AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

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PAGE CONTENTS

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PRESIDENTS NEWSLETTER

In my world of academia the changes continue rapid fire and days for quiet reflection or reading in the library is increasingly scarce. Some would doubtless say "about time too", but continued attrition of time and staffing is taking its toll on the basic strengths of many disciplines and pathology in particular. Consider that not so long ago, mainly via the medium of "Pathology of Domestic Animals", the world saw Australia as one of the source areas for veterinary pathology. Now, as I said in my last newsletter, we see a discipline which some might regard as battling for survival. I also said in that, the Executive gave high priority to some form of political lobbying as a first step in addressing this issue.

I can report to you that the Executive has continued lo work on documenting a case for a new professional arm of the Society designed to supersede the Registry. This is now in the final draft stage. If successfully launched, the new entity will have major functions in quality assurance, continuing education, and professional advice to agencies on appropriate career and reward structures for veterinary pathologists and consultancy roles. It is proposed that an experienced and respected pathologist be appointed to run it and that appropriate funding be secured for a three year period in the first instance prior to a review. A case has been submitted and will be argued as forcefully as possible. The Executive sees the success of this proposal as being a litmus test of the ability of the Society to establish a political "beachhead" for the campaign to promote the important national role of veterinary pathologists. Let us all hope for success.

Planning is actively under way for the annual conference with the Local Arrangements Committee (Tony Ross, Malcolm France and Paul Gill) well into the tasks of securing a venue, drafting a program and obtaining sponsorships. Hope to see you there.

EDITORIAL

AVA SIG ballot - You could not have failed to sense that that arrangements for the ballot on affiliation with the AVA did not go according to plan. Due to circumstances beyond my control, envelopes were not sent out with the ballot papers and I suppose I should be grateful that only one letter of complaint and several phone enquires was received.

It was my intention to publish the results of the ballot in this issue of the report. However, because of the failure to provide envelopes and because of the low number of completed ballot papers received by the Secretariat, Committee has instructed me to run the ballot again.

With this issue of the report are an initialed ballot paper and two envelopes. Please complete them immediately and return them to The Secretary, ASVP, Animal Health Laboratories, Agriculture WA, Locked Bag No. 4, Bentley Delivery Service WA 6983. You are reminded again, that only financial members may vote. If you have not paid your subscription for this year, you are not entitled to vote.

Conference - Don't forget our Annual Conference is Saturday 16 and Sunday 17 May. The venue is the new Veterinary Science Conference Centre at the University of Sydney. Tony Ross and Malcolm France have put together a great program on the pathology of the GI system.

Registration fees are $75 for both days and $40 if you wish to attend for one day only. The Conference Dinner will be held at the acclaimed Cafe Otto and will cost $27/head. Send Registration fees to Tony Ross at EMAI.

The program has just about been finalised and 10.00 a.m-4.00 pm on Sunday has been made available for members' contributions (15-20 minutes each). If 45 minutes is allowed for lunch, by my calculations there is room for 15-16 papers covering either the theme or topics of the speakers' choice? Abstracts must be with Paul Gill by Monday 27 April so he can prepare the Proceedings.

STATE REPORTS

VICTORIA - Malcolm Lancaster

Idiopathic encephalomyelitis and demyelinating cranial neuritis in laying chickens - MJ Lancaster VIAS Attwood

Many 33 to 41 week-old laying hens on a farm in southern NSW developed ataxia with marked eating/drinking impairment. The high case fatality rate may have been due to dehydration. There was a diffuse encephalomyelitis and ganglioneuritis in all birds examined histologically. Most birds had blobs of degenerate myelin in the vestibulocochlear nerves. Other cranial nerves were similarly affected in some birds. Degenerate myelin was seen beside intact axons in the medulla of one bird. Occasional necrotic neurones were present in the pons and medulla of several birds. No viral or bacterial agent was isolated from affected brains. Has anyone seen this clinicopathological syndrome in layers?

Purkinje cell necrosis in broiler chickens - D Humphrey VIAS Attwood

Eight well grown broiler chickens were presented with mild head tremor, mild incoordination and low head carriage with neck extension. Histologically, many Purkinje cells were eosinophilic and surrounded by prominent glial cells. The owner reported that coccidiosis had been a problem in this batch of birds. Di-orthotoluamide (DOT or dinitolmide), a known neurotoxin, was used as the coccidiostat.

Atypical Mycobacteriosis in a long footed potoroo - Dr Judith S Nimmo Wilkie, Victorian Veterinary Pathology Services

A six -year old female long footed potoroo had a chronic suppurative osteoarthritis and osteomyelitis involving the right stifle joint. A Mycobacterium was cultured from purulent material from the joint. This was identified as *Mycobacterium terrae* complex. The animal was euthanased a few months later due to deterioration in its condition. At necropsy, the only lesions, other than the affected joint, were focal caseous areas in the dorsal parts of the lungs. Microscopic examination revealed multiple pyogranulomas in the stifle area, with club colonies of acid-fast bacteria in the purulent centres. Foamy, epithelioid macrophages were present in the outer layers of the pyogranulomas as were occasional multinucleate giant cells. Scant numbers of single acid-fast bacilli were present within the epithelioid macrophages. The lung lesions involved marked consolidation of an extensive area of lung due to a focal granulomatous pneumonia containing areas of caseous necrosis. The lesion of foamy macrophages similar to those in the joint and a peripheral lymphoplasmacytic infiltration. Very rare acid-fast bacilli were present in the macrophages. Long footed potoroos are known to be susceptible to infection with *Mycobacterium avium* but in that condition the bacteria and giant cells are numerous and lesions in the liver and the spleen are usually present.

Cryptococcosis in Western Victoria - Dr Judith S Nimmo Wilkie, Victorian Veterinary Pathology Services

Cryptococcosis has been a relatively common diagnosis in the past 12 months, with most cases originating from western Victoria. Both cats and dogs have been affected although most cases have been in cats. Lesions in the nasal cavity have been the most common manifestation but other sites have been skin lesions at the base of the ear and an unusual case of a large mass in the intestinal wall of a dog. This mass involved the full thickness of the intestinal wall and almost completely encircled the intestine. It was, not surprisingly, initially diagnosed clinically as an intestinal tumour.

Northern Territory - Anton Janmaat

Dental abnormalities in two weaner calves with persistent Pestivirus infection - Helen Parkes, Berrimah Veterinary Laboratories, NT Department of Primary Industries and Fisheries

In the Veterinary Pathology Report, Volume 46, July 1997, Bruce Hill described dental abnormalities in a 2 year old Murray Grey steer with persistent pestivirus infection. We have recently seen persistent pestivirus infection as the apparent cause of abnormalities in the milk teeth, resulting in excessive wear of the teeth and associated ill-thrift in two weaner calves.

The two weaners were from a group of 24 heifers, aged between 5 and 8 months, in a parasitology trial at the Berrimah Agricultural Research Farm, Darwin. One heifer maintained her weight, but gradually deteriorated in condition (harsh coat, faecal matting of tail and hindquarters, mild anaemia, weak and separated from the herd) from week 2 of the trial to week 14, when she was euthanased. The second heifer showed weight loss (22 kg over 18 weeks), diarrhoea and weakness. By week 18 of the trial she was thin, weak and dehydrated, was not responding to treatment with antibiotics and fluids, and was euthanased. Both these heifers had low faecal egg and oocyst counts until the final stages of their illness, when faecal oocyst counts rose. The remaining animals in the trial showed weight gains and appeared healthy.

At post-mortem examination there were no specific findings in either heifer apart from severe dental abnormalities. The incisors were discoloured (dark brown), with patchy erosion of the enamel. The premolars and molars were discoloured and irregularly worn, resulting in a "saw-tooth" appearance, and in the second heifer sharp edges and spurs on the teeth had caused ulceration of the mucosa of the hard palate. Both heifers were positive for pestivirus antigen by ELISA, while the AGID test for pestivirus antibody was negative. The remaining weaners in the group were all pestivirus antigen negative and pestivirus antibody positive. No dental abnormalities have been noted in these animals.

If the dental abnormalities in this case were caused by pestivirus infection, then damage to the developing enamel must have occurred *in utero*, i.e. at the time of infection of the dam and subsequent persistent infection of the foetus. The dental abnormalities certainly appeared to be severe enough to cause the general ill-thrift seen in both these calves.

Anaplasmosis in a Herd of Cows on the Douglas Daly Research Farm - Helen Parkes, Berrimah Veterinary Laboratories, NT Department of Primary Industries and Fisheries

A recent diagnosis of anaplasmosis as the cause of death in 16 cows from a herd of 170 Brahman breeders demonstrated to us that anaplasmosis can occur as an outbreak, showing apparently sudden deaths in mature, healthy animals born and raised in an area where cattle ticks are endemic. Furthermore, *Anaplasma* organisms may not be evident in erythrocytes of severely affected or dead cattle.

Anaplasmosis was diagnosed as the cause of death in 16 cows from a breeding herd of 170 cows at the DPIF's Douglas Daly Research Farm. Deaths commenced in late August and continued until early December, 1997. The cows were calving over this period and several stillborn full-term calves and small, weak calves were born. The outbreak was characterised by sudden death, with cows either found dead, or showing clinical signs including weakness, reluctance to move, dull coat, heavy breathing, slobbering from the mouth and mucus discharge around the nose, for at most 2-3 days before death. Examination of blood from affected cows showed severe anaemia with evidence of extensive extra vascular erythrocyte destruction and marked attempts at regeneration. Serum chemistry was relatively normal except in cows close to death, when evidence of liver and kidney damage was present. At post-mortem examination, increased abdominal and pericardial fluid, jaundice and "raspberry jam" spleens were noted. The most consistent histopathological findings were periacinar necrosis of hepatocytes and renal tubular changes, consistent with hypoxia.

Anaplasmosis was high on the list of differential diagnoses, but initially we tended to rule it out for several reasons. Firstly, these were locally bred, mature Brahman cows on a property well above the "tick line", suggesting they should have good acquired immunity to the tick fevers. Secondly, we thought of anaplasmosis as a chronic disease, not one causing high mortality and sudden deaths in apparently healthy cows. Thirdly, we were unable to find any *Anaplasma* organisms in the erythrocytes of dead cows or cows with severe clinical disease.

It was not until 30 cows were separated from the main mob, bled twice 12 days apart and kept under closer observation that we were able to make a diagnosis. On the first bleed, about one third of this group of 30 cows had *Anaplasma marginale* present at low levels with one cow having about 5% of erythrocytes parasitised. Mild to moderate regenerative anaemia, with a range of red cell changes including anisocytosis, crenation, spherocytosis and basophilic stippling, was present in most of the cows that were positive for *Anaplasma*. The repeat bleed showed increasing numbers of organisms in two cows with worsening anaemia. Paired serum samples sent to the Tick Fever Research Centre at Wacol in Queensland showed seroconversions or increasing reactivity of the serum in the Card Agglutination Test for *Anaplasma* antibodies in cows with developing disease. Affected cows recovered after treatment with tetracyclines.

Why didn't we pick this earlier? As with the other tick fevers, calves are relatively resistant to disease but develop strong, long-lasting immunity if exposed to ticks carrying the organism. However, older cattle that have not come in contact with infected ticks can be highly susceptible to disease; *Box indicus* cattle are just as susceptible as *Bos taurus* breeds. This was a closed herd on a well-run property with low tick burdens, so these cows may have remained naive. A mob of weaner steers from another property were kept briefly in a laneway next to these cows at the end of April 1997, then spent a month in a paddock that was subsequently used for some of the cows. It is suspected that this contact may have introduced the infected ticks.

Why the sudden deaths and high mortality rate? Russell Bock from the Tick Fever Research Centre suggests that in animals newly infected with *Anaplasma*, increasing numbers of red cells become parasitised over a period of days, but it is not until the development of an immune response and the appearance of circulating antibodies that large numbers of red cells are suddenly removed from the circulation, resulting in severe anaemia. These were heavily pregnant or early lactating cows under difficult environmental conditions (build up and early wet season), which may have contributed to the severity of the disease. Presumably we couldn't find any *Anaplasma* in the erythrocytes of the severely ill or dead cows because the infected cells had by then been removed from the circulation.

We will now be sure to include anaplasmosis as a differential diagnosis for sudden death in apparently healthy, mature, locally bred cattle!

Queensland - Jim Taylor

Lead poisoning in a heifer - Anita Gordon, QDPI Yeerongpilly Veterinary Laboratory

Nine of nineteen 10-month-old Friesian heifers in a small suburban paddock died during a three week period. Most were found dead without premonitory signs, although one animal observed prior to death was apparently blind, panting and drooling. The ninth heifer to die was found in extremis and died shortly afterwards. This animal was submitted for necropsy.

Necropsy revealed a poorly-grown, malnourished carcase with widespread petechial and ecchymotic haemorrhages of the thoracic subcutis, parietal pleura and subepicardium. The most significant necropsy finding was a large volume and variety of foreign material in the forestomachs and abomasum, including several plastic shopping bags, lengths of hemp rope and thick black rubber, numerous metal staples (possibly fencing clips), flakes of metal and small rocks. There was no evidence of perforation.

Histology of the kidney revealed a few, possible acid-fast intranuclear inclusions in tubular epithelial cells. Sections of cerebral cortex revealed moderately severe laminar cortical necrosis, consistent with subacutce lead poisoning.

Lead levels in the liver and kidney were 27.2 and 87.7 ppm (wet weight) respectively. Analysis of the metal flakes from the stomachs revealed 50% lead. The owner originally claimed the heifers' paddock was clean, but a subsequent search revealed a battery which had been chewed. He was mystified as to the origins of the other foreign material. The heifer's ill-thrift was attributed to a combination of chronic indigestion and helminthiasis (epg = 6900; larval culture: *Cooperia* sp. 96%, *Trichostrongylus* sp. 4%).

Suspect Epizootic Ulcerative Syndrome (Red spot) in Farmed Silver Perch - Jim Taylor, QDPI, Toowoomba Veterinary Laboratory

An aquaculturalist reported red ulcers on the fins and bodies of 80% of about 1000 farmed Silver Perch (*Bidyanus bidyanus).* The fish were held in groups in floating cages on a large dam. A fish was submitted for necropsy with multifocal red ulcerative lesions. Wet mount preparations revealed large numbers of sessile protozoa and broad aseptate fungal hyphae. Histologically the lesions were deep ulcerative and granulomatous dermatitis with broad aseptate fungi extending deep into the subcutaneous musculature. The protozoa were restricted to the surface of the lesion.

Pneumonic pasteurellosis in bovine pestivirus, persistently infected cattle - Jim Taylor QDPI, Toowoomba Veterinary Laboratory

Two 18 month old steers from a group of 150 had a history of wasting and laboured respiration. At necropsy both animals were in poor condition, heavily infested with lice and Barber's pole worms and had extensive consolidation of the lungs with a marked fibrinous pleural effusion. Histologically the lungs had a severe diffuse subacute fibrinopurulent pleuropneumonia and *Pasteurella multocida* was isolated. Both animals tested positive on the bovine pestivirus antigen capture ELISA.

Salmonellosis in meat chickens - Jim Taylor, QDPI, Toowoomba Veterinary Laboratory

An estimated 2000 birds were reported dead with another 2000 sick in a group of 250 000, 10-13 week old broiler chicks. Birds would become recumbent and die with a few showing nervous signs. At necropsy numerous birds had fibrinous peritoneal and pericardial exudates. Histologically there was fibrinous peritonitis, pericarditis, perimyelitis and meningoencephalitis. *Salmonella typhimurium* was isolated in pure heavy growth from primary cultures.

Chronic fowl cholera in quail - Jim Taylor, QDPI, Toowoomba Veterinary Laboratory

A commercial game bird producer reported deaths and illness in up to 10% of a group of 3000 brown quail. Twelve birds were submitted for examination. Lesions included pyogranulomatous pericarditis, perihepatitis, meningitis, pneumonitis and arthritis. *Pasteurella multocida* was isolated in pure heavy growth from numerous sites.

Periorchitis associated with Glasser's disease in pigs - John Gibson, QDPI, Toowoomba Veterinary Laboratory

A large Darling Downs piggery had been experiencing problems with Glasser's disease. At one stage a number of grower pigs died suddenly overnight and a number were observed sick. A noticeable feature in entire male pigs was marked swelling and reddening of the scrotum which was hot to touch in live animals. These pigs had a marked fibrinopurulent periorchitis and *Haemophilis parasitis* was isolated in pure culture. Other lesions typical of Glasser's were found in other sites of the carcasses.

Johne's disease exclusion in a ewe - Jim Taylor, QDPI, Toowoomba Veterinary Laboratory

An aged Merino ewe originally introduced from NSW had a history of weight loss and was submitted for necropsy to exclude Johne's disease. At necropsy there was a moderate serous peritoneal and pleural effusion with the intestinal serosa covered with multifocal coalescing white plaques. These plaques were associated with some cording of the serosal lymphatics, mild enlargement of the mesenteric lymph nodes and cicatrisation of the intestinal mesentery. Histologically the lesions were scirrhous adenocarcinomas with metastases in the mesenteric lymph nodes.

Western Australia - David Forshaw

Neosporosis in a dog - Mandy O 'Hara; Murdoch University

A 9-month-old male boxer dog was referred to the Murdoch University Veterinary Hospital with a 4-week history of progressive hind limb weakness and marked muscle pain. On clinical examination the dog was found to have hind limb areflexia and abnormal EMG activity in the hind limbs and caudal lumbar epaxial muscles. Nerve conduction tests were normal. The dog was euthanased and submitted for post mortem examination. Significant gross lesions were restricted to the myocardium and skeletal musculature. There was diffuse muscle atrophy over the hindquarters and multifocal pale streaking within the proximal hindlimb and forelimb muscles, tongue and myocardium.

Histopathological examination identified a subacute, moderate to severe, multifocally necrotising lympho-histiocytic to pyogranulomatous polymyositis and myocarditis with intralesional tachyzoites. Sections from the midlumbar spinal cord and nerve roots demonstrated a subacute, moderate to severe, lymphohistiocytic meningoradiculitis and leukomyelitis with intralesional tachyzoites. A mild multifocal and subacute lymphocytic neuritis was observed in the sciatic nerve.

Indirect immunoperoxidase histochemistry on paraffin sections from the heart was positive for *Neospora caninium*. Neosporosis has been reported before in Australia as the cause of neurological signs in greyhounds (Munday *et al*. 1990) and a boxer pup (Gasser *et al* 1993). However, in cattle Neosporosis has been principally reported as a cause of abortion (Boulton *et al*. 1995). Neosporosis has also been reported as a cause of abortion and neuromuscular disease in other species but not humans. Although *N. caninum* is known to be transmitted transplacentally, the life cycle of *N. caninum* is as yet undetermined.

Munday BL, Dubey JP and Mason RW (1990) *Aust VetJ67*: 76.

Gasscr RB, Edwards G and Cole RA (1993) *Aust Vet Practit* 23: 190-193.

Boulton JG et *al* (1995) Aust *Vet J* 72: 119-120.

Mucor amphibiorum infection in tree frogs - Cleve Main and John Creeper, AHL Agriculture WA

Readers are referred to the last issue of this report which described disseminated granulomatous lesions in tree frogs from a local zoo. Since then, the infectious agent has been identified as *M. amphibiorum*, a fungus that at the time of writing was incorrectly thought of as a culture contaminate.

Note: Publication in preparation.

Necrotising enteritis in piglets - John Creeper, AHL Agriculture WA

An outbreak of mortalities in 2-week old piglets was investigated. Some deaths were preceded by diarrhoea, however in several cases affected piglets appeared stunted and showed no premonitory signs.

Necropsy changes were restricted to the small intestine from the jejunum to the ileum with "skip lesions" in which grossly normal sections of intestine intervened between bowel affected by thickening and the presence of a yellow-white diphtheritic membrane overlying the mucosa.

Histologically lesions showed extensive deep infarctive-type mucosal necrosis, an intense necrotising inflammatory reaction in the submucosa and a variable inflammatory infiltrate extending through the muscularis to the serosal surface.

In one piglet several transverse intestinal sections showed this transmural
inflammatory reaction in one half of the circumference of the bowel with a sharp transition to near normal mucosa in the opposite side of the mucosal lumen.

Whilst anaerobic culture was negative, the predominance of clostridial-like organisms deep within the mucosal necrosis was highly suggestive of Necrotic Enteritis in Pigs, a disease we believe to have been rarely diagnosed in WA.

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