THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Regional Veterinary Laboratory.
Private Mail Bag. Wagga Wagga. N.S.W. 2650
069 230920

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OFFICE BEARERS 1986-87

ELECTED EXECUTIVE.

PRESIDENT:	Dr. John Glastonbury, BVSc., MVSc.. MVS.	*	(069 230929)
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Minutes A.S.V.P. Annual General Meeting. Brisbane, 18th May, 1986.

Minutes of the 3rd Annual General Meeting held in the Veterinary School, University of Queensland, Sunday 18th May, 1986, at 8.30

1) Present

J. Glastonbury (President), J. Allen, J. Boulton, C. Bourke, K. Cook, T. Ellis, W. Hartley, S. Hum, R. Jones, W. Kelly, P. Ladds, I. Links, L. Melville, D. Obendorf, P. Phillips, R. Rahaley, W. Robinson, R. Rogers. T. Rothwell, W. de Saram. J. Searson, L. Stephens, R. Sutton, F. Trueman, K. Walker. D. Williams, R.Webb, R. Whittington.

2) Apologies

R. Campbell, M. Carrigan. R. Badman, A. Fahy. E. Gardner, R. Giesecke, J. Howell. C. Huxtable, R. Mason. B. Munday, D. Pass, J. Seaman, L. Sims, V. Tham.

3) Minutes of the 2nd A.G.H. held In Melbourne, Sunday, 19th May, 1985, accepted as a true record. (Links/Rahaley)

4) Business arising from Minutes - none.

5) Presidents Report
Moved that the report be accepted (Glastonbury/Ross). Carried.

6) Treasurers Report
Moved that the report be accepted (Whittington/Phillips). Carried.

7) Membership Report (Secretary)
Current membership is 156 (125 in Australia, 21 in North America, 6 in Asia, 2 in New Zealand, 2 in Europe). Moved that the report be accepted (Links/Robinson). Carried.

8) Elections

President:	J. Glastonbury
Secretary:	I. Links
Treasurer:	R. Whittington
Committee:	J. Searson
Committee:	R. Cooke

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

9) General Correspondence

a) Postgraduate Refresher Course "Through the Naked Eye the gross pathology of domestic animals". Correspondence with the Director, Post Graduate Foundation and related correspondence in organising speakers.

b) Establishment of the Australian Registry of Veterinary Pathology. The President read excerpts from correspondence regarding applications for funding from the McGarvie Smith Trust, Rural Credits and the Animal Health Committee. Applications to date unsuccessful but not final.

c) Organisation of the Annual Conference, Brisbane, May 1986.

Moved that General Correspondence be accepted (Glastonbury/Searson) Carried.

10) Membership Fee

Moved that it remain at $10 (Whittington/Stephens) Carried.

11) Veterinary Pathology Report/State Representatives

Current format acceptable. Owen Williams resigned as representative for Northern Territory. Meeting expresses appreciation for his contribution. Lorna Melville elected new representative for the Northern Territory.

12) Membership Fee for Overseas Members

Moved that overseas members have a choice of sea mail ($10/year) or air mail ($15/year) subscription (Ladds/Rahaley) Carried.

13) Slide of the Month

Suggestion that slides be labelled with the month of issue.

14) Australian Registry of Veterinary Pathology

Hartley spoke on the cost of establishment with initial costs approximately $20,000 1st year. $15,000 succeeding 2-3 years. Subsequently would fit in with day-to-day work of diagnostic laboratory at Macarthur Institute with minimal cost. Currently on hand 5000 photographic slides, 1000's of tissue blocks. Hartley located at Taronga Park Zoo for next couple of years. He appreciates the enthusiastic support for the Wildlife and non-Domestic Animal Registry. 12000-15000 sections examined in first 6 months including 200 macropod/250 native bird submissions. Currently 1200 zoo animal submissions (500 sections) in registry.

Material from exotic diseases - no satisfactory collection in Australia. Liaise with P. Hooper, Animal Health Lab (Geelong). Seek assistance of Australian Agricultural Health and Quarantine Service (Ladds). Training courses on exotic diseases (Ross).

- benefits - accessible (East Coast, proximity to Camden campus of Univ. of Sydney), reference collection, teaching collection, exchange sets, histological quality control for all veterinary laboratories in Australia, proper cataloguing of pathological entities, benefits to specific animal industries. Similar role as Armed Forces Institute of Pathology (Searson/Ladds/Walker)

- control to be vested in the A.S.V.P. and payments of staff through A.S.V.P. (Walker).

President to write to Principal Laboratory Officers from each state outlining details of registry, estimated cost and envisaged benefits (Rahaley, Obendorf, Rothwell). Veterinary pathologists to lobby their stated representatives on PLO subcommittee.

President to approach private companies utilising laboratory facilities for financial support (Hartley).

15) Post Graduate Refresher Course "Through the Naked Eye - the gross pathology of domestic animals".

- Videos of post-mortem technique for major animal species:
  President to approach Director PGF for provision of viewing facilities during lunch breaks. Low cost video currently available, or to be made by interested parties, (D. Williams/S. Friend - sheep. A. Ross - Vetlink. N.Z.)

- Colour atlas of slides - not pursued by Director PGF because of coat (Hartley)

- Kodachrome exchange - requests for slides to illustrate talks to be co-ordinated by J. Glastonbury.

- Kodachrome collection - Every speaker to provide duplicates of slides from all talks for inclusion in the pathology registry (Ladds). Currently 5,000 transparencies of gross pathology available for registry (Hartley).

- Session on submission of specimens to the laboratory is essential. President to approach Director P.G.F. (Rothwell).

- Moved that copying of slides, videos of post-mortem technique and session on specimen submission be pursued (Walker/Searson) - Carried.

16) Crest and Motto for A.S.V.P.

A suitable price will be awarded for the best entry in a competition to select a crest and motto. The competition is to be judged by the committee of the A.S.V.P. with the assistance of a reputable artistic adviser(s). Entries to be submitted to J. Glastonbury, Regional Vet. Lab., Wagga 2650. Judging to be finalised by the A.G.M.. 1987.

17) General Business

Establishment of a professional body for laboratory diagnosticians including veterinarians, scientific officers and technical officers - (Rogers).

- Currently a range of professional bodies based on disciplines.

- Subcommittee of Principal Laboratory Officers doesn't get representation from staff at the workplace.

- Should the A.S.V.P. broaden its role?

- After lively discussion the general consensus of the meeting was that the A.S.V.P. supported the concept of establishment of a separate professional society of veterinary laboratory scientists but does not wish to expand its own role.

- Moved that general business be closed (Links/Searson) - Carried.

18) Next Meeting and Annual Conference

Moved that it be confined to 1 day of general presentations to be held the day before or after the Post Graduate Refresher Course (Glastonbury/Links) - Carried.

19) There being no further business the President declared the meeting closed at 10.00 a.m.

PRESIDENT'S REPORT.

Your Executive commences its second year in office much encouraged by the response to the Annual Conference in Brisbane. Many people contributed to its success. Both our invited guests and our members gave informative and lucid presentations. Roger Kelly's local arrangements were top class and the initiative of John Searson in organising the Trade Exhibit is to be commended.

For those of you who didn't make it to Brisbane, your copy of the proceedings is enclosed with this newsletter. Ian Links' innovative approach to editing has produced a marvellous result.

We succeeded in making a modest profit from the conference. It was extremely well attended for such a distant venue with a total registration of 63, made up of 48 members, 12 students and 3 guests. Ten companies displayed their wares at the Trade Exhibit and being thrilled with the response have indicated a keen desire to participate next year in Sydney. Two of our guests, Chris Bishop and Mark Gorrell, were so impressed with our Society that they have become Associate Members.

This year the activities of the Society will focus upon communication, the Postgraduate Refresher Course and Annual Conference in Sydney on Saturday 23rd May 1987. Organisation for the latter two is well in hand and will be described in greater detail elsewhere in this edition of the "Veterinary Pathology Report".

I see the fostering of communication between Veterinary Pathologists as the major aim of our Society. Three vehicles for the exchange of information are provided. These are the "Veterinary Pathology Report". the Microscopic Pathology Club and our Annual Conference. The mainstays of the "Veterinary Pathology Report" are our State Correspondents who all do an excellent job. However they largely depend upon individual members to provide then with information. Please support them in their endeavours by readily passing on information regarding interesting cases, projects, research findings and even gossip. In addition if you would like to contribute a short communication, solicit material for a research project or advertise a vacant position please feel free to provide details directly to Ian Links for inclusion in our newsletter. To date we have received no comments regarding "The Slides of The Month". Surely you don't agree with all the diagnoses, be controversial and stand up for your views by contributing them to the "Veterinary Pathology Report". In short keep writing and talking. We need to hear from you.

EXAMINATIONS FOR ENTRY INTO THE AUSTRALIAN COLLEGE OF VETERINARY SCIENTISTS.

Applications to sit for the membership examination in 1987 close on the 1st September 1986. The written papers will be taken in your own centre on the 27th January 1987 and the oral/practical examinations will be held on the 16th & 17th February 1987 in New Zealand and on the 19th & 20th February 1987 in Australia. The fee to undertake these examinations is $200:00.

If you have not yet taken the plunge I would implore you to give these examinations your serious consideration. It is a wonderful exercise in self-discipline allowing you to bring yourself up-to-date with the latest findings in Veterinary Pathology. The Pathobiology Chapter can only be strengthened by an increased membership of experienced practising pathologists. If you have any queries regarding these examinations please feel free to contact John Glastonbury.

For the membership examination in 1988 and beyond the College has decided that candidates must register their intent. At this time a fee of $50.00 is required and the Chapter will appoint a mentor to guide and counsel you during your preparations for the examination. The Chapter has indicated that they intend to refund the registration fee in situations where the expenses of the mentor are minimal. With our good spread of qualified pathologist throughout the country this should prove to be the case n most instances.

Applications for the fellowship examinations in 1987 have now closed. All enquiries regarding fellowship examinations would be best directed to the Chief Examiner (Professor C.R. Bellenger). Australian College of Veterinary Scientists, P. 0. Box 34, Indooroopilly, Queensland, 4068.

THE REGISTRY OF IMMUNOPATHOLOGICAL AND CYTOCHEMICAL TECHNIQUES.

At our recent Annual Conference it was decided that the Australian Society for Veterinary Pathology should establish the above registry. Our committee man, Roger Cook, and the staff of the Regional Veterinary Laboratory Wollongbar have kindly agreed to undertake this important task for us.

To establish this registry could you please supply to Roger the following details:

What your technique is.
The tissues upon which it is used and the required methods of fixation.
The diseases or structures being investigated.
An outline of the procedures used.

This information should be forwarded to the Officer-in-Charge, Regional Veterinary Laboratory, Bruxner Highway, Wo1longbar, N.S.W. 2480.

"THROUGH THE NAKED EYE - THE GROSS PATHOLOGY OF DOMESTIC ANIMALS".

There has been a slight hiccup regarding our organisation for the above refresher course. Regrettably the Australian Veterinary Association attempted to hold their Annual General Meeting for 1987 in Launceston during the same week as the refresher course. However Tom Hungerford has succeeded in convincing them of the error of their ways and our refresher course will definitely be held in Sydney from the 18th-22nd May 1987.

In addition I have had some success with Tom Hungerford in regard to the points raised about the refresher course at our Annual Conference. The Poet-Graduate Committee will be happy to make available a venue for the display of video films depicting post-mortem techniques during the lunch hour sessions. Therefore it is imperative that you let John Glastonbury at the Regional Veterinary Laboratory, Private Mail Bag, Wagga Wagga, N.S.W. 2650, know as soon as possible if you have such a film which would be suitable for the course.

Could you please supply us with the following details of such films:

Title.
Length of film·
The format in which the film is recorded.
The address and telephone number of the supplier of the film.

These films will prove to be a vital adjunct to the more formal aspects of our course so please volunteer your productions as soon as possible.

The Post-Graduate Committee was not prepared to alter the program to allow for a session on the submission of specimens to the laboratory. However a compromise was reached when it was decided that comprehensive notes on this subject would be included in the proceedings. Terry Rothwell has been approached to prepare these notes. Other speakers should also keep this aspect in mind during the preparation of their notes in order that points regarding the submission of specimens to the laboratory can be covered in the sections on individual diseases or body systems.

Could those speakers who need Kodachrome slides for their talks please advise John Glastonbury of their requirements. We will include a list in the next Vet. Path. Report so that the haves and have nots can get together.

To ensure that this course is a success we would appreciate you encouraging your colleagues to attend in Sydney from the 18th-22nd May 1987.

1987 ANNUAL CONFERENCE OF THE AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY.

Our Annual Conference for 1987 will be held in Sydney on Saturday 23rd May 1987 which is the day immediately following the Post-Graduate Refresher Course. Note this date in your diary and begin to make plans to attend and contribute now.

Preliminary arrangements are well in hand with Tony Ross kindly undertaking to make the local arrangements. The J. D. Stewart Lecture Theatre has been booked and a venue for the Trade Exhibit arranged. We intend to have a dinner on that evening, possibly with a financial contribution from Bio-Scientific.

The format for the scientific session will be similar to that for the second day of our previous conferences. Presentations could include interesting cases, descriptions of unusual pathological findings, the results of research projects and any other relevant material. Time slips by rapidly so you should be gathering your thoughts together for possible presentations in the near future. A stronger demand for presentations and the format for typing of abstracts will be included in the next edition of the "Veterinary Pathology Report".

THE AUSTRALIAN REGISTRY OF VETERINARY PATHOLOGY.

Negotiations for the establishment of this registry are proceeding slowly. Money is required to provide Bill Hartley's salary and consumables for the registry until such time as it is transferred from Taronga Park to the John Macarthur Agricultural Institute at Camden Park.

Malcolm Smeal, the Director of Animal Health Research in N.S.W., presented our case at a recent Laboratory Leaders' meeting held in Perth. His proposals met with a mixed but largely favourable response. As a consequence of this meeting we are to prepare a more detailed proposal with accurate costings and advantages of the registry for presentation at the next Laboratory Leader meeting. If our proposal survives this hurdle it will then be presented to the Animal Health Committee with a request for definite financial support from each of the States. At this stage you can assist the cause by extolling the virtues of such a registry to the principal Laboratory Officer in your State.

Aims/Industry Significance of Project:

The aim is to establish an Australian Registry of Veterinary Pathology containing good examples of all disease conditions which affect domestic animals in Australia.

Points of significance of this Registry to the Livestock Industries are:-

1. A rapid and accurate reference source will be available to all Veterinary Pathologist allowing:

    a. a definitive diagnosis to be given to owners quickly,
    b. delineation of new, unusual or emerging conditions,
    c. identification of exotic diseases.

2. An ideal avenue for the training of Veterinary Pathologists through:

    a. courses conducted at the Registry by Dr. Hartley,
    b. individual visits to the Registry to discuss problems with Dr. Hartley or study relevant material in the Registry.
    c. distribution of "study-sets" of slides from the Registry to various laboratories throughout Australia.

3. Availability of excellent material for use as lecturing and teaching aids.

4. Accurate documentation of all diseases affecting domestic livestock in Australia.

5. Quality control over the preparation of histological sections and staining procedures at the Nation's Veterinary Laboratories.

To ensure effective use of the Registry Dr. Hartley will:

1. offer a consultative service to Veterinary Pathologists throughout Australia,

2. prepare study-sets of slides for circulation to Veterinary Laboratories,

3. visit Veterinary Laboratories to conduct seminars and training programs,

4. organise and conduct short courses and seminars at the Registry,

5. distribute material from the Registry for use as teaching aids,

6. maintain a system of quality control over the preparation of histological sections and staining procedures which will be available to Laboratory Technicians throughout the country.

Also Bill Hartley intends to approach various private organisations for financial contributions. He will concentrate his attention on those companies which made a commitment to the non-Domestic Animal Pathology Registry being established at Taronga Park.

If we are successful along either of these avenues a fresh approach will be made to the McGarvie-Smith Trust. They have indicated that they would be interested in giving financial support provided that we could prove that the ultimate users of the registry were a1so contributing.

MEMBERSHIP SUBSCRIPTIONS

The membership fee is still $10.00 for those resident in Australia. For those overseas you have s choice of a $10 sea mail or $15 airmail membership. Please fill in the invoice attached to the bottom of the application for membership form. Those members who attended the Boat Cruise at the Annual General Meeting will be credited with the 1986-87 membership covered by the $10 refund. Receipts are included with the Vet. Path. Report.

NEW MEMBERS

Under our constitution, new members must be nominated and seconded by current members. An application form detailing membership criteria is attached. Please make new members of staff at your institution aware of the ASVP and encourage them to join. We currently have only two members in New Zealand. We believe that the A.S.V.P. has a lot to offer our colleagues across the Tasman and would welcome new members from that direction.

STATE REPORTS.

NEWS FROM VICTORIA - S. FRIEND. (May, 1986)

Bendigo

R.V.L. - Bob Jones.

Swine Dysentery.

An upsurge in swine dysentery diagnoses has occurred over the past 6 Months. During the first 8 months of 1965 there were 3 confirmed cases. In the subsequent 8 months, 8 separate properties, several of them well managed "closed" intensive piggeries have experienced problems with this disease. The reason for this apparent increase is yet to be determined. Diagnosis has been based on the gross post mortem findings of colitis along with culture of beta haemolytic treponemes from the large intestine, and has been facilitated by the submission of freshly dead, or killed pigs.

Mareks Disease

Mareks disease continues to be a problem where no vaccination is carried out. Three live 16-week-old chickens submitted from Marong were some of the 10 affected out of 450 at risk.

The gross, lesions seen were enlarged mottled livers, enlarged spleen and ovaries. Histological changes included the full range of Mareks lesions with lymphocyte invasion of liver, spleen, ovary, brain and nerves.

A more unusual manifestation of Mareks disease was seen on a property at Strathfieldsaye. Seven birds out of 8000 37-week-old layers were affected with swollen pale combs. One bird was exhibiting ataxia and torticollis. Cellulitis of the comb invoking initial fears of Fowl Plague was the provisional diagnosis. This was proved wrong by the massive lymphocyte invasion of the comb, and involvement of the brain, spleen, liver and kidney.

Vaccination failure in this flock is being investigated.

Tick Paralysis - a reality

In March there were 5 submissions of poultry where there was tick infestation (Argus persicus). One such case submitted from St Arnaud was typical of all cases. Three out of 24 fowls had died and 6 remained affected. The fowl received was unable to walk and had laboured breathing, but was otherwise bright and alert. According to the owner 5 other fowls were similarly affected and first showed leg weakness, followed 48 hours later by laboured breathing, and died 2-3 days later.

At autopsy no gross lesions were found except for heavy tick burden. No histological lesions were found and no Borellia anserina were demonstrated. The bird was not anaemic.

With subsequent submissions, all similar history and clinical signs and lack of any demonstrable pathological changes, we are now of the opinion that Argas is capable of causing tick paralysis in fowls. A recent issue of the University of Sydney Post Graduate Foundation Control and Therapy mentioned similar cases in Central Western N.S.W. Even an old poultry vet like Tom H. had to be convinced before it could be published.

V.R.I. - John Finney

Transfer of disease between poultry and pigeons.

The 1985 virulent avian influence outbreak in Central Victoria was a strong reminder that commercial poultry should not have direct or indirect contact with wild birds or their excreta. The contamination of feed ingredients at Liverpool docks in U.K. 1984, by faeces from pigeons infected with Newcastle Disease was associated with the spread of virulent Newcastle Disease to poultry flocks via prepared feed-stuffs (Alexander et al 1984 Vet Rec. 115: 601). In recent times, we have isolated two poultry pathogens from pigeons; Mycoplasma synoviae (Reece et al 1986 AVJ 63: 166) and infectious bronchitis virus serotype B (DA Barr unpublished data).

These episodes indicate that poultry and pigeons do intermingle and the potential for the transfer of pathogens is important.

Fortunately, the racing pigeons of Australia do not appear to be infected with virulent Newcastle Disease or Avian Influenza. However, adequate wild-bird proofing of commercial poultry farms should be carried out.

Mycoplasmosis in racing pigeons.

Pigeons must be in good health in order to endure long distance races. We have investigated the disease status of quite a number of racing pigeon lofts which have had a history of poor performance, excessive non-return rates from races and respiratory distress. Unlike chlamydiosis, salmonellosis, trlchomoniasis or Pigeon herpes virus, there was little or no morbidity or mortality in the young stock or breeders. Pigeons which had returned from a race in the previous day or so were usually presented with mild respiratory distress. Some also had conjunctivitis. Necropsy usually revealed mucoid tracheitis with or without some air sacculitis. Mycoplasma columborale and M. columbinum were commonly isolated from these lesions. Their significance in that respiratory disease syndrome is not known.

However, the response of these flocks to tetracycline and tylosin therapy was remarkable. Some other mycoplasmas, including M. synoviae were also isolated from some affected pigeons.

R.L. Reece, L. Ireland and P.C. Scott (1986) AVJ 63: 166.

BAIRNSDALE R.V.L. - Rob Seiler

POOR WOUND HEALING WITH VICRYL SUTURE MATERIAL

Samples were submitted from the edges of poor healing, or dehiscent laparotomy wounds from goats following ovum transplant surgery. Many animals were affected with reactions ranging from excessive swelling to herniation of intestine. Some animals were lost because of this response.

Histology of 3-week-old lesions showed a profound granulomatous response (with many giant cells) associated with the presence of suture material in the tissue. In one case a necrotic segment of small intestine was embedded in the centre of the lesion, and surrounded by organising granulation tissue.

It is not possible to infer that the strength of the wounds was less than expected, or that the suture material had broken down prematurely. However, there was a profound cellular response to the material and this was not anticipated with a suture that is supposedly absorbed by hydrolysis with minimal tissue reaction. Whether this reaction was due to an abnormal response by the goats, or to a defective batch of vicryl is not yet determined. In the meantime, a little caution is suggested in the use of vicryl for this purpose.

ACORN POISONING IN CALVES

Outbreaks of acorn poisoning in cattle were confirmed on 3 South Gippsland properties during April. Cattle affected have been dairy replacement or crossbred heifers 6 to 18 months old and had been run in paddocks containing English Oaks for 2 to 3 weeks prior to the onset of disease.

Usual signs are depression, anorexia, nasal discharge and diarrhoea which may become bloody. Marked elevation of serum urea (50 to 86mM) and creatinine levels (1.16 to 1.90 mM) were consistently present. Necropsies revealed variable perirenal oedema, pallor of kidneys, colonic haemorrhage and uraemic erosions of the GI tract. Pre-mortem platelet reduction and marked tissue petechiation was present in some calves. A distinctive, subacute, severe renal tubular necrosis with interstitial fibrosis was evident on histological examination.

The toxin responsible for acorn poisoning is not known. Tannins are present in acorns but are unlikely to cause significant nephrotoxicity. A blue-green mould (Penillium sp) found beneath the husks of acorns on all three properties is being tested for production of nephrotoxins.

CITRULLINAEMIA IN A NEONATAL CALF

A 5 day old Friesian calf was reported to be wandering aimlessly, walking through fences and appeared to be blind. Within 6 hours the calf was in lateral recumbency with severe muscular tremors, but no nystagmus end normal pupillary light reflex. The calf was killed end histological examination of the brain, liver and kidney revealed no abnormalities. Blood forwarded to the Glenfield Veterinary Laboratory, however, was tested by Peter Healy and Peter Harper end found to have a plasma citrulline level (2.7 mM) approximately 40 times the normal range for calves (0.06 to 0.02 nM)

Citrullinaemia is an inborn error of amino acid metabolism resulting in an excessive amount of citrulline in the blood, urine and cerebrospinal fluid and an associated urea intoxication. The disease is well recognised in human medicine but this is only the second confirmed case in calves in Australia. It is likely that at least some of the previously undiagnosed cases of neurological disease in Friesian calves seen in Gippsland have been cases of citrullinaemia.

HAMILTON R.V.L. - Cor Lenghaus

DEATHS OF LITTLE PENGUINS (EUDYPTULA MINOR)

During March-April 1986 many thousands of dead and dying Little (or Fairy) Penguins were washed ashore along 50 km of beach in Southwest Victoria. These must have been migratory birds, as local penguin colonies are quite small. Only a handful of banded birds were recovered.

Birds submitted to the laboratory were emaciated and had markedly swollen livers. Most had massive intra-abdominal haemorrhage following liver rupture. Sectioning the liver revealed only a thin rim of normal tissue. The remainder consisted of irregular, small channels and tracts within "chewed-up" tissue, from which hundreds of small trematodes, 2-4 mm long, could readily be expressed.

Within the glandular stomach (equivalent to the avian proventriculus, although these penguins didn't have a discrete gizzard) there were large numbers (50 to 100's) of stout, round worms 1-3 cm long, either free in the lumen mixed with host blood, or partly embedded in the thickened, ulcerated stomach wall.

The intestinal tract did not contain any food, but was choked with cestodes up to 5cm long.

Histologically there was massive disruption to the normal hepatic architecture, with trematodes present singly or in multiples, in dilated bile ducts or free in the hepatic parenchyma surrounded by maturing host connective tissue. There was marked bile ductular hyperplasia and moderate heterophil and mononuclear cell response. In the stomach there were craterous ulcers with nematodes embedded in them. Some lesions had penetrated deeply into the muscle layers, causing a localised peritonitis.

There was minimal reaction to the cestodes present in the intestinal tract.

The trematodes were identified as Mawsonotrema eudyptulae, the nematodes as Contracaecum spiculigerum and the cestodes as Tetrabothria sp. While Contracaecum may have a direct life-cycle the other parasites are more likely to follow indirect life-cycles, possibly using pilchards and anchovies (the penguin's main food source) as intermediate hosts.

Other points of general interest from Southwest Victoria include the first local diagnosis of Johne's Disease in goats, an outbreak of Brucellosis in cattle, widespread fascioliasis of sheep and also perennial ryegrass staggers in sheep throughout the district in autumn. The latter conditions in sheep usually only occur sporadically here. On a more esoteric note, we diagnosed Arizona sp. infection in a taipan and examined a dolphin which presumably died from old age!

BENALLA R.V.L. - Geoff Mitchell.

TUBERCULOSIS/BRUCELLOSIS BREAKDOWNS

Within the past month there have been 3 confirmed cases of tuberculosis in cattle herds in the Goulburn Valley. This has so far resulted in the depopulation of one herd and the testing of 32 contact herds to date. The tracing of possible contact has yet to be completed and it is anticipated many more herds will have to be tested. This activity has stretched resources of the remaining V.P.S. staff to the limit.

During May, Bovine Brucellosis was confirmed on 2 properties in the Ballarat area and one in Gippsland. Both Ballarat herds have been depopulated. Abortions are known to have occurred in one herd since February but were not reported by the owner. The infection was detected following testing of cull cows from one of the properties at an abattoir.

The recent breakdowns of both T.B and Brucellosis in Victoria should serve as a salutary lesson that the eradication programs for these diseases are not yet completed. Whilst investigations are not yet finalized the monitoring systems which are in place appear to have detected these infections before the disease spread to other herds. It is not believed that these breakdowns have jeopardized the state's target of freedom from these diseases in 1987.

Veterinary practitioners should be aware that these diseases are still present in the state's cattle herd albeit at a very low level, and not delete them from their list of differential diagnoses. They should promptly advise their local district veterinary officer whenever they have reason to suspect the presence of TB or Brucellosis in a client's herd.

HYPOMAGNESAEMIA - GRASS TETANY:

The first clinical cases of Grass Tetany have occurred (20 May 1986) for this season. Surveillance of high risk animals by field services and laboratory staff is in progress.

MYOPATHY IN SHEEP:

Acute skeletal muscle necrosis was the significant finding in paretic weaner lambs from two farms. Muscle pallor was evident grossly, along with serous fluid accumulation in fascial planes. The one lamb necropsied from a third farm had chalky white lesions in the heart, with fibrosis and mineralization apparent histologically. As glutathione peroxidase levels were within the normal range from lambs on all three farms, vitamin E deficiency was considered probable.

LUPINOSIS IN A DONKEY:

Two donkeys died out of a group of twenty grazing wheat and lupin stubbles. Clinical signs noted were depression and apparent blindness. At necropsy the liver was small, firm and friable. Histologically, the hepatic cords were disordered with groups of degenerate or hypertrophied hepatocytes. Moderate biliary hyperplasia was also present. The diffuse renal tubular fatty change was consistent with lupinosis. However, there were also a few oxalate crystals in tubules as well as bacterial colonies and neutrophils in a few glomeruli. Lupinosis was considered to be the underlying problem; with hepatic encephalopathy and a terminal septicaemia.

PORCINE POLYSEROSITIS OUTBREAK:

Suckers, weaners and sows died after a short febrile illness usually with ataxia or convulsions. Lesions included pleuritis, synovitis and meningitis. Haemophilus parasuis was eventually isolated when an untreated pig was submitted to the laboratory. Polyserositis or Glassar's Disease is caused by either Haemophilus suis or Haemophilus parasuis two very similar organisms differing in their growth requirements for X factor.

BOVINE SPOROZOAN ABORTIONS:

A dozen cows slipped calves of varying ages and condition within a couple of weeks of each other. Multifocal gliosis and necrosis was seen in the one foetal brain examined, and many organisms resembling Sarcocystis sp. were seen in a variety of organs from this and another foetus.

A single abortion from a different farm had similar organisms in the lung. The brain was not examined.

The identity and life cycle of this putative sporozoan is not known. Dog and cat faeces from the first property did not contain oocysts.

SALMONELLOSIS IN OTHER SPECIES:

The usual autumn break cases of salmonellosis have occurred this season with the disease being diagnosed in calves and Salmonella sp organisms being isolated from aborted foetuses.

CANINE AUTOIMMUNE HAEMOLYTIC ANAEMIA:

A dog with regenerative anaemia and thrombocytopaenia was found to be positive on both the direct and indirect haemaglutination tests. Antinuclear antibody was not detected.

PERSONAL:

Dr. Robert Rahaley is at Purdue University on long service leave until March, 1987.

UNIVERSITY OF MELBOURNE V.C.C. - J. Spillman

ANTHRAX IN A DOG

A five month old male Collie in breeding kennels north of Melbourne died suddenly overnight. The dog had been normal until the day before when it was noticed to be a 'little off colour'. There were many dogs on the property. This dog had never been vaccinated. One litter mate had died in the same way two months ago.

On gross examination, the carcass was in good nutritional condition but was extremely autolysed. Pharyngeal tonsils were enlarged and erythrematous. The lungs were congested and oedematous and there was an excess of serosanguinous pleural fluid. No oedema was present over the head or in the pharyngeal region.

Histologically, the lesions were characteristic for anthrax. There were areas of necrosis through the liver, splenic follicles, mediastinal lymph nodes and tonsils. There was marked reactivity of lymphoid tissue. Thrombosis of vessels was evident at the adrenal corticomedullary junction. A vasculitis was evident in the subcutis adjacent to the external nares but no oedema was present. A fibrinous pleuritis was present. Large, square-ended Gram positive bacilli were in large numbers through the pleura and lungs and present in other tissues.

Lungs, pleural fluid and tonsils were submitted for routine bacteriology but B.anthracis was not cultured. Old methylene blue stains of smears from these tissues did not reveal the characteristic pink staining capsules. These negative results can be explained by degeneration of the bacillus due to autolysis and overgrowth of other encapsulated bacilli.

The Department of Agriculture was notified of our diagnosis and follow up investigations did not reveal any immediate source of infection. However, the property on which the dogs were kept had formerly been owned by a meat-processing enterprise.

WESTERN AUSTRALIA (David Pass, April 1986)

MURDOCH UNIVERSITY

During 1984 and again in the last few weeks, we have seen an apparently new disease in young purebred Persian cats, usually around 6-8 weeks of age. Cats affected have all been well grown and outwardly healthy. There is sudden onset of disease with salivation and episodes of trembling and then collapse. Death can occur within hours of the onset of signs.

Clinically, OP poisoning was strongly suspected but has not been confirmed. At necropsy, the major feature is pulmonary congestion, often with focally variable intensity.

Histologically, there is evidence of acute severe alveolar capillary damage, with fibrin exudation and red cell extravasation. There are also numerous perivascular nodules and cuffs of lymphoid cells and multifocal pneumonitis with septal lymphoid cells and a few neutrophils. About 30% of cats have also had a multifocal non-suppurative myocarditis. To date, we have been unable to demonstrate bacteria, virus or toxoplasma as the cause, but feel it is probably a viral infection.

Has anybody else seen it? (Ref. – Huxtable, C.R. 1986, Proc. Ann Conf. A.S.V.P.: 2.14)

W.A. DEPT. OF AGRICULTURE

Caltrops Poisoning In Sheep and Goats - by Ron Peet

Hepatogenous photosensitisation associated with ingestion of Caltrops (Tribulus terrestris) was diagnosed in sheep and goats on 4 properties in the North Eastern wheat belt area. The field diagnosis with the characteristic "geeldikop" appearance of swollen heads and jaundiced animals was confined in the laboratory by the apparently pathognomic lesions in the livers and kidneys. These were characterised by the variable presence of acicular clefts of varying size in hepatocytes. Kupffer cell nodules, bile ducts and nephrons in the kidneys. This is only the second time the disease has been diagnosed in W.A. (M. Rose and R. Peet 1978 unpublished) and both outbreaks are associated with unseasonal summer rain followed by a hot period wilting the Caltrops.

Reference

Glastonbury J.R.W., Doughty, F.R.. Whittaker, S.J., Sergeant, E.

Australian Veterinary Journal (1984)

61 : 314 – 316

Myopathy in a young Llama – by J. Dickson

A well grown 6 month old female llama died unexpectedly in the Perth Zoo. It was seen to be lying a lot and straining as if to urinate or defecate but it could still get to its feet. In any event when it was removed from the pen to take it to the hospital area, it died. It was noticed that it bad spit marks about the back of the head. These may have been due to the mother harassing it when it was unwilling to rise (U. Gaynor, Zoo Vet). Post mortem examination revealed pale musculature about the colour of the semi tendonosis muscle in sheep. (What is the colour of normal llama muscle?). The muscles were not noticeably wet. The endocardium of the right ventricular wall in particular was creamy white but white plaques were also seen in the left chamber. Histopathology showed a myopathy in both skeletal and cardiac muscles similar to that seen in ovine nutritional myopathy.

Liver and feed selenium and Vitamin E levels are still to be assessed. The feed ration consisted of approximately 80% lucerne hay and lucerne and oaten chaff and 20% of commercially prepared pellets which were supposed to have an increased selenium content of 4ppm and 23ppm Vit E. The previous batches of pellets contained 1ppm selenium.

However, it is of interest that in the Llama medicine workshop proceedings, June 23/24 1984, it is mentioned that dietary levels of 125ppm Se have been tried and that 10ppm Se appears to be acceptable. This suggests that the llama has higher requirements of selenium than is generally known.

This animal was penned with its sire and dam which are about 7-10 years old. They were kept in a small enclosure measuring approximately 20 x 40 feet of irrigated kikuyu grass with access to a stable.

A single lamb had been produced every year for 3 years. The first, a male died after handling sometime later and the second, also a male died with a ruptured bladder, but the cause was not determined. Comments would be welcome.

Reference.:

Llama medicine workshop proceedings, June 23/24, 1984.
Editor M.E. Fowler, Santa Cruz (presumably Zoo), America.

WESTERN AUSTRALIA - Prepared by C Huxtable (June 1986)

ALBANY REGIONAL LABORATORY - (Ruth Reuter)

SUSPECTED ERGOTISM IN CATTLE: For several seasons an unexplained lameness has occurred in a herd of Murray Grey crossbred animals near Albany. The animals show varying degrees of abnormal gait ranging from mild stiffness which disappears on exercise, to reluctance to rise. The most severely affected animals graze on their knees. No other signs of clinical illness are present. There is no evidence of pain, heat or swelling around the joints and the musculature is grossly normal. Various combination· of limbs are affected.

Abattoir specimens of brain, spinal cord, liver, kidney, lymph node and lower limbs have been examined with little reward, with one exception. The horn growth on affected legs has been irregular and the coronary bands appear depressed. Histologically the depressed areas show thrombosis of some small vessels, necrosis of areas of epidermis with acanthosis of adjacent sections and perivascular oedema in the dermal papillae. In two cases the horn had sloughed from one limb.

The paddock on which the animals graze was managed conservatively, resulting in lush growth of a mixture of pasture including ryegrass. A relatively heavy infestation of ergot has been found in these areas, suggesting that this could be a case of low grade ergot toxicity. Any other suggestions are welcome!

CEREBELLAR HYPOPLASIA IN POLLED SHORTHORNS: For the past five years three or four abnormal calves have been produced by a herd of 160 purebred polled shorthorn cows. These animals have shown signs of stiffness in the legs and necks, heads bent to the side, hyperaesthesia and a weak sucking reflex from birth. A number of Angus/Shorthorn crossbred cows on the same property have produced normal calves. A three day old live calf submitted to the laboratory was bright and alert, but unable to stand. The head was turned to the right side and could not be straightened without resistance and evidence of pain. The four legs were rigid and extended. However, the hind legs could be flexed with some effort. The animal was hyperaesthetic to sound and needle pricks.

On post mortem examination, the major finding was cerebellar hypoplasia with complete absence of the vermis and reduction of the folia to fine thread-like folds. Histologically, the sections showed severe reduction in the granular layer and failure of migration of Purkinje cells. The changes appeared similar to those described previously in Queensland (0'Sullivan and McPhee, A.V.J. 51:469-471, 1975). Unfortunately, the possibility of a genetic disorder could not be investigated in this case, since all cows producing defective calves had been removed from the herd, and a new bull introduced after the last breeding season.

BRANHAMELLA OVIS IN GOATS: The current upsurge of interest in the raising of goats for "fun and profit" has led to an increasing number of laboratory submissions from this species. One of the organisms consistently isolated from eye swabs taken from animals with or without a history of clinical disease is Branhamella ovis. This Gram negative diplococcus was previously included in the Neisseriae classification. It is haemolytic on blood agar, grows readily in 24 hours, and has colony morphology similar to Moraxella bovis. It appears to be part of the normal flora of goats in this area.

MURDOCH UNIVERSITY - (Clive Huxtable)

POSSIBLE BOVINE TUBERCULOSIS IN CAPTIVE SEALS: A group of Hew Zealand fur seals (Arctocephalus forsteris and Neophoca cinerea) has been maintained at a marine park north of Perth since 1980. The animals were originally captured in the wild on the Recherche Archipelago off Esperance, WA. Over the last two years, five animals (after necropsy at Murdoch University) have been diagnosed as having mycobacterial infection. It is only recently, however, that the infection has been shown to be due to an organism which has the characteristics of M. bovis. In all cases, there was extensive multifocal granulomatous pneumonia: in one case accompanied by diffuse thoracic serositis, and in the last case by involvement of the liver, spleen and kidneys. At this stage, the source of the infection has not been identified but, as can be imagined, it has provoked a flurry of activity through several State bureaucracies. (As part of the show at the park, the seals came into contact with the public). All surviving seals reacted positively to intra-dermal tuberculin, but no evidence of disease was found in one such animal sacrificed for examination. The survivors are currently under a 60-day quarantine, with their ultimate fate in the hands of the State Health Department.

Anecdotal evidence is accumulating that a tuberculous disease is probably widespread in wild seals and that old-time sealers were frequently infected. This suggests that the organism may not, in fact, be M. bovis, but s closely related strain.

PERIACUTE PNEUMONIA IN GREYHOUNDS: A total of five young adult greyhounds died over a period of three days. Animals were first observed to be stiff and depressed and went rapidly into a state of collapse with death occurring within 24 hours. Terminally, there was marked pyrexia, a degenerative left shift and dyspnoea. At necropsy, large areas of cranial and middle lung lobes were plum red and consolidated. Histologic examination revealed an acute haemorrhagic bronchopneumonia and a Streptococcus was isolated in pure culture. The kennel contained a total of about 20 dogs and was well managed but had been suffering an outbreak of “kennel-cough”. Viral culture was attempted from one animal but with negative results. Acute pneumonia is uncommon in dogs, especially in an outbreak form, and it is difficult to believe that a Streptococcus infection was the primary cause. Any comment· would be appreciated.

QUEENSLAND

University of Qld. - Roger Kelly

Those who remember the equine case of Micronema deletrix nematodiasis, which was presented at the I985 conference as having been put in remission by anthelmintic treatment, will be saddened to learn that the animal had to be killed after recrudescence of the disease. At necropsy, there was invasion and partial destruction of lower lumbar vertebrae, as well as involvement of most lymph nodes in that area.

On reviewing our files, we have noted a disproportionately large number of cases of squamous cell carcinoma of flanks and underline of Dalmatian hounds; bull terriers are also over-represented in this group. The tumors occur in unpigmented skin, are often multiple and are locally very invasive and accompanied by a florid fibrous tissue response. The working hypothesis is that sunlight is a predisposing factor, as these animals often lie on their sides in the sun.

Animal Research Institute - Mike Hill

Infectious bovine rhinotracheitis

A feedlot at Roma had 1200 cattle ranging in age from 5-15 months. Of 326 steers 5-8 months old, 7 had died and 30 showed clinical signs of severe dyspnoea, harsh cough and purulent nasal discharge. They had been introduced to the feedlot approximately 1 month before the first case appeared, and this morbidity and mortality was recorded during the following week.

Significant lesions included severe necrotizing tracheitis and bronchopneumonia both of which had numerous bacterial colonies in the degenerating tissues. IBR serum neutralizing titres were available on 10 animals sampled 10 days apart (unfortunately animal identification was not given). Titres were; negative(x6). 2(x2), and 8(x2) on the first bleed; and negative (x2), 2(x2), 4(x2), 8(x3) and 16(x1) on the second bleed.

Clinical, serological and pathological findings, together with isolation of bovine herpesvirus type (1) from tracheal mucosa, were diagnostic of IBR. The condition was made more severe by secondary infection with Pasteurella sp. Antibiotic feed additive (based on sensitivities to these organisms) prevented further mortality.

Theileriosis

In two instances Theileria buffeli was considered as significant in producing severe anaemia. An aged Santa Gertrudis cow at Warwick displayed anaemia, jaundice and haemoglobinuria of insidious onset. She had been introduced to the property 18 weeks previously. A heavy T. buffeli parasitaemia was seen on smear examination.

Endosulphan Toxicity

Eleven dairy cows at Boonah displayed a sudden onset of trembling, lateral recumbency, paddling and convulsions. One cow died while the others slowly recovered. Endosulphan, a chlorinated hydrocarbon, was found in the liver of the dead cow and also on the recently-consumed pasture, which had been sprayed with Endosulphan 28 days previously. Endosulphan is generally considered a safe insecticide, as it is rapidly broken down by UV light, and has low mammalian toxicity.

Suspected Claviceps sp. poisoning

Two hundred head of breeders and calves were grazed at Wowan on water couch for 6 weeks. Twenty developed muscle twitching, incoordination, salivation and aggressive behaviour. Claviceps sp. was identified in large amounts in the seed heads of the grass. Cattle recovered within 2 weeks of being removed from the pasture.

A. James Cook University - Pathology Section - Phil Ladds

Interesting cases have included diffuse septic peritonitis due to a penetrating foreign body in a saltwater crocodile (Crocodylus porosus) and subcutaneous granuloma in a dog caused by Curvularia sp.

Research in Progress or Commencing

Continuing projects include genital pathology and immunopathology in the ram, immunopathology of ovine aural squamous cell carcinoma - with particular interest in cell-mediated immune mechanisms; diseases of macropods in north Queensland; heavy metal (especially cadmium and nickel) toxicity in prawns; Crotalaria toxicity; Newcastle disease pathogenesis studies (being undertaken in collaboration with pathologists at Bogor, Indonesia); diseases of the crown-of-thorns starfish (Acanthaster planci), and diseases of barramundi (Lates calcifera).

In addition, studies on hatchling mortality in farmed saltwater crocodiles, and diseases of cane toads are commencing.

Preparation of my 'Colour atlas of Lymph node Pathology in Cattle' is now complete and the atlas should be available in August - September. I would like to thank the numerous A.S.V.P. Members who provided me with material for the atlas.

B. Animal Health Station, D.P.I. Oonoonba

1. Reports of interesting cases and outbreaks

Bovine
- Sudden death of 15 cattle occurred close to a goldmine at Ravenswood. Analysis of gut content confirmed cyanide poisoning as the cause but the actual source was not ascertained.
- A metastatic sertoli cell tumour was observed in a medial iliac lymph node of an old bull and was associated with osseous metaplasia in the node. We are unaware of any previous report of such sertoli cell metastases in the bull.

Ovine
- Extremely severe infestation with the nodule worm Oesophagostomum columbianum was the cause of death of five Border Leicester sheep and i11thrift in many others in a group of 50. In some areas nodules were so prevalent that no normal intestinal wall could be seen. Affected sheep also had widely disseminated eosinophilic granuloma due to larval migration.

Goats
- Melioidosis was confirmed in one goat which had aborted and which had abscesses in the spleen and udder. The causative bacteria were isolated from the milk, thus emphasising the potential public health risk of providing goat's milk to invalids.

Pigs
- The false castor oil plant (Datura stramonium) was confirmed as the cause of bloody diarrhoea in several weaner pigs.

Feline
- A further case of gnathostomiasis was observed in a 3 year old cat which died after showing signs of convulsions, diarrhoea, and haemorrhage from the mouth. Necropsy revealed massive pulmonary and pleural haemorrhage and, on microscopic examination, a marked eosinophilic vasculitis of lung vessels was evident (see previous reference, Trueman and Ferris (1977) A.V.J. 53:498).

2. Research in Progress

Continuing and new projects include studies on bovine stephanofilariasis, pathogenesis of non-infectious abortion in cows on the Atherton Tableland, life cycle studies on coccidiosis in calves, and (in collaboration with other laboratories), development and application of a direct immunoperoxidase labellir procedure using an affinity-purified antibody to Mycobacterium bovis for the early identification of cultures.

NEW SOUTH WALES - prepared by Mark Carrigan

Avomec toxicity in cattle

Since the January issue of this Journal the RVL Orange has investigated two further cases of avomec toxicity.

Property A -
The property was visited on the afternoon of 24th May. Two days previously the owner had treated 70 stud Hereford cows and their calves with Avomec. At the time of the visit, 2 calves had died and up to 7 of the calves were showing clinical signs. The cows appeared normal. Mortality and clinical signs were confined to the smaller calves. Clinically, the two worst cases showed unco-ordination and ataxia, were blind, slobbering and one showed fine visceral muscle fasciculation. There was no evidence of tongue paralysis although one of the calves had a slight permanent protrusion of the tongue.

Property B -
Two 2 month old Hereford calves in a mob of 20 cows and calves showed clinical signs 2 days after treatment of the cows and calves with Avomec. One calf was dead and the other was euthanased and autopsied by the attending private practitioner. Clinical signs included blindness, muscle fasciculation, ataxia and slow heart rate. The head was held down, eyes had lateral strabismus and nystagmus, and the tongue was flaccid but not protruding from the mouth.

In both these cases there was no significant necropsy or histopathological findings. It must be noted that in both these cases there is the strong possibility that the young calves were overdosed with Avomec, however, this was definitely not so with the case reported in January. Merck, Sharp and Dohme informed us that 7 similar episodes had been reported/investigated in calves treated with Avomec in South Australia.

Dermatosis vegetans in pigs

Distinctive skin lesions were observed in 4 ex 50 three week old Landrace x Large White piglets on a farm near Orange. Lesions were present over the flanks, abdomen and back and consisted of brownish brittle material with a hard horny surface. This developed cracks and fissures giving an almost papillomatous appearance. There was also marked swelling in the coronary region of all digits and the erythematous surface was covered with yellowish brown greasy material. The walls of the hooves were thickened and had horizontal furrows. The piglets exhibited shallow rapid respiration. Histologically the respiratory dysfunction was due to a characteristic giant-cell pneumonitis.

The epidermis showed rapid irregular growths giving an uneven external surface with great variation in depth and integrity of all layers. Acanthosis and acantholysis were present together with intraepidermal micro-abscessation. There was hyperkeratosis and parakeratosis on the external surface, some keratin pearls in the epidermis and glandular retention cysts in the dermis.

Cameron (1981) indicates that this condition has not been reported in Australia, for a full description refer to Done et al {1967} who believe that dermatosis vegetans is a semi-lethal hereditary disease of pigs due to an autosomal recessive factor originating in the Danish Landrace breed.

Cameron, R.D.A. (1981). University of Sydney Post-Graduate Foundation Proceedings No. 56 - Refresher Course on Pigs. p. 463.

Done, J.T.. Loosmore, R.M. and Saunders, C.N. {1967). Vet. Rec. 80 : 292.

Osteodystrophia fibrosa (secondary) in pigs

This involved illthrift and death of 10% of wearers on a sma11 intensive piggery. Four weaner pigs were necropsied; all were ‘pot bellied’ and low in body condition. All had weak bones and obvious enlargements of the epipyseal plates in the region of the costo-chondral junctions (i.e. ‘rosary bead’ formation). Loss of tone in the smooth muscles of the bowel was associated with excessive bowel dilatation. The ribs of all pigs snap easily under moderate hard pressure and can be readily distorted by light pressure.

Histologically the principle lesions were observed in the ribs. The most dramatic change was tunnelling of the cortex associated with massive numbers of osteoclasts and the presence of replacement fibrosis. Elsewhere the primary spongiosa appeared to have thin trabeculae and there was slight fibrosis of the marrow cavity particularly near the cortices. The physis appeared normal.

The sections of long bone revealed few changes apart from mild osteoporosis.

This condition is due to a deficiency of calcium, a deficiency of Vitamin D or excessive amounts of phosphorus in the diet. It is not uncommon in young, rapidly growing pigs that are fed a grain based diet and kept indoors.

The main characteristics of the lesion are increased numbers of osteoclasts and replacement of the marrow cavity by cellular fibrous tissue and these changes are best observed in the flat bones particularly of the skull and in the ribs. The degree of fibrosis in pigs is usually not great.

Protozoal encephalomyelitis in sheep

Four out of 30 nine month old Poll Dorset lambs exhibited CNS signs. Clinically the sheep were Uncoordinated, had hypermetria of the hind legs and often fell over. At necropsy there were no significant changes.

Histologically there was extensive and severe non-suppurative myelitis and active Wallerian degeneration in major motor tracts of spinal chord. In the brain there was focal non-suppurative encephalitis and meningitis. Changes were severe throughout the spinal chord and less severe in the brain. Changes typical of an infectious cause, possibly protozoa1.

Dr H. Hartley commented: "The pathology in this case is indistinguishable from that reported from protozoan infection – probably not Toxoplasma. I did see a couple of structures, one which may have been early endopolygony and one almost certainly a clump of mature zoites."

Serum had a Toxoplasma IFAT of 16.

Pneumonia in sheep - during April, eight outbreaks of ovine pneumonia, three in adults and five in lambs, were investigated by RVL Wagga. Mortality rates were mostly of the order of 2-3%. Pasteurella haemolytica Type A was responsible for six causing severe fibrinous pleuropneumonia. One outbreak in 8 to 10 week old lambs was found to be due to Pasteurella haemolytica Type T and pathologically the lesions in the lung were more focal and suppurative leading to early abscess formation. Corynebacterium pyogenes was found to be responsible for acute embolic pneumonia in adult rams leading to a 10% mortality rate.

Histophilus ovis - scrotal palpation of rams in a Broucella ovis accredited flock detected three animals with abnormalities. Scrotal contents from two animals were submitted to the laboratory and in each case there were multiple abscesses containing green purulent material throughout one epididymis. Moderate growths of Histophilus ovis were recovered from these abscesses.

Braxy in goats

Diarrhoea leading to death within 48 hours was observed in two out of twelve 6 year old Angora goats. Pathologically severe segmental haemorrhagic erosive/ulcerative inflammation was detected in both the abomasum and colon. Fluorescent antibody staining of sections of these areas of the alimentary tract revealed the presence of extremely large numbers of organisms typical of Clostridium septicum.

TASMANIA - Prepared by David Obendorf

Perennial ryegrass staggers has been widespread and severe throughout Tasmania during the autumn. In many instances severe neurological signs have resulted, in sheep becoming cast in swampy terrain or drowning in dams. Tasmania experienced a mild wet summer in 1985-86 followed by a humid but relatively rain free autumn. These conditions favoured the germination of ryegrass with a high endophyte content. For large areas of pastoral Tasmania, Loliurn perenne represents the predominant if not the 'sole' pasture species.

Little Penguin Mortalities - Every autumn from about March onwards penguins are found washed up on beaches of Victoria and Tasmania. Parasitism and emaciation appear to be the major post mortem findings. As the slides of the months of May 1986 and March 1984 demonstrate, at least two parasites are associated with considerable pathology. The Victorian Ministry for Conservation has instituted a 3 year research program into various aspects of the little penguin biology. These include:

i) An investigation of the areas used by penguins as feeding sites. This will depend on successful development of transmitters/tracking devices, and is to be associated with various transects run from boats (University of Tasmania, FWS).

ii) Studies of breeding, and food taken, at sites other than at Phillip Island (Monash University).

iii) Investigation of correlations between sea surface temperatures and aspects of breeding success or mortalities (Marine Science Laboratory) (MSL).

iv) Pathological examination of penguins to determine causes of death where appropriate. (Karl Harrigan, Department of Veterinary Paraclinical Science, University of Melbourne).

v) Studies of potential food populations (to be conducted by MSL).

If anybody has information to contribute to this study please write to Karl Harrigan.

Personal: Dr Judith Handlinger has joined the veterinary laboratory at Mt. Pleasant.

Organophosphate poisoning in geese - Roy Mason

Nine of 15 free-range geese died as a result of grazing pasture which had been inadvertently sprayed with an OP Insecticide. Apparently the farmer had cleaned out a spray pump, refilled it with insecticide for jetting sheep but failed to fully close the venting tap. When operating the pump some insecticide sprayed out over a patch of grass. The rest is history!

Hemlock (Conium maculatum) poisoning in pigeons - Roy Mason

A pigeon fancier submitted several dead birds for examination. Birds were dying soon after returning to the loft. No gross, histopathological, microbiological or common toxic cause could be determined, however, Conium sp. seeds were found in the crop and proventriculus. Apparently the owner of the birds had been pulling up hemlock C. maculatum from a creek verge and put the plants in a heap for burning. It appears that the birds were feeding on the unripe hemlock seed, probably from these pulled plants and died soon after returning to the loft. Unripe seeds are more toxic than ripe or dried seed.

SOUTH AUSTRALIA - Prepared by P. Phillip·

CENTRAL VETERINARY LABORATORIES - Peter Phillips

MURPHY'S LAW IN OPERATION: A Friesian cow inseminated to a bull suspected of being a carrier of citrullinaemia gave birth to a calf which over 3 days became progressively ataxic and recumbent. We were advised of the calf after lunch on a Friday and raced to the property to collect it to find that it had died before we arrived. It was brought back to the lab as a suspect citrullinaemia case and autopsied. The blood had clotted and very little was collected. The brain was removed and half placed into formalin and the other half frozen. Over the weekend the freezer broke down and enthusiastic staff disposed of all contents on the Monday morning as some had "gone off". The brain, however, was retrieved but the blood was lost.

In the meantime histopathology on the brain revealed pathology consistent with Haemophilus somnus infection. The retrieved, thawed-out brain was sampled for culture but nothing grew. So we had a case that might have been citrullinaemia and was possibly H. somnus!

LIPIDOSIS AND CARDIOMYOPATHY IN IMPORTED TEXAN ANGORA GOATS: (R. Giesecke, E. Gardner)

In September 1984, 69 Angora goats were brought onto the Terrene Island Animal Quarantine Station and placed under intensive husbandry conditions. On the original diet of small lucerne pellets, lucerne chaff and oaten chaff some of the does became excessively fat and their diet was modified in October-November 1985. The bucks remained on the original diet with lupin grain supplementation. Over the following three months some of the does lost weight progressively and became severely depressed and anorectic over a terminal 48 hour period. A ketotic syndrome was suspected and a treatment regime which included propylene glycol, glycerine, antibiotics or corticosteroids, was instituted. Material submitted to the laboratory from non-survivors showed severe generalised fatty infiltration of the liver and kidney. Liver copper levels lay between 2.0 and 3.2mmol/kg and no bacterial pathogens were isolated.

Detailed necropsies were undertaken on a further 4 does and 1 buck which died over a 4 week period. The majority were in good to fat condition with extensive accumulation of fat in the peritoneum. Varying degrees of jaundice accompanied by haemorrhages in the peritoneal fat, pancreas, liver, kidney and heart were seen on necropsy. Livers were pale, fatty and friable and kidneys pale and soft. There was no evidence of fat necrosis. The rumen volume appeared reduced and there was little content present. Rumen and/or urine pH was 6.0 - 6.5 (most animals had been treated prior to death). In one buck a distorted, fibrotic liver, ascites and hydrothorax were seen and in the other a purulent pneumonia was seen on necropsy. Helminth and coccidial populations were not significant and liver copper levels, ranging from 1.1 to 7.8mmol/kg were considered within normal limits. The biological test for Clostridium perfringens Type D toxin was negative in each animal. The most consistent microscopic lesions were again generalised fatty infiltration of the hepatocytes and renal tubular epithelium. Interstitial oedema, haemorrhage and fibrosis were seen in the myocardium of 2 does; a proliferative cardiomyopathy was present in the buck with purulent pneumonia and myocardial fibrosis and atrophy in the atrial walls of the buck with hepatic cirrhosis. The cause of death was considered to be hepatic lipidosis and/or cardiomyopathy resulting from complex nutritional and stress factors in the majority of these goats .

Biochemical analyses were undertaken on a further 6 goats, treated and untreated, from 'at risk' groups. In these, blood glucose ranged from 2.5 to 4.0mmol/L; serum B12 from 250 to 700umol/L and B-hydroxybutyrate from 0.06 - 0.32mmol/L, with no significant differences between treated and untreated goats in this small group. However, blood copper levels bordering on potentially toxic (40umol/L) and narrow Ca:P ratios were seen in some individuals.

Investigations are continuing into the improvement of nutrition in the quarantined goats and no further deaths from the 'ketotic' syndrome have occurred since the addition of lopped branches for 'browse' and the increase of feeding time allowed.

SOUTH EAST REGIONAL VETERINARY LABORATORY - Mike Hindmarsh

WHITE MUSCLE DISEASE: White muscle disease was seen in 6 flocks of sheep on long term stubble-grazing over summer and autumn. Lack of rain meant no growth of volunteer weeds and the only supplement was grain. In one flock deaths occurred. Other animals were lame and had a high plasma LD. All flocks had low (<1 p.p.m.) Vitamin E, while half had adequate selenium.

LATE-SEASON LUPINOSIS: Thirty percent mortality was seen in a flock of ewes close to lambing, where the ewes had access to lupin stubble severely infected with Phomopsis spp. The lupin stubble had been raked into windrows and burnt. Following rain lush lupin seedlings dominated the pasture, however, these were unpalatable to the ewes which sought out the remaining unburned stubble. Lupin stubble, according to local opinion, has an increased palatability when wet.

PORCINE ENCEPHALOMYOCARDITIS (E.M.C): Recently, from a piggery at Kaniva, Vic. which had a history of stillbirths, we found a titre of 1/64 against E.M.C. over a month after farrowing. Tests for parvovirus, leptospirosis toxoplasmosis and brucellosis were negative. In collaboration with the CVL and Hamilton Regional Lab. we are undertaking to confirm the spread of the virus to the area by virology studies and paired serology.

CLAYTON'S T.B.:

Six heifers at Struan Research Centre reacted positively to a routine tuberculin check test. All were found to have no visible lesions when slaughtered. Subsequently when the dams of these heifers were tested all reacted positively to the tuberculin test. It was then realised that both cows and heifers had taken part in a trial of Somatostatin growth regulating hormone. On investigation it has been discovered that Freund's Complete Adjuvant had been incorporated in the trial injection and that the killed Mycobacterium tuberculosis component bad sensitised the reactors to the test. No lymph node cultures were positive for Mycobacteria and a lesion from the injection site of one cow showed giant cells, macrophages and fibro-lymphocytic reaction consistent with the use of Freund's Complete Adjuvant. A further test to establish the longevity of the sensitivity to tuberculin in the treated animals is to be carried out.

From Diagnostic Lab. Services - (Tammy Utteridge)

Disseminated intravascular coagulation: Prolonged prothrombin time, PTTK time, increased fibrin degradation products and thrombocytopenia consistent with D.I.C. were seen in a bitch following a caesarian, and a cat with azotemia and hepatocellular damage.

Hypoadrenocorticism: Seen in a dog whose owner had been diagnosed and treated for leptospirosis. The owner had a history of having accidentally deeply wounded himself, and the dog had licked the bleeding wound. The dog became ill afterwards, and the biochemistry suggested hypoadrenocorticism, which fitted the clinical symptoms. Leptospiral serology {done at the I.M.V.S.) revealed a titre of 1:100 for Leptospira interogans var copenhageni1. The dog died before repeat serology could be done. Leptospirosis has been described as causing hypoadrenocorticis by producing large haemorrhages in the adrenal cortex. However, the source of infection is unclear as according to the I.M.V.S. Leptospira interogans var copenhagenii has not been diagnosed in the occupational group of the owner for some time.

Feline mammary hypertrophy: A case was seen in a young cat which was clinically symptomless apart from the swelling. We see this condition very infrequently, with most ventral abdominal swellings in cats being either chronic active steatitis or mammary adenocarcinomas.

Systemic lupus erythematosus: A positive antinuclear antibody test was obtained in a dog with a history of severe lameness.

NORTHERN TERRITORY - Prepared by L. Melville

BERRIMAH AGRICULTURAL LABORATORY - (L. Melville)

SIXTH BLUETONGUE SEROTYPE FROM NORTHERN AUSTRALIA: A blue tongue virus isolated from sentinel cattle near Darwin has recently been identified as Type 9 by the Bluetongue World Reference Laboratory. Onderstepoort, South Africa. This new identification is the sixth bluetongue serotype found in Northern Australia, the others being types 1, 15, 20, 21 and 23.

The virus was isolated from heparinised blood samples collected from two animals on 28th March 1985 and one animal on 18th April 1985. The primary isolation was in 10 day old embryonated eggs followed by passage into Aedesa albopictus (C6/36) cell culture then passage into BHK 21 and hamster lung cell cultures. After 7 days the normal monolayers were passaged to further mammalian cells and cytopathic effect found.

The viruses were identified as blue tongue by immunofluorescence but were not neutralised by immune rabbit serum to blue tongue types 1, 15, 20, 21 and 23. Further work at AAHL confirmed the viruses were different to the known Australian blue tongue serotypes. After considerable difficulty with the Transport Workers Union the virus and its immune sheep sera were finally consigned to South Africa and identification as Type 9 made.

Type 9 is an African serotype which has been isolated in India where it was responsible for the deaths of Australian sheep imported into that country. Antibody to Type 9 had also been reported in S.E. Asia.

Further work will he carried out to determine the -virulence of Type 9 in sheep in Darwin.

BLOOD FLUKE IN FRESHWATER CROCODILES - (C. Johnston)

Since the establishment of crocodile farms near Darwin, monitoring of hatchling and yearling mortalities has shown parasites to be a major cause of death.

For several months severe generalised histological lesions were found in tissues from freshwater crocodiles at Letaba Croc. Farm. The lesion initially seen was generalised granulomatous inflammation associated with eggs in kidney, lung, liver and spleen. The eggs appeared to resemble fluke eggs and a closer examination of the tissues involved showed the presence of a very small parasite in small capillaries. There appeared to be minimal reaction to the parasite. Careful dissection of fresh tissue in saline and examination under a dissecting microscope finally enabled the entire parasite (about 1mm long) to be found.

Blood flukes have not previously been described in crocodiles so this new parasite is currently being subjected to taxonomic description end naming.

The ponds where the crocodiles were housed were infested with large numbers of snails. These were collected and sent to J. Boray at Glenfield who identified the snails as Amerianna sp. Typical spirorchid cercariae were found in 11% of the snails.

Further work is continuing to try to establish times involved for development of the parasite. It appears that most of the tissue damage occurs from the release of large numbers of eggs which lodge in tissues, rather then from the presence of the adult in small capillaries

NEWS FROM VICTORIA - S. Friend (March 1986)

Bendigo

Foreign body pneumonia in pigs - R.T. Badman

A large commercial pig breeding establishment has a policy of monitoring its claimed enzootic pneumonia-free status by routine slaughter checks of lungs of baconer pigs passing through abattoirs.

In September 1985 lungs were seen with minor degrees of consolidation of the right cardiac lobes and with lesser involvement of left cardiac lobes and apical lobes.

Histopathology was carried out on randomly selected lung sets and a marked giant cell alveolitis and granuloma formation in bronchioles was found. Marked lymphoid cuffing of bronchioles and larger blood vessels was seen. While individual lungs had some features of mycoplasma infection these were not so pronounced to be diagnostic of enzootic pneumonia.

Further lung checks reinforced the uniform involvement of the right cardiac lobes and the foreign body reaction in bronchioles and alveoli. Plant material was found in many granulomata. A small number of lungs had features of Mycoplasma hyopopneumoniae infection - peribronchial lymphoid proliferation, bronchiolar epithelial hyperplasia and macrophage reaction in alveoli.

Fluorescent antibody studies on tissue from a number of affected lungs were carried out at I.M.V.S, Adelaide (Andrew Pointon). Fluorescence was seen against M. flocculare but not to M.hypopneumoniae or M.hyporhinitis.

Foreign body reactions in the lungs of pigs seen at this laboratory are rare whereas presumed Mycoplasma pneumonia is quite common (histopathological diagnosis). The regular occurrence of foreign body reaction in the lungs of pigs from one piggery caused speculation as to the reasons for its presence.

These include upper respiratory tract disease - Atrophic Rhinitis and Inclusion Body Rhinitis, and agents that may have damaged the mucocillary escalator - ammonia and mycoplasmas of low pathogenicity (M.flocculare).

The third possibility that M.hypopneumoniae exists on the property does not fit well with the fact that multiplier herds supplied regularly with stock from this property have had no enzootic pneumonia lesions (slaughter checks).

Any comments you may wish to make would be appreciated.

The Australian Animal Health Laboratory - P. Hooper

The Australian Animal Health Laboratory, our high security laboratory, is now operational and should be working with some exotic diseases from 1986. It will be possible to send out fixed material and slides from infected animals and in a relatively short time, there should be a good range of material available. Emphasis is not often placed on the histopathology of many of these diseases but they often produce distinct lesions useful in diagnosis.

The first good samples of an exotic virus disease received by the laboratory were fixed small intestines from a goat with peste de petits ruminants, the rinderpest-like disease. This goat had developed a remarkable degree of villus atrophy with eosinophilc cytoplasmic inclusion bodies in epithelial cells. Another goat with the same disease examined at Plum Island (U.S.A.) had a remarkable number of intranuclear inclusion bodies.

V.R.I. - John Finney

Recent departures:
Peter Hooper to senior pathology position at Australian Animal Health Laboratory. Mary Barton to academic position in biomedical sciences at Warrnambool Institute of Advanced Education.

The V.R.I. is increasing its capacity in ultrastructural pathology directed to the improved diagnosis and investigation of animal disease (R. Condron).

HAMILTON - Peter Beers

Enzootic ataxia in goats

Progressive posterior ataxia and weakness developed in 5 goat kids aged 9-12 weeks. The goats were running on basalt soil types known to have adequate levels of copper. Mean serum Cu levels of does was 3.6µm and kids 0.9µm (<8µm considered deficient).

Post-mortem, one kid had a liver Cu level of 0.03mmol/kg (<0.8 deficient) and histologically, had demyelinating lesions in the brain and spinal cord. Secondary or conditioned copper deficiency was confirmed by analysis of herbage which contained 7.6mg/kg Cu (dry weight) and 4.1mg/kg Molybdenum. Herbage levels of Cu >6mg/kg are considered adequate, however a Cu:Mo ratio of <5:1 has the potential to induce Cu deficiency, and a ratio of <2:1, as seen in this case, is considered critical. No Mo has been applied to these pastures during the last 4 years.

Mastocytoma

A 6 year old multiparous Aberdeen Angus cow had lost weight and had severe diarrhoea. A large number of "wart-like growths" were present on the udder, hind legs end perineum. A biopsy showed these growths to be a primitive mastocytoma. The animal was slaughtered and the tumour was found disseminated throughout the body. Growths were found in the capsule of the spleen, kidney, lymph nodes, mammary gland, skin, skeletal muscles and the abomasum which weighed 18kg. Many other tissues contained individual or small groups of similar neoplastic cells. According to Moulton mastocytoma is a rare tumour in cattle but almost invariably metastasises widely.

BAIRNSDALE - Steven Hum

Since October Steven Hum has been replacing Steven McOrist while he is in Edinburgh. Dr. Keith McSporran, a New Zealander from the Whangarei Animal Health Laboratory has been here since November and will be finishing up in March. Or. Ian McCausland is going to take up his new position in mid March as Executive Director of the Australian Meat and Livestock Research and Development Corporation in Sydney.

White liver disease (WLD)

WLD occurred on several properties in late spring. The unusually wet season has resulted in rapid pasture growth, and intake of soil (an important source of cobalt) has been reduced by the abundant feed on offer. The liver cobalt values were consistently low and varied from 0.03 to 1.19 um/kg (normal 1.7-6.8 um/kg).

Mainly young fast growing lambs were affected and the clinical signs consisted of illthrift occasionally accompanied by serious ocular discharge and photosensitization. Sometimes apparently healthy lambs were found dead.

At necropsy enlarged pale friable liver was noted. Histologically there was moderate to severe diffuse hepatocellular lipidosis consistent with acute WLD. Biochemical analysis of blood samples from affected flocks revealed low serum B12 levels (<200pp/ml) compatible with low cobalt intake.

Castor oil plant toxicity in an Angora goat

A mature Angora goat in good condition was presented dead at the laboratory with a history of vague gastro-intestinal and respiratory signs over a period of two days. The animal had been presented in extremis by the owner and treated symptomatically but died an hour later. On necropsy foam was visible at the external nares and throughout the respiratory tree. The lungs were moist and heavy. Pulmonary oedema was evident when the lungs were examined histologically along with a mild diffuse granulomatous cardiomyopathy.

On questioning the owner it was established that the goats had browsed on a number of Caster oil shrubs (Ricinus communis) which were growing along a river border in the paddock. Some of the branches had been knocked down by their activities. There was evidence that both foliage and fruit had been eaten. One other goat from the group had died prior to the present case. The toxic principle of this attractive purple/green plant is Ricin, one of the most toxic proteins known. It is concentrated in the seeds but is also present in leaves and stems. Affected animals develop diarrhoea and a 'tumultuous' heartbeat that may be heard a short distance away. Goats are reported to be more resistant to ricin than other domestic liverstock but sudden exposure to the toxin may prove fatal. (K. McSporran)

Abomasal ulcers in calves

There have been several calf deaths lately due to perforation of an abomasal ulcer with resultant peritonitis. These are usually in dairy calves, although affected beef calves have also been seen. Most are young (2-8 weeks) and are on milk replacer diets. Deaths are sudden and necropsy findings are dramatic with a severe, diffuse, fibrinous peritonitis, sometimes with evidence of free ingesta. The ulceration and perforation is usually acute with little evidence of inflammatory changes. Fungi may be demonstrated in the margins of some ulcers, although it is unlikely that fungal infection is the primary cause. Causative factors are unclear, but, the feeding of certain diets (milk replacers, concentrates and coarse roughage) is sometimes associated with multiple cases in a herd.

JOBLINE - Training and employment opportunities for Veterinary Pathologists at home and abroad.

A) UNIVERSITY OF TENNESSEE

INSTRUCTOR - Department of Pathobiology, College of Veterinary Medicine, University of Tennessee, Knoxville, Tennessee has an opening for an instructor in pathology. This is a nontenure-track position of 12 months duration. Candidates should possess a D.V.M. or equivalent degree and have completed either formal training in a residency or graduate program or have documented extensive experience under supervision in anatomic pathology. Candidates should have partially or completely met the eligibility requirements to allow them to sit for the examination for Diplomat, American College of Veterinary Pathologists. The individual in this program will have approximately 15 weeks per year in the necropsy laboratory, and will be responsible for the supervision of that class which involves 4-5 senior students and 1-2 junior residents. Other service responsibilities include biopsy service which is rostered evenly amongst all pathologists, and usually occurs one week in six. The salary is US$28,500.00 per year (approximately equivalent to A$40,000.00) per year. Starting date is July 1, 1966 or as soon as possible after that date. For further information, please contact Dr. M.D. McGavin at the above address, or by phone at area code 615-546-9230 ext, 221.

Applicants should send letters of application, curriculum vitae and the names of three referees to:

Dr. M.D. McGavin
Department of Pathobiology
College of Veterinary Medicine
The University of Tennessee
P.O. Box 1071
Knoxville, Tennessee 37901-1071

The University of Tennessee is an equal opportunity/affirmative action employer.

B) NORTH CAROLINA STATE UNIVERSITY

The School of Veterinary Medicine at North Carolina State University is seeking applications for a graduate student position leading to the M.S. /Ph.P. degree with a major in either Microbiology or Pathology. Applicants should have the D.V.M. or an equivalent degree.

The successful applicant will undertake research on the pathogenesis of bluetongue virus infection of cattle.

The annual stipend for the first year will be $16,800.00. Positions are renewed on an annual basis subject to satisfactory progress. Those interested should contact Dr. N. James MacLachlan, Department of Microbiology, Pathology and Parasitology, Telephone (919) 829-1274 for further information. Application material (Ref: Position # 030} should be obtained through the Office of Research and Graduate Studies, School of Veterinary Medicine, North Carolina State University, Raleigh, NC 27606. Closing date for completed application is November 1.

North Carolina State University is an equal opportunity employer.

CREST AND MOTTO FOR A.S.V.P

The first entry has been received in the competition and is reproduced below. As much as we would like to see Geoff Mitchell win the Gold Medal, we are sure that there are other members out there just dying to show their artistic skills, so keep those entries rolling in!

THE FILTER OF KNOWLEDGE

UNABLE TO ADD DIAGRAM

Mitchell's
Complexes

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

DIARY OF COMING EVENTS

1st September, 1986 - Close of applications to sit membership examinations, Australian College of Veterinary Scientists.

17th-20th September, 1986 - International Meeting of Veterinary Pathology Cordoba, Spain.

27th January, 1987 - Written examination MACVSc.

19th -20th February,1987 - Oral/Practical examination MACVSc.

18th-22nd May, 1987- Post-Graduate Refresher Course in Gross Pathology, University of Sydney.

16th-21st August, 1987 - World Veterinary Congress, Montreal, Canada

24th-28th August 1987-ANZAAS Congress, Townsvi1le, "Science and Life in the Tropics". Contact the Hon. Organising Secretary, ANZAAS Conference, James Cook University, TOWNSVILLE, QLD, 4811.

VET. PATH. REPORT PUBLICATION DATES.

JULY, OCTOBER, JANUARY, APRIL.

ADDRESS LABELS ON MEMBERSHIP RENEWAL FORMS

A) Please advise if address has altered.

B) Red dot in the top left corner indicates that you attended the 1986 AGM of the ASVP and have collected your copy of the proceedings).

C) Black dot indicates that you are credited with $10 refund from the boat cruise at the 1986 AGM which covers your 1986/87 membership subscription - no further action is required to renew your subscription.

D) Blue dot indicates a receipt is included for 1986 AGM/membership payments.

E) Green dot indicates you have not yet paid your 1985/86 membership subscription and it is now overdue.

Please advise if we are incorrect in any of these matters.