THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
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DIARY OF COMING EVENTS.

17th-18th May, 1986 - Annual Conference ASVP Veterinary School, University of Queensland, Brisbane.
17th-20th September, 1986 - International Meeting of Veterinary Pathology, Cordoba, Spain.
18th-22nd May, 1987 - Post-graduate Refresher course on Gross Pathology, University of Sydney.
16th-21st August, 1987 - World Veterinary Congress, Montreal, Canada.

PRESIDENT'S REPORT.

During the past quarter the activities of the Executive have been directed towards the organisation of our Annual Conference in Brisbane during May 1986, The Australian Registry of Veterinary Pathology and the post-graduate course on Gross Pathology set down for May 1987. This edition of "The Veterinary Pathology Report" contains accounts of our progress to date in these vital spheres which affect Veterinary Pathologists throughout the country. If you have any queries regarding our plans and aspirations or constructive suggestions for future action please let us know. Don't wait until the Annual Meeting because the sooner your bright Ideas can be considered the better.

If this newsletter achieves nothing else, we hope that it stimulates you all into masterly activity with regard to our Annual Conference. Our yearly meetings depend entirely upon your participation either through merely attending and sharing your pearls of wisdom during periods of discussion or the contribution of interesting material for the scientific sessions. Judging from the wealth of unusual outbreaks of disease already described by our State correspondents in these pages you should have absolutely no problems in further sharing your experiences with us on the second day of the conference. A preliminary flutter at this forum allows you to assess the worth of the material for later publication in the scientific literature and to glean future directions of investigation. So make sure you are there, Brisbane, on 17th and 18th May, 1986 and that you have your paper with Ian Links by 4th April, 1986.

Finally I would like to make a plea for you all to support The Pathobiology Chapter of The Australian College of Veterinary Scientists. For the sake of young Veterinary Pathologists it is essential that we set worthwhile goals for them to achieve and provide them with a body of recognised experts in their discipline with which they can identify. On 22nd February, 1986, I have to represent the Chapter at a meeting of the Executive Sub-Council of the College called to discuss a number of important topics. These include:

- Fellowship of the College by invitation.

- The relationship of the College and its Chapters with the Advisory Committee on Specialist Registration, including

    a. a possible consultative mechanism to assist the Advisory Committee gain the views of practising specialists in the various fields of veterinary practice;

    b. the clarification of the level of specialisation which should be registered.

The only way which you can truly influence these events is by becoming a member of the College. This year we have eight intrepid Pathologists who have placed their reputations on the line. Let's have double that number in 1987. Your Australian College of Veterinary Scientists needs you now!

ANNUAL CONFERENCE 1986.

The New Year is already upon us but fortunately the plans for our conference are progressing very well. Professor Peter Doherty, from the John Curtin School of Medical Research, has kindly agreed to present the key-note address on day 1 in line with the conference theme, "Immunopathology and Immunohistochemical Techniques". Our special thanks go to Roger Kelly who has done a sterling job organising the venue, arranging accommodation and refreshments and attracting two noted guest speakers. Professor Halliday and Dr. Chris Bishop, from Brisbane.

The conference will be held at the Veterinary School, University of Queensland, Brisbane on 17th and 18th May, 1986. Accommodation has been arranged at Cromwell College for Friday & Saturday nights at a cost of $30.00 per night (including breakfast and lunch on Saturday & Sunday). The conference registration fee, which will cover morning and afternoon teas, as well as other expenses, has yet to be decided but will probably be in the order of $20 to $30.

A river cruise has been booked for the Saturday evening at a cost of about $25 per head including a smorgasbord dinner. There is a good chance that the Aviary and Caged Bird SIG of the AVA will be holding their meeting in tandem with ours, and they have expressed an interest in sharing costs in some of our activities, including the boat trip.

Further details and registration forms will be included in the next "Veterinary Pathology Report".

Day 1 - To date we have been able to formulate the following tentative program:-

. - KEYNOTE ADDRESS - Professor Peter Doherty, John Curtin School of Medical Research, CANBERRA, "Experimental analysis of the inflammatory process in immunopathological disease".

- Dr. Chris BISHOP, Queensland Institute of Medical Research, Brisbane, "Morphological aspects of cell-mediated immunity".

- Professor V. HALLIDAY, Department of Microbiology, University of Queensland, "The latest developments in tumour immunopathology".

- Dr. Wayne ROBINSON, School of Veterinary Studies, Murdoch University, “The immunopathology of caprine retrovirus infection".

- Dr. Dieter PALMER. School of Veterinary Studies, Murdoch University. "The localisation of viral antigen in formalin fixed paraffin embedded sections".

- Dr. David PRITCHARD, School of Veterinary Studies, Murdoch University, "Applications of the avidin - biotinylated peroxidase (ABC) complex in immunohistochemistry".

- Dr. Tony ROSS. Regional Veterinary Laboratory, Glenfleld, “Immunoperoxidase staining of papilloma virus infections in cattle and sheep".

- Dr. Geoff MITCHELL, Regional Veterinary Laboratory, Benalla. Title to be announced.

- Two staff members from the James Cook University of North Queensland, Townsville. Topics to be announced.

- CLOSING ADDRESS - Dr. Phil LADDS, James Cook University of North Queensland, Townsville, who will draw together the many aspects of immunopathology covered during the day.

There are still a few spots available, so if you would like to contribute please let us know as soon as possible (Contact John Glastonbury on 069-230929). Presentations will be selected in the following order of priority:

1. demonstrate the use of immunopathological or immunohistochemical techniques.

2. demonstrate diseases where these techniques could be used to define a condition further but are not currently available.

3. diseases which have an underlying immunological pathogenic mechanism.

If time permits we are considering having our Annual Meeting at the end of this day,

DAY 2 - In a similar vein to previous years the second day of our Conference will be devoted to approximately 15 short contributions from members on a fruit salad mixture of topics. Reports of findings in research, interesting cases or useful diagnostic techniques will be gleefully received. Roll up your sleeves; we would appreciate receiving your contribution in the near future. Speakers should aim to speak for 10 minutes, leaving 10 minutes for discussion. Photographic slides of salient lesions should be prepared.

Presentation of Manuscripts - To allow for printing and distribution of our Conference Proceedings we will require your contributions (for both day 1 and day 2) by Friday 4th April. 1986. Don't let us down. For the second day your abstracts should be 1 to 2 pages in length including a brief introduction, methods, conclusions and possibly citing several major references on the topic. The manuscript should be prepared as follows:

* Type single spaced on A4 paper, using Letter Gothic 12 (or Prestige 12 or similar) type with a carbon ribbon.

* Leave 3.5cm Margin on the left and 2.5cm margin on all other sides to allow for binding and trimming.

* Even if the text is brief, begin at the top of the page, and leave the remainder blank for notes.

* Remember the copy you send will be photocopied exactly as it appears for binding into the booklet. No corrections or errors please!

* Mail flat to the Secretary. ASVP, C/- Regional Veterinary Laboratory, Private Mail Bag, Wagga Wagga, N.S.W. 2650.

Suggested lay-out for abstracts (Day 2):

AN OUTBREAK OF NEPHROSIS IN GOATS

J.A. Smith
Regional Veterinary Laboratory, Dimboola, VIC. 3414.

Twenty-five of a group of 100 goats died after eating silage
……

References

Smith. J. (1983) Aust. Vet. J. 101:290.

Trade Exhibit - John Searson has done a marvellous job organising a Trade Exhibit related to Immunopathology for Day 1, of the Conference. It will be mounted close to the lecture theatre and adjacent to where the lunches and morning teas will be served. We will devote an additional 1 to 2 hours at the end of the day to liquid refreshments in order that you may have sufficient time to savour their wares. The following companies marketing reagents and equipment relevant to immunopathology have been invited to participate:

* Flow Laboratories, Australian Monoclonal Developments,
* Medical Dynamics,
* Australian Laboratory Services,
* Commonwealth Serum Laboratories,
* Integrated Sciences,
* Medos.

If you know of any other company which may be interested in mounting a display, then John Searson is the contact person at the RVL, Wagga Wagga on 069-230928.

THE AUSTRALIAN REGISTRY OF VETERINARY PATHOLOGY.

Bill Hartley is now ensconced at Taronga Park working diligently upon the establishment of our Registry. He is presently employed by the Zoological Parks Board of New South Wales to develop a pathology registry for non-domestic animals. It has been decided that Bill will work on our registry in tandem with his duties at the Zoo. When the John Macarthur Agricultural Institute at Camden is completed in 1968, the Australian Registry of Veterinary Pathology will be permanently moved to that location. To provide Bill with financial remuneration for his endeavours we have applied to the McGarvie Smith Institute for funds to supply one half salary for 3 years. The Board of the Institute will make the big decision on 10th February, 1986. Keep your fingers crossed.

We feel that the establishment of this Registry is a most vital initiative for Veterinary Pathology in this country. Good examples of all disease conditions at various stages of the pathogenic process will be accurately stored and filed. The advantages of the Registry will be many and include the availability of an accurate reference source, an ideal avenue for training budding pathologists and the ready access to good quality lecturing and teaching aids.

When the executive of our Society moves from New South Wales I feel that it will be necessary to establish a committee to oversee the management of the Registry. It should have representatives from most states to ensure that the Registry is a truly national collection available to, and used by all.

You should have all received a circular from Bill regarding The Native Animals Registry. For positive reinforcement it is reproduced below:

SPECIMENS REQUIRED FOR THE NATIVE ANIMALS REGISTRY.

A letter is to be sent from Taronga Zoo to interested organizations outlining the aims and objectives of the native Animal Pathology Registry. This is a request for materials to be included in it.

It is probable that many veterinary diagnostic laboratories and other institutions will have on file at least some materials on diseases of native species for which they have no further use or which they would care to share with others. It is to be hoped that much of this material will be made available to the Registry where it will be examined and filed for reference and for teaching purposes.

The Registry will be pleased to accept as much as you feel could be useful for inclusion. We will accept colour transparencies depicting clinical signs, gross and microscopic lesions (any you want returned can be duplicated) from any freshly dead or non-decomposed animals; please send any tissues you have (we also need normals as well as diseased). Paraffin blocks are preferable but stained or unstained sections are acceptable. Stained blood smears with parasites will be welcomed. Also, if you have reprints of published papers or unpublished reports on diseases of native animals, please send these as well.

Samples for the Registry should be posted to Dr. W. J. Hartley, Taronga Zoo, P. 0. Box 20, Mosman, N.S.W. 2088. With the specimens should also be included the species of animal, location, age if possible, sex, brief clinical history, post-mortem findings and results of any laboratory examinations.

With the co-operation of everyone, we should have, within two or three years, a very comprehensive and valuable collection.

It is important to note that the Registry while initially concentrating on native species is designed to include exotic non-domestic animals also. This material will initially come from the zoos of Australia but if you have anything of interest in this area, we would be pleased to receive details in the same format as with native species.

THROUGH THE NAKED EYE - The Gross Pathology of Domestic Animals.

Under the guiding hand of Tom Hungerford the Post-Graduate Foundation in Veterinary Science is well ahead with plans for our refresher course to be held in Sydney from 18th to 22nd May, 1987. He hopes to have the program finalised for inclusion in the next ASVP newsletter. In addition 5 people have been asked to prepare short video films demonstrating post-mortem techniques for sma11 ruminants, horses, small animals, birds and fish. These will be shown at the commencement of each day's proceedings.

We will probably organise our Annual Conference for either the weekend before or after the course. At this stage we would appreciate you spreading the word amongst your colleagues in practice and field services so that we can put on a fine display to the remainder of the profession, educating the heathens into the bargain!

MEMBERSHIP FEES.

A reminder to the 70 members who have not yet paid their annual subscriptions, that the fee of $10 for 1985/86 was due on the 30th June, 1985. Members who do not pay promptly will have their names removed from the mailing list. For overseas subscribers we hope to organise airmailing of ASVP newsletters (including conference proceedings) commencing in June, 1966, but this will require an increased subscription, probably an extra $Aust 4.00 (New Zealand), $Aust 5.00 (Malaysia) or $Aust 8.00 (D.S.A.) per annum.

The new airmail subscription rates for overseas members will be advised after the 1986/87 membership fee is set at the 1986 Annual General Meeting.

STATE REPORTS.

WESTERN AUSTRALIA (prepared by David Pass).
MURDOCH UNIVERSITY.

Virus diarrhoea in Galahs

Profuse, mucoid diarrhoea occurred in captured wild Galahs that had Psittacine Beak and Feather disease. The disease spread rapidly to our caged Galahs and seven died. An in-contact Sulphur-crested Cockatoo developed diarrhoea but did not die. Diarrhoea lasted several days in Galahs and did not respond to therapy. Direct EM examination of faeces revealed “wall to wall” virus particles 30-35mm diameter. The type of virus has not been determined but they have a calici-like appearance. At PM, intestines were dilated, flaccid and contained very mucoid contents. Histologically there was mild villous atrophy, fusion of villi and epithelial hyperplasia.

Psittacine Beak and Feather disease has been successfully reproduced in nestling budgies and galahs by Sarah Wylie. The galahs were hatched from eggs collected in the wheat belt of Western Australia where there is no evidence of this disease (the only known cases in Western Australia are in the flock referred to above and they probably originate from aviary escapes). The inoculum used was prepared from affected feathers and contained virus particles. The work has demonstrated that the disease is infectious.

DEPARTMENT OF AGRICULTURE (W.A.)

Hairy or Woolly pod vetch poisoning by: R. L. PEET

Three cows and calves died at Esperance In November 1985. The Angus herd of 150 had been grazing a pasture of vetch with cotton fire-weed since early October 1985. Some animals started to lose condition and develop scabby circular lesions on their skin. One icteric cow was initially post-mortemed by the district veterinary officer who suspected cotton fire-weed poisoning. Histological examination of samples submitted to A.H.L. showed a severe granulomatous eosinophilic inflammatory reaction in heart, liver, kidney, lymph nodes, spleen and the wall of the small intestine. The inflammation was characterised by marked giant cell, eosinophil, plasma/lymphoid cell and macrophage infiltration. Hairy vetch (Vicia villosa Roth) poisoning was suggested and the vetch plants subsequently submitted to the herbarium were identified as Vicia villosa Roth subsp. dasycarpa. This is also known as "Namoi" in the eastern states since it was originally tested in the Namoi district of New South Wales. Poisoning has only previously been reported in the southern states of U.S.A. and South Africa but was suggested as the likely aetiology of the kidney lesions in Case No. 2 A.S.V.P. conference proceedings 1985 submitted by L. M. Badcoe. A subsequent cow post mortem at Esperance showed identical histological lesions.

The hairy vetch pasture at Esperance had been planted in 1984 and grazed by cattle over the dry summer of 1984-85 with no ill effects and the animals had "done well". This conforms with the reported overseas experience of toxicity only occurring when the plants are green and "in the full zenith of their growth".

Refs. 1) Panciera R.J., Johnson L.. Osburn B., J.A.V.K.A. (1966) 146 : 804-808.
2) Burroughs G.W., Neser J.A., Kellerman T.S. and Van Niekerk F.A. (J.S.A.V.A.) (1983) 54 : 75-79.

EDITOR'S COMMENT: Lyndon Badcoe advises that some errors crept into the summary of his case report presented to the 1985 ASVP Annual Conference (see Vet. Path. Report No. 9, July 1965, p.15). There was no amyloid present in the kidney sections, while the plant implicated in the condition vas Vicia dasycarpa (Woolly pod vetch). Presumably this is the same plant as described above.

ALBANY REGIONAL LABORATORY – RUTH REUTER

PARAKERATOSIS IN SWINE

A three month old piglet was submitted from a group of 120 animals. Over a period of two months 80-90% of the animals had developed thickening, wrinkling and crust formation of the skin on the back, ears and legs. In the most severe cases the entire body was affected. Treatment for mange had produced no response.

On histology there was irregular hypertrophy of the stratum corneum with retention of corneal nuclei; acanthosis, basal cell proliferation, and dilation of sweat glands. Dermal oedema and mild mononuclear cell infiltration were present as well. The lesions were considered characteristic of swine deficiency. The animals had not been receiving any vitamin/mineral supplementation in the diet.

ASPHYXIATION IN A DOLPHIN

A dolphin found by the Wildlife Service was brought in to determine if the cause of death could be identified. The body was covered with abrasions and areas of skin loss. A large amount of blood-stained fluid was found in the abdominal cavity; however the most striking changes were seen in the lungs. The airways were filled with frothy fluid, and there was severe parenchymal congestion and oedema. The changes seen at post mortem were consistent with asphyxiation due to drowning. This, plus the multiple abrasions present on the skin, suggested the dolphin had been caught in a fisherman's net and had been unable to escape.

NEW SOUTH WALES prepared by Mark Carrigan

Suspect Mycotoxicosis in Pigs (C. Bourke, Orange)

Three days after water damaged wheat was offered to pigs at Gulargambone, an outbreak of ataxia, tremors, and death commenced. The wheat constituted the only feed available to the pigs and after an initial period of reluctance, most pigs appeared to eat it. During the following 14 days, 75 of 100 pigs (all greater than six months of age), developed clinical signs and 48 of the affected pigs subsequently died. Autopsy of three pigs failed to demonstrate any significant pathology and histological examination of tissues from these pigs failed to demonstrate any convincing pathology. Throughout the brain sections there were scattered foci of gliosis and some add patchy vacuolation. In the liver sections there was very mild to moderate centrilobular to midzonal necrosis. Other tissues were unremarkable.

A sample of the suspect wheat was fed to four pigs at the R.V.L. Orange. These pigs were eight weeks old and weighed approx, 23 kg. For the first 48 hours all pigs refused to eat the wheat. After another 48 hours one pig had developed clinical signs and 24 hours later it died. Within a further five days the other three pigs had developed clinical signs and died. The average quantity of wheat eaten prior to death was 7 kg. The same wheat was fed to rats for more than two months without affect. The clinical signs, autopsy and histopathology findings in the experimental pigs were identical to those of the field cases. The clinical signs were specifically those of ataxia, tremors, convulsions and death, there was no vomiting and no scouring.

The sample of wheat fed consisted of a mixture of rotted grain, fermented grain, and apparently normal grain. The rotted and fermented grain was odorous and extensively colonised by fungi. The following fungi were identified in the sample: Rhizopus stolonifer, Scopulariopsis brevicaulis, Aspergillus flavus, Penicillium citrinum, Aspergillus glaucus and Chaetomium spp. Assay for aflatoxins A & B was less than 10 ppb. No verruculogen, penitrem A or paxilline could be detected 1n the sample. The wheat was water damaged 1n storage and came off the floor of an Australian Wheat Board Silo the day before it was first fed out on the piggery. On the available evidence this case would appear to represent a previously unrecorded mycotoxicosis.

Suspect Avomec Toxicity (J. Seaman, Orange)

A significant mortality in Murray Grey cattle following the use of the new injectable parasiticide "Avomec" (Merck Sharp and Dohme) was investtigated at the R.V.L. Orange. A total of eight cattle died (2% mortality rate) with five live steers (400-450 kg) being received at the laboratory for full clinical and pathological examinations. Affected animals showed severe central nervous system derangement with clinical signs developing 48-60 hours after the "Avomec" injections. Early signs included inco-ordination, knuckling over of fetlocks, swaying gait, muscle fasciculation, apparent blindness with drooling from the mouth and sometimes tongue paralysis (2/8 cases). As the condition progressed animals became depressed, unable to rise and eventually comatose before death in 24 hours. One steer was less severely affected and survived for 2 ½ weeks but showed marked weight loss, mild ataxia and was unable to lift its head.

Autopsy examination on the five steers was disappointing with patchy emphysema of the lungs the only consistent gross finding. Histopathology was equally unrewarding with no significant pathological changes to explain the severe clinical neurological syndrome.

A double blind trial was conducted by Merck Sharp and Dohme on the property with one steer (out of 50) developing neurological signs approximately 40 hours after injecting with "Avomec" at the standard dose (1 ml per 50 kg). Tissues from the affected animals and a control steer have been sent to the U.S.A. for "Avomec" analysis.

The active ingredient of Avomec (Avermectin Bl) stimulates release of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) from nerve terminals and potentiates GABA binding to the post-synaptic receptors so paralysing nematodes. In cattle signs of toxicity involve depression and ataxia but in toxicity trials up to five times the therapeutic dose was tolerated without clinical signs developing. It appears there is an adverse reaction to the product in Murray Grey cattle on this particular property.

Salmonellosis

Salmonella typhimurium was implicated in the death of 30 ex 700 Merino ewes. The mob had been in a paddock for six days and prior to this had been held in a hay shed for three days. All affected sheep developed clinical signs (lethargy and watery scours) and died within 24 hours. Two sheep presented for autopsy had mildly inflamed abomasums and intestines. Histologically there was a moderately severe enteritis with stunting and bridging of villi, dilation of crypts and infiltration of the lamina propria with mononuclear inflammatory cells. S. typhimurium was isolated from the intestines of both sheep.

Salmonella bovis morbificants was recovered from the intestines, liver and kidney from two foals. The stud had 32 mares and foals running in a paddock and over a period of 2-3 weeks approximately 50% of the foals developed a profuse watery diarrhoea. Salmonella typhimurium was recovered in pure growth from joint fluid and blood from a two week old foal with polyarthritis and depression.

Salmonella dublin was recovered from a three month old AIS calf with diarrhoea which died from a group of 30; four other calves were sick. The same organism was also recovered from the faeces of three very ill-thrifty Friesian cows and from a wide range of organs of another two month old calf.

Neonatal mortalities

Specimens for histological examination were received from 1 of 10 calves which was weak and died within 5-10 minutes of birth. Sections of the brain and spinal cord showed a severe disseminated meningoencephalitis characterised by the plugging of many blood vessels with lymphocytic type cells, neutrophils and large mononuclear cells, and multiple small foci malacia with polymorph infiltration. There was a mass outpouring of neutrophils into the neuropi1. In the liver there was disruption of the hepatic cords and a heavy sinusoidal leucocytosis. Inflammatory foci were also detected in the kidney and heart. The aetiology of the encephalomyelitis is not clear; however, it would most likely be viral in origin. Foetal heart blood was negative for Akabane and Aino virus antibodies. The pathology was not considered typical of a sporozoan abortion.

Purulent leptomeningitis with polyarthritis was diagnosed in two new born calves, born a month apart on a Lismore property. Bacterial agents were not demonstrated in, or recovered from lesions in either animal.

Septicaemia (Klebsiella pneumoniae) - a neonatal mortality problem in a small Clydesdale stud was investigated. Affected new-born animals failed to suckle and succumbed during the early days of life. A six day old colt submitted to the Laboratory had excessive volumes of yellow blood-stained serous and synovial fluids together with marked visceral congestion. Heavy pure growths of Klebsiella pneumoniae were recovered from a variety of internal organs and the synovial fluid from one joint. Septic fibrin thrombi were prominent microscopically.

Miscellaneous

A severe, chronic active liver fluke infestation caused sickness in 5 animals and death of 1 ex 31, 12 month old Australian Grey bull. Signs included rapid weight loss with some animals scouring. The worst affected animal that did not die had excessive salivation, was inappetent, lost weight, had brown watery faeces and temperature of 40 C. At necropsy of the dead animal, there was excessive pleural and peritoneal fluid and extensive severe hepatic fibrosis with haemorrhagic tracts and liver fluke infestation including 3mm length flukes in the haemorrhagic tracts.

Yersiniosis - severe outbreaks of diarrhoea in yearling Merino sheep on 2 properties were investigated. In the first there was a 33% morbidity rate with a case fatality rate of 5% while the corresponding figures for the second farm were 14 and 8%. Yersinia pseudotuberculosis was isolated from the small intestinal contents of a case from the first property and in both instances acute segmental erosive enteritis associated with the presence of prominent bacterial colonies was observed histologically.

Erysipelas - of 2,200 3-4 month old cross-bred lambs 1.4% were found to have polyarthritis. Similar occurrences had been noted in previous years and the male lambs had been marked with "elastrator" rings. Pathologically the affected joints were swollen, contained purulent synovial fluid and were surrounded by thickened joint capsules. No significant growth was obtained on primary culture but light pure growths of Erysipelothrix rhusiopathiae were recovered following passage through cooked meat medium. Chronic hyperplastic suppurative synovitis was found histologically.

Verminous Pneumonia - an aged female Macaque circus monkey found the harsh environment of West Wyalong to be a little too much. It died suddenly five days after arrival from sunny Queensland. Histological examination of the lungs revealed very severe diffuse fibromuscular hypertrophy, stenosis of the lumens of the bronchioles, vesicular emphysema and the presence of many parasites in the lumens of the respiratory tract. The good books tell us that Filaroides cebus is the most likely culprit.

SOUTH AUSTRALIA Prepared by Peter Phillips.

LEG WEAKNESS IN CHICKENS Robin Giesecke

Between 7 and 11 days of age 20% of a flock of dwarf chickens were noticed to have 'leg weakness'.

Eleven-day-old chicks presented for post-mortem were depressed, with flaccid paralysis of both legs, but no body tremor. No gross lesions were noted on post mortem, but histological sections of the brain and spinal cord showed perivascular lymphocytic cuffing, with chromatolysis of neurones. Multiple, lymphoid nodules were seen in the pancreas, but no lesions were seen in the musculature, sciatic nerves or limb bones. Avian encephalomyelitis (AEM) was confirmed by yolk sac inoculation and ELISA.

Vaccination of the parent flock had been allowed to lapse.

YERSINIOSIS IN FINCHES Robin Giesecke

Yersinia pseudotuberculosis was recovered from all organs of two finches which showed bilary abscesses in the livers and consolidation of the left lungs, on post mortem. The isolate was sensitive, in vitro to streptomycin, tetracyclines and neomycin.

IRON POISONING OF PIGLETS Robin Giesecke

A litter of 10 piglets dying within 24 hours of receiving an oral dose of a commercial iron preparation (actual formulation not known) showed marked oedema of the soft tissues of the throat, pallor of heart and lungs excess clear serous fluid in the thoracic cavity and softening of both kidneys, on post mortem

Histologically there was also a generalised cytoplasmic necrosis of hepatocytes and necrosis of the epithelium of the proximal and distal loops of Henle, accompanied by marked interstitial haemorrhage. There vas marked submucosal oedema of the upper trachea, interstitial oedema of the myocardium and septal and alveolar oedema within the lungs.

The piglets ranged in body weight from 0.6 to 1.6kg. The toxic oral dose for iron is 0.6gm/kg.

TOXOPLASMOSIS IN YELLOW-FOOTED ROCK WALLABIES Peter Phillips

Lungs from two juvenile yellow-footed rock wallabies were submitted for histopathological and microbiological examination following death of the animals from apparent respiratory failure. No bacteria were isolated. Histologically there was inflammatory exudate with mixed polymorph lymphocytic cellular content. There were many alveolar macrophages, only one of which in one section was seen to contain small basophilic round bodies.

A third animal died of a similar syndrome four days later and was submitted for necropsy. The findings were excess clear straw-coloured fluid in the thoracic and abdominal cavities. The lungs were uniformly darker than normal with white flecking across the pleura. The cut surface was moist. Histopathology was of a similar pneumonia to the other two animals, multiple malacic foci in the brain with occasional protozoan "cysts" and focal non-suppurative interstitial myocarditis with young intracellular protozoan "cysts".

Toxoplasmosis was diagnosed.

QUEENSLAND Prepared by Roger Kelly

Animal Research Institute

Rotavirus has been detected by EM in faeces from cases of post-weaning diarrhoea in pigs; this was associated with typical intestinal histology in one animal.

Significant mortalities in goats on two properties have been attributed to poisoning by Trema aspera. Liver lesions were severe periacinar hepatocellular necrosis (Poison Peach).

There have been two heavy mortalities in chickens due to avian encephalomyelitis.

Clostridium botulinum Type B has been isolated from the intestinal content of a foal with classical clinical signs of botulism; the toxin itself seems not to be demonstrable in gut content or serum in such cases, nor is there any apparent extrinsic source of toxin in the feed.

TASMANIA Prepared by David Obendorf

1. AVITAMINOSIS A IN REPLACEMENT LAYERS

A Departmental Poultry Officer visited a breeder establishment in Southern Tasmania and in passing became aware that 5,000 replacement layers between 10 and 14 weeks old were not doing well. It was reported that the birds had "bunged up eyes" with a flock morbidity of 100% and a mortality of 15%. The owner had suspected fowl cholera and was presumably self-treating the birds.

Eight birds were forwarded to Mt Pleasant for examination. They were in poor physical condition and all had plugs of white caseous material in the conjunctival spaces. On autopsy, the birds also had white pustule- like foci in the pharyngeal and oesophageal mucosa and some also had distended ureters.

Pasteurella galindrum was recovered from the conjunctival sacs but not Haemophilus sp or Mycoplasma sp. Histological changes consisted of squamous metaplasia in the glandular mucosa of the pharynx, oesophagus and of the ureteral epithelium. The trachea! Epithelium was hypoplastic, flattened and semi squamous.

Liver Vitamin A levels were all <0.05 mmol/kg.

Vitamin A deficiency was provisionally diagnosed on gross lesions and Vetemul was given to the birds. A significant improvement was noticed in 3-4 days and all remaining birds subsequently recovered and were sold.

The owner mixes his own rations. The date on the invoice of the premix in use was for 9 months previously. The premix was kept in a woven fabric bag on the floor of a galvanised iron shed. It would have suffered from exposure to damp and high temperature. (Roy Mason)

2. SUSPECT INFECTIOUS BRONCHITIS DEPRESSED EGG PRODUCTION

In early September 1985, 1,500 day old layer chicks were obtained from a large commercial enterprise. These chicks were placed in a cleaned and rested shed.

The property is very isolated from other poultry keeping enterprises and the owner operates 2 sheds on an all in all out basis. The sheds are about 100 metres apart and each holds approximately 1,500 birds under a raised slatted floor natural ventilation environment. The practice is to run one shed with active laying birds while the other shed is "down" for cleaning, resting and rearing a replacement flock.

On 22 October 1985 layers in the nearby shed were noticed to be showing mild respiratory distress with some gasping, gurgling and voice change. The birds were a little depressed but could be easily roused. At this stage egg production was about 75%.

Three birds were submitted for examination during this acute phase. They had laryngeal and upper tracheal mucosal hyperaemia with excess pharyngeal, laryngeal and tracheal mucus. Pasteurella haemolytica was recovered from the trachea and not Mycoplasma sp or Haemophilus sp. An embryo lethal and dwarfing agent was isolated in fowl eggs from a tracheal homogenate. The chorioallantoic fluid did not agglutinate fowl erythrocytes. Histologically there was epithelial hyperplasia, inflammatory cell infiltration and a mucopurulent exudate in the tracheal lumen.

Within 7 to 10 days of the first signs the egg production had fallen to 40% and there was also a fall in feed consumption. Egg production had returned to normal within 50 days of the detection of the first signs.

This property has a long history of freedom from infectious bronchitis maintained by its isolation and the all in all out management. Although TB vaccination was not requested by the owners, it is likely that the day old chickens had been exposed to TB vaccine which is known to be used at the supply source. It would appear the vaccine virus remained active within the birds to at least 6 weeks of age then spread across to the other shed infecting the unvaccinated layers. (Roy Mason)

3. LEUCOMYELITIS IN GOATS

Two 2-month old Toggenberg kids from a Southern Tasmanian property developed a rapidly progressive paraplegia. Clinical signs were noticed only a few weeks prior to presentation, commencing as proprioceptive deficits and swaying of the hindlegs, through to paresis, loss of co-ordination and recumbency and finally flaccid paralysis and urinary incompetence. The tendon stretch reflexes were present in both hindlegs, however, withdrawal pain responses were poor or unapparent.

The spinal cord in the lower thoracic and upper lumber region of both kids was grossly enlarged, soft and greyish in colour. From transverse sections of formalin fixed spinal cord it was apparent that one side was more enlarged with greater disorientation of the grey matter and white matter tracts than the other. This may have explained the somewhat hemiplegic nature of the nervous sign in these kids and the tendency of the animals to prefer lying on one side. Histologically there was an extensive infiltration of lymphocytic and round cells especially around blood vessels but also extending into the open spaces between axon sheaths. Axonal degeneration, leucomalacia with macrophage activity was evident through the white matter. The grey matter tracts were affected but to a lesser extent.

The property of origin has a long history of caprine retrovirus pneumonia and arthritis. Caprine retrovirus gel diffusion tests were conducted on sera from both kids using the two distinct antigens of CAE. A strong positive reaction to Antigen I occurred to both sera and one of two reacted positively to Antigen II. The possibility of colostral transfer of doe antibodies cannot be discarded in the interpretation of these results. (David Obendorf).