THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology S.A. Department of Agriculture Central Veterinary Laboratories, G.P.O. Box 1671, Adelaide, S.A. 5001 08-228 7271

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- PAGE <u>CONTENTS</u>
- 2 OFFICE BEARERS 1987/38
- 3 PRESIDENT'S REPORT
- 4 1987 A.G.M. MINUTES

STATE REPORTS

- 7 South Australia
- 7 Victoria
- 11 Queensland
- 13 New South Wales
- 14 Western Australia
- 18 Tasmania
- 19 JOBLINE

21 DEADLINE FOR OCTOBER ISSUE

OFFICE BEARERS 1987/88

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PRESIDENT'S REPORT

I am both honoured and humbled by being nominated and elected to the office of President of the A.S.V.P. I see so many erudite and energetic young veterinary pathologists around the country that I can only wonder as to why I should have been elected. I only hope that I and my team in South Australia can match the performance of previous office bearers. We will try.

I intend so far as is possible to restrict the activities of the A.S.V.P. to the pursuit of excellence in veterinary pathology and refer matters of political, industrial and regulatory matters to more appropriate bodies such as the Australian Veterinary Association and State/Commonwealth Public Service Associations.

South Australia is about to enter a most interesting and challenging period for veterinary laboratory services. As from 1st September 1987, free "herd and flock" laboratory testing will end and such cases will be fully charged. There are exceptions, these being:

- 1. When the disease is a notifiable disease.
- 2. When the testing is part of an approved prescheduled disease survey.
- 3. When the disease or condition is one in which the Chief Inspector of Stock has specifically requested the submission of samples.
- 4. When the samples originate from a Department of Agriculture research or extension project, and
- 5. When the samples originate from animals on Department of Agriculture research centres.

No doubt the number of submissions from the rural community will decrease markedly at least in the first instance, however it is hoped that there will be benefits in that practitioners will be more selective in specimens submitted and that we as pathologists will be more able to undertake better investigative pathology rather than the more traditional 'fire brigade' work.

The situation will be watched with great interest both from within and undoubtedly by interested parties interstate.

P. H. Phillips PRESIDENT

Minutes A.S.V.P. Annual General Meeting. Sydney

23rd May 1987

Minutes of the 4th Annual General Meeting held in the Veterinary School, University of Sydney, Saturday 23rd May 1987 at 8.30a.m.

1. <u>Present</u>

J. Glastonbury (President). I. Links (Secretary), R Whittington (Treasurer), A Crowley, L Badcoe, M. Carrigan, J Charles, J. Handlinger, W. Hartley. R .T. Jones, P. Ladds, C Lenghaus, D. McEwan, J. Marshall. L. Melville, P. Rahaley. R. Reece, T. Ross, J. Rothwell, J Seaman, P. Staples, G. Stone, R. Sutton, V. .L Tham, K. Thompson, J Thomas, T. Utteridge, K. Walker. D. Watt, R. Webb.

2. <u>Apologies</u>

R. Badman, C. A Bourke, J. Boulton, J. Finnie, R. Giesecke, C. Huxtable, R. Kelly, M. Lancaster, T. Nicholls, P. Phillips, W. deSaram, L. Sims, F. Trueman.

3. <u>Minutes</u> of the 3rd A.G.M. held in Brisbane, Sunday 18 May, 1986, accepted as a true record. (Webb/Ross)

- 4. <u>Business arising from Minutes</u> none.
- 5. <u>Presidents Report</u>

Moved that the report be accepted (Glastonbury/Ross) - Carried.

6. <u>Treasurers Report</u>

Moved that the report be accepted (Whittington/Rahaley) - Carried.

7. <u>Membership Report</u> - (Secretary)

Current Membership is 169 (136 in Australia, 19 in North America, 8 in Asia, 3 in Europe, 3 in New Zealand). This is an increase of 13 since 1986. Moved that the report be accepted (Links/Carrigan) - Carried.

8. <u>Elections</u>

Nominations must reach the Secretary 7 days before the meeting.

President:	P. Phillips
Secretary:	V. L. Tham
Treasurer:	T. Utteridge
Committee:	P. R. Giesecke
Committee:	W. deSaram

There being no other nominations the above officers were declared elected unopposed.

9. <u>General Correspondence</u>

- a) <u>Post graduate Refresher Course on Gross Pathology</u> Correspondence with the Director, Post Graduate Foundation and related correspondence in organising speakers.
- b) Australian Registry of Veterinary Pathology

Considerable correspondence. (See below 13 for details)

c) <u>Annual Proceedings</u> - <u>Deposit in Libraries</u> - <u>Communication with the librarian at the RVL Wagga established that to increase the recognition of the ASVP Annual Proceedings a copy should be deposited with each of the State Libraries, the Veterinary School Libraries, the National Library and for New South Wales - the Parliamentary Library and the Fisher Library (University of Sydney).</u>

Moved that correspondence be accepted (Links/Glastonbury) - Carried.

10. <u>Membership Fee/Non-financial Members</u>

Moved that Australian membership fee be set at \$12.50 (Whittington/Hartley) - Carried.

Membership fee for overseas members will be \$12.50 (sea mail) or \$17.50 (air mail). Moved that members unfinancial for greater than 2 years be removed from list of members (Lenghaus/Ladds) - Carried.

11. <u>Veterinary Pathology Report</u> - Format and State Representatives

The membership was happy with the current format of the Veterinary Pathology Report. The President thanked the State Representatives for their efforts. The State Representatives nominated for 1987-88 were Jim Rothwell (NSW), Sue Friend (VIC), Fraser Trueman/Roger Kelly (QLD), Lorna Melville (NT) and Clive Huxtable (WA). Judith Handlinger was to organise a representative for Tasmania.

12. <u>Slide of the Month</u>

Clive Huxtable advises that he is happy to continue as co-coordinator. The slide of the month with its new format (more information on diagnosis etc.) has proven very successful. Anyone who would like to receive the slides is to contact Clive.

13. <u>Australian Registry of Veterinary Pathology</u>

A submission was made to the Laboratory Leaders Group within the Animal Health Committee requesting funding for Dr. W. J. Hartley for 3 years plus operating expenses (a total of \$70,000) to establish the registry. This submission emphasised the benefits of the registry as a reference source and avenue for training and enabling documentation of all diseases affecting domestic livestock in Australia.

Correspondence with local Members of State end Federal Parliament regarding establishment of the registry and requesting their co-operation. The reply from the Federal Minister for Agriculture advised that funding depended on support from the states and that it was currently considered beyond their resources.

The submission to the A.H.C. was subsequently (September 1986) revised to a request for \$53,500 to be funded by the various states in the ratio: NSW and VIC 30%; SA, WA and QLD 10%; TAS and NT 5%.

Letters were sent to the Australian Agricultural Health and Quarantine Service, the various Veterinary Schools in Australia and to CSIRO requesting financial support (\$500/annum) for the registry to establish the "bona fides" of grass root support. To date donations have been received from the Commonwealth

Bureau of Rural Science (\$500) and the Graduate School of Tropical Veterinary Science, James Cook University of North Queensland (\$250).

In March 1987 an application for a research grant was made through the NSW Dept of Agriculture to the Rural Credits Development Fund with the total amount requested of \$38,270 for the first 2 years to establish the registry. The Rural Credits Development Fund meets in June with results known in October 1987.

There was a suggestion that we approach private companies for funds (Webb) however, it was considered better to wait until we had it established (Hartley). Access is available to the N.Z. registry in the interim (Thompson). Registry to be based at Camden Park (to be finished in 1988) with room allocated. NSW Dept of Agriculture has provided \$5,000 one-off allocation for purchase of Nikkon Microscope/Teaching Head/Computer /Slide-block-Kodachrome storage facilities.

A subcommittee was formed to handle the affairs involving the Registry. In view of the necessity for local liaison the following were nominated - Keith Walker, Tony Ross, Bill Hartley, John Glastonbury (Ladds/Lenghaus) - Carried.

14. <u>Award for the Best Presentation at the Conference</u>

Due to perceived difficulties in implementing such an award, the matter lapsed.

15. <u>General Business</u>

None.

16. <u>Next Meeting and Annual Conference</u>

Suggested piggy-back on a larger Conference e.g. Canberra 1988 (Ross) Topics suggested for 1st day were pathology of the respiratory system, foetus, liver or general pathology. A need was seen for improved understanding of the basic mechanisms of the latter.

17. There being no further business, the President declared the meeting closed at 9.30a.m.

Ian J Links Hon. Secretary

STATE REPORTS

SOUTH AUSTRALIA - Peter Phillips

Cryptococcosis Cases - (Tammy Utteridge, Diagnostic Lab. Services)

While we normally see only one or two Cryptococcus cases a year, we have seen three within a fortnight in early winter. The first was isolated from cerebrospinal fluid of a fitting dog after detecting yeasts in a Gram Stain of the CSF. The second was from a nasal swab of a cat with miliary pulmonary nodules. The third was in a lymph node aspirate submitted as the vet. queried lymphosarcoma. The smear contained massive numbers of yeasts, and excision biopsy of the lymph node for histopathology confirmation and culture yielded a heavy pure growth of <u>Cryptococcus neoformans</u>.

MUSCOVY DUCK MORTALITY- (Mike Hindmarsh, Robin Giesecke, Peter Phillips, Vui Ling Tham)

In April/May this year 2 four-month-old Muscovy (<u>Cairina moschata</u>) ducklings were submitted for necropsy. They were from an inbred flock (consisting of 4 matured and 46 four-month-old birds) from the Port MacDonnell area. These birds were kept on free range on water from a natural spring and had contact with water hens and blue cranes. Other poultry kept on the same property were chickens, cross-bred ducks (<u>Anas spp</u>) and quails. Only the Muscovy ducklings were affected and mortality (up to 50%) was noted when the birds reached four months of age. No mortality had previously been noted. Lameness was initially observed followed by bill pallor, facial caruncles, bluish fluid from nostrils and mouth, and death. Postmortem examination revealed excess fluid in trachea, marked pulmonary congestion and oedema and fibrinous deposits in thoracic air sacs.

Histologically, there was marked congestion, moderate infiltration by mononuclears of inter-air-capillary septa and clusters of rod-shaped organisms in vascular endothelial cells in the lung. Intracellular rod-shaped organisms were also present in endothelium of some blood vessels in the liver, renal glomeruli and intestine. Further changes were hepatic periportal mononuclear infiltrates, intestinal serosal mononuclear infiltrates and splenic congestion. The rod-shaped organisms were identified by electron microscopy as intracellular rod-shaped bacteria indistinguishable from those described by R.J. Julian <u>et al</u> (1985). (Journal of Wildlife Diseases. 21:335-337).

VICTORIA - Sue Friend

REGIONAL VETERINARY LABORATORY, BENALLA - Rob Seiler

General:

Current seasonal animal health problems in northeastern Victoria include hypomagnesaemia in cattle (sheep have also been affected), helminthiasis in all ruminant species and malnutrition in young lambs. This latter syndrome appears to be related to poor pasture quality, overstocking of ewes and consequent agalactia.

Suspect pyrrolizidine alkaloidosis in parrots

Two parrots owned by a local breeder died several weeks after the introduction of new seeds into their diet. Both birds had marked hepatic megalocytosis and biliary proliferation. Examination of the new seed showed it was contaminated with seed of Blue Heliotrope (<u>Heliotropium amplexicaule</u>), a prohibited species in Victoria.

Ovarian hypoplasia in sheep

Ovarian hypoplasia was identified in a large number of ewes in a flock with a low lambing percentage (marking percentage less than 50%). Some ewes had apparently never lambed although the youngest ewes were 3 years old. The highest incidence of ovarian hypoplasia was in these younger ewes. Bracken fern was abundant on the farm. Clover phytoestrogen levels were negligible.

A genetically similar flock of ewes run on a different farm by the same farmer has normal lambing and lambing percentages. There is little bracken on this second farm.

Malcolm Lancaster (057-62-2933) would be interested to learn of similar syndromes if they have been observed by personnel in other laboratories.

Hepatopathy in Drysdale Ewes

Over the past 4 years we have necropsied 12 ewes from a local Drysdale stud. The affected ewes had become depressed and inappetent. None had recovered. The consistent histological lesions are scattered hepatocellular necrosis and focal aggregations of neutrophils in the liver. Variable numbers of macrophages, containing PAS positive material, are present around blood vessels. In addition, there is some karyomegalocytosis and biliary hyperplasia. Bile plugs are sometimes present in canaliculi and haemoglobinuric nephrosis has been seen in some animals.

Liver and blood copper levels are elevated in most sheep. The problem appears to be more than simple phytogenous or hepatogenous copper poisoning, however, because the earliest and persistent biochemical change is elevated gamma glutamyl trasferase.

REGIONAL VETERINARY LABORATORY, BENDIGO - Rod Badman

Canine Heartworm

A 6 year old GSH Pointer bitch was treated for roundworms with piperazine tablets at 3 pm in the afternoon. By 10 pm the dog was cold and shaking and was found dead at 6.30 am the following morning. Haemorrhagic faeces were found where the dog died. Gross pathology revealed massive haemorrhage into the small intestine and considerable splanchnic congestion. The lungs were moderately congested and one pulmonary arterial branch contained fibrillary thrombotic material. Numerous large heartworms (<u>Dirofilaria immitis</u>) up to 25 cm long were present in the right ventricle and extended into the pulmonary artery.

Acute cardiopulmonary failure and shock due to heartworm was the diagnosis.

The effects of piperazine on adult heartworms were speculated upon. The circumstantial evidence suggests that piperazine initiated the course of events leading to the dog's death.

AUSTRALIAN ANIMAL HEALTH LABORATORY - Peter Hooper

AAHL has paraffin-embedded blocks and fixed tissues of a number of exotic diseases of animals obtained from overseas laboratories and other sources.

Examples of tissues held include those from cases of Foot and Mouth Disease, Rabies, Rinderpest, Peste des petits ruminants, Swine Fever (and comparable Salmonella encephalitis), African Swine Fever, pulmonary Sheep Pox, Lumpy Skin Disease, and Pseudolumpy Skin Disease (Bovine Herpesvirus 2), Aujeszky's Disease (CNS, PNS, and pulmonary), Avian Influenza (H_1N_1 , $N_6 N_2$) and Newcastle Disease.

If anybody is interested in obtaining sections of any of these diseases, contact Peter Hooper (052-265 222]

VETERINARY RESEARCH INSTITUTE - Robin Condron

The CSIRO Animal Health Laboratory, Parkville is the managing agent of the China Australia Poultry Project under the auspices of the Australian Development Assistance Bureau. Dr. T.J. Bagust is the Project Director.

The Veterinary Research Institute, Parkville is collaborating in this project particularly with regard to the training of Chinese veterinarians from the Poultry Disease Diagnostic and Training Centre, Tianjin. Dr. D.A. Barr is one of the external consultants to the project.

The major objectives of the project are:

- a. The establishment of a Poultry Disease Diagnostic and Training Centre at Tianjin.
- b. The establishment of a Nucleus SPF Poultry Facility at Harbin.
- c. Provision of advice for the establishment of a demonstration SPF Poultry Production Unit probably at Beijing.
- d. Trials of Australian commercial poultry stocks in China.

The first 3 trainees, Mrs. Lili Guo, Mrs. Fugui Li and her husband Wensheng Zhang had arrived in Melbourne in June and will spend the next 9 months studying avian bacteriology, pathology and virology.

REGIONAL VETERINARY LABORATORY BAIRNSDALE

Cryptococcosis in Two Dogs and A Cat (87/3286, 87/3300, 87/3733)

The laboratory diagnoses cryptococcosis only once or twice a year (on average) so it was something of a surprise recently to see 3 cases in 2 weeks, and 2 of these on the same day.

A 6 year old cat seen by Steven Postlethwaite had chronic, upper respiratory infection with nasal discharge, sneezing and regional lymphadenopathy. It was unresponsive to a range of treatments and was eventually euthanased. There was a large gelatinous mass in the nasal cavity, regional lymph nodes were enlarged and there was a discharging ulcer on the skin over the rump. Histologically there were accumulations of typical <u>Cryptococcus</u> organisms in each of these sites. The first dog, a 6 year old Labrador was seen by John Brennan after a short history of coughing and weight loss. Retropharyngeal lymph nodes, and subsequently pre-scapular lymph nodes were enlarged. One of these nodes was biopsied and showed a profound, granulomatous lymphadenitis in which numerous, small, often budding forms of <u>Cryptococcus</u> were seen.

The second dog, a 1 year old Rottweiller seen by Mark Neilan also had a brief history of weight loss and lethargy. It went on to develop nervous signs of hyperaesthesia about the neck and loss of the withdrawal reflex before it was euthanased. Necropsy showed the cranial meninges to be slightly wet and glistening, but did not indicate fully the extent of the histologic lesions. These consisted of large meningeal accumulations of Cryptococcus organisms in most sections of the brain and spinal cord. There was minimal inflammatory response (as is usual) but marked degeneration of axons in the spinal cord.

Cryptococcosis may be a difficult clinical diagnosis, although the pathologic diagnosis is straight forward. In animals with discharging lesions e.g. in the nose of cats (or more rarely in the skin) a smear should provide a clue. The laboratory also has the facility to culture the organisms if desired. Discharging lesions also raise the question of the public health risk. There are no known reports of transmission from animals to man, or even to other animals, however, the infection is acquired from the environment/soil, manure and dust. It is often suggested that infected animals may be immunodeficient. We have no way of thoroughly assessing the immune status in these 3 cases but the cat was negative to a FeLV test (Rob Seiler).

Coccidiosis in Wombat (87/4414)

We recently diagnosed an interesting case of intestinal coccidiosis in a 12 month old wombat (Graeme Best, Merimbula). The animal was found dead in a wildlife park following 2 days of slight diarrhoea.

Gross post mortem examination revealed an empty stomach and brown-yellow intestinal content. The wall of the small intestine was thickened; the mucosa appeared necrotic and was covered by a fibrin-like exudate.

No bacterial pathogens were isolated from small intestinal contents, but examination of faeces yielded 76,000 coccidial oocyst per gram. Oocysts were ovoid, thick-walled, brown, 50 x 30 microns. Histology of the small intestine showed severe epithelial and mucosal injury caused by heavy parasitism with <u>Coccidia</u> <u>sp</u>.

Although Munday in the Postgraduate Committee Proceedings (No. 36, 1978) states that there is no evidence that coccidiosis causes significant illness in wombats we believe that intestinal coccidiosis caused the death of this animal. It may have been immunosuppressed because no other wombats kept in the same enclosure snowed signs of illness. (Steven Hum, Nick Barton)

VETERINARY CLINIC AND HOSPITAL, UNIVERSITY OF MELBOURNE, WERRIBEE - Jenny Charles

Suspected amyloidosis in a Standard Bred Gelding

A tentative diagnosis of nasal and laryngeal amyloidosis has been made in a 6 year old racing standard bred gelding. The horse was referred to the clinic after a 6 month history of intermittent epistaxis and more recent nasal stridor with dyspnoea having failed to respond to treatment with antibiotics and sodium iodide for suspected nasal zygomycosis. The animal was euthanased after rhinolaryngoscopic examination revealed extensive lesions.

Necropsy revealed severe ulcerative and proliferative lesions over the arytenoid cartilages and epiglottis, and similar friable haemorrhagic and ulcerative nodules over the mucosa of the anterior nasal conchae, alar folds, and rostral nasal septum, without involvement of the lower respiratory tract. Histological examination of these tissues revealed an intense foreign-body type granulomatous response to large extracellular and intracellular submucosal masses of homogeneous eosinophilic material, displaying strong birefringence following Congo Red staining.

We are awaiting electron microscopic confirmation of a diagnosis of amyloidosis and special staining with Congo Red and KMnO₄, to determine amyloid type.

Cryptococcosis in a Border Collie Dog

Disseminated Cryptococcosis has also been diagnosed at Werribee, in a 2 year old Border Collie which presented with marked submandibular lymphadenomegaly, lethargy and progressive neurological dysfunction.

Necropsy revealed numerous <u>Cryptococcus</u> yeast forms in lungs, kidneys, pancreas, spleen, adrenal glands, gastric mucosa, visceral and peripheral lymph nodes, eyes, ethmoid turbinates, brain and spinal cord. There was also a severe secondary bacterial bronchopneumonia and a moderate duodenal infestation by Sarcocystis species, the latter presumably a consequence of immunosuppression.

10.

11.

QUEENSLAND - Fraser Trueman

Northern Queensland (P. Ladds)

<u>Queensland DPI, Animal Health Laboratory, Oonoonba Fluoracetate Poisoning</u> - four, six month old beef weaners in a mob of 80, died. Necropsy of one weaner, killed when sick, revealed myocardial necrosis and early calcification. It was suggested that the calves had access to plants such as <u>Castrolobium</u> grandiflorum.

<u>Bluetongue virus research</u> - the laboratory has been advised of support from the Wool Research and Development Fund to continue this important work which has now been discontinued at the CSIRO Longpocket Laboratories in Brisbane.

Graduate School of Tropical Veterinary Science, James Cook University.

<u>Crocodile hatchling mortalities</u> - in conjunction with staff of the PNG National Veterinary Laboratory at Kila Kila. investigations were made on the causes of high hatchling mortalities in both freshwater (<u>Crocodylus novaeguineae</u>) and saltwater (<u>C. porosus</u>) farmed crocodiles.

Detailed necropsies on 55 hatchlings revealed that metazoan parasites, coccidiosis, and bacterial septicaemia were the probable cause(s) of sickness or death in 13 (23%), 13 (23%) and 10 (18%) hatchlings, respectively. Other observed changes were non-suppurative meningitis in several animals, ulcerative dermatitis (important commercially) associated with <u>Dermatophilus-like</u> filaments in lesions, and a high occurrence of mostly intraerythrocytic, but sometimes also extraerythrocytic parasites. These studies in particular agents and appropriate control procedures are continuing.

Biological control of the cane toad (Bufo marinus)

In collaboration with other sections of the University, the DPI and the N.T. Government, work on this project has commenced. One aspect concerns diseases that affect toads. Preliminary surveys have revealed some of the following diseases: abromoniasis, cestodiasis, fungal granulomatosis, parasitic pneumonia and gastritis, "Coccidiosis", ? inclusion body enteritis, and ulcerative enteritis of undetermined aetiology. Tissue culture studies have commenced. The School is currently advertising for a Research Fellow (Animal Diseases) to handle much of this cane toad work.

Other

Other interesting cases included a further case of mycotic (probable zygomycotic) pneumonia with extensive lymph node metastasis in a slaughtered ox, and glycogenosis which caused nervous signs, exhaustion and death in a brahman crossbred weaner.

Animal Research Institute - (Fraser Trueman)

Regional diagnostic laboratories at Rockhampton andToowoomba commenced processing specimen submissions in May. Both laboratories are coping well with a good supply of diagnostic samples. The diagnostic group at Yeerongpilly now provides regional services for the Brisbane division. The Yeerongpilly laboratory will continue to provide support for accreditation and export testing and virological services for Toowoomba and Rockhampton laboratories.

<u>Kidney damage</u> was the main feature of the significant mortality at Cheepie (Quilpie area). Out of a group of 44 two-year-old steers, 41 died. The others were sick and another one subsequently died. Symptoms included dullness, reluctance to move and inco-ordination of the hindquarters. Laboured respiration was noticed followed by recumbency and death within 2 days. Clinical pathology revealed moderate anaemia and marginally elevated serum creatinines in 2 clinically sick animals.

Necropsy of these two steers revealed extensive transudates in the thoracic, abdominal and pericardial spaces. The liver and renal cortices were pale. Histologically; there was severe interstitial fibrosis in the kidneys. Tubules were collapsed with thickened basement membranes, and some contained yellow pigment in epithelial cells. There was also quite severe interstitial infiltration with inflammatory cells.

The 6000 acre paddock containing these animals was heavily timbered with an abundance of Pimelea and Ellangowan poison bush. The cause of the mortalities remains unknown, but the kidney lesions were considered significant. Abortions occurring between 3 and 7 months gestation were investigated on a property in the Ipswich area. The one foetus examined had both cerebellar hypoplasia and internal hydrocephalus. Abortion due to mucosal disease virus was diagnosed on the basis of these lesions, plus a titre or 1/4 in pleural fluid from the foetus.

Selenium Poisoning

A mistake in addition of selenium supplement to a ration caused disease in pigs 24-26 weeks of age. Depression, paralysis and coma affected 20 pigs and 2 died. Lesions of the spinal cord have been described for selenium toxicity but were not seen in the one spinal cord examined in this case.

Two feed samples had 50 and 79 mg/Kg Selenium and 8 blood samples have levels of 2.1 to 5.4 mgm/Kg. Liver and kidney samples collected from a pig at the onset of the problem had levels of 105 and 13mg/Kg FW respectively.

Brucellosis

Serological evidence for <u>Brucella suis</u> infection was present in a 50 sow herd with a history of summer abortions at Wandoan. Dry sows were run with a boar in a 20 acre paddock and there was possible contact with feral pigs. Tube agglutination titres in 8 sows ranged from 4/50 to greater than 4/400 and were confirmed by mercaptoethanol and 56° tube agglutination tests.

<u>Clostridium septicum infection</u>. Three hundred of a mob of 6,000 mixed-sex Merino weaners, mulesed and marked 11 days previously, developed lameness and anorexia progressing in many cases to death. Mulesing and marking wounds appeared infected and malodourous. Histological examination revealed a severe purulent inflammatory reaction and necrosis of superficial tissue with inflammation, necrosis and fibrous tissue replacement of underlying muscle. Numerous Colostridial-like bacteria were visible. <u>Clostridium septicum</u> was isolated from affected tissue.

<u>Copper Deficiency</u>. Forty three of 400 Angora goats had died on a property near Brisbane. The clinical findings were marked anaemia, poor body condition and poor fleece growth. The post mortem findings were unremarkable apart from anaemia. Histopathology on liver and kidney revealed heavy deposition of haemosiderin in Kupffer cells and in the epithelium of the proximal convoluted tubules. Liver copper levels from one animal were 18 mg/Kg which is less than half the normal value.

<u>Cassia occidentalis</u>. Six 9-month-old Brahman heifers died and 10 others were affected during mustering of 250 head at Emerald. The cattle staggered and collapsed. Necropsy of one revealed all skeletal muscles pale and dry with the larger muscle groups mostly affected. Severe necrosis of muscle fibres was seen histologically and serum CPK and GOT were 130,000 IU/L and 6677 IU/L respectively. Three dogs at the mustering camp ate part of the carcase of the necropsied heifer and 2 dogs later could work for only half the day, becoming very stiff and unable to continue with the muster. Serums obtained from the dogs were too haemolysed for analysis.

Toowoomba Veterinary Laboratory

<u>Polioencephalomalacia</u> was diagnosed as the cause of death of 30 two-tooth Merino wethers from a flock of 650 grazing forage sorghum. Clinical signs included frothing salivation, lateral recumbency,

opisthotonus and hyperaesthesia. Histological changes were typical of polioencephalomalacia and included lamina necrosis of cerebral cortex with pink necrotic neurones and prominent perivascular and perineuronal spaces.

<u>Rhodococcus equi</u> was isolated from multiple hepatic abscesses and a spinal abscess in a 2 year old Saanen doe. The doe presented with severe paraplegia, no anal reflex and poor skin sensation caudal to the first lumbar vertebrae. The spinal abscess approximately 30mm in diameter was ventral end lateral to T9-T10 intervertebral space and impinged dorsally on the vertebral canal causing compression and narrowing of the spinal cord. Histological lesions in the cord were severe. Wallerian degeneration, numerous spheroids and chromatolysis of lower motor neurons distal to the lesion.

Erysipelas/Suspected Aflatoxicosis

<u>Erysipelothrix rhusiopathiae</u> was isolated in pure culture from the lung, liver, kidney and spleen of one of eight 16 week old large white pigs that had blotchy discoloration of the skin, fever (41.5°C), rapid respiration and terminal convulsions. Interestingly liver lesions consistent with chronic aflatoxicosis were present and it has been shown that subclinical toxicity from aflatoxin enhances the susceptibility of swine to acute erysipelas (Am.J. Vet. Res. (1978) <u>39</u>:445)

Suspect <u>Papova virus</u> infection was diagnosed in a 3 month old Peachface parrot that died suddenly. At necropsy the bird had a pale mottled liver. Histological lesions included severe acute hepatic necrosis. Hepatocyte nuclei were enlarged with marginated chromatin and eosinophilic intranuclear inclusions which completely fill the nucleus. Virus particles were observed by electron microscopy on negatively stained preparations of ground formalized liver. The lesion is consistent with a previous report of the disease (AVJ (1985) <u>62</u>:318)

NEW SOUTH WALES - Jim Rothwell

Vibrio anguillarum infection in prawns

This was diagnosed as the cause of death of about 200,000 hatchery prawns (Penaeus plebejus). They were affected at the mysis and zoeae stages of development, (up to 1 mm body length). Death was preceded by a period of sluggish movement. The prawns' appendages were darkly discoloured, and bacteria were detected histologically in them. V. anguillarum, which is a recognised prawn pathogen, was isolated on a marine medium. Infection by virus or by the fungus Lagenidiua sp. had been suspected, but was not confirmed.

<u>Degenerative axonopathy of neonatal calves</u> - P.A.W. Harper, Veterinary Laboratories, Glenfield, New South Wales, 2167.

Lesions of moderate to severe diffuse axonal swelling and loss, with Wallerian-type degeneration, myelin depletion and gliosis in the spinal cord, brain-stem and occasionally mid-brain, and peripheral nerve roots has been recorded in eighteen neonatal calves in the past 12 months at Glenfield. The lesions suggest a prenatal insult affecting mainly motor areas of the foetal neuraxis.

Clinical signs of weakness and recumbency from birth, and variable neurological deficits including hyperaesthesia, depression, limb extension, head tremor, nystagmus, apparent blindness, and opisthotonus when stimulated have been observed. Thirteen of the calves have been Holstein-Friesians with a sire to daughter mating in one case, and two calves having a common sire used widely in AI programmes, however, the aetiology remains undetermined. We would be interested to receive advice and material from others who may have had experience with what we believe may be a previously unrecognised disease entity.

Sow Wastage

A 250 sow piggery at Goulburn, New South Wales with history of uninvestigated wastage of about one sow per week: A moribund but conscious large white hybrid sow was presented for autopsy 28 days post mating. Ante-mortem haematology was as follows: PVC 33%, Total WCC 40 x $10^{9}/1$, differential, 42% band neutrophils, 35% segmented neutrophils, 3% lymphocytes, 20% myeloblasts (1%) or metamyelocytes (19%).

Post mortem examination indicated 20 litres of foul smelling brown fluid in the uterus. The posterior cervix was dilated, closed and contained 50 mls of creamish exudate. <u>C. pyogenes</u> was isolated from both sites, plus a Group D <u>Streptococcus sp</u>. from the main body of the uterus. The uterine wall was necrotic with a severe suppurative diptheresis. Liver and kidney were pale and friable. Histologically there was acute iliac lymphadenitis with marked lymphoid hyperplasia, however, the spleen showed severe toxic depletion of white pulp and acute splenitis. Renal and hepatic toxic degeneration was apparent. (GN87/2163). Can anyone comment critically on pyometron, metritis etc. as a prime cause/contributor to sow wastage problems in Australia?

Dairy Cattle

Adenovirus enteritis

A case of haemorrhagic enteritis and death in an 18 month old Friesian heifer from Wingham submitted in September last year was reviewed as part of an enterotoxaemia project. Large inclusion bodies were found in endothelial cell nuclei in the cortex of the kidney. On further investigation they were also seen in the endothelial cells of the small intestinal mucosa and in the mesenteric lymph node. No inclusions were seen in the liver or lung. The history and presence of inclusions were very similar to a case of adenovirus enteritis reported on the North Coast last year by RVL Wollongbar. A comparison of the 2 cases showed similarities in distribution sire and colour of inclusion bodies. Both cases had a history of a haemorrhagic enteropathy. Both cases involved single animals. Adenovirus enteritis is a recognised entity according to Jubb and Kennedy. However, to our knowledge it is rare in Australia. (GN86/3688)

WESTERN AUSTRALIA - David Pass

DEPARTMENT OF AGRICULTURE. PERTH

Copper Poisoning in Goats - J. Dickson

Copper poisoning in goats has occurred in Australia following therapeutic injections of copper preparations. Here and elsewhere copper poisoning as it is known in sheep appears to be uncommon.

The case described here concerns two 6 month old (sisters) unmated Saanen does which had been kept separate from the herd (which included the male triplet) to ensure freedom from CAE virus. They were kept in what had been an old chicken pen and there was no history of stress. The corrugated iron shed had a concrete floor that had been thoroughly cleaned and the surface soil of the pen had been dug out to a depth of approximately 25 cm and replaced by clean sandy soil. The owner insisted that they shared the same food and water as the rest of the herd. No copper blocks were used in water troughs and there was no access to any other plant material.

The first animal was off colour for a week before it died on a Saturday. In that time it was given an injection of Vitamins B and C, a worm drench (Systamex), iron tonic, trolyte and sulphamezathine (10 ml). The sulphamezathine was given as some blood stained faecal material had been passed. The viscera were removed at death and kept refrigerated until submitted to the laboratory on the following Monday.

At gross inspection the liver was swollen and had a nutmeg appearance and poisoning was suspected. The other organs appeared normal and all bacteriology and parasitology examinations were negative. By this time the owner noticed that the second goat was less bright than usual and it died the next day, Tuesday. At post mortem examination the animal was in fat condition with a full rumen. The omental fat showed a faint pinkish colour - a very indistinct change. There was a lot of fibrinous fluid in the thoracic and abdominal cavities, approximately 4 litres. The liver was swollen, had a nutmeg appearance and fibrin strands were adherent to the capsule. The lungs were firm and oedematous and the right side of the heart was distended. Kidneys appeared to be normal.

Histopathological examination of both livers showed massive centrilobular haemorrhages which was often confluent between lobules. The hepatocytes bordering the haemorrhages were variably vacuolated and some cells were undergoing apoptosis. Portal areas were unaffected. Kidney sections showed some interstitial congestion but no significant pathology, and were negative for haemosiderin. The lungs showed generalised interstitial congestion with oedema and release of macrophages into some alveoli.

Circumstantially the possibility of plant poisoning and mouldy food was excluded and tests for heavy metals such as arsenic and lead were negative. The liver changes, however, could still be consistent with copper poisoning.

The results of copper assays on the livers and kidneys of the two goats and later of six other apparently normal goats obtained from the abattoir were:-

	Goat 1 Goat 2				<u>6 goats ex abattoir</u>				
Liver copper ppm	66	45	41	158	171	115	366	130	
Kidney copper ppm	110	73	14	14	15	14	17	14	

These levels, while not particularly high, show greater content of copper in the kidneys than in the liver in goats 1 and 2 and suggest some impairment of copper metabolism.

Whether or not the two goats died from copper poisoning is controversial, but the chemistry lends support to the histological picture.

If the diagnosis is correct the lack of post mortem signs compared to those commonly seen in sheep could explain why copper poisoning has not been reported more commonly in the goat.

There have been no further deaths, the source of copper has not been found and the episode is something of a mystery.

If anyone has any information, observations or experience to air on the subject I would appreciate hearing from them.

LITERATURE

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ALBANY REGIONAL LABORATORY- Ruth Reuter

Selenium responsive myopathies in lambs and calves.

In association with the driest summer conditions experienced in the Southern Coastal region of Western Australia since the 1940's, the laboratory was presented with numerous cases of myopathy and ill thrift in weaner lambs, and sudden death due to cardiomyopathy in 1-2 month old calves. The spectrum of post mortem lesions in lambs ranged from paleness and streaking of skeletal muscles to cardiac haemorrhages and effusions reminiscent of Mulberry Heart Disease in pigs. In calves the striking gross changes were white streaked cardiac muscles, particularly affecting the left ventricle, and pulmonary oedema. Microscopic changes were typical of a Nutritional myopathy. Calcification was not present in the bovine cases. Selenium levels in the liver were low (around 0.21ppm in the sheep) and Vitamin E was not detectable by routine analysis. Treatment with selenium Vitamin E preparations resolved the problem.

Haemorrhagic enteritis in Calves associated with Rotavirus.

Sporadic report of losses of calves in the first few weeks of life due to haemorrhagic enteritis finally culminated in submission of a depressed, diarrhoeic 10 day old calf to the laboratory. On gross examination there were infarcts in the abomasal mucosa and grossly distended intestinal loops containing watery blood stained faecal material.

On microscopic examination the villi in the small intestine were shortened with blunt tips, covered by flattened to cuboidal epithelium. The lamina propria was congested with a mixture of inflammatory cells present. Rotavirus was identified in both small and large intestinal contents, haemolytic <u>E. coli</u> were isolated on culture and sections of abomasum contained large numbers of fungal elements in the area of the infarcts.

Lupinosis in Weaner Lambs.

Approximately 50% of a line of 472 lambs slaughtered at the abattoir in Albany was condemned for jaundice ranging from mild to severe in degree. Histologic lesions of hepatocyte necrosis, abnormal mitosis, retention of pigment, bile duct proliferation and fibrosis were characteristic of toxicity associated with phomopsin poisoning.

Brain Abscess due to Streptococcus in a pig.

Four weaner piglets from a group of 90 developed nervous signs including ataxia, opisthotonus and terminal convulsions. One piglet submitted live showed a head tilt to the left, inability to stand and trembling. A large greenish coloured abscess was present in the right lateral ventricle of the brain extending into the adjacent parenchyma. On culture a heavy growth of a Streptococcus biochemically similar to Str.suis was isolated. The relation of this finding to the other losses reported was not determined.

Other conditions seen in the laboratory included

- Campylobacter infection in pigs causing a range of conditions from necrotic enteritis and haemorrhagic bowel syndrome to porcine intestinal adenomatosis.
- Chronic interstitial nephritis with severe pulmonary calcification in a kangaroo.
- Polioencephalomalacia, enterotoxaemia and squamous cell carcinoma in goats.
- Losses associated with toxic algae in sheep of various ages.

MURDOCH UNIVERSITY

Mycobacteriosis in Brush-Tailed Bettongs - J. Thomas

Ongoing mortalities have occurred in captive marsupials at Murdoch University due to atypical Mycobacteria. A report of osteomyelitis in a brush-tailed bettong (<u>Bettongia</u> penicillata) due to Mycobacterium intracellulare was published last year (Richardson, K. C. and Read, R. A. - Journal of Wildlife Diseases, 22:3, 1986, pp425-429). Two further cases have occurred this year in the same colony. One animal had extensive severe lung and thoracic abscessation and chronic pericarditis. Acid-fast organisms were present throughout the lesion and Mycobacterium intracellulare has been isolated. A second animal had a more typical history of a swollen tail with discharging sinuses present for several weeks. A severe pyogranulomatous osteomyelitis had obliterated several coccygeal vertebrae and acid-fast organisms were identified throughout the lesion. Multiple abscesses, containing similar organisms, were present throughout the liver. A mild erosive pyogranulomatous arthritis and periarthritis was present in both hock joints. Bacteriology is still progressing in this case.

Whilst there are reports of Myeobacteriosis in native marsupials, few involve the musculoskeletal system and they are considered rare in wild populations. Superficial abrasions associated with housing are considered the possible route of entry for this organism. However, the case with thoracic involvement and no cutaneous involvement suggests inhalation as a possible route of entry.

Cage Birds

Pigeons: An outbreak of Salmonellosis occurred in fancy pigeons in Perth, resulting in the loss of 80-100 birds. The owner purchased two new flocks of birds and mixed then with his own flock but did not increase his loft space. Birds exhibited lameness, wing-drops and posterior paralysis due to arthritis of various joints and generalised debility due to septicaemia. <u>S. typhimurium</u> was isolated from joints or liver from a number of birds.

Galahs: Diarrhoea associated with the presence of 30mm diameter virus particles has again been seen in captive and wild galahs over the last 6 months. Birds have profuse, mucoid diarrhoea and lose weight rapidly. All birds to date have also had psittacine beak and feather disease. Contrary to our earlier statements about lack of survival, we have observed one bird that did survive. This bird was submitted because of depression and regurgitation. The bird bad chewed new wire and was diagnosed as having zinc poisoning. Pieces of metal were seen in the gizzard on X-rays. The bird responded well to Ca-EDTA and Maxalon therapy. Several days after it was returned home, it returned with mucoid droppings that were loaded with virus particles but it recovered in 3 days. It also had mild PBFD. The type of enteric virus, which is quite distinct from the virus of PBFD, is not clear. It was originally suggested that it may be a calici-like virus but sera from affected birds did not contain antibodies against human, chicken, feline, canine or bovine caliciviruses or human astroviruses 1, 2 and 4. We have been unable to reproduce disease, or see evidence of virus propagation in normal or PBFD-affected galahs and lovebirds and SPP chickens.

Budgerigars: We have recently had cases of coccidiosis in budgies. The disease occurs regularly in Perth, or at least we diagnose it every year.

Affected birds lose weight and droppings accumulate around the vent. Diagnosis can be tricky as birds that die from it usually do not have oocysts in faeces. They die from an ulcerative enteritis that appears to be induced mainly by sexual stages rather than asexual stages and by the time they die oocysts have been passed. If birds are examined early in the course of the clinical disease, oocysts, some of which may be sporulating, may be found in faeces stuck to the feathers around the vent or on the cage floor. Because of the size of the budgie's gut, gross lesions are very difficult to detect but lesions are obvious histologically in the intestine. Coccidial stages are present in surface epithelial cells and deep in the lamina propria. Single oocysts may be seen in the inflamed lamina propria of ulcerated gut.

18.

TASMANIA - Judith Handlinger

1. Dieldrin poisoning in dogs kenneled over old dip site

Samples from the second of two dogs dying with depression and convulsions were submitted to the Government analyst for poison analysis after brain histopathology and lead analysis gave no diagnosis. A diagnosis of dieldrin poisoning was made when 80 ppm dieldrin was detected.

History revealed that 8 months earlier their kennel had been relocated on the cement draining area of a disused plunge dip. Soil around the dog kennels contained 2200 ppm dieldrin. Even at 10 metres out from the dip dieldrin levels in the soil were 35 ppm. The dieldrin had apparently persisted from use as a dip in the 1950's/early 1960's. (B. Jackson/R. Mason)

2. <u>Eye lesions and Woolly Coated Poll Hereford cardiomyopathy</u> - a word of caution. (J. Handlinger)

During May, a Poll Hereford breeder submitted to the laboratory, on veterinary advice, a calf showing sudden bilateral corneal opacity (of one day's duration in one eye, two days in the other). This was shortly after another calf had died suddenly two days after developing similar symptoms. The calf showed a curly coat and extensive cardiac fibrosis on post mortem and a diagnosis of genetic recessive Woolly Coated Poll Hereford cardiomyopathy was made. Eye lesions have commonly been observed with this condition, but the reason for the association is not known (R. Cook, pers, comm.). In this case <u>Moraxella bovis</u> was isolated from one eye, though the eye lesions were of corneal oedema.

A third calf with eye lesions was submitted without further veterinary advice. This calf did not have a curly coat or abnormal heart sounds, but did have an elevated temperature. <u>Moraxella bovis</u> was isolated from both eyes, and both temperature and eyes responded to tetracycline treatment. There were no cardiac lesions at post mortem. Meanwhile a fourth (curly coated) calf was found dead in the paddock with extensive recent myocardial necrosis but no eye lesions. All calves showed normal selenium and/or GSH-PX and Vitamin E levels. All were sired by the same bull.

In this climate Moraxella bovis is usually seen in the hotter dryer months, with greater insect activity, but the second submission (third calf) does seem to be a simple Moraxella case, and these findings raise some questions about the nature of the eye lesions in these myocarditis affected calves.

3. E. coli 078 septicaemia has been seen in 5 month old weaner lambs as distinct from the usually much younger affected lambs. (R. Mason)

4. An investigation into poor egg quality suggested a relationship to the strain of bird used for the production of coloured eggs, and poorer quality of eggs from older birds. (R. Mason)

5. <u>Edwardsiella tarda</u> was isolated from a stranded sperm whale. This may have significance for the farmed fish industry. (R. Mason)

6. Ragwort poisoning was diagnosed in cattle two months after they left a heavily infestated property.

7. The breakdown of surgical wounds has been seen in goats with "VICCRYL" stature material (as reported in other States). (B. Munday)

8. Listeria monocytogenes was diagnosed as a cause of bovine abortion. (J. Hanlinger)

9. Giardia infection was diagnosed in an orphan monkey.

POSITION VACANT

JAMES COOK UNIVERSITY OF NORTH QUEENSLAND RESEARCH FELLOW (Animal Diseases)

Biological Control of the Cane Toad (Bufo marinus)

In cooperation with the Commonwealth Council of Conservation Ministers and the Conservation Commission of the Northern Territory, a project on the biological control of the cane toad is being carried out by James Cook University and the Queensland Department of Primary Industries.

The project consists of a control agent program supervised by the Graduate School of Tropical Veterinary Science and a population dynamics study in the Department of Zoology.

Applications are invited for the position of Research Fellow in the Graduate School of Tropical Veterinary Science. Appointment will be for one year in the first instance with prospects of renewal for a further period.

Applicants will be expected to have a veterinary postgraduate degree and/or appropriate experience in one or more aspects of animal disease research (pathology, microbiology, parasitology, etc) and be capable of pursuing research at the postdoctoral or equivalent level.

The appointee will be required to undertake comprehensive pathological and disease studies with the assistance of the Animal Health Division of the Graduate School. Some epidemiological field work in Queensland and the Northern Territory may be involved.

The salary range for the position is \$27,859-\$36,600 per annum and the commencing salary will be determined in accordance with qualifications and experience.

Applications in writing stating age, qualifications, experience, the names and addresses of three referees, and telephone contact number (if any) should be forwarded to the Registrar, James Cook University, Townsville, Qld. 4811. In reply please quote reference number A87038. Interested persons seeking more information please phone Phil Ladds 077-81 4428.

FACULTY POSITION ANNOUNCEMENT

Position:	Veterinary Anatomical Pathologist
Location:	Department of Microbiology, Pathology and Parasitology, School of Veterinary Medicine, North Carolina State University, Raleigh, North Carolina, 27066.
Rank:	Commensurate with qualifications/experience.
Qualifications:	DVM and Ph.D degrees with evidence of interest in teaching and research in area of veterinary anatomical pathology. ACVP Board Certification or eligibility preferred.
Responsibilities:	The individual will contribute to the teaching of anatomical pathology to professional veterinary students, be expected to train graduate students and residents, participate in service functions of the department, and develop a high quality research program in area of interest. Research interests in toxicologic pathology, gastrointestinal pathology or immunopathology are preferred.
Application Closing Date:	December 31, 1987, or until a suitable candidate is chosen.
Position Availability Date:	July 1, 1988.
Application Procedure:	Submit a curriculum vitae with the names of three (3) references to Dr. Talmage T. Brown, Professor of Pathology, School of Veterinary Medicine, Microbiology, Pathology and Parasitology, 4700 Hillsborough Street, Raleigh, North Carolina 27606

North Carolina State University does not discriminate on the basis of race, sex, or handicap and is an Affirmative Action/Equal Opportunity Employer.

VETERINARY PATHOLOGIST URGENTLY REQUIRED

The Regional Veterinary Laboratory at Bairnsdale, Victoria has a vacancy for a Veterinarian, preferably with pathology experience or training. The position is available for a minimum of one year.

Bairnsdale is a pleasant country town with a population of 10,000 set on the Gippsland Lakes and within a few hours drive of Melbourne and well known skiing fields.

The laboratory has a staff of 30 people and processes 12,000 diagnostic submissions per year.

Interested veterinarians should contact the Acting Director, Dr. Kit Button, phone (051) 522 751 or write to him at P.O. Box 483, Bairnsdale, Victoria 3875.

Watch for advertisement in the weekend press.

DEADLINE FOR OCTOBER ISSUE

11 September 1987