The virologists contributed to the investigation. The offending mares yielded titres of between 16 and $\geq$ 512 in the virus neutralisation test (VNT) for EHV 1. All tissues cultured, one lung from the first submission and liver as well as lung from the other foetuses, yielded EHV 1.

This outbreak of abortion was attributed to EHV 1 infection on the basis of the detection of consistent histopathological lesions and the virological findings. No correlation could be discerned between the location and severity of the foci of necrosis as well as the presence of inclusion bodies and the stage of gestation at which the foetuses were expelled. However, it was interesting that the last foetus to be aborted died approximately 1 month after the others, came from a mob in which there had been no previous abortions and did not have focal necrosis or inclusion bodies. Perhaps its dam possessed a degree of immunity to the virus, which prevented the acute necrosis but encouraged non-suppurative inflammation, still leading to the same inevitable conclusion.

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

Just when we thought that we had seen everything bird brains have to offer, the most recent occurrences of Newcastle Disease have provided us with really spectacular lesions in the central nervous system.

As an example, shaking and paralysis were observed in early January 2000 in a shed of 39-day-old broiler chickens which had been vaccinated "in water" against Newcastle Disease at 14 days of age. The brains of three of four clinically affected chickens submitted to the laboratory had extremely severe multifocal non-suppurative meningoencephalitis. The meningoencephalitis was characterised by:

* perivascular cuffing, with predominantly small lymphocytes, which was most severe in the brain stem caudal to the red nucleus and in the cerebellar white matter. Mild to moderate degrees of cuffing was noted in the leptomeninges over the cerebellum and the ventral brain stem and about the vessels radiating across the molecular layer of the cerebellar cortex. Cuffs encompassed the vessel wall and were up to 10 to 15 cell layers deep;

* neuronal necrosis in the nuclei of the caudal brain stem and in the Purkinje cell layer of the cerebellar cortex. Degenerating neurones exhibited loss of Nissl substance, cytoplasmic eosinophilia, an angular profile and shrunken pyknotic nuclei;

* severe and diffuse gliosis in the Purkinje cell layer, deep cerebellar white matter and the ventral medulla oblongata. In the more severely affected areas, it was accompanied by oedema of the neuropil.

Our colleagues at the Australian Animal Health Laboratory (AAHL) confirmed the aetiological diagnosis by means of immunohistochemistry, virus isolation and PCR. From a brain pool from the four brains, they reported "a PCR product with the amino acid sequence at the cleavage site of the F protein of RRQRRF was detected following nested PCR amplification".

The inflammatory changes in these brains are certainly much more severe then those we observed during the outbreaks at Blacktown and on Mangrove Mountain; a view shared by AAHL - "These were more chronic (older) lesions then mostly experienced in experimental cases and earlier in the outbreaks".

Why is this so? Perhaps the local Newcastle Disease virus is becoming more neuropathogenic or the vaccine may have exacerbated the immunological inflammatory response.

Brown C, King DJ and Seal BS. Pathogenesis of Newcastle Disease in chickens experimentally infected with viruses of different virulence. Vet Pathol 1999;36:I25-132.

Epizootic Haematopoietic Necrosis Virus (EHNV) Infection of Rainbow Trout - John Glastonbury, Regional Veterinary Laboratory Camden. Elizabeth Macarthur Agricultural Institute, NSW Agriculture.

Rainbow trout, *Oncorhynchus mykiss* Walbaum, fingerlings on a large trout hatchery in southern New South Wales were dying at the rate of 1% per day over a 10-day period. The 20,000 fingerlings were not overcrowded and were contained in 15 ponds, in which the rate of water flow was good and the temperature was maintained at 11°C. Copper sulphate was added to the water and the feed medicated with oxytetracycline in an endeavour to stem the mortality.

Other than finding the fish dead, the manager noted no particular clinical signs.

Twenty fingerlings were submitted to the laboratory and detailed histopathological examinations were carried out on four. There were no significant gross pathological findings.

Histologically, the kidneys and gills were of most interest. The most striking observation was the presence of large intracytoplasmic basophilic inclusion bodies, which displaced the nucleus, within the cells of the haematopoietic kidney. Single cell necrosis/apoptosis was present in this tissue in three fish, while in the fourth; there were multiple foci of acute necrosis, containing basophilic debris. The gills displayed hypertrophy and segmental hyperplasia of the branchial epithelium associated with a very mild infiltration of mononuclear leucocytes.

The histological findings are considered to be pathognomonic for EHNV infection, which was first detected in rainbow trout in Australia in this hatchery. Foci of necrosis were not found in the liver, spleen and heart and immunological techniques were not used to demonstrate the presence of EHNV antigen in vascular endothelium. Reddacliff and Whittington (1996) were of the opinion that the causative iridovirus is endotheliotrophic and many of the lesions result from ischaemia.

Reddacliff LA, Whittington RJ. Pathology of epizootic haematopoietic necrosis virus (EHNV) infection in rainbow trout (*Oncorhynchus mykiss* Walbaum) and redfin perch (*Perca fluviatilis* L). J Comp Path 1996; 15:103-115.

Northern Territory - Anton Janmaat

Ivermectin toxicity in dogs - Helen Parkes, Berrimah Veterinary Laboratories (BVL), NT Department of Primary industry and Fisheries

Three, four week old, greyhound pups from a litter often were submitted to the BVL for post-mortem examination. Apparently the owner had given each pup 0.5mls (by subcutaneous injection) of cattle ivermectin (l0mg/ml). The pups were weighed at PM and they varied between about 1.4 and 1.7kg, giving an approximate dose rate of about 3,000µg/kg. The pups started showing signs about five hours after dosing, including tremors, ataxia, blindness, mild seizures, aimless wandering and head pressing, which advanced to collapse and coma. The first deaths occurred about 12 hours after dosing. In all, six pups from the litter died over three days.

There were no significant post-mortem findings. Histological examination showed generalised oedema of the brain. The other four pups went on to make a full recovery, but required intensive supportive therapy (nursing care, i/v fluids, corticosteroids, antibiotics) for four days. The apparent blindness continued for about seven days, but had completely resolved after ten days.

Ivermectin is approved for use as an anthelmintic in cattle, sheep, pigs and horses (at 100-20Cµg/kg), and for the prevention of heartworm infection in dogs (at 6µg/kg monthly). However, extra-label use by vets, at higher doses, is common for the control of other internal and external parasites, particularly in dogs. Ivermectin works by enhancing the activity of the inhibitory neurotransmitter GABA (γ-aminobutyric acid). In invertebrates, GABA acts peripherally, but in vertebrates it is only found in the central nervous system. In general, the blood-brain barrier in mammals is very effective at excluding ivermectin. However, in some breeds of dog (particularly collies and related breeds) the blood-brain barrier appears to be less effective, and at high enough doses, ivermectin can cause toxicity in any breed. Perhaps the age of these pups also contributed to their susceptibility.

The many faces of melioidosis - Helen Parkes, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

We had three really diverse cases of melioidosis within a week, in December 1999.

The first was a four-week-old goat with a history of hindleg paralysis of one week's duration. Post-mortem examination showed small abscesses in several lymph nodes in the throat area, as well as an abscess in the spleen. *Burkholderia pseudomallei* was isolated from both these sites, as well as from a swab of the lumbar spinal cord. Histological examination showed extensive mixed inflammation in the membranes around the lumbar spinal cord, as well as increased mononuclear cells in the central canal, with thick perivascular cuffs of neutrophils, lymphocytes and plasma cells, and focal abscessation, in the lumbar cord. Both the mother and father of this kid are serologically positive for melioidosis, but we were unable to culture the organism from the mother's milk.

B. pseudomallei was also grown from a sample of fluid taken from the scrotum of a dog, several days after castration due to a swollen testicle and high temperature. Histological examination of the testis and spermatic cord showed an epididymitis and multiple small abscesses in the spermatic cord, obviously extending into the abdomen.

The third case was an aged gibbon (about 40 years old), that had arrived in Darwin less than six months before and died after being noticed depressed and not eating for about a week. Post-mortem examination showed extensive pneumonia, with areas of dark red consolidation, a large (4cm diameter), thick-walled abscess filled with thin, brown pus in one lobe, and several small nodules (l-2mm diameter) scattered throughout the lungs. The liver was enlarged, with multiple pale foci. *B. pseudomallei* was cultured from

the lung and liver. Histological examination of the liver showed focal necrosis, with abundant Gram negative rods, and the lung had extensive lytic necrosis and haemorrhage, again with large numbers of organisms present. The changes were acute to sub-acute, with limited inflammatory response.

Wasting syndrome and deaths in farmed barramundi Lates calcarifer: peritonitis, enteritis and gill parasitism - John Humphrey, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

A syndrome characterised by wasting, poor condition, abdominal distension and low-grade mortalities in farmed juvenile barramundi approximately 10-12cm in length was investigated. The syndrome occurred in October 1999 in fish reared in earthen freshwater ponds fed a commercial pellet ration. Water quality examination showed oxygen at 4.7mg/1, T30°C and pH 8.1. Nitrite and ammonia were reported as satisfactory. Fish were submitted fixed in formalin for examination. A history of exposure to re-circulated effluent water was indicated.

Gross examination showed poor muscle development and a tucked in appearance of the abdominal musculature in some fish and marked abdominal distension caused by excess fluid in others.

Histological examination showed a range of changes and infectious agents, including:

* Colonisation of the intestinal lumen by large numbers of Gram negative rod-shaped bacteria, intense mucosal hyperaemia and dilation of the intestinal lumen.
* Poorly resorbed yolk sacs with a massive colonisation by Gram negative bacteria similar to those in the intestinal lumen.
* Ulceration and erosion of the integument with underlying necrosis of skeletal muscle associated with colonisation by mats of long, filamentous Gram negative bacteria consistent with *Flexibacter* sp.
* Low to moderate numbers of *Myxobolus* sp. cysts in the gills.
* *Trichodina* sp. in the gills of some fish, with generalised clubbing and hypertrophy of secondary lamellae epithelium.
* Moderate to high numbers of the pathogenic protozoan *Ichthyobodo necatrix* associated with the gill epithelium.
* Occasional cystic bodies typical of the rickettsial infection, epitheliocystis, present in the secondary lamellar epithelium.
* Sub-peritoneal oedema, fibrin deposits, haemorrhage, inflammatory cell infiltration and numerous Gram negative bacteria in the inflammatory exudate in fish showing abdominal distension.

A number of diagnoses were made including:

* Bacterial enteritis, Gram negative coccobacilli
* Bacterial yolk sac inflammation, Gram negative coccobacilli
* Myxoboliasis, gill
* Epitheliocystis, gill
* Ichthyobodiasis (Costiasis), gill
* Trichodiniasis, gill
* Epidermal ulceration (Flexibacter-like sp.)

The submitter was advised that the fish showed a range of serious or potentially serious infectious agents and that these agents will be present in the population as a whole, presenting an on-going threat to the health of the fish. Under the existing conditions, chemotherapy was problematic and strict attention to stress minimisation and optimisation of water quality were recommended.

Queensland - Jim Taylor

Presumed infectious canine hepatitis - John Mackie, Veterinary Pathology Services, Brisbane.

A 9-week-old unvaccinated Border collie pup died after being depressed and febrile for 24 hours with late development of neurological signs. The vaccination status of the dam was unknown. At necropsy the referring veterinarian noted visceral congestion.

Microscopically there was multifocal hepatocellular necrosis, mild non-suppurative encephalitis with vasculitis and mild interstitial pneumonia. Large intranuclear inclusion bodies were present in hepatocytes and in endothelial cells in the liver, brain, lung, kidney and lymph nodes. The combination of history, gross lesions and microscopic lesions was highly suggestive of infection with canine adenovirus-1. This disease appears to be very rare these days, presumably because of widespread vaccination, but as this case demonstrates, it is still out there!

Salinomycin toxicity in turkeys - Anita Gordon and Ross McKenzie Yeerongpilly Veterinary Laboratory, Queensland Department of Primary Industries

Forty out of 50 turkeys died over five days. There was a history of a change of feed seven days prior to the first deaths. The suspect feed came from an unlabelled bag at a produce store. One affected bird presented for necropsy showed gross, histological and biochemical evidence of widespread, severe myonecrosis.

Serum CPK and AST were 1,314,000.00 and 11,200.00 IU/L, respectively. At necropsy there was bilaterally symmetrical, streaky to diffuse pallor of limb musculature. Histological examination revealed swollen, glassy or fragmented muscle fibres, a few of which were mineralised, accompanied by evidence of early repair. Myocardium was unaffected. A presumptive diagnosis of ionophore toxicity was made.

The presence of salinomycin was confirmed by Pfizer Animal Health in Sydney. Two separate assays at different Pfizer laboratories yielded salinomycin at 30ppm and 630ppm respectively.

There are several reports of salinomycin intoxication of turkeys inadvertently fed chicken broiler rations containing 50-60ppm salinomycin as a coccidiostat (Harries and Hanson, 1991). Although the two widely different concentrations obtained by Pfizer suggest a mixing error at the feed mill, salinomycin has also been demonstrated to be toxic to turkeys at low inclusion rates (as little as 15-30ppm) (Stuart, 1983).

References

Harries, N. and Hanson, J. (1991). Can. Vet. J. 32:117. Stuart, J.C. (1983). Vet. Rec. 113:597.

Acute renal failure in a dog with a shiny coat - Richard Ploeg, University of Queensland

An eighteen month old male Shetland Sheep dog was presented to the referring veterinarian for the sudden onset of lethargy, depression, and anorexia four days after attending a show. The owner was concerned about sabotage given that she knew of at least three other dogs dying due to acute renal failure following show attendance over the last twelve months. At physical examination the animal was clinically dehydrated and had a stilted gait. Haematology and biochemistry revealed a PCV of 65L/L, TPP of l0lg/L, BUN of 61.6mmol/L and serum creatinine of 0.72mmol/L. A diagnosis of acute renal failure was made and intravenous fluid therapy commenced.

Twenty-four hours later the animal began to vomit repeatedly and pass large volumes of dilute urine (Usg 1.009). Intravenous fluid therapy was continued and blood work repeated, now three days after presentation. The azotaemia had improved (BUN-39.1mmol/L, serum creatinine- 0.38mmol/L) and there was now evidence of a mild hepatopathy (no details given). The dog was taken home only to be presented

the following day severely dehydrated and perpetually vomiting. The owner elected for euthanasia and necropsy, given the suspicious circumstances surrounding the animal's death.

The referring veterinarian carried out the necropsy and reported irregular pale blotching within the renal cortex. No other gross lesions were noted. Fragments of liver and kidney were presented. Histologically the proximal convoluted tubules contained irregular lightly basophilic amorphous material resembling shed epithelial cells as well as isolated epithelial cells undergoing coagulative necrosis. There was evidence of recent marked regenerative activity in the surrounding tubular epithelial cells. Also evident was marked renal papillary necrosis and diffuse dilatation of the distal convoluted tubules. No significant abnormalities were detected in liver sections.

A diagnosis of acute tubular nephrosis and papillary necrosis was made and the referring vet was asked to question the owner further regarding any possible exposure of the dog to medication/toxins at home. The owner assured the veterinarian that the only medication the animal had received was a parentally administered dose of a proprietary organic arsenical, which is used frequently by Sheltie breeders to improve the sheen of their coat prior to shows. The dog had received the medication two weeks prior to presentation to the veterinarian. The drug is registered in horses as an appetite stimulant and also as a remedy for a dry coat. The recommended dose is approximately 1mg/kg. The dog received a dose of 2.7mg/kg.

Although arsenic levels are pending on renal tissues the histological findings are consistent with those reported in dogs following parenteral arsenical administration (1). The referring veterinarian assures me that the dog did indeed have a shiny coat.

(1) Tsukamoto, H , Parker, H.R., Gribble, D.H., Mariassy, A., and Peoples, S.A. Nephrotoxicity of sodium arsenate in dogs. Am J Vet Res, 1983 **44**(12), pp 2324-2330.

Nitrate poisoning associated with Malva parviflora in hay - Anita Gordon and Ross McKenzie Yeerongpilly Veterinary Laboratory, Queensland Department of Primary Industries

Seven of 350 10-mth-old Brahman X calves died suddenly one week following weaning. The weaners had been yarded and fed on hay sourced from Biloela and containing up to 25% marshmallow (*Malva parviflora*). The hay had been produced on land irrigated with meatworks effluent.

Field necropsies of three calves revealed subcutaneous petechiae, and pulmonary congestion and oedema. Pale hindquarter musculature was evident to some extent in all three, and was also noticeable in the fixed samples submitted. However, there was no histological lesion to account for this pallor. Qualitative testing was positive for blood nitrate, despite the absence of gross methaemoglobinemia (brown discolouration). The hay was found to contain 8% $KN0_3$ on a dry matter basis. (Concentrations of $KN0_3$ >1.5% DM are considered dangerous).

M. parviflora is known as a potential nitrate-accumulating plant, but actual cases are rare. In this case the incorporation of the plant into hay made it more likely that poisoning would occur. There is evidence that *M. parviflora* will cause skeletal muscle damage in sheep, particularly in the hindquarters, but this effect was not demonstrable in this case.

Syngamus trachea in brush-turkey poults - Anita Gordon, Yeerongpilly Veterinary Laboratory, Queensland Department of Primary Industries

An experimental rainforest aviary in Maleny was used to house newly hatched brush-turkeys (*Alectura lathami*). Only six birds at a time were reared, and several cohorts passed through the aviary uneventfully. All six birds in the final cohort were observed to sneeze, wheeze, become lethargic and die between the ages of 1-8 weeks. The last dead bird was submitted for necropsy.

Necropsy revealed partial obstruction of the distal half of the trachea by 20-30 *Syngamus trachea*. Histological changes comprised hyperplasia and squamous metaplasia of the tracheal epithelium, together with early fibrosis of the lamina propria. Inflammatory cell infiltration was not a major feature.

Although this parasite has a wide host range in both domestic and wild birds (Harrigan 1978), our parasitologist, Peter Green, can't remember when he last saw this parasite, and would be interested to hear about other laboratories' experience. In this case, the practice of providing food at the same site in the aviary probably contributed to faecal contamination and subsequent heavy infection in the last cohort of birds.

Reference:

Harrigan, KE (1978). Parasitic Diseases of Birds. Pp 489-536 in PGCVS Proc. No. 36.

Argentine Report - Roger Kelly, University of Queensland

I had an unusual Christmas break this year: two months giving workshops and working on enterotoxaemia in lambs, based at the INTA laboratory at San Carlos de Bariloche, on the shores of a lake tucked up under the eastern foothills of the Patagonian Andes. The program was organised by Paco Uzal who had done his PhD with us at UQ on enterotoxaemia in goats, and the aim was to see if the goat model is applicable to sheep. It seems to be, and when the ELISAs are done we should have an accurate idea of what levels of anti-epsilon antibody in lambs are protective.

The sheep industry in Patagonia has many problems paralleling those in Australia: Sow wool prices, of course, coupled with overgrazing, poor roads and infrastructure. Some are trying feedlotting of lambs which raises all the disease spectres associated with intensification (coccidiosis, PE, enterotoxaemia, Cu poisoning, etc). The laboratory support available for diagnosis in the more remote areas is poor, and in this sense remoteness begins not all that far from Buenos Aires.

There is a more hopeful air about the beef industry with the progressive declaration of foot and mouth free zones and they are looking forward to access to a wider range of export markets. They are also looking at farming their indigenous camelids like the guanaco and vicuña and the native ratites (rhea or choique) for people with more money that wisdom to invest in. But cattle and horses remain the status species in the animal industries.

Bovine Ephemeral Fever in a feedlot - John Gibson, Toowoomba Veterinary Laboratory, Queensland Department of Primary Industries

Ephemeral fever was diagnosed in a 17,000 head feedlot in central Queensland. Fifteen animals died and a further 25 were sick at the time of initial sampling. Affected animals were 20 to 30 month old Brahman cross steers weighing 400 to 450kgs that had been on feed 10 to 21 days. Early clinical signs included fever, lethargy and lameness, progressing to recumbency. Some animals remained recumbent for 8 days but a majority responded to phenylbutazone treatment. Other clinical signs observed in a few animals included knuckling of fetlocks and hyperexcitability. Cattle in paddocks surrounding the feedlot were reported to have similar clinical signs. Ephemeral fever virus was isolated from EDTA blood samples from 7 of 21 animals cultured during the course of the outbreak. Many early samples were drawn from recumbent afebrile animals and results for virus isolation were poor. But when blood was collected from febrile, ambulant animals, virus was recovered from 6 of 11 animals sampled. Hyperfibrinogenaemia was a consistent finding with 19 of 22 samples > 0.7g/dl (mean l.0lg/dl). Paired samples were collected from 10 animals for serology with 7 animals showing a five fold increase in litre of serum neutralising antibodies to ephemeral fever virus.

Myxosporidiosis in eels - Greg Storie, Yeerongpilly Veterinary Laboratory, Queensland Department of Primary Industries

Three hundred 500 gram, long fin eels died and another 550 were affected in a pond of 6,000 on an eel farm in southeast Queensland. Affected eels had numerous, white to pink, 1-10 mm diameter smooth round and fleshy nodules covering the skin and fins. The nodules were confluent in some areas, with some ulceration. Squash preparations revealed numerous myxosporean spores morphologically consistent with those of the genus Myxidium.

The method of transmission for most myxosporeans is unknown, but most probably has an indirect life cycle involving a fish and an annelid host, with both sexual and asexual stages in each host. Light infestations are usually relatively harmless and cause only moderate host reaction, but very heavy infestations such as this can be associated with very high mortalities

South Australia - Ruth Reuter

Murine Pulmonary Histiocytosis (crystal pneumonitis) - John Finnie, Veterinary Services Division, IMVS, Adelaide

Six cases of this pulmonary disorder have been diagnosed in transgenic (Eμ-Pim I) mice. At necropsy, large areas of lung were consolidated and, microscopically, many alveoli were filled with macrophages and a few multinucleated giant cells. In some of these, mononuclear cells were large, eosinophilic, needle-like crystals, which are the hallmark of this condition. In addition, there were a few neutrophil aggregations and patchy type II pneumocyte metaplasia. Previous ultrastructural studies have shown that these crystals are produced in lysosomes of macrophages, but their aetiology is unknown. These lesions were originally described in mice with an autosomal recessive mutation (motheaten gene) and these animals died from this unusual pneumonia at 4-8 weeks of age.

Ward JM (1978) Pulmonary pathology of the motheaten mouse. Vet Pathol 15:170-178

Spontaneous corneal opacity in mice - John Finnie, Veterinary Services Division, IMVS, Adelaide

All mice in one box from a shipment of many boxes from the U.S., were found dead on arrival in Australia. These animals invariably had corneal opacities due to acute keratitis and early corneal ulceration. The development of these lesions has been attributed to exposure to excessive ammonia and it is probable in this case, that the ventilation areas of the box were inadvertently covered over when the boxes were stacked for shipment.

Van Winkle TJ and Balk MW (1986) Spontaneous corneal opacities in laboratory mice. Lab Anim Sci 36:248-255

Rhodococcus equi lymphadenitis and abscess at ion in a 3 month old foal - Steve McOrist, Veterinary Pathology Services, Adelaide

A three month old colt foal had been "off colour and colicky", and had been treated symptomatically with antibiotics. The animal appeared to make a complete recovery. It was then found with complete posterior paralysis approximately two weeks later. On clinical examination there was an area of anaesthesia in the lower lumbar region and groin. The temperature was normal. A spinal abscess was suspected and the foal was euthanased and submitted to the laboratory for post mortem examination.

On post mortem there were numerous abscesses ranging from 5 to 80 mm diameters in most of the mesenteric lymph nodes. The abscesses were surrounded by 2 to 5mm thick fibrous capsules, with thin yellow purulent fluid in the centre. There was local serosal inflammation. The larger abscesses were located in the upper abdomen adjacent to the spinal column. On culture, a heavy growth of *Rhodococcus equi* was isolated from the abscesses. On histopathology, large numbers of macrophages containing intracellular bacteria were seen in granulomas in the affected tissue.

Rhodococcus is a relatively common cause of infections in foals, with pneumonia the most commonly recognised presentation. However, necrotising lymphadenitis, joint and eye infections can also be seen. *R. equi* is largely a soil borne organism, whose growth requirements appear to be met ideally by herbivore manure in temperate climates. Numbers of organisms on infected pastures can increase many thousands of times under ideal conditions, making contaminated foal pastures a significant health hazard on affected farms. *R. equi* are able to evade macrophage attack by preventing phagosome fusion with lysosomes. Virulence factors are thought to be more active at elevated body temperatures than at lower ones.

Necrolytic Migratory Erythema - Julia Lucas, Veterinary Pathology Services, Adelaide.

A 9 year old fox terrier presented for a chronic dermatopathy. The referring clinician did not provide any clinical description of the lesions, but submitted three punch biopsies. Samples were labelled: periscrotal skin, elbow and paw.

Four small punch biopsies of skin were examined. In each section there was hyperplasia and acanthosis of the epidermis and a severe interface dermatitis. There was moderate parakeratotic hyperkeratosis and focal areas of vacuolation and swelling of the upper layers of the epidermis. There was exocytosis of numerous neutrophils through the epidermis. On the surface of the epidermis the degenerate neutrophils formed a crust over areas of ulceration and there was colonisation by large numbers of coccoid bacteria consistent with *Staphylococcus spp.*

In the dermis the inflammatory reaction varied from areas composed predominantly of macrophages to areas composed of large numbers of plasma cells and lymphocytes with moderate numbers of neutrophils and occasional eosinophils.

Several weeks later the veterinarian reported that the lesion had not responded to antibiotic or corticosteroids and the dog died. The referring vet had performed a necropsy and the liver was small and fibrotic.

The lesion is histologically consistent with necrolytic migratory erythema (NME) and the necropsy findings supported this diagnosis. This is a rare condition usually associated with liver disease, diabetes mellitus or glucagon-producing pancreatic neoplasms [1,2,5,9.10,11,12]. Synonyms include hepatocutaneous syndrome, diabetic dermatopathy, and superficial migratory dermatitis. A comparable syndrome is described in humans usually associated with glucagon-secreting tumours, but a few cases have been described with other disease including liver disease [4,6,7,8,9,12]. In dogs only 3 cases reported in the literature were associated with glucagon-secreting neoplasia [1,2,10,11]. Most cases in dogs are associated with other disorders such as Diabetes mellitus or liver disease [1,5,9].

The pathogenesis of the lesion is not understood but it has been suggested that it may be related to metabolic abnormalities involving proteins, biotin, essential fatty acids or zinc [3,9]. Although it is postulated that glucagon is not the cause of the lesion, in one case removal of the tumour lead to resolution of the skin lesions[10].

Clinical presentation is typically old dogs, without breed or sex predilection [9]. The skin disease is usually the presenting complaint and only in rare cases is the systemic disease the reason for initial presentation [9]. In one case the dog developed skin disease several months before developing hyperglycaemia (Diabetes mellitus) [2].

Lesions are found in areas of trauma such as the mucocutaneous junctions, muzzle, distal limbs and footpads [9]. Lesions may also occur on the ventrum, elbows and hocks. In some cases antinuclear antibodies may be detected [9].

Biochemical results are variable and may reflect the underlying systemic disease of diabetes or liver failure [9]. However, hypoaminoacidaemia seems a more consistent finding [9,10]. Glucagon levels are usually elevated in cases associated with pancreatic islet neoplasms [9]. In skin cytology and histological sections, bacteria, yeasts or dermatophytes may be present [9].

Differential diagnoses include other dermatopathies characterised by parakeratosis and superficial inflammation. However, the oedema, vacuolation and swelling of epithelial cells in the upper layers of the epidermis is characteristic of NME [9]. This feature may be subtle or absent in chronic lesions [9]. Differential diagnoses include Pemphigus foliaceous, zinc deficiency and generic dog food dermatosis, which feature parakeratosis [9]. In chronic lesions NME may have colonisation by yeast, dermatophytes or bacteria, crusts

on the surface and an interface inflammatory pattern [9]. Thus it must differentiated from auto-immune disease such as systemic or discoid lupus erythematosus as well Pemphigus foliaceous.

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Tasmania - Philip Ladds

Mount Pleasant Laboratory Report - Phil Ladds, DPI, Mount Pleasant

Interesting and varied submissions have been received at the Mount Pleasant Laboratory during recent months. Most bovine lesions examined as part of the National Granuloma program are "club-forming", especially actinobacillosis, but *Rhodococcus equi* was isolated from several lymph node granulomas - one of which had microscopic changes largely indistinguishable from tuberculosis.

Ovine cases included severe nutritional muscular dystrophy with obvious myocardial lesions in 2-month-old lambs that died suddenly, and deaths of lambs with anaemia and jaundice, and prevalent *Eperythrozoon ovis* in erythrocytes - seemingly incriminating *E.ovis* as an important, primary pathogen? Necropsy of three young rams from a flock in which 18 had died soon after they were placed on lush (clover) pasture, revealed intense pulmonary congestion, haemorrhage and emphysema, perhaps suggestive of a 3 methyl indole-type toxicity. Such toxicity has been described in sheep in Scandinavia.

High mortality in free-ranging poultry (~200 birds over several months) was associated with fibrino-suppurative serositis, and *Pasteurella multocida* was isolated. Deaths of 20 cage and aviary birds, in a group of 90, were due to *Yersinia pseudotuberculosis* infection; lesions were those of multi-focal necrotising hepatitis and encephalitis. Attempts to farm Cape Barren geese have been severely compromised by fatal pulmonary aspergillosis in goslings.

Interesting wildlife cases included *Toxoplasma gondii* myocarditis and encephalitis in a brush-tail possum, salmonellosis with intussusception in a wallaby, and a severe multi-nodular enteritis in a pademelon caused by infestation with a spirurid nematode, tentatively identified by Dr Ian Beveridge, Univ. of Melbourne, as *Cylicospirura heydoni*. Work in the Mt. Pleasant Fish Health Unit has included an ongoing study of mudworms (*Boccarda knoxi*) in abalone, health assessment of cultured rock lobsters, and jellyfish toxicity in farmed salmonids.

Victoria - Malcolm Lancaster

Concurrent Bovine Chlamydiosis/Perennial Ryegrass Staggers - Malcolm Lancaster VIAS Attwood

Several property owners in north-eastern Victoria reported cattle with head tremors and stiff forelimb movements, associated with the grazing of perennial ryegrass. Three animals from one farm were necropsied after low level CFT titres to Chlamydia were found. Gross changes were restricted to the body cavities. All three animals had increased fibrin levels in the fluid of various body cavities as demonstrated by the formation of loose clots before or after the body cavities were opened. Slightly roughened areas of parietal pleura were seen in all three animals, with other serosal areas inconsistently affected.

Histologically, serosal changes were minimal, with small accumulations of mixed mononuclear cells in the parietal pleura and villous epithelial hyperplasia. The animal with head tremors had diffuse irregular vacuolation of the molecular layer in all cerebellar lobes and scattered swollen Purkinje cell axons characteristic of perennial ryegrass staggers. Cerebellar lesions were not seen in the two animals without head tremors and meningitis was not evident in any animal.

As increased vascular permeability and pleuritis are not recorded as part of perennial ryegrass staggers, these animals may have suffered a mild chlamydial infection as well as lolitrem intoxication. Chlamydial elementary bodies were not demonstrated in serosal smears, and no Chlamydia grew following egg inoculation, but all three animals had been treated with long-acting tetracycline.

Capillariasis in Domestic Turkey - Judith S. Nimmo Wilkie. Idexx/CVDL

Several young birds (3.5 months of age) from a free-range flock of domestic turkeys exhibited signs of lethargy, weight loss and a hunched posture. One was noticed to regurgitate fluid. Two birds had died.

Microscopic examination of the crop of an affected bird showed a marked hyperplasia of the epidermis and a severe ingluviitis with a broad submucosal infiltrate composed of heterophils and mononuclear inflammatory cells. In burrows in the epithelium there were numerous adult nematodes and clusters of nematode eggs with the bipolar plugs characteristic of *Capillaria spp*. There is some heterophilic infiltration around some of the parasites and eggs.

Capillariasis in turkeys and other wild birds has been associated with significant mortalities. Death occurs secondary to emaciation because affected crops are unable to drain effectively and become filled with fetid fluid.

Capillaria annulata and *C. contorta* are the two species that affect the crop and oesophagus. These species are similar morphologically (annulata has a cuticular swelling behind the head) and may be a single species. The life cycle is believed to involve earthworms as an intermediate host for the former species and to be direct for the latter. It was not evident from the cross sections of the adult worms in this case which species was involved.

Western Australia - David Forshaw

Haemagglutinating Encephalomyelitis Virus in sucker pigs - David Forshaw and Barry Richards Agriculture WA, Albany

20/250 two-week-old piglets died over two weeks. A total of 40/500 in the batch eventually died. Fixed tissue and blood from two piglets was submitted. The submitting veterinarian investigated a syndrome he described as "piglets that were scouring mildly before dying". No nervous signs were noted but subsequent discussion with farm workers established that vomiting was seen in some piglets and some surviving piglets were emaciated. The workers commented that affected piglets could be identified by the "sound of fluid sloshing around in their guts" when they were picked up.

The brain of one piglet had a severe non-suppurative menigoencephalitis consisting of neuronophagia, neuronal necrosis, gliosis and lymphoplasmacytic cuffing. Lesions were particularly severe in the ventral hippocampus and adjacent cerebral cortex. The other piglet had mild encephalitis with similar distribution and characterised by thick vascular cuffs of lymphocytes and plasma cells. There was also an unusual enteritis in one piglet which consisted of necrotic cells beneath the lumenal enterocytes and a layer of mixed inflammatory cells in the submucosa of the colon. Bacterial culture failed to demonstrate the presence of recognised pathogens.

An additional five ill thrifty animals were necropsied and sera collected. Apart from emaciation, no gross or microscopic lesions were identified. Twelve serum samples sent to the Victorian Institute of Animal Science, including samples from the two original piglets with encephalitis, were tested for haemagglutinating encephalomyelitis virus (HEV) in the haemagglutination inhibition test - all were strongly positive, 7 with very high titres (>1:8192).

Sera sent to AAHL was negative for Aujeszky's disease antibodies by Latex Agglutination Test and no Aujeszky's disease virus was isolated from tissues from ill thifty piglets.

The piggery is an extensive 1000 sow unit split into two 500 sow farms operated separately and physically separated by a distance of approximately 200m. Piglets were affected in concurrent batches on both farms but after the initial necropsies were done subsequent batches were unaffected and older piglets remained unaffected. The grower pigs are moved to various grow-out facilities in different parts of the state. Breeding stock come from a single source.

The differential diagnosis included Aujeszky's disease, Haemagglutinating Encephalomyelitis Virus (vomiting and wasting disease) and encephalitis caused by Enterovirus. HEV infection was suspected because vomiting and wasting was seen, lesions were not seen in other systems (apart from the unusual gut lesions, the significance of which is not clear) and the disease did not spread as rapidly as would be expected with Aujeszky's disease. Inclusion bodies were not seen in the acute severe CNS. Strongly positive HEV serology confirmed the diagnosis.

HEV has not previously been diagnosed in WA. We understand it to be endemic and widespread in Victoria where it causes sporadic losses and does not constitute a serious industry problem. We would be interested to hear of the situation in other states.

Sorghum Neurotoxicity in Weaner Sheep - J Creeper Animal Health Laboratories - Perth

Of 800 weaners running on a 45cm high sorghum crop, 8 were seen to be exhibiting nervous signs of muscular tremors of the head and neck, nystagmus and extension of the forelimbs. Another weaner was seen to be aimlessly running about, then collapsing. Formalin fixed sections of brain and upper cervical spinal cord from the worst affected animal revealed axonal spheroids throughout the brain, being most numerous adjacent to the cerebellar roof nuclei. Additionally, there was a focal area of Wallerian

degeneration within the cerebellar white and several axonal spheroids within the ventral gray matter of the cervical cord adjacent to the motor neurones.

Similar changes have been previously described in NSW in sheep, goats and calves. A good description by John Glastonbury and Barbara Maloney can be found in the 1997 ASVP Proceedings.

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