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VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
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DEADLINE FOR NEXT VET. PATH REPORT IS JANUARY 30, 1995

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Chairperson (Registry of Domestic Animal Pathology)	Tony Ross
Newsletter Editor	Jim Taylor
Coordinator (Training Committee)	Robin Giesecke

CONVENOR - SLIDE OF THE MONTH

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EDITORIAL

The letters addressing the possible affiliation of the ASVP with the AVA have been limited. The two letters received appear later in the report. There have also been some comments submitted. These are basically along the line of no objections provided there are no disadvantages such as increase in costs to the membership, loss of the low key administration of the society and restrictions on membership. Although there was little correspondence on this issue I feel that there has been extensive discussion on this matter over the last couple of years and members generally have well established views on this matter. In line with the resolution passed at the last AGM the next VPR will contain the conditions for affiliation and a postal vote for all members to finally decide the matter.

Next year the current executive and committee will see out their term in office and by convention the new executive and committee will be formed in Victoria. In the next VPR I will be seeking nominations and ask all Victorian members to consider positions on the executive and committee.

I also need the assistance of a local organiser for the conference in Melbourne in May. This basically involves obtaining some prices and information on possible venues and accommodation. We would like to keep this in line with the two previous conferences with package deals for both venues and accommodation, but I am open to suggestions. If you can help please contact me.

I have been speaking to Robin Giesecke and Robin hopes to have a questionnaire for membership on the training needs for circulation early next year.

Finally on behalf of the executive and committee I would like to wish all members the best for the coming festive season.

Regards to all.

Jim Taylor.

QUEENSLAN D - Greg Storie

Acute Respiratory Disease Syndrome in Horses - Peter Ketterer (Yeerongpilly Vet Lab), Peter Hooper (AAHL).

On 22 September 1994 an emergency situation was recognised at a Hendra stables in Brisbane. Onset of severe illness in 8-9 horses had occurred on 19 September 1994. Signs observed were depression, anorexia, weakness, high fever of 40 to 41 and severe respiratory difficulty in later stages with frothy discharge from the nose terminally. Eleven animals were euthanased or died naturally between 20 September 1994 and 26 September 1994. One animal in an adjacent neighbouring stable was euthanased after showing signs of severe illness for only several hours. Another animal which had left the stables on 16 September 1994 died on a Kenilworth property after showing similar signs. The index case was suspected to be a mare which entered the stables from a spelling paddock at Cannon Hill on 7 September 1994. This animal died on 9 September 1994 after showing severe respiratory difficulty and terminal frothy discharge from the nose.

The consistent necropsy findings in all animals were cyanosis and petechiation of the buccal mucosa, stable froth in the trachea and bronchi and severe congestion and oedema of the lungs with multiple focal haemorrhages on the pleural and cut surfaces. The cut surface of the lung exuded copious blood stained fluid. Some animals showed mild to moderate jaundice, petechiation of the peritoneum and epicardial haemorrhage. One horse showed petechiation of the gastric mucosa.

Histologically, lung was congested and had large focal or extensive haemorrhages which were frequently associated with fibrin thrombi in capillaries and arterioles. Oedema was extensive in ventral lung sections and alveolar lesions were larger, more advanced lesions showed alveolar necrosis and fibrinous alveolar exudate containing some neutrophils. As the lesion progressed a cellular exudate consisting of macrophages occurred as well as small foci of hypertrophy and hyperplasia of type 2 pneumocytes. Multinucleate syncytia were present in the alveoli in some lung sections in both peracute haemorrhagic and later stages. Multinucleate or polymorphonuclear cells with little cytoplasm were numerous in arterioles and capillaries in some early lesions. These resembled megakaryocytes but in arterioles they appeared to be derived from endothelium. Possible eosinophilic intracytoplasmic and intranuclear inclusions were seen very rarely in a few macrophages and septal cells. The lung lesion was interpreted as a peracute or acute interstitial pneumonia which was associated with intravascular coagulation in arterioles and alveolar capillaries.

A paramyxovirus causing prominent CPE with syncytium formation in equine dermal cells and other cell lines has been isolated from lung specimens of 5 of 6 animals sampled at necropsy. Antibodies to this virus have been demonstrated in several horses which recovered. Transmission of lung material an of cell culture isolate at the Australian Animal Health Laboratory, Geelong has reproduced an acute fatal respiratory disease in horses and confirmed that the virus is the cause of the condition. Further Morphological characterisation at AAHL has demonstrated that the virus is a morbillivirus (measles, distemper, rinderpest group) but no relation ship to known morbilliviruses has been shown as yet.

Serological survey of horses in Queensland has so far demonstrated no positive animals outside the three infected sites. Several cases of human infection have been verified serologically including the trainer Vic Rail who died with an acute lung disease.

Acknowledgments

Thanks to Geoff Mitchell, Veterinary Pathology Services and Roger Kelly, University of Queensland for input to this report.

YEERONGPILLY VETERINARY LABORATORY

Idiopathic Heart Failure/Liver Necrosis Syndrome

Grower pigs on a Beaudesert piggery began suffering from dyspnoea and sudden death during November 1993. Only that group which had suffered an incident of nutritionally induced diarrhoea later developed the syndrome. Necropsy findings were dominated by dilation of the heart, excessive volumes of fluid in body cavities and swollen congested liver (chronic venous congestion). Pulmonary oedema and extensive pneumonia was present in some but not all affected pigs. Histologically there was extensive centrilobular, in some cases almost panlobular hepatocyte loss with variable replacement haemorrhage (anoxia induced?) in livers. Lungs frequently showed severe interstitial pneumonia and oedema. No significant histopathological lesions were seen in the heart muscle. The cause of the syndrome is presently unresolved.

The scouring which preceded this syndrome occurred in weaners (4-10 weeks) held in 2 pens adjacent to the farrowing shed. A nutritionally dense diet high in energy and protein and comprised of canola meal, full-fat soya meal, fish meal and vegetable oil was considered the cause of the scour. A low specification feed was introduced and also (a week later) medication of the feed with lincomycin (44g/ton) and neomycin (300g/ton) was carried out for a week. Canola meal still comprised 25% of the ration. The scouring ceased but 2 weeks later the sudden deaths commenced. At this time the pigs had been shifted from the weaner pens to the nearby grower shed consisting of pens with solid walls on each side of a central walkway. Excreta was collected beneath the pens and removed, draining (without flushing) every 2-3 days. Pigs died at the rate of 1-2 per day and a total of 63 pigs died in the 2 month period to 10-1-94.

Biochemical estimations on samples from 10 pigs of the affected group were carried out. Serum vitamin E and blood glutathione peroxidase levels were normal; serum iron was low in 2 pigs, 299 and 704 ug/L (ref 730-1400) and serum copper was high in 6 pigs, 1347 to 1726 ug/L (ref 800-1200). Further biochemistry was done on tissue samples from 3 pigs with typical liver and heart lesions. Liver and kidney selenium levels were normal; liver copper was low in 2 pigs, 11.4 and 6.7 ppm (ref >15) but kidney copper levels were normal. Hydrogen sulphide poisoning from the effluent pit, blue green algae toxicity from the drinking water (an on-site dam) and plant seed toxicity including pyrrolizidine alkaloids were also considered but not implicated in the disease syndrome.

Avian pulmonary tuberculosis in broiler breeders

Avian pulmonary tuberculosis was diagnosed histologically in a fixed lung specimen submitted from one of a flock of male broiler breeders in the Lockyer district in mid December 1993. Acid-fast bacilli were present in moderate numbers in the multiple small caseous granulomas in lung tissue. Thirty of 500 birds had died in a flock of 30-week-old birds. Clinical signs described were anorexia, lethargy and loss of weight. Diarrhoea was also seen in this flock, but the connection with the tuberculosis was uncertain. The flock was receiving chlortetracycline medication. Further samples from the flock for laboratory study have had no gross or histological lesions of tuberculosis. Tuberculosis in poultry caused by *Mycobacterium avium* serotypes 1 or 2 is typically seen as intestinal, liver and spleen lesions. These lesions have not been reported from the flock in question. It appears possible that the unidentified acid-fast bacilli seen in the lung lesions were an opportunistic pathogen of environmental origin.

Tail Rot in Eels

Heavy losses occurred in eels (Anguilla reinhardti) in a holding tank on Reddacliff Peninsula after harvest at Bjelke Petersen Dam and transport to Brisbane. Losses commenced 4 days after introduction and by day 7 about 200kg of a total of 400kg had died. Severe tail erosion with extensive skin ulceration progressing forward on both sides was seen in 5 eels (length 72-132cm) examined at the laboratory. A thin translucent membrane which readily peeled off was present on affected skin areas. This was seen to be composed of a mat of fungal hyphae consistent with Saprolegnia sp on histological examination. Internal organs and gills were not affected on gross and microscopic examination and bacterial cultures of skin and kidney resulted

in isolates of Aeromonas hydrophila from skin only. The diagnosis made was tail rot due to Seprolegnia sp and Aeromonas hydrophila and this was considered secondary to stress caused by capture, transport, high stocking density and water quality problems.

Crotalaria dissitiflora (pyrrolizidine alkaloidosis)

Typical pyrrolizidine alkaloidosis was seen in 4 stock horses grazing Mitchell grass downs containing a large population of *Crotalaria dissitiflora* (grey rattlepod) near Richmond in mid-February 1994. All 4 horses died or were killed humanely. The last recorded case of horses with pyrrolizidine alkaloidosis associated with this Crotalaria species was in 1947 by C. T. White, then Government Botanist. Plant specimens from the current case were placed in the Queensland Herbarium as vouchers (AQ623846; AQ624771). Curiously, the test for pyrrolic metabolites on the liver of one horse with typical lesions was negative.

Nitrate-nitrite poisoning from button grass

Nitrate-nitrite poisoning was diagnosed in a group of 152 1-2 year old Brahman steers trucked from Charters Towers, dipped in Barricade S at Maxwelton on 10 May, trucked to a property outside Maxwelton and placed into cattle yards full of dried-off button grass. On the morning of 11 May, 5 were found dead and a further 6 were sick of which 2 died within a few hours. Necropsy revealed dark brown discolouration of the blood. An aqueous humour sample from one dead steer contained 75mg nitrate/L (normal ca 5mg/L). A sample of the remaining button grass (*Dactyloctenium radulans*) contained 4.8% KNO3 in dry matter. Concentrations over 1.5% KNO3 are hazardous to ruminants. The important point about this case is that even *dried-off* button grass is a high-soil-nitrate situation, such as cattle yards is a serious hazard to stock.

Oleander poisoning

Poisoning by oleander (*Nerium oleander*) was diagnosed in a group of 50 20-month-old Santa Gertrudis-cross weaners introduced 5 days previously to a property at Nanango in early June 1994. The group had access to oleander trees in a house yard and the pasture available was of poor quality. Severe dysentery was observed. Five animals died. Necropsy of one animal that died over 24 hours after onset of illness revealed haemorrhage and congestion in the large intestine, cyanosis and ecchymoses over an enlarged heart. Oleander leaves were recognised in the rumen. Multiple scattered foci of degeneration and necrosis were seen in cardiac muscle submitted for histopathology.

VETERINARY PATHOLOGY SERVICES (BRISBANE)

Cardiomyopathy and Atrial Thrombosis in a Foal

Clinical examination of a 6-months old thoroughbred foal indicated loss of condition, depression, recumbency and exaggerated heart sounds.

The most prominent post gross autopsy finding was hydropericardium. On histopathological examination of the right ventricular myocardium and atria, there were multifocal areas of myocardial degeneration. Associated with the atrial endocardium there was fibrin and platelet thrombus formation with evidence of early organisation.

These changes lead to a diagnosis of cardiomyopathy with atrial thrombosis occurring secondarily to disordered myocardial contractility of some duration.

Aetiological suggestions centred on the possibility of a congenital cardiomyopathy of an acquired condition, possibly associated with a selenium/vitamin E responsive syndrome.

Herpes virus pneumonia in a feedlot steer

Portions of lung from a feedlot steer with laboured breathing showed extensive grey consolidation with pale bronchogenic patterns. Microscopically there was a necrotising bronchiolitis and interstitial pneumonia with prominent hyperplasia of Type II pneumocytes to give alveoli an epithelialised or pseudoglandular appearance. There was abundant accumulation of neutrophils within airways and alveoli. Numerous intranuclear eosinophilic inclusion bodies typical of herpesvirus were found in airway and alveolar epithelium.

These changes are typical of those associated with the more severe respiratory manifestations of bovine herpes virus, Infectious Bovine Rhinotracheitis (IBR) infection which is common in feedlots in North America and being encountered with increasing frequency in Australian feedlots.

Dermatophyte infection as a cause of Pyogranulomatous Dermatitis in a Cat

Microsporum canis was cultured from chronic suppurative cutaneous lesions in the lumbosacral area of a cat which has persisted for more than a year. The provisional diagnosis was cutaneous mycobacteriosis.

Histopathologically there was a severe deep dermal inflammatory reaction characterised by a multifocal and coalescing granulomatous response to numerous, relatively large club colony formations of non-pigmented yeast and hyphal forms of a fungus with the microns in diameter. Focal aggregations of foamy macrophages were a feature of the granulomatous response. There was sinus tract formation with ulceration of the overlying epidermis to give the lesion characteristics of a mycetoma.

Cutaneous Nodular Dermatofibrosis of German Shepherds associated with Renal Cystic disease and Neoplasia

Several cases of nodular dermatofibrosis (a recently described reputedly rare syndrome in German Shepherds) have recently been diagnosed by VPS.

This syndrome presents clinically as multiple firm well circumscribed dermal and subcutaneous nodules varying from several millimetres to 4cm in diameter. The overlying skin may show irregular thickening and hyperpigmentation, and larger lesions are often alopecic and ulcerated.

The lesions are found primarily on the limbs, head and ears, usually middle aged or older.

The skin lesions are of particular interest because they may act as a marker for an internal malignancy, specifically renal cystadenocarcinoma. A metastatic tumour of this type has been confirmed histologically in a Sydney German Shepherd dog affected with dermatofibrosis.

Also, in keeping with overseas findings, non-neoplastic renal cystic disease has also been associated with this condition in some of our cases.

The disease is reported to have a dominant mode of inheritance and overseas has been seen in cross bred dogs with German Shepherd parentage. The relationship of the skin lesions in the development of renal neoplasms is not understood, but in humans, several familiar syndromes of multiple cutaneous tumours associated with specific internal malignancies exist. Cutaneous nodular dermatofibrosis is the first such entity to be documented in animals.

Chlamydiosis in an Ostrich

Chlamydial infection has received little attention as a potential disease problem in Australian ostriches, but the recent finding at VPS of a positive chlamydial antigen rest on a nasal swab from a 12 months old ostrich with catarrhal rhinitis and sinusitis may indicate the desirability of closer surveillance for this disease. The bird was serologically negative for *Aspergillus sp* infection.

Catastrophic ILT mortality

Tissues for histopathological examination were received following an investigation of the deaths of 130 of a group of 140 bantam chickens which had dyspnoea as the salient clinical finding.

On histopathology, there was a severe diphtheritic tracheitis with formation of a necrotic exudative cast containing exfoliated tracheal epithelial cells, many of which contained intranuclear inclusion bodies. The tracheal mucosa was haemorrhagic, ulcerated and had mixed leucocytic infiltration. Numerous bacterial colonies were also present in the cast. Similar changes were evident in larger airways in the lung.

A diagnosis of ILT (Infectious Laryngotracheitis) was made on the basis of the distinctive necrotising and inflammatory changes in the trachea and typical herpes virus-type inclusion bodies.

This disease outbreak illustrates the catastrophic nature of the mortalities which can be associated with infection of a virulent strain of ILT virus. In this case there was perivascular mononuclear cuffing in the brain and heart reminiscent of that which may occur in fowl plague and avian influenza, possibilities which are being investigated by the relevant authorities.

DEPARTMENT OF BIOMEDICAL AND TROPICAL VETERINARY SCIENCES, JAMES COOK UNIVERSITY, TOWNSVILLE - PW Ladds

Current research projects in pathology in the Department concern diseases of hatchling crocodiles and disease-handling-husbandry relationships in green sea turtles (*Chelonia mydas*) in Indonesia.

Interesting diagnostic cases seen recently were:

Equine: Colitis X, Neonatal septicaemia with enteritis in a foal, pyogranulomatous rhinitis due to pigmented fungi.

Canine: Babesiosis (in pups), viral (probably adenoviral) hepatitis with frequent inclusions (It’s years since we've seen a case of ICH, how about elsewhere in Australia?), granulomatous meningoencephalitis, Hodgkin's-like lymphosarcoma, gastric leiomyosarcoma and metastasising sarcoma possibly primary myocardial rhabdomyosarcoma.

Porcine: Severe non-suppurative encephalitis in a number of weaner pigs. Material sent from these to AAHL was negative for swine fever, African swine fever and Aujeszky's.

Crocodile: Cutaneous mycosis, poxvirus, dermatitis, probable dermatophilosis, coccidiosis, septicaemia and suppurative meningitis due to *Providencia rettgeri*, and chronic enteritis/pneumonia/septicaemia in a 70 year old crocodile that also had mineralisation and Sertoli cell hyperplasia in the testes.

Guinea pig: Probable aflatoxicosis, severe neonatal cryptosporidiosis.

Other: Severe necrotising enteritis (of undetermined primary cause) in a jungle carpet python, haemoglobinuric nephrosis in a Major Mitchell parrot, and ulcerative typhlitis in a koala.

L Owens

7.

A retail aquarist from central Queensland was having large scale losses of goldfish (*Crassius auratus*), both at his premises and at customers' premises. The fish displayed cloudy eyes, voluminous mucus production and epidermal haemorrhages, including the fins. Many treatments prescribed and licensed for hobby-fish use were trialed with no effect.

Histopathology of surviving fish was inconclusive with few apparent changes except for some suggestion of bacteraemia and very early granulomas.

Bacterial isolations from fish with cloudy eyes produced almost pure culture of *Citrobacter freundi* with multiple antibiotic resistance, notably to erythromycin, tetracycline and sulphamerazine. This supports the current observations from the salmonid and crayfish industries that *C.freundi* is an emerging pathogen of some interest.

SOUTH AUSTRALIA - Ruth Reuter

VETERINARY PATHOLOGY SERVICES (ADELAIDE) - Ruth Reuter

Larval Cyathostomiasis in Horses

Several properties in the Adelaide area reported cases of chronic progressive weight loss in horses during July and August of 1994.

The affected animals ranged in age from yearling to 3 years. The major presenting signs were weight loss, with or without diarrhoea, anorexia and periodic elevation in body temperature.

Consistent findings were hypoproteinaemia, hypoalbuminaemia with severe hyponatraemia and hypochloridaemia. Haemograms revealed low grade anaemia, leucocytosis with relative neutrophilia, high fibrogen levels and, in some cases mild eosinophilia. Faecal floatations were negative and faecal cultures did not yield significant pathogens. There was variable response to treatment with drenches, antibiotics and steroids. However, the missing piece of the puzzle was found when one animal began excreting very large numbers of small strongyle larvae in the faeces.

Small strongyles (cyathostomes) are ordinarily considered of little pathologic significance, particularly in the adult form in the large intestine. The larval stages migrate into the intestinal mucosa and submucosa for development, usually causing a moderate inflammatory reaction. However, in horses which have been heavily infected in the previous year, a massive emergence of larvae during late winter and early spring can cause profuse diarrhoea, ventral oedema and severe weight loss.

When the larvae are in the mucosa, faecal egg counts are usually negative. Once they emerge into the lumen, treatment with febendazole or invermectin can be helpful. Management procedures, including grazing horses with cattle, can help reduce the exposure of the horses to large burdens of larvae.

Reference

Becht, JL (1993) In “Equine Internal Medicine”, Post Grad Comm Vet Sci, Proc 206, pp 149-50.

Gastric Botryomycosis in a Rhodesian Ridgeback

A 15 month old Rhodesian Ridgeback dog was submitted to a local veterinary clinic with a history of weight loss, vomition of bile and occasional melena. The dog was still eating and was bright and alert on clinical examination. A range of biochemistry and a full blood count was done. Significant changes are listed below:

		Reference ranges
Total protein g/L	106	55-81
Globulin g/L	80	23-50
Eosinophils X 10^9	1.0	0.1-0.5

The elevated globulin indicated an antigenic response, with the eosinophilia suggesting hypersensitivity or necrosis. On laparotomy multiple firm white nodules were found in the wall of the stomach and scattered along mesentery and intestinal serosa. The dog was euthanased.

Histopathology revealed marked proliferation of dense connective tissue in the gastric submucosa with granulomas composed of eosinophils, macrophages and lymphocytes scattered throughout the stomach wall. In the centre of the granulomas were "sulfur granules" containing Gram positive cocci. The muscle layers were hypertrophied.

"Botryomycosis" due to persistent low grade infection with *Staphylococcus aureus* has been reported in horses and pigs. It is usually associated with skin wounds on the neck and pectoral region in the horse; and castration wounds and mammary lesions in the pig. Dissemination to the lymph nodes and viscera has been described in some early reports in the literature.

There were no obvious skin lesions in this dog at the time of presentation and no record of ingestion of a foreign body. However, such portals of entry would need to be considered in a possible pathogenesis, particularly in a young active puppy. I have been unable to find any descriptions of this condition in dogs in the literature to date.

VETLAB (ADELAIDE) - John Finnie

Australasian Society of Experimental Pathology (ASEP)

At the recent meeting of the ASEP in Adelaide, I was invited to nominate for Council membership of this organisation. I accepted this position to foster links between the ASVP and AESP and promote the discipline of comparative pathology. It is self evident that animal models are important in understanding diseases in man and the study of animal diseases assists in the elucidation of mechanisms of general pathology.

Several veterinarians contributed to this meeting in Adelaide and the ASEP is keen to broaden the input to these annual gatherings. It is also an excellent forum for postgraduate students to present their research findings in an informal setting and establish contacts with the wider pathology community in this country.

I would therefore invite feedback from veterinary pathologists, encourage ASVP members to consider joining ASEP (at a similar fee to ASVP) and contribute papers in the context of comparative pathology to their meetings. The Society is also proposing to affiliate (at no extra cost to ASEP members) with the American Society for Investigative Pathology which publishes the "American Journal of Pathology".
Paul Gill

NEW SOUTH WALES - Paul Gill

WHAT'S HAPPENING TO THE TARONGA PATHOLOGY REGISTRY?

In the Veterinary Pathology Report (No 39, July 1994) Rod Reece mentioned that the Registry housed at Taronga Zoo was being reviewed for possible restructuring. This has happened with repercussions for several people and institutions.

After the review process, the Zoological Parks Board of NSW approached the Veterinary Pathology Department of The University of Sydney with a proposition relating to the management of the Registry. In a nutshell, the Registry will continue to be housed at, and funded by, Taronga Zoo but will be managed for a three year period by the Department of Veterinary Pathology. It is anticipated that Bill Hartley will continue on as consultant veterinary pathologist to the zoo and the Registry. The Department has designated a person to work with Bill and the Registry which, at this time, happens to be me. Rod Reece is no longer associated with the zoo registry but is currently employed on a part-time basis at EMAI and will continue to service the National Registry for domestic Animal Pathology (NRDAP) for the time being.

So what? What changes? Well, at present Bill and I are reviewing all aspects of the Registry. But what we do know is that the zoo Registry (now officially called the Comparative Pathology Registry for Zoo Animals and Wildlife, but to be commonly known by the short title of Taronga Pathology Registry) is an important facility that needs to be preserved. This means that transparencies (especially of gross specimens), glass slides and blocks of interesting cases still need to be submitted to the Registry. That depends on your interest and goodwill.

Obviously, the terms under which this material is presented to the Registry need to be reassessed for mutual acceptability. Individuals and organisations will be contacted with this in mind. The Registry will continue to serve as a reference collection for teaching and training. This seems to be universally acceptable. The question of publications from the Registry will need to be clarified. This needs to be resolved to the contributor's satisfaction.

The Registry, started by Bill Hartley and ably continued by Rod Reece, will continue to fulfill its role of disseminating information to the veterinary profession. At this stage, the way(s) this is achieved is under discussion. Provision of knowledge via computer discs or through newsletters are just two possibilities.

Bill and I shall keep you informed of all developments; developments to which, hopefully, all of you will be willing to contribute.

(Paul Canfield, Department of Veterinary Pathology, University of Sydney, 2006. Tel (fax) 02: 692 3099.

REGIONAL VETERINARY LABORATORY WAGGA WAGGA - John Glastonbury

Sheep

Balanitis was observed in 70% of 35 Border Leicester rams following an 8-week period of joining. Bacterial and viral cultures were unrewarding and 3 serums gave negative reactions in viral neutralisation tests for herpes viruses - the caprine and bovine varieties. Severe, subacute, segmental, necrotising and ulcerative balanoposthitis was found histologically. Exudation was fibrinohaemorrhagic and the underlying inflammatory response was dominated by plasma cells.

Toxoplasmosis was diagnosed as the cause of late term abortion in 15 of 320 Crossbred ewes. Microscopic lesions included multifocal leukoencephalomalacia, mild multifocal non-suppurative encephalitis and non-suppurative placentitis associated with numerous *Toxoplasma gondii*.

Pasteurella haemolytica Type T caused bacteraemia in weaner sheep.

Associated histopathological lesions included acute multifocal necrotic hepatitis, sub-acute multifocal suppurative embolic pneumonia and acute segmental fibrinous pleuritis.

Cattle

Malignant catarrhal fever was diagnosed in 2 of 25 Angus cattle. Depression, pyrexia, dyspnoea, bilateral nasal discharge and opacity of the sclerocorneal junction were observed clinically. The pathognomonic non-suppurative and fibrinoid vasculitis was found histologically in portal triads, brain, leptomeninges, oral mucosa, oesophagus and lymph nodes. In the more acute of the two cases, moderate, acute, multifocal necrotising lymphadenitis was an additional observation.

Native Animals

Coccidiosis caused disease in a 14-month-old male wombat and a 16-month-old female Eastern Grey kangaroo. The former had numerous very large oocysts, schizonts and macrogametocytes within the lacteals of the ileum. *Eimeria (Globidium) tasmania* is the most likely aetiological candidate. *Eimeria kogoni* was the possible cause in the latter. The kangaroo also had evidence of a disseminated **T. gondii** infection in the form of moderate to severe, chronic, multifocal, non-suppurative myositis and interstitial myocarditis. Lesser non-suppurative inflammation was also noted in the intestinal tunica muscularis, portal triads, lungs, kidneys and thyroid gland.

Cats

Dermatophytosis was associated with interesting pathological changes in two cats.

Material for histological examination, only, was submitted from the first case, which was a 4.5 year-old speyed Domestic Long Hair. It had severe chronic granulomatous peritonitis associated with prominent mycetomas. A mycetoma was found in the hypodermis of one skin section , along with mild hyperplastic dermatitis and numerous fungal hyphae and spores in the superficial epidermis. The Department of Microbiology at the Royal North Shore Hospital placed *Pseudallescheria boydii* "at the top of the list of suspects" for the peritoneal fungi. *Microsporum canis* was recovered from lesions of severe chronic granulomatous lymphadenitis and chronic granulomatous nodular dermatitis in a 4.5 year-old male Persian.

REGIONAL VETERINARY LABORATORY WOLLONGBAR - Roger Cook

Ectopia cordis in a calf - Roger Cook

A neonate Angus-cross calf which had died 1½ hours after birth was presented to the laboratory. The heart was globose and displaced ventrally outside the body through an 8cm long by 2cm wide defect in the sternum. Developmental abnormalities in the heart included:

* an atrial septal defect (3cm diameter foramen ovale)

* dilation of the right ventricle with thickening of interventricular septum and right ventricular wall. The dorsal and ventral surfaces of the cusps of the tricuspid valve were thickened, red and granular with tangles of red thrombi extending over the cusp surfaces and around the corda tendinae.

* There were three circular interventricular septal defects 5 to 10mm in diameter; one in the craniodorsal septum under the anterior of the a/v valve adjacent and caudal to the pulmonary outflow; the other two were near the ventricular apex.

* the aorta was completely transposed to the right of and anterior to the conus arteriosus. The aortic trunk exited in front of the pulmonary artery and extended as a 14cm long vessel to the dorsal thorax where it branched to form the brachiocephalic trunk and descending aorta.

* Left ventricular hypoplasia (50mm deep, 40mm wide). It had a narrow a/v opening (15mm in diameter) and drained to the right ventricle through the three interventricular septal defects.

It is interesting that the calf survived for 1½ hours after birth, considering the severity of the abnormality.

Rhabdomyosarcoma in a koala

An adult female koala had haemathorax with collapse of the right lung and left displacement of the thoracic organs. The source of the haemorrhage was a solid pale grey multinodular tumour - 2cm x 3cm in right dorsal diaphragm, adjacent to the costal wall. It infiltrated lumbar muscles. The tumour was composed of whorls and fascicles of closely packed plump, round to fusiform cells with even eosinophilic cytoplasm and a single nucleus. Multinucleated giant cells were evident in several areas through the tumour. Cross striations were not evident in these cells when stained by PTAH: whether the freeze/thaw affected this result is unknown. However, the site of the tumour and its pleomorphism is consistent with rhabdomyosarcoma. Rhabdomyosarcomas are very unusual tumours in koalas; we are unaware of any previous records of rhabdomyosarcoma in koalas.

DEPARTMENT OF VETERINARY PATHOLOGY UNIVERSITY OF SYDNEY

Fatal anaphylaxis in a kitten - Malcolm France

A 4-month-old kitten was found dead in the early morning after being apparently normal the night before. At necropsy the lungs were diffusely dark red and on histological examination there was marked congestion, thick proteinaceous fluid in many alveoli and larger airways, dilation of pulmonary lymphatic vessels, and a moderate increase in alveolar macrophages; no haemosiderin was seen. Dilation of lymphatic vessels was also seen histologically around the mesenteric lymph node. Sections of liver, intestine, kidney, spleen and brain were normal.

The thickness of the alveolar fluid seen here suggested a substantial increase in capillary permeability. This is in contrast to the more familiar low protein pulmonary oedema fluid commonly seen with congestive heart failure. On further discussion with the owner, it was learned that there was a bee hive on the neighbour's property. It was felt, therefore, that the kitten had suffered a type I hypersensitivity reaction to a bee sting, presumably following sensitisation from previous stings, and that death was due to the ensuing exudative response possibly accompanied by bronchospasm. While the main organ manifesting anaphylaxis in the cat is said to be the lungs, the gastrointestinal tract can also be involved; this can cause vomiting, although this does not appear to have occurred here because the stomach was still full.

Castration-responsive dermatosis

A 6-year-old male Keeshond had been developing symmetrical alopecia and hyperpigmentation over the back and ventrum for 18 months; four skin biopsies were submitted. The most striking changes were hyperpigmentation of the basal layer of the epidermis, diffuse pigmentation of all other epidermal layers, follicular hyperkeratosis, and the presence of numerous 'flame follicles' (follicles in which bright orange spikes of keratin radiate from the centre through the outer root sheath). Other changes included slight acanthosis, moderate hyperkeratosis, and dilation of the apocrine sweat glands; all hair follicles were in telogen.

Individually, these changes can be seen in a range of endocrinopathies and other 'atrophic' skin diseases. However, the breed, history, prominence of flame follicles and finely granular (as opposed to clumped) melanin led to a tentative diagnosis of castration-responsive dermatosis. This is a condition to which the Spitz-type breeds are predisposed; it is also referred to as adult-onset growth hormone-responsive dermatosis. This dog was castrated and within a few weeks, extensive regrowth of hair had occurred, much to the owner's delight.

Cerebellar abiotrophy in laboratory mice

Occasional mice in a colony of C57BL/6 mice homozygous for the severe combined immunodeficiency (*scid*) gene were seen to display a continuous rapid fine motor tremor (possible intention tremor), ataxia and a tendency to fall to one side. They were in good body condition and had been shown to be fertile. On necropsy the cerebellum was found to be about 2/3 normal diameter; histological examination revealed thinning of the cerebellar molecular layer, at least a 50% reduction in the number of Purkinje cells, and occasional shrunken, markedly basophilic Purkinje cells.

Several mutations are described in mice in which cerebellar abnormalities are a major finding; these are given descriptive names such as lurcher, reeler, teetering, jolting and vibrator. It is uncertain at this stage whether the condition in these mice is a previously undescribed mutation.

REGIONAL VETERINARY LABORATORY ARMIDALE

Multifocal hepatic necrosis - Poultry - Steven Hum

There was a sudden increase in mortalities and decrease in production in a 26 week old layer flock near Tamworth. Birds appeared febrile and often died quickly and quietly. There was a good response to antibiotics but the problem recurred when treatment stopped.

The typical gross lesions were multiple whitish foci of varying size, 1 to 5mm in diameter throughout the liver. Some birds had petechial haemorrhages in some visceral organs.

On histopathology the significant lesion was found in the liver and consisted of acute multifocal hepatic necrosis infiltrated by low to moderate number of heterophils and macrophages. Some of these foci were progressing to form granulomas. In other places in the liver there were small foci of coagulative necrosis of hepatocytes with no or very little inflammatory cell response.

Routine bacteriologic examination of the livers was negative.

The findings suggested a bacterial aetiology and more livers were cultured using selective media for Salmonella, and selective media and microaerophilic atmosphere for Campylobacter without success.

Silver and gram stains of liver sections were also negative.

The aetiology is not known although avian spirochetosis is suspected because the shed was infested with Argas persicus.

Visceral larva migrans - Pig - Steven Hum

Formalin fixed tissues were submitted from a 13 week old piglet which was euthanased following ill thrift and inco-ordination affecting all four limbs. At post mortem there were large numbers of *Ascaris* worms in the small intestine. Histologic examination of the spinal cord revealed extensive multifocal haemorrhages in the ventral horn of the grey matter, surrounded by macrophages and a few eosinophils. Many large neurons were necrotic. In the white matter there was axonal swelling and Wallerian degeneration especially

in areas close to the grey matter. In the liver there were multifocal granulomas with eosinophils. Lung was not submitted.

A diagnosis of possible "visceral larva migrans" was made.

Internal parasitism caused by *Ascaris suis* is rarely seen today. The ingested eggs hatch in the intestine, larvae reach the liver through the hepatoportal blood stream, and are carried by the blood to the lungs. Most of the larvae are arrested in the capillaries in the lungs but some may pass through into the arterial circulation and reach other organs where extensive migration may occur.

The piggery is semi-intensive, sows farrow in the paddock and weaners are brought into the shed at 6 weeks. Deaths have occurred sporadically since weaning; clinical signs included lethargy and progressive weight loss over a number of weeks. The weaners were drenched several times with Nilverm. Re-infestation, faulty drenching or drench resistance is being investigated.

Drought, Metabolic Diseases and Plant Poisonings - Stephen Love

According to the August 1994 Weather Review for Armidale (compiled by Peter Burr, UNE), the winter total of 57mm of rain was the lowest since the last major drought in 1982 when only 27mm was recorded for the three months. The clear skies and lighter than usual winds resulted in warmer than normal day temperatures and very cold overnight minimums, with minus 10.2°C being recorded one day in East Armidale.

Predictably the weather conditions pedisposed pregnant and post-partum ewes and cows to metabolic diseases such as pregnancy toxaemia (with losses up to 10% in some flocks) and hypocalcaemia and this was reflected in submissions to the lab.

There have been reports of small losses due to enterotoxaemia in sheep on grain diets, especially in lot feeding situations. Grain poisoning has also been encountered. In one case, sheep on one brand of pellet suffered severe acidosis when a new batch was used. Sixteen died and up to 30% were sick out of a mob of 1200 ewes.

The drought has also resulted in more submissions and enquiries relating to plant poisoning, particularly in the north west slopes and plains of NSW. Shaun Slattery, District Veterinarian at Narrabri, has seen a number of cases of "Mother of Millions" (Bryophylum sp) in cattle and also suspected Quinine Bush poisoning. There have been reports of rock fern poisoning in cattle also. Bill Hetherington (DV Moree) has had his share of suspected plant poisonings as well. One case (AN94.3023.SL) involved 12 deaths in a mob of 170 cattle at Gravesend on drought affected "pasture". Tree Tobacco (Nicotiana glauca) was the main suspect and the cattle had been eating this as well as a variety of other plants, including Narrow Leaf Cotton Bush (*Gomphocarpus fruticosus*). In South Africa, ostriches are reportedly the animal most commonly poisoned by *N glauca*.

Of interest also have been cases of suspected "Agave" poisoning in cattle.

Suspected *Agave* Poisoning in Cattle - Stephen Love, Bob Cloverdale

We have had two cases of suspected *Agave americana* poisoning recently.

Peter Gough, private practitioner at Moree, rang the lab in August to discuss suspected *Agave* toxicity in cattle on a property at Bellata. Four out of 13 Hereford cows in a particular paddock were first affected in July. There was little feed and the cattle had clearly spent some time eating an *Agave* plant in the paddock. Peter describes the hind limbs as being affected, the appearance being not unlike that of obturator paralysis in 3 post-partum cow. The feet are flat on the ground but the pastern and the fetlock are further forward

than normal. Of the 4 affected cows, two have completely recovered, one still walks with an abnormal hind limb gait and the fourth is still down. This fourth cow is quite bright in demeanour, has no pressure sores, and can half raise herself from the ground using her forelegs.

The second case occurred early in September and was investigated by Barraba veterinarian Lee Morris and handled by Bob Coverdale at the lab (AN94.3452.ORC). Five to ten out of 23 Hereford cows on a Barraba property were affected. There was no green feed available and some obviously had been eating *Agave*. Lee describes the affected animals as being bright and walking on the hind toes with contracted fetlocks. Urine was submitted from one animal and blood from three. Urinalysis and the haemograms were unremarkable. Ca, Mg, P, creatinine, AST, CPK, GLDH, and AP estimates were done on the 3 serums. CPK's were 1322, 271 and 916 (reference range: <200 U/L). Other analytes were unremarkable apart from marginally elevated AST and GLDH in the animal with a CK of 916 U/L.

Everist tells us that the Avavaceae family has more than 650 species, mostly tropical or subtropical in origin. One species of *Agave* has been reported as poisonous to livestock in North America (presumably *A. lechequilla,* referred to by the Merck Veterinary Manual (MVM)) but this plant is not known to occur in Australia. Several species are common garden plants in this country and two (*Agave Americana,* Century Plant, and *A. sisalina,* Sisal) are semi-naturalised, the first in tropical and subtropical inland areas, and the second in coastal central Queensland (Everist 1981).

According to Everist there is no positive evidence of toxicity for either *A. americana* or *A. sisalina* apart from reports of persistently painful skin wounds in people caused by the leaf tips. Blood and Studdert (1988) state that *A. americana* contains a toxic saponin and can cause poisoning. Regarding *A. lechequilla* , they say it contains an exogenous photodynamic agent and causes dermatitis but without signs of liver damage. The MVM records *A. lechequilla* as containing an unidentified hepatotoxin (causing photosensitivity) and a toxic saponin with an abortifacient action. (Camp et al (1988) state that the toxin has been identified as a steroidal saponin, similar to those identified in *Panicum* spp, and responsible for hepatogenous photosensitisation). Sheep, cattle and goats are affected, usually during drought. The course is subacute with signs such as listlessness, inappetance, icterus, yellow oculonasal discharge, photosensitisation, coma and death occurring.

Ross McKenzie (Principal Veterinary Pathologist, Animal Health Laboratories, DPI, Yeerongpilly, Qld. advises us that he has one case of suspected *A. americana* on file. This involved 7 out of 100 cattle "on the road" at Roma during a drought in December 1991. Stiffness, abnormal gait and tucking of the hindlegs under the body were observed. All affected animals recovered. Clinical pathology revealed markedly elevated CK and AST. Ross adds that Steyne (1950) produced paralysis of the hindquarters in a rabbit with 100ml of *A. americana* juice twice daily for 3 days. After no improvement on day 6, the rabbit was killed and necropsied, the findings being gastric hyperaemia and a pale liver, Ross McKenzie further informs us that Watt and Breyer-Brandwijk (1962) make reference to Steyne producing bloat, cyanosis, dyspnoea and tachycardia in sheep using *A. americana*. Leaf extracts are apparently used in folk medicine in Africa as purgatives and diuretics.

It appears likely then that, when consumed in sufficient quantities, *Agave americana* can cause poisoning in cattle and other animals, this toxicity presumably manifesting itself as a myopathy and mainly affecting the hindlimbs. Most cases seem to recover.

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Calves with Spongy Brains - Stephen Love

Shaun Slatterv, District Veterinarian at Narrabri, investigated an interesting nervous syndrome in week old "dairy" calves at Pilliga. The cattle are a mixture of Jersey and Guernsey with a fair degree of "line" breeding. Over the last 3 years the owner has had a dozen out of 40 or so calves die from a nervous syndrome. Cases occur in winter-spring. Calves are usually 7-10 days when first showing nervous signs and die usually within 2-4 days. Signs include proprioceptive defects with forelimb hypermetria ("goose-stepping") and constant head nodding. Withdrawal (front and back legs) and menace reflexes were absent; a patella reflex was present.

Haematology and biochemistry (AM94.2463.BV 24.6.94) was unremarkable apart from indications of dehydration. (Although able to suck, the ataxia and head nodding made this well nigh impossible). The blood was negative for pestivirus antigen and antibody.

The calf deteriorated and was necropsied. Bochemistry and haematology results (AN94.2505.SL 28.6.94) were similar to those of a few days earlier. Analysis of the blood indicated the animal was homozygous normal for maple syrup urine disease. Histologically, severe spongiform changes were evident in the brain and spinal cord (AN94.2505.SL. We sent histoslides to Dr. Peter Harper, neuropathologist of note and currently Senior Field Veterinary Officer for NSW Agriculture at Grafton. Peter commented that the oedematous CNS lesions were among the most widespread he has observed, with severe spongy changes in both the myelinated areas and in the cerebrocortical grey matter.

Investigations are continuing.

Cholera in turkeys - Stephen Love

A turkey farm in the Tamworth district suffered heavy losses from cholera in August. At the time birds were submitted to the lab, approximately 10 a day were getting sick and dying in a group of 500 near marketable birds (22-23 weeks of age). The birds receive a cholera vaccine at 12 and 16 weeks. However, this vaccine has not been changed for 10 years and the strain of P. multocida isolated at the lab from cases of cholera in May was not represented in the vaccine. Deaths start about 2 weeks after antimicrobials are withdrawn from the feed.

Necropsy and histological changes were in keeping with cholera, isolated (heavy pure culture) from various sites. (AN94.3313.SL).

Toxicity due to "Colleague" (SmithKline) drench - Barbara Vanselow

In December last year, after the commercial release of the drench "Colleague", deaths were reported in sheep shortly after drenching. Carcasses, tissues and blood samples were sent to the laboratory from 4 separate properties at Armidale, Glen Innes and Uralla. These samples were from lambs but deaths were also recorded in adult sheep.

Property 3, 55 weaners died out of 3026 drenched. 7 ewes died out of 3555 drenched.
Property 4, A total of 39 died out of 1697 drenched with deaths occurring in each mob of 3 mobs drenched. These mobs were :- 1010 ewes and wethers, 317 hoggets, 370 lambs.

Five lambs were post-mortemed at the lab. Their weights ranged from 9.5-16 kg. From the history there was no evidence of overdosing, they were dosed at the recommended level of 1ml per 5kg body weight.

The earliest recorded death occurred 4-6 hours after drenching while most deaths occurred within 1-2 days of drenching. Clinical signs observed included excess salivation, muscle fasciculation, abdominal pain, bloat, laboured breathing, and diarrhoea. Post-mortem findings included vascular congestion of the lungs with froth in the trachea, and vascular congestion of the small intestine. These changes were not found

consistently in all carcasses. Histopathological changes were non-specific and included vascular congestion and oedema in the lungs and vascular congestion in the liver. Two lambs from one property had evidence of acute myocardial and hepatic necrosis.

Haematological changes included elevated haematocrit and plasma protein consistent with dehydration and elevated muscle enzymes including CPK, LDH AND SGOT, which may have been related to the muscle fasciculation observed. GSHPX levels were measured from single blood samples from 2 separate properties and were 42 and 113 U/gHb respectively. Brain and blood samples were sent to Macquarie Vetnostic Services, Leichardt 2040 for measurement of cholinesterase levels. These were :-

BRAIN Animal 1, 0.01 IU/kg
" Animal 2, 0.008 IU/kg
" Animal 3, 0.03 IU/kg

NORMAL BRAIN Approx.0.22 IU/kg

WHOLE BLOOD Animal 3, 0.34 IU/I

NORMAL BLOOD Approx. 0.4 IU/I

These low cholinesterase levels are consistent with organophosphate toxicity. The cholinesterase levels in brain tissue are the most reliable measure of organophosphate toxicity. The organophosphate component, Pyraclofos, was known before clearance to be potentially toxic under some circumstances, particularly with liver damage.

The product was commercially released in December 1993 and by 6th January 1994 SmithKline Beecham had identified 409 deaths Australia-wide from 96,000 doses used on 37 farms. The product has been withdrawn from the market pending further investigations.

Lamb deaths caused by penetration of oesophageal wall by slow release capsules - Barbara Vanselow

One week after the administration of "Proftril" slow release anthelmintic capsules to 2700 lambs, 20-30 lambs had died and another 30 were ill. Three live lambs were submitted for clinical and post-mortem examination. Clinically they showed slobbering, nasal discharge depression and stiff gait. In all 3 lambs the capsule had ruptured the oesophagus at the back of the pharynx with the capsule completely penetrating and passing through the wall of the oesophagus into the neck tissue behind the oesophagus. This was associated with a severe cellulitis and in all 3 cases food material had passed through the rupture in the oesophageal wall into the surrounding tissue. The oesophageal walls in these 3 lambs appeared to be quite fragile and could be easily ruptured with a finger. These lambs' weights ranged from 15-18 kg just below the recommended cut-off of 20kg.

Thyroid insufficiency in a stillborn Anglo Nubian kid - B Vanselow.

Three stillborn kids from one property were sent to the laboratory for post-mortem examination. Their weights were 3.8, 4.5 and 4.25kg respectively and the postmortem findings suggested the deaths were attributed to difficulties at birth with the first 2 showing marked oedema of the head most likely resulting from dystocia and the 3rd kid being a twin may have died from asphyxiation. The first kid examined had partial alopecia along the back and the back of the neck. The thyroid glands could not be found at post-mortem examination and serum collected from heart blood indicated hypothyroidism. From the 2 subsequent kids, both with normal hair growth, the thyroids were grossly and histologically normal. Serum samples from the affected kid's dam, a second stillborn kid (without alopecia} and it's dam indicated normal thyroid activity.

Animal Identification	Alopecia	Free T4 (pica Moles/I)	TSH (uU/ml)
94/433 doe	No	30.3	0.06
" kid	Yes	<1.3	14.6
94/589 doe	No	22.3	0.24
" kid	No	12.8	<0.02
Human adult	-	9.8-23.1	0.35-5.5

TSH (Thyroid stimulating hormone) is a pituitary hormone which increases in a situation of thyroid insufficiency. Elevation of this hormone level is the best indication of insufficiency and was markedly elevated in the affected kid. Free T4 (Thyroxine) also indicates thyroid function and in the case of the affected kid T4 was not detectable. TSH and T4 tests were done by New England Pathology.

Suspect Pithomyces chartarum toxicity in 2 Murray Grey calves - Barbara Vanselow

Two moribund Murray Grey calves were submitted alive for clinical and post-mortem examination after another calf had been found dead, but was too decomposed for a post-mortem examination. One calf was submitted in late March and the second in mid-April. The first calf was approximately 4 months old and the second 2 to 3 months old and although they had been running with their mothers the stomach contents were all plant material; grass, and small amounts of mistletoe, bracken and wattle seeds, indicating that these calves were no longer sucking from their mothers. Both calves appeared to be small for their age.

Clinically they were recumbent, and severely jaundiced. An enlarged liver was easily palpated through the abdominal wall.

The post-mortem findings were virtually the same for both calves:-
- severe jaundice
- enlarged brown liver
- gall bladder virtually empty
- no evidence of photosensitization, both Murray Greys with well pigmented skin.

The first calf was also anaemic (PCV 10%) and adult Haemonchus were visible in the abomasum (EPG 3040). The second calf had been drenched (PCV 28%, but with many nucleated red blood cells in the smear suggesting a previous anaemia) and EPG of 0. This second calf also had very pale intestinal contents.

Both calves had neutrophilia:-
94/1165TWCC - 14.4 x 10/I
Segmented neutrophils - 11.66 x 10/I

94/1487TWCC -31.2 x 10/l
Segmented neutrophils - 26.8 x 10/I

Urinalysis was similar for both calves, positive for bilirubin but negative for urobilinogen indicating that there was an obstruction to the flow of bile into the small intestine.

Both calves had elevated serum total bilirubin and AST and the second calf also had elevated GGT.

	Calf 1	Calf 2	Normal Values
Total bilirubin	187.2 umol/l	229.7 umol/l	<10 umol/l
AST	293 U/I	243 U/I	<150 U/I
GGT	32 U/I	267 U/I	<70 U/I

Histopathological examination of livers from both calves revealed massive damage to bile ducts, and canaliculi with ballooned and disrupted epithelial cells with bile accumulated in these structures and within hepatocytes. There were focal areas of hepatocyte necrosis with an associated neutrophil infiltrate.

This lesion is the same as that caused by the mycotoxin, sporidesmin present in the saprophytic fungus, Pithomyces chartarum. Sporidesmin is excreted in the bile causing inflammation of the bile ducts and progressive obliterative cholangiolitis resulting in a slowing down of the rate of bile flow to negligible levels over a period of about 14 days. In the second calf it was obvious from the paleness of the intestinal contents that bile was not getting through. The urinalysis results confirmed this.

Sporidesmin toxicity has been reported only very rarely in New England and an investigation of the paddock failed to reveal the source. One Pithomyces spore was identified on a sample of dead grass but their was very little dead grass when the paddock was examined in late April. Perhaps there had been large numbers of spores in March when the toxic insult occurred. The climatic conditions at that time were suitable for the fungus to proliferate. In New Zealand the disease is common and occurs when pasture is short and contains recently killed plant material in abundance, and under climatic conditions of warm, humid weather, which favours a heavy infestation with the fungus. This is most likely to be a problem in autumn when the summer has been hot and dry, the pasture well eaten back and good rains fall when the ground is still warm.

China Australia sheep research project - Barbara Vanselow

An investigation of lamb mortality.

As part of a one month assignment to the CASRP in Urumqi, Xinjiang Province in north-west China in May/June this year, severe lamb mortality was investigated at the Nanshan stud farm in the foothills of the Tienshin mountains.

Nanshan stud farm belongs to the Xinjiang Academy of Animal Science. The sheep, predominantly Merino, are herded by the local partly-nomadic Kasak people. The 5000 breeding ewes are divided into 17 flocks cared for by separate families.

Average lamb mortality (over 9 years) was 12% but for 1994 mortality was approximately 30%. Lambing in February and March in sub-zero temperatures, takes place indoors in crowded, contaminated and often muddy pens. Mismothering and poor milk supplies are common, as is the level of human intervention in feeding the lambs. At approximately 2 days of age, lambs are separated from their mothers except for feeding twice per day.

In this environment, it was not surprising to find infectious disease. Clinical and post-mortem signs included ulcers in the mouth, scabs on the muzzle, proliferative lesions on the gums, pneumonia, pleurisy, pericarditis, diarrhoea, foci of suppuration and necrosis in the liver. Parapox virus was identified in scabs by electron microscopy and Fusobacterium necrophorum was isolated from ulcerative lesions of the mouth. The youngest lamb affected was 7 days old and the oldest over 1 month old.

The sequence of events leading to this unusually high mortality was believed to be as follows :-

i An outbreak of scabby-mouth, spread rapidly because of crowding and contamination of the environment.

ii Secondary infection, particularly with *Fusobacterium necrophorum*. This organism is a normal inhabitant of the gut and is usually present in faeces. It can cause necrotic stomatitis visible as hard circumscribed swellings covered with necrotic material. The infection may extend systemically to the lungs, stomach, liver, kidneys or spleen.

Drunken horse grass

The plant Drunken Horse Grass (*Achnatherum inebrians*) occurs in the grazing pastures of northern and western China, particularly on degraded soil. It has been observed to be toxic to sheep, goats, cattle and horses and particularly to animals recently introduced into the area. Clinical signs include incoordination, gangrene of the extremities during cold weather, and death.

Grass and seeds were sent to the New Zealand Agricultural Research Institute by the CASRP. It was determined that these specimens were endophyte infected and contained high levels of ergot alkaloids. Thus the toxicity is most like fescue poisoning. Control measures are being investigated.

The quick detection of the toxic principle exemplifies what can be achieved by cooperation between international research groups.

RVL MENANGLE

Blindness in kangaroos - WJ Hartley (Taronga Zoo), G Curran (NSW Agriculture, Cobar), GL Reddacliff (RVL Menangle) & K Filmer (NPWS)

Cases of blindness in kangaroos occurred in western NSW and North-western Victoria, mainly in flood plain and sandhill country along the Murray-Darling river system. The first cases were reported from about mid-May in NSW and about a month later in Victoria. At the height of the outbreak in July up to 10% of Western grey kangaroos were thought to be affected, with a lower prevalence (about 1%) in Red kangaroos. By mid-August few new cases were occurring. In most areas there was ample pasture and no unusual or predominate weed species were present. Despite the time of year mosquito activity was noted.

Clinically the animals were simply blind, and stumbled into bushes and other objects, especially when disturbed. Otherwise they were apparently normal, could hear, move and feed freely, and generally maintained body condition. There were no external signs of eye disease.

Fixed material from animals collected early to mid-outbreak was received through the Wildlife Pathology Service at Sydney University. Subsequently 10 fresh heads and one sedated blind roo were received at RVL Menangle at the end of July. Cultures were established for viral examination, and sera stored for future arboviral analysis. Grossly the retinas of several animals were pale, sometimes apparently detached, and white foci 1-2mm across were seen in two. Otherwise there were no gross lesions, except for bilateral lens opacities in one animal.

Eyes and brain from 19 animals (including 4 Red kangaroos) were examined histologically. Eyes from acute cases had a fairly diffuse, moderate to severe, non-suppurative inflammation of the choroid, often in association with sloughing of the retina. The retina sometimes appeared normal, sometimes had a mild to moderate mononuclear cell infiltrate, and sometimes had focal necrosis. There was often an attached mixed cell inflammatory exudate. Less acute cases were similar, but with less severe chordoiditis, and focal necrosis, with mineralisation, of the rod and cone basal segments of the retina , and often diffuse atrophy elsewhere. Chronic cases (those animals collected later in the outbreak, and in poorer condition) usually

had mild to moderate multifocal non-suppurative choroiditis, and atrophy of retina. Where retinal architecture was reasonably preserved there was an apparent reduction in the number of ganglion cells, and the surface layer of ganglion cell axons was markedly atrophied, vacuolated or entirely absent. Lesions in the optic nerves varied from moderate recent to severe old-standing Wallerian degeneration. Often there was virtually no normal staining myelin left. Some nerves had some associated mild non-suppurative multifocal inflammation. These lesions continued through the optic chiasm into the optic tracts, but white matter elsewhere in the brain was unaffected. Several animals had a mild to moderate multifocal non-suppurative encephalitis, characterised by focal gliosis and some perivascular cuffing. There were no significant lesions in other tissues.

So far nothing has grown on the viral cultures, although the microscopic findings and epidemiology are suggestive of an infectious cause. Uveitis, sometimes leading to complete blindness with optic nerve involvement and retinal detachment, possibly associated with *Borrelia burgdorferi* has been found in captive kangaroos at the St Louis zoo in the USA. Silver-stained sections from eyes from our cases were negative for spirochaetes. The optic nerve lesions are similar to those reported for *Stypandra* poisoning in livestock, but the associated choroidal and retinal changes seem unique to this outbreak.

Other causes of apparent blindness in kangaroos that were uncovered during field investigations into this problem included “lumpy jaw” in euros at Fowler’s gap, possible polioencephalomalacia associated with clover or medic pastures at Coonabarabran, and blindness associated with ocular discharge, corneal opacities and emaciation from drought affected areas around Brewarrina and Bourke (possibly nutritional deficiencies).

scjl:newsltr.994

REQUEST FOR HAIR SAMPLES FROM CALVES WITH MSUD

Dr Don Rivett of CSIRO, Division of Biomolecular Engineering, 343 Royal Parade, Parkville, Victoria 3052, would like to obtain hair samples from calves affected with Maple Syrup Urine Disease.

Dr Rivett can be contacted on phone 03 342 4200.

Newborn Poll Hereford calves that develop severe neurological disease within 2 days of birth are possibilities. Calves which are apparently normal for the first day of life then become depressed, show disorganized limb movements, and finally develop opisthotonus by day 2-4 would be good starters. Perhaps hair samples could be clipped from the body and held frozen until MSUD is confirmed by amino acid analyses on serum, plasma or eye fluids or by PCR analyses on blood, tissue or hair roots or by histology.

(Tony Ross)

WESTERN AUSTRALIA - David Forshaw

WESTERN AUSTRALIAN DEPARTMENT OF AGRICULTURE

ANIMAL HEALTH LABORATORIES SOUTH PERTH

Equine pyrrolizidine alkaloidosis. copper toxicity and haemoglobinuria

A three year old stock horse trucked from Queensland to Kununurra and yarded in the local dip yards was seen to have a sudden onset of depression, inappetence and port wine urine. The horse died shortly after.

Histologically the liver was extensively fibrosed with megalocytosis and regenerative nodules. Kidney and liver copper levels were 4 and 3 times normal limits respectively.

Pyrrolizidine alkaloids associated with copper toxicity and intravascular haemolysis is not commonly recorded in horses. The horse was provided access to lick blocks containing copper; this presumably had some role in the pathogenesis of this case. (John Creeper)

Lymphosarcoma in a dolphin

An adult female bottlenosed dolphin was presented for post mortem examination after being found washed up on a beach south of Perth. Necropsy revealed large multilobular masses on the diaphragmatic surface of the liver, caudal lung and involving the mesenteric lymph nodes. There was a severe peritonitis with numerous abdominal abscesses and extensive adhesions involving all organs in the lower abdomen. The uterus was distended with approximately 1.5 litres of fluid. Histopathology revealed a well differentiated lymphosarcoma involving the mesenteric lymph nodes with metastases to the liver and large intestine. The cause of the peritonitis could not be identified. (Martin Robertson)

ALBANY REGIONAL VETERINARY LABORATORY

Trichostrongylus axei in sheep

Trichostrongylus axei infection in sheep is extremely rare in our experience, and then only in sheep in close association with cattle. Within the space of two weeks we had three submissions with T.axei in the abomasum of sheep. The first case was from a departmental trial and only a few worms were seen. The next two were case submissions from private practitioners:

Case 1 -
Hoggets scouring since early winter. Drenched and improved but scouring again in early October. No summer drench had been given.
Total Worm Counts
A. total 4150 including 1020 T.axei, 2340 L4 and 740 immatures.
B. total 59760 including 10,515 T.axei, 43110 L4's and 6135 immatures.

Case 2 -
Two year old wethers scouring.
Total Worm counts
A. total 110 including 660 T.axei, 400 L4's and 50 immatures.
B. total 12280 including 10880 T.axei, 900 ostertagia, 200 L4's and 300 immatures.

In these cases, neither property had any cattle on the place for over ten years. In case 1, the sheep came from a closed flock where only rams were bought. In case two, sheep were bought and sold regularly. Both properties are in wheatbelt areas which had dry summers and less than average rainfall over winter and spring.

Osteoporosis associated with low liver copper and high iron levels in lambs

Lameness was noticed in a flock of lambs at the Mount Barker Research Station 50km north of Albany. Five lambs were necropsied; one had a fractured femur, all had very fragile bones and two had healing rib fractures. Apart from this, they all had score 2 footrot lesions and one had focal areas of myocardial necrosis. All had very low plasma glutathione peroxidase levels and liver selenium levels. All had low liver copper levels (3,5,6,7,and 7mg/kg) and high liver iron levels (2421-4120mg/kg). Rib ash content varied from 45.2-49.2%, rib Phosphorous 8.2-8.8% and rib Calcium 17-18.5%. Pasture copper levels were within our normal ranges (5.8-6.9mg/kg) and molybdenum (1.5-1.6mg/kg) and sulphur (0.34-0.32%db) levels were also judged to be OK. Pasture iron levels appeared high at 2900-3000mg/kg, in fact so high we are wondering if the samples were contaminated somehow.

Possible causes of osteoporosis include poor nutrition and milk intake but the ewes in this case were reported to be in good condition and this must be one of the few parts of the country enjoying a good season. Heavy intestinal parasitism is also reported to cause osteoporosis and these lambs had reasonably high parasite burdens but not more than other flocks in the district with none of these bony problems.

Could the low copper levels in these lambs have been induced by high iron intake? Experimentally high iron intake in cattle can induce low liver copper levels but does not result in lesions of copper deficiency . Could this be a case of copper deficiency induced osteoporosis? It seems unlikely as no other signs of copper deficiency were seen but otherwise, we haven't got a diagnosis.

Reference

Caple, I (1990) Sheep medicine; Sydney postgrad proceedings 141. pg 369-370
Phillipo,M. (1987) The effect of dietary molybdenum and iron on copper status and growth in cattle. J.Agric Sci, Camb. 315-320
Bremner I. et al (1987) Iron induced copper deficiency in calves: Dose - Response relationships and interactions with molybdenum and sulphur. Animal Production 45: 403-414

Septicaemic pasteurellosis in pet rabbits

Two large white rabbits were necropsied. They were the 3rd and 4th rabbits to die in five days from a group of six. . There were neither recent introductions nor any history of respiratory disease. The owner had found the first two dead and the third showing signs of "stiffness with its eyes rolling back in its head". The fourth rabbit was also found dead. At necropsy both were extremely fat. The only lesions were multiple pinpoint white foci distributed throughout the liver. Histologically, these were focal areas of necrosis surrounded by polymorph infiltrates. Bacterial colonies could be seen in the sinusoids. We grew a pure growth of Pasteurella multocida from liver, lung and kidney of one rabbit and the liver of the other. In a 1993 report on diseases in commercial rabbits in Western Australia, Trott cites Pasteurella multocida as a cause of upper and lower respiratory tract infections, body abscesses, middle ear infections and mastitis/metritis.

Reference

Trott, D. in Commercial Rabbit Production. Still some rabbits in the hat? Misc publication 23/93 WA Dept of Agriculture 1993.

(David Forshaw)

NORTHERN TERRITORY - Anton Janmaat

Avulsion of Femoral and Humeral Tuberosities in Chickens - David Pritchard

Four caged layer hens were submitted to the laboratory for post mortem examination. There had been a recent drop in production by about 8% and a 20% increase in the prevalence of thin shells. A similar type of problem earlier in the year apparently had responded favourably to vitamin/mineral supplementation (no details given). Some hens were described as having "the wing joints appear necrotic".

On examination in the laboratory the hens were in excellent condition and had extensive purplish and green discolouration of tissues around the scapulo-humeral and femoro-tibial joints, a consequence of periarticular haemorrhage and subsequent degradation of extravasated blood. At necropsy the haemorrhage was found to be due to avulsion of the proximal tuberosities of the humeri and lateral condyles of the femurs, points for insertion of the great flight muscles and massive thigh muscles, respectively. In one case the entire distal end of the femur had separated from the shaft.

The history of increased prevalence of thin-shelled eggs and the multiple skeletal fractures point fairly compellingly to a vitamin/mineral deficiency or imbalance. Alas, we did not get any follow-up on epidemiological details or nutritional history.

Johne's Disease in the Northern Territory - Anton Janmaat

A presumptive diagnosis of Johne's Disease (JD), in a cow from a dairy in Katherine, was arrived at after serological and histopathological examinations. The animal’s serum was reactive in both the CFT and ELISA (CSL) and the intestinal lesion was typical with many acid fast bacilli present. The diagnosis was confirmed when VIAS reported a positive radiometric culture of post mortem tissues. The post mortem had been performed by a local private practitioner.

The cow originally came from either Victoria or New South Wales. The NT has now lost its JD-free status and CFT testing for JD of breeders destined for export has been re-introduced. In a mob of 417 breeders for export from a station in the Tennant Creek district 48 (11.5%) reacted in the CFT. Three of these (strong reactors in both the CFT and ELISA) were slaughtered. There were no gross or histological lesions suggestive of JD. Culture [now performed at the Berrimah Veterinary Laboratory (BVL)] of faeces and tissues is still in progress. The cattle had been grazing the "lakes paddock" and it is tempting to speculate that the serological tests were detecting antibodies to avian mycobacteria. Trace forward from the Katherine dairy has so far failed to detect spread of the disease

Disseminated Haemangiosarcoma in a Dog - Anton Janmaat

It is nice to find textbook (Moulton in this case) examples of disease when performing a post mortem examination in front of veterinary students on work experience.

Necropsy of a dog is a rare occurrence at BVL. On this occasion the owner was worried because an apparently healthy dog was found dead in the morning. The animal was in good nutritional condition. Six red tumours from mm to cm size were detected in the skin. In-situ examination revealed haemopericardium and red tumours in many organs and tissues.

The spleen had multiple tumours from mm to 8cm in diameter. The mesentery was affected and the tumours were mainly located near the intestines. The intestinal wall itself was also affected. There were multiple small (mm to cm) tumours in the lung and there was one large (8cm) tumour in the wall of the left atrium. The epicardium overlying this tumour appeared torn suggesting a rupture causing the haemopericardium which was considered to be the immediate cause of death. There were a few heartworms in the right pulmonary artery.

Microscopic examination revealed that the tumours were indeed haemangiosarcomas. In many tissues there was involvement of blood vessels suggesting haematogenous spread.

Crocodile Crawlers - Lois Small

A wild saltwater crocodile (Crocodylus porosus) had been kept on a commercial crocodile farm, but had not thrived. During slaughter, worms were reported to have crawled out of its mouth, much to the disgust of the female packer. An examination of the worms showed that they were *Geoeltascaris australiensis.* Further examination of the stomach contents revealed about 20 adult nematodes.

In two other recent submissions of healthy saltwater crocodiles from the wild, *Geoeltascaris* were found in the stomach and appeared to be the cause of deep ulcerations in the stomach wall. Ulcers were up to 4cm in diameter and appeared to contain a plug of material which was composed of nematodes, cellular debris and exudate. Up to 50 nematodes were found in one plug. Plugs of similar material were also free in the stomach contents. The effects of these nematodes on the health of the crocodile are not clear. [Ladds and Sims, AVJ 67:323 (1990)].

Arbovirus Monitoring in the Northern Territory in 1994 - Loma Melville

Weekly blood sampling for virus isolation was made at Coastal Plains Research Station throughout the year. The sentinel herd was changed in July to increase the chance of virus isolation during the early wet season and this has proved successful, with virus activity being detected following the first rains in October.

Bluetongue serotype 1 was isolated from January to June and Bluetongue serotype 21 in May to June. This is the first isolation of BLU 21 since 1984. EHD 5 and EHD 1 were also isolated during this period. Serology on the other sentinel herds confirmed that Bluetongue activity was restricted to the top part of the Northern Territory.

Apart form Bovine Ephemeral Fever, arbovrius activity was very low throughout the NT due to drought conditions. There was widespread BEF activity throughout the NT during the wet season. Seroconversions occurred at all sentinel sites except Alice Springs, with clinical cases reported from as far south as Tennant Creek and the Barkly Tablelands. One group of export steers had 150 head rejected for export due to BEF.

This year an entomology program has been run in association with sentinel herds. None of the important Bluetongue vectors were found at Alice Springs or Victoria River Research Station during the year. *Culicoides brevitarsis* was only at Katherine with *C. brevitarsus* and *C. actoni* at Douglas Daly Research Farm. *C. brevitarsus, C. actoni*and *C. wadai* were identified at Berrimah, while all vectors were present at the Coastal Plains in all months with *C. fulvus* the dominant vector species during the wet season and *C. brevitarsus* during the dry season.

TUMOURS IN BIRDS

I am writing the chapter on:

TUMOURS OF UNKNOWN ETIOLOGY, for the next edition of **DISEASES OF POULTRY** by Calnek et al.

The emphasis of this textbook is on poultry i.e. domestic fowls and chickens, turkeys, ducks, game birds and pigeons, but other species are dealt with as warranted. I have some examples of naturally occurring tumours of non-commercial birds (see Avian Pathol **21**:3-321 and domestic fowls from my time at the Veterinary Research Institute, Victoria. However, I would like to obtain other cases to include in this chapter.

Could you please assist by supplying?

TRANSPARENCIES

of the gross appearance of tumours in birds which might be useful. If you send originals I can copy these and return them. It would also be an advantage to obtain

STAINED HISTOLOGICAL SECTIONS

that might be suitable for photography. Any material received would be entered into the National Registry of Domestic Animal Pathology (NRDAP) and I would obtain specific permission from each submitter before inclusion into the manuscript.

I do not need examples of Marek's or leukosis.

I am particularly interested in obtaining examples of tumours of:

nervous system;
testes, ovary and oviduct - esp arrhenomas, dysgeminomas, ovarian Sertoli cell tumours, and ovo-testes;
thyroid and other endocrine organs;
pancreas, proventriculus and intestines;
skin and feathers;
muscle, bone and cartilage;
teratomas.

Please phone, fax or write in the first instance if you think you have something suitable.

Thank you.

Rod Reece BVSc, MSc, FACVSc. Registrar, NRDAP EMAI,
PMB 8, Camden, NSW 2570, AUSTRALIA
ph 046 293333; fax 046 293400

MINISTRY OF AGRICULTURE AND FISHERIES **MAF**
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Newsletter Editor
Australian Society for Veterinary Pathology
Toowoomba Veterinary Laboratory
P.O. Box 102
Toowoomba, 4350
QLD Australia

14th Sept, 1994

Dear Jim,

It is with interest that I read of the proposed amalgamation of the ASVP with the AVA. The draft paper published in the July issue of the Newsletter clearly sets out the perceived advantages and disadvantages in a balanced way and, as invited, I take this opportunity to comment on them.

The biggest disadvantage is clearly one of cost; not only in subscription but also in future conferences. Although it is pointed out that less than 50% of members of ASVP are not members of the AVA, I wonder what exactly is the proportion? I am concerned that a proportion of the ASVP members will be alienated by a requirement to join the AVA and in particular would request consideration be given to overseas members of the ASVP. As of 1992, this was only a small proportion (18/160). In a country the size of NZ, membership of the ASVP is very important for continuing education, training requirements (NZ recently took on two pathology trainees in the laboratory network) and for keeping abreast of pathology news in Australasia. May I suggest that associate membership be considered as a way around the financial disadvantage. No doubt this issue will come up again from other non-AVA members and possibly non-veterinary members.

With recent restructuring exercises in the MAF animal health laboratory system in NZ, there is some empathy here for the Victorian situation and understanding for concern about inadequate political lobbying and representation. However, considering the costs that will develop, this must be a real and practical, not just theoretical advantage. How effective was the AVA in addressing members concerns during the Centaur tender? Would having the ASVP as a special interest group have made it more effective? Just how effective can these large organisations really be when it comes to Treasury decisions in Parliament? Hopefully members of the negotiating team will have positive answers. However, considering the immediate reaction is likely to be one of disenchantment of "less than 50%" of the membership they had better be answers that are brought into the current debate.

The other advantages mentioned in the draft include "structured access to a wider range of views" (surely this can be sought by individuals interested in whatever wider range of views they like) and "income to support better speakers at conferences". This latter would be an advantage if there truly were the money to bring respected international speakers. Does this happen with other SIG's now? “Increased memberships from this happen with other SIG's now?”Increased membership from 'non-specialised' AVA membership" should be regarded with some caution. Does the ASVP want non-pathologist members? As was the situation two years ago in the NZSCVP, admitting non-pathologists as members does raise some issues on voting rights and majorities.

It is suggested that being an SIG will increase support for members to get to meetings (by their bosses presumably?). In recent years I believe the ASVP meetings have been held to coincide with the AVA AGM, so why is there any additional advantage to being part of the AVA meetings as a SIG? Is there a reason that in future years the ASVP meetings couldn't be held regularly at the same location and similar time as the AVA if people do wish to attend both?

After reading the draft summary, I would suggest that the fact that there are 4 listed disadvantages to the ASVP and yet only one for the AVA that the proposal has less merit than may appear at first glance.

Yours faithfully.

Bronwyn Smits

MAF Quality Management
Ruakura Animal Health Laboratory, Ruakura Agricultural Centre. East Street. PO Box 14103, Hamilton, New Zealand
Telephone 64-7-856 5522. Facsimile 64-7-856 8797

THE UNIVERSITY OF EDINBURGH

DEPARTMENT OF VETERINARY PATHOLOGY

Jim Taylor,
ASVP
Queensland DPI
O Box 102
Toowoomba Qld 4350
Australia

Dear Jim,

I write in response to the draft report on a possible reversion to our Society becoming a SIG of the AVA. as published in VPR 39.

As someone who was present during the 1983 meeting in Sydney where Ian McCausland, Bill Hartley and others formed the ASVP, can I say that the recommendations put forward run the great risk of repeating history, by ignoring it. Some of the reasons the ASVP was formed are contained in the "Disadvantages" section of the draft. Other reasons included the complete apathy on the part of the AVA towards, veterinary graduates employed in laboratories, their inability to see a conference as something where the advancement of science took place at a reasonable cost to participants and its patrician practitioner leadership. The ASVP could usefully ask Ian McCausland his views on the draft.

In the 14 years I have worked as a veterinary scientist, I have not felt the lack of joining a practitioner based organisation and will not do so now. I sincerely wish to remain a member of the ASVP and look forward lo doing so for many years to come.

Yours faithfully

Steven McOrist
BVSc MVSc PhD MACVSc

Pathmail: an electronic mailing list to link veterinary pathology laboratories.

This is an invitation for all Australian government and university veterinary laboratories to join pathmail - an electronic mailing list to link veterinary pathology laboratories.

We have been operating pathmail on a trial basis and have successfully linked a number of government laboratories. We would now like to expand the service. Subscribers to the system can post information about diseases or request comments or help on specific disease investigations. This information is automatically distributed to all other subscribers on the mailing list. Subscribers can then post responses directly back to the system for distribution to all other subscribers. The system is of great benefit in rapidly sharing and distributing information to a large number of institutions.

If you are interested in subscribing to the mail facility could you please provide me with a contact name and a full E-mail address e.g.

Contact name: Marc Kabay
E-mail address: mark@aidpoa.agric.wa.gov.au
Fax: 09 474 1881
Phone: 09 368 3473

You will need to have E-mail facilities at your laboratories and access to national electronic mail facilities through AARNET or INTERNET. The service is provided free of charge. I will forward additional information on using the system on receipt of your subscription.

Looking forward to your support for this initiative.

Marc Kabay
Veterinary Pathologist
Animal Health Laboratories
Department of Agriculture
3 Baron Hay Crt
South Perth. W.A.

How to apply

Complete the enrolment form overleaf and return it with your payment to:

David "Dos" O'Sullivan
National Key Centre for Teaching and Research in Aquaculture, University of Tasmania PO Box 1214, Launceston, TAS 7250.
Fax: (003) 243-804 Int. fax: + 61 03 243-804

Closing date for enrolments is two weeks before the start of the course, that is:

Wednesday 1st February 1995

Please note

Pre-registration is required. If this workshop is cancelled, a full refund will be paid. If you wish to withdraw from the workshop, a full refund (less a service fee of 10%) will be paid provided you notify David "Dos" O'Sullivan, National Key Centre for Teaching and Research in Aquaculture, University of Tasmania at Launceston, no less than **seven days** before the start of the workshop. No refunds will be granted after this time.

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Dos O'Sullivan
Tel: (003) 243-816, Fax: (003) 243-804

Speakers

Dr. Barry Munday D.V.Sc., M.A.C.V.Sc
Senior Research Fellow, National Key Centre for Teaching and Research in Aquaculture.

Dr. Barbara Nowak M.Sc. PhD.
Lecturer, National Key Centre for Teaching and Research in Aquaculture.

Murray Frith
Laboratory Manager, University of Tasmania

Dr. Judith Handlinger M.Sc. PhD
Fish Health Unit, Department of Primary Industry and Fisheries, Tasmania

About the workshop

This 3 day workshop has been designed in direct response to the increased interest in the investigation of fish kills, their causes and prevention. This workshop will address the causes of fish kills, procedures for their investigation and the history of fish kills in Australia. Various causes of fish kills, including diseases, environmental factors, toxicants and toxins, will be examined in detail. Several case histories of fish kills will be investigated in discussion groups.

A number of internationally recognised specialists will provide the papers, practicals and example, including Dr. Barry Munday, Dr. Judith Handlinger and Dr. Barbara Nowak. These will be supported by Murray Frith and experts from the National Key Centre for Teaching and Research in Aquaculture and the Mt Pleasant Laboratories of the Tasmanian Department of Primary Industry and Fisheries.

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AQUACULTURE

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Presented by the staff of the
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and the
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15-17 February, 1995
Department of Aquaculture

University of Tasmania at Launceston

PROGRAM

Day 1 - Wednesday 15 February

8.00 am Registration - Applied Science Building

9.00 am Welcome - Prof Nigel Forteath

9.30 am Introduction (Dr. Barry Munday) What is a fish kill? Why is it important to know the cause? History of fish kills in Aust.

10.15 am Morning tea.

10.45 am Environment factors as causes of fish kills. Part I (Dr. Judith Handlinger)

11.45 am Environmental factors as causes of fish kills. Part II (Dr. Barbara Nowak)

12.45 pm Lunch

2.00 pm Toxins and Toxicants as causes of fish kills (Dr. Barbara Nowak).

3.00 pm Diseases as causes of fish kills. (Dr. Barry Munday)

3.45 p.m Afternoon tea

4.15 pm History-taking, collection of information, etc. (Dr. Judith Handlinger)

5.00 pm Finish of lectures for day

6.30pm Dinner (Japanese), University Bistro

8.00 pm Forum discussions on day's activities. Evening snacks, teas, coffee, etc.

Day 2 - Thursday, 16 February

9.00 am Legal proceedings. (Dr. Barbara Nowak)

10.00 am Collection of evidence/samples. Use of fish kill kits. (Dr. Judith Handlinger)

10.45 am Morning tea

11.15 am Collection of samples from fish (i.e. fish `dissection, etc)

12.30 pm Lunch

1.00 pm Water quality testing. Part I Theory (Murray Frith, Laboratory Manager)

1.45 pm Water quality testing. Part II Practical (Murray Frith, Laboratory Manager)

3.00 pm Afternoon tea.

3.30 pm Use of fish kill kits. (All lecturers)

5.00 pm Finish of lectures for day

6.00 pm Dinner (Mediterranean)

8.00 pm Forum discussions on day's activities Evening snacks, teas, coffee, etc.

Day 3 - Friday, 17 February

9.00 am Presentations of actual fish kills. All participants are invited to give a case history. This is an informal presentation of 20 mins. to cover points such as: What was done to make the investigation effective? What has been done to prevent a further occurrence?

10.15 am Morning tea

10.45 am Presentations (continued)

12.45 pm Lunch

2.00 pm Presentations (continued)

3.45 pm Afternoon tea

4.15 pm Review and wrap up (Dr. Barry Munday)

5.00 pm Finish

6.30 pm Pre-dinner drinks, University Bistro

7.00 pm Workshop dinner (Tassie Seafood Feast).

Enrolment Form

Name and address of participant

(As individual requirements may differ, please photocopy form and use a separate one for each delegate or phone for more copies).

Title: …… First Name: ……

Surname: ……...

Postal Address: ……..

…… Postcode: ……

Phone: Bus: (……) …….

Fax: (……) …….

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Registration, notes, lunches morning/afternoon teas, dinners.	$370
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Accommodation Thursday 16th	40
Accommodation Friday 17th	40

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33.

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The second tenure track position is available at the rank of Assistant Professor. Applicants will have DVM (or equivalent) and PhD degrees. Preference will be given to those who are certified by the American College of Veterinary Pathologists (ACVP). The individual selected for this position will commit approximately 45% effort to graduate education, and instruction of veterinary students in classroom and teaching hospital rotations. Approximately 5 % effort will be devoted to the betterment of the department, college, and university through participation in departmental meetings and service on committees. The remaining 50% time will be devoted to toxicology research, for which the individual will acquire extramural support. Salary for both positions is negotiable. Starting date is July 1, 1995. Applicants should send letter of application, curriculum vitae, and names of 3 references to Dr Talmage T Brown, Jr. Department of Microbiology, Pathology, and Parasitology, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough Street, Raleigh, NC 27606, (919) 829-4258 or (919) 829-4455 (FAX). Application closing date is January 10, 1995 or until suitable candidates are chosen.

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