# THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
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## PRESIDENT'S REPORT

The Executive is pleased to report that the Australian Society for Veterinary Pathology has continued on an even keel since the last edition of "The Veterinary Pathology Report". Final organisation for the forthcoming Annual Conference and Post-Graduate Refresher Course has been plain sailing whereas some further tacking is still required to bring the Registry to safe waters. Damn that silly America's Cup!

Our Treasurer, Richard Whittington, was delighted with the response to his request to members regarding outstanding subscriptions. At the Annual Conference you will have the pleasant task of deciding how our expected balance of approximately $1800.00 can be best utilised for the betterment of Veterinary Pathology. On the negative side of the ledger a decision will have to be made about the mall percentage of recalcitrant people who have not been financial for longer than two years.

The majority of our members are employed in state veterinary laboratories. A seminar on "The Role of the Veterinary Laboratory in the 90's" organised by Martin Copland at the I.M.V.S. Adelaide on 12 and 13 November 1986 was of vital interest to them. In this issue of "The Veterinary Pathology Report", Terry Nicholls, Director of the Regional Veterinary Laboratory Benalla, gives his impressions of the seminar and thoughts regarding the survival of state laboratories. We would gladly welcome your comments on the subject for future publication in the newsletter. Surely many of you must have firm opinions on the entrepreneurial approach, restriction of services to economic livestock, contract research, charging of livestock producers and many others so please share them with your colleagues.

Recently I have been indirectly involved in the selection of veterinarians for employment in laboratories. One feature that immediately became obvious was a shortage of people with appropriate post-graduate training. With five Veterinary schools in Australia I think that this is a poor situation. There are several possible reasons for the deficiency. Perhaps undergraduates are not being sufficiently encouraged in the direction of “the ultimate truth”. Current post-graduate course work degrees may not be applicable for veterinarians seeking a career in state laboratories. Planning of courses is probably directed more towards the obtaining of cheap labour, which is not necessarily a bad thing because of the experience derived, and maintenance of student quotas and funding. Financial constraints both in terms of intending students and the tertiary institutions are of undoubted importance. Finally our branch of the veterinary profession like other fields of scientific endeavour suffers appreciably from the “brain drain”. Over the years many Australian Veterinary Pathologists have trodden the path to North America, obtained extensive training and experience in their chosen field and in turn have been rewarded with intellectually and financially attractive positions. Why come home?

It is important that we find answers to this problem. A trained Pathologist is at least three to four years more advanced than an inexperienced Veterinarian, being ready to assume responsibilities in the area of applied research and necessitating time-consuming on-the-job training. Veterinary Schools should orientate their course-work type post-graduate degrees more towards what is required out in the market place. It is no doubt inefficient to have the schools competing for the relatively small number of potential students and one “centre of excellence” would serve Australia's needs. For their part State Departments of Agriculture should recognise the work of trained personnel, pay them salaries commensurate with their skills and provide career structures which allow continued pursuit of Veterinary Science. The Australian College of Veterinary Scientists and Australian Registry of Veterinary Pathology have integral parts to play in our continuing education and are all deserving of our full support.

### The Australian Registry of Veterinary Pathology

Plans for the establishment of our Registry appear to be headed in the right direction. Three possible sources of funding are presently being explored and we are quietly confident of success.

At the Animal Health Committee Meeting held during October 1986 somewhat ambiguous support was agreed to by the States. Basically they agreed to financially support the Registry provided that we made approaches to other potential users such as the C.S.I.R.O. and the various veterinary schools. The laboratory leaders from the different states are presently working on a basis for pro-rata funding in response to the A.H.C. decision. On our part we have written to the other possible users suggesting a contribution of $500 but with limited success. To date we have had a positive response from the James Cook University of North Queensland (Graduate School of Tropical Veterinary Science) with a donation of $250. The C.S.I.R.O. and the University of Queensland were very sympathetic to the cause but unable to provide financial support because of financial stringency. We need financial support from the Universities to prevent a catcb-22 situation arising - i.e. the A.H.C. needs to be shown that there is widespread support for the Registry to encourage funding from the States. Now is the time to lobby your faculty or State Department of Agriculture.

Like a bolt out of the blue the N.S.W. Department of Agriculture very recently allocated $5000 to the Registry for the purchase of capital items. Bill Hartley is currently planning the cost efficient use of this allocation. The Society would like to express their gratitude to Mr. George Knowles, Director General of Agriculture in N.S.W. for his continuing generosity.

Finally we have submitted an application to the Rural Credits fund to pay Bill's salary for 2-3 years. This application will be placed before the Reserve Bank under the auspices of the N.S.W. Department of Agriculture and we should hear of the result before the Annual Meeting.

THROUGH THE NAKED EYE - The Gross Pathology of Domestic Animals

Our Post-Graduate Refresher Course to be held at the University of Sydney from 18 to 22 May 1987 is almost upon us. Tom Hungerford assures me that the lecture notes are rolling in and that they are of an extremely high standard. The Foundation has commenced advertising the course in earnest and our members should supplement this at the local level wherever possible. The more veterinarians who realise our true worth as a consequence of this course the better.

Visual aide for the course are still creating some problems. With regard to the preparation of video films depicting post-mortem techniques John Seaman has produced an excellent one for sheep, Roger Kelly has one available for poultry and John Boulton has promised to produce one for pigs. The foundation has gone to a deal of trouble to co-operate with us in this venture by hiring a large screen and a VHS recorder. Please if anyone has available, or would be prepared to produce one on small animals or horses could they notify John Glastonbury at the Regional Veterinary Laboratory, Private Mail Bag. Wagga Wagga N.S.W. 2650 as soon as possible.

Having rushed to meet Tom's deadline for the preparation of notes I am sure that some lecturers may have overlooked the availability of suitable Kodachromes. If anyone is still requiring Kodachromes could they please let John Glastonbury know of their requirements?

1987 Annual Conference of the Australian Society of Veterinary Pathology

We should all be excitedly looking forward to the 1987 Annual Conference which is to be held in the J.D. Stewart Lecture Theatre in the Veterinary School at the University of Sydney on Saturday 23 May 1987.

At this stage we are soliciting contributions from members for the scientific segment of the meeting. As our conference will only be of one day's duration we will only have time for approximately 12-16 presentations from members. They may cover such things as interesting cases and research projects. The presentation will be selected upon the basis of their order of receipt. They should be forwarded to the Secretary, Ian Links, at the Regional Veterinary Laboratory, Private Mail Bag, Wagga Wagga, N.S.W. 2650. The manuscripts should be prepared in the following manner:

* 1-2 pages in length including a brief introduction, methods, conclusions and possibly citing several major references.
* Typed single spaced on A4 paper using Letter Gothic 12 (or Prestige 12 or similar type with carbon ribbon).
* Leave a 3.5 cm margin on the left and 2.5 cm 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 in the proceedings. No corrections or errors please!

Local arrangements are in the capable hands of Tony Ross. Remember that Tony is organising a dinner which will be held on the evening of 22 May 1987. The trade exhibit will be held in conjunction with the Post-Graduate Foundation "Gross Pathology Course" on Thursday 21 and Friday 22 of May in the foyer of the Stephen Roberts Theatre. Tony Ross and John Searson will be assisting with the coordination of the exhibit. The exhibitors have agreed to make a donation to both the Post-Graduate Foundation and the A.S.V.P. so please give them your support.

For accommodation we suggest that you consult Tom Hungerford's advice on the subject which is set out below. As the majority of you will also be attending the Post-Graduate Refresher Course you will merely require one or two nights additional accommodation

Accommodation for the Gross Pathology Course

Please make your own bookings. Don't ask us to do so, when you do it yourself you know you're booked in, exactly where and how. PRICES ARE CURRENT AS AT 1/10/86.

International House, 96 City Road, Chippendale, 2008. Tel: (02) 692 2040 - Single: $45 per night including 3 meals, Twin: $40 per person per night Including 3 meals.

Metro Gateway Motor Inn. 1/9 Meagher St., Chippendale, 2008. Tel: (02) 699 4133 and ask for Gavin or Margaret Reid - Single: $44, Double/twin.- $49 per night.

University Motor Inn. 25 Arundel St., Glebe, 2037. Tel: (02) 660 5777 - Double/twin: $68 per night.

Travelodge Motel, 9-15 Missenden Road, Camperdown, 2050. Tel: (02) 516 1522. - Single or Double: $65 per night.

Wesley College, Uni. Syd., Western Avenue. Tel: (02) 51 5268 or 51 4801 and ask for Wendy Fothergill - fullboard $32, B & B: $24 per day. Lunch $4 and Dinner $4.

St Andrews College, Carillon Avenue, Newtown, 2042. Tel: (02) 51 1449 and ask for Rachel Guest - fullboard: $35, B & B: $25 (cooked breakfast) per day. Lunch $6 and Dinner $7.50.

Women's College. Carillon Avenue, Newtown 2042. Tel: (02) 51 1195 and ask for Manager, Len Cupitt - single/fullboard: $32, B & B: $24. Twin rooms are available on application - Twln/fullboard: $52, B & B: $36.

Suites with your own kitchen

City Gardens, 1 Myrtle St., Chippendale, 2008. Tel: (02) 690 9100 - Studios: $65, Bedrooms: $75 per night per person and $8 for each additional person.

As most of us prefer a spartan existence might I suggest that St. Andrews College would be preferable mainly because of its proximity to the Veterinary School.

Registration forms for the Conference will be included with the next edition of "The Veterinary Pathology Report" which is due to be circulated in April 1987. However commence to make your plans now!

The Next Executive of the Australian Society of Veterinary Pathology

Your present Executive has been honoured to serve you for the past two years but our term expires at the Annual General Meeting on 23 May 1987. We have put out some preliminary feelers and hope to be successful. However you should give this matter your earnest consideration remembering that nominations must be with the Secretary, Ian Links, at least seven days before the Annual meeting.

The Examinations for Entry into the Australian College of Veterinary Scientists

Six of our members sat for the written examination on 27 January 1987 and are no doubt looking forward with keen anticipation to the oral/practical exams on 19 and 20 February 1987. I would like to congratulate these candidates on their endeavour and wish them every success.

Those members who have yet to take the plunge should seriously consider offering themselves for examination in 1988. What better way to celebrate our bicentenary than by undertaking a rigorous program of revision and strengthening an organisation which gives due recognition for expertise?

The Head and Assistant Examiners this year are Phil Ladds and John Glastonbury respectively. Phil had a very poor response from members of the college for Kodachromes to be used during the practical segment of the examination. This is a most important part of the exam and we would be moat grateful if you could give some thought to contributing for next year.

STATE REPORTS

**NORTHERN TERRITORY** - Prepared by L. Melville

BERRIMAH AGRICULTURAL LABORATORY (Lorna Melville)

MORE BLUETONGUE SEROTYPES FROM NORTHERN AUSTRALIA:- 1986 has been a particularly good year for virus activity in the top end. 137 isolations have been made so far this year from sentinel animals. Of these 58 have been identified as blue tongue viruses on immunofluorescence.

Among the blue tongue isolations were a number which were not neutralized by immune rabbit sera to types 1, 9, 15, 20, 21 or 23. These isolates were sent to AAHL where four were identified as Type 3 and fifteen as Type 16 using the plaque inhibition neutralisation test and microneutralization.

The viruses v«r« Isolated from heparinised blood samples collected from ten animals in May and June 1986. The primary isolation was in 10 day old embryonated eggs followed by passage into Aedes albopictus (C6/36) cell culture then passage into BHK21 and hamster lung cell culture.

Type 3 and type 16 are both African serotypes which have been isolated in India. Type 3 it considered very pathogenic in Africa and Type 16 moderately pathogenic. The isolation of these two serotypes brings the number of Australian serotypes to eight.

GREYHOUND TURNIP TOPS – CONTINUED:- The case of greyhounds poisoned by a turnip top stew was described in the previous report. Further work on the stomach contents of these dogs showed the presence of an alkaloid which was also present in the turnip tops. This alkaloid is indistinguishable from strychnine on HPLC. The alkaloid was probably responsible for the hypertrophic tubular epithelium seen in the kidney. The exact chemical nature of this alkaloid is unknown at present.

AFLTOXICOSIS IN CATTLE AND HORSES:- Six cows died and 40 horses showed symptoms of colic after being fed peanut hay. Liver lesions in one cow examined consisted of fatty infiltration and hepatocellular necrosis. Aspergillus flavus was isolated from peanuts in the hay and aflatoxin analysis showed 1500 micrograms per kg.

CLOSTRIDIUM AND MORE CLOSTRIDIUM:- The first rains of the wet season appear to have stimulated the appearance of clostridial disease in a number of places.

Two cows in a group of 20 experimental animals on Berrimah Research Farm died suddenly. Clostridium septicum and Clostridium novyii were isolated from heart blood.

Fifty buffalo in a group of 2000 undergoing export testing for Cubs died over 4 days. Clostridium novyii and Clostridium septicum were isolated from liver and heart blood of four animals examined.

The problem has even extended into a large commercial piggery. A sow died with clinical jaundice following a brief illness. On autopsy the left lobe of the liver was swollen, hard and full of gas while the right lobe appeared soft, yellow and toxic. There was extensive serosanguinous fluid in both thoracic and abdominal cavities. Clostridium novyii was isolated in pure heavy growth from the liver.

**QUEENSLAND**

University of Qld - Roger Kelly

"Chemical & Plant Poisons" : Alan Seawright is at present updating this valuable text, and some of the regional labs have already had visits from him on the trail of new or upgraded information. He is particularly keen to hear from people who can (a) point out errors and omissions in the first edition (b) bring to his notice any examples of new intoxications, preferably we11-documented, which need to be included in the new work. There is a deadline in March, so, if you have anything to contribute, please ring Alan on 07 377 3666 (work) or 07 1666902 (home)

Closantel toxicity in goats: Accidental and preliminary experimental intoxications with this anthelmintic have produced blindness in goats; the limited studies so far have revealed degeneration of the outer nuclear layer of the retina. Sheep also appear to be susceptible. Sometimes the problem is due to rank overdosage; n others it is idiosyncratic.

Indospicine poisoning in dogs: In 1984 there were 30 or more deaths in the Alice Springs district in dogs which had eaten meat from horses which had been shot after developing "Birdsville horse disease" (Indigofera linnaei poisoning). Analysis has revealed up to 50mg/kg indospicine in the meat, and subsequent work has shown that low oral intakes of pure indospicine (of the order of l-2mg/kg bw/day) will produce significant liver damage within a few weeks. To date, the naturally occurring syndrome, which was of complete liver failure and death, has not yet been experimentally reproduced. One of the curious features of this intoxication is that, while "Birdsville horse disease" is a neurological disorder, with no apparent lesions, affected dogs show only liver disease.

Suspected phenol intoxication in dogs: Richard Miller, of Central Diagnostic Laboratories, Wooloongabba, recently diagnosed severe interstitial pneumonia: of the "atypical" or paraquat-induced type, in a dog which had recently had its anal sacs cauterised by instillation of phenol. After very faint bells were heard, a similar case was isolated from our own files; the victim was from the sane practice as Richard's case. The diagnosis offered in the earlier case was paraquat poisoning. There is an account in the Vet.Record (Linklater.K. et al,1982,110:33-6) of similar lung lesions being produced in sheep by the phenols in some old-fashioned carbolic sheep dips in Britain.

**NEW SOUTH WALES** - Mark Carrigan

Stypandra glauca poisoning in goats

On a large station property 420 Angora wethers of mixed age had been consigned to rough hill country 3-4 months ago. When recently mustered 50% were noted to be depressed and 5-10% appeared blind. Detailed clinical examination revealed bilaterally dilated pupils, a high stepping gait and abnormal carriage of the head. Histologically there was severe status spongiosis of white matter tracts within the brain and mild Wallerian degeneration in the optic/tracts and nerves. Early retinal degeneration in the outer nuclear accompanied by hypertrophy of the pigment epithelium was detected in some goats.

A considerable amount of Stypandra glauca R. Br., "nodding blue-lily", was present in the paddock and there was evidence of it having been consumed by the animals. This plant is a close relative of Stypandra imbricata R. Br., "blind grass" which is a well documented cause of blindness in various aspects in Western Australia. There appears to be no previous reports of S. glauca causing a similar syndrome and the Soil Conservation Service of N.S.W. is promoting its planting for the control of soil erosion.

Suspect Cedar Berry poisoning in sheep

Numerous berries of Melia azedarach L var australasica were observed in the rumenal contents of yearling wethers which had died suddenly. The mortality rate was 1% of 600 and gross pathological findings were a swollen, mottled, congested and friable liver. Severe acute periacinar necrosis was found histologically along with polioencephalomalacia in the cerebral cortex. It is uncertain whether the two pathological findings are related and we have been unable to find detailed accounts of this toxicosis.

Chronic Phalaris poisoning in goats

Seven out of 30 two year old Angora goats developed clinical signs of slight ataxia, proppy hind limb gait and low exercise tolerance in April-May 1986 after grazing phalaris for the majority of their lives. The goats were removed to a phalaris free pasture and clinical signs slowly progressed until September when one goat with ataxia blindness and convulsions was necropsied and a range of tissues collected for histopathology. The remaining clinically affected goats were shorn during a cold snap and all died.

Histologically changes were in keeping with chronic phalaris intoxication. Neurones in the lateral geniculate bodies and in nuclei throughout the brain stem contained fine intracytoplasmic golden-brown pigment granules. Similar brown granules were present in Kuppfer cells throughout the liver and in epithelial cells lining collecting tubules in the renal medulla.

Systemic Isospora sp. infection in canaries

A small aviary lost 12 out of 18 4-5 week old canaries over a week. Clinically birds showed signs of depression, inappetence and were fluffed up for 12 hours prior to death. In the majority of birds necropsied the liver was swollen and contained pale irregular lesions.

Histologically there was a severe diffuse hepatitis with congestion, extensive infiltration with large macrophage inflammatory cells and large areas of coagulative necrosis. The Intestines had a severe proliferative enteritis with a heavy mononuclear cell infiltration of the lamia propria. In the birds that died early in the outbreak protozoal schizonts were detected in the liver and lamina propria of the small intestines. Low numbers of protozoal schizonts were present in the lamina propria of the small intestine and enterocytes, but not in the liver of birds dying late in the outbreak.

Dr W. Hartley commented that this case probably involved an Isospora sp. which is highly pathogenic to canaries. (E.D. Box. J. Protozoal. 17 : 391-394; 22 : 165-169; 25 : 57-67).

Necrotic enteritis in lorikeets

Necrotic enteritis was diagnosed in rainbow and scaly breasted lorikeets during October. Affected birds have been found at Byron Bay, Iluka/Yamba and Coffs Harbour. Outbreaks of this condition have been reported in free-flying and caged rainbow and scaly-breasted lorikeets; signs include weakness and inability to fly ("paralysis") and in some cases diarrhoea. See PGF Control and Therapy Number 953, by Janeen L. Samuel. Cl. Perfringens is a suspected but unproven cause.

Streptococcus suis

Sporadic cases of Streptococcus suit infection have been seen throughout N.S.W.

(1) Strep. Suis Type II was isolated from the CSF and lungs of an 8 week old pig that was submitted for post-mortem examination with a history of nervous signs for a brief period prior to death. Pathologically cloudy cerebrospinal fluid and leptomeninges. Internal hydrocephalus, visceral congestion and strands of fibrin in the peritoneal cavity were noted. The histological findings consisted of diffuse subacute suppurative ependymochoriomeningoencephalitis and acute haemorrhagic pneumonia.

(2) Fifteen 4 week old suckers developed hind limb inco-ordination progressing to paresis and had intentional tremors. Meningeal oedema of the brain and spinal cord was the only necropsy finding. Histologically there was a severe diffuse suppurative meningitis with heavy infiltrations of the leptomeninges with polymorphs. A second piglet from the same litter was submitted to the laboratory two days later. Gross pathology was similar to the first piglet. Histologically there was a pyogranulomatous meningitis.

Strep, suis Type I was isolated in moderate pure growth from the CSF.

(3) Strep, suis (non-typeable) was recovered from lesions of extensive bilateral pleurisy and pneumonia in a 15 week old unthrifty pig.

Osteoporosis in sheep

On a property on the south west slopes 2% of the 4 month old Corriedale lambs grazing lush oats were found to have multiple fractures of the limbs. In various bones submitted to the laboratory there appeared to be normal physes, deficient primary spongiosa, pathological fractures at the junction of the primary spongiosa and the zone of provisional mineralisation, and absence of osteoclasts in the metaphyses and apparently inactive osteoblasts. Calcium deficiency was offered as the most likely cause.

Disseminated mycotic infection with cerebellar, pulmonary, splenic and hepatic infarction was diagnosed as the cause of death of a Charolais cow with necrotic rhinitis and tracheitis.

**TASMANIA** - Prepared by David Obendorf

Contributions by Roy Mason

1. E. coli 078 septicaemia in young, lambs is again occurring, although numbers of submitting properties less than in previous years. Of interest, and perhaps developing concern, was that occurrence of the disease in 2 calves one of which had severe meningoencephalitis and polyarthritis.

2. Micronutrient deficiencies are currently prevalent. This is probably a function of the good seasonal conditions. Low level of copper, GSHPX and vitamin B12 have bean associated with bone fragility, white muscle disease and ill thrift respectively.

3. Cryptosporidium sp has bean associated with enteritis in young stock, goat kids being particularly prevalent amongst affected animals.

4. Lead poisoning in calves. In one instance following ingestion of sump oil, the other following 'penning up" between old painted doors used as pen partitions.

5. Severe anaemia in young sheep, with deaths in some, has been associated with Eperythrozoon ovis on two properties in Northern Tasmania. Seasonal conditions would be conducive to an abundance of mosquito activity.

6. Marked yellow skull bone fluorescence was observed in a lamb on examination under U.V. light for brain fluorescence which is often associated with cerebrocortical necrosis. The lambs had been treated the proceeding day with systemic oxytetracycline.

7. Seven geese had been found dead over a 7 day period. The geese had been fed Ovaltine waste by the owner. It is thought that the birds probably died from theobromine poisoning. Theobromine is an alkaloid present in cocoa product used in chocolate, and Ovaltine production. Theobromine is a cardiac stimulant and it is well absorbed but excreted slowly so that continual ingestion can have a cumulative effect with death delayed until a critical level of theobromine is reached. Few signs are usually seen, death occurring suddenly from heart failure.

8. Strychnine poisoning. A pack of 7 farm dogs was found dead on their chain first thing in the morning. The owner had fed them pellets the previous evening, although a little later than usual.

   On autopsy the stomach of each dog contained a slice of bread and associated pink crystalline substance. The bread was well away from the oesophageal inlet adjacent to the gastric mucosa and the remainder of the stomach lumen was filled with dog pellets.

   The crystalline substance was confirmed as strychnine and from the location of the bread in the stomachs it would appear the poison bait was given shortly before the owner fed his dogs.

   Strychnine acts rapidly, within 30 minutes to two hours of ingestion. It is believed, in this instance, that death would have occurred soon after the owner fed the dogs.

Polioencephalomalacia in Dairy Calves. Twenty of 110 4-month-old Friesian calves died over a period of two weeks. The calves had been bucket fed and subsequently weaned onto good quality pasture. Many animals were found dead, however some showed CNS signs (blindness, nystagmus and opisthotonus) prior to death.

Examination of three brains revealed swelling, yellowing and softening of the dorsal and lateral cerebral cortex. Under ultra-violet light these affected areas fluoresced. The restriction of fluorescence to the lamina cortical areas was obvious on cut surface. The aetiology of this outbreak was not specifical1y determined, however, two recently affected animals were successfully treated with thiamine and the remaining calves in the mob were prophylatically given thiamine. No further deaths ware encountered. Liver Cobalt levels in affected animals were low to marginal 1.7 and 1.3 µmol/kgm (David Obendorf).

Laminitis in Cattle. A recent weekend "neighbourly" call was to a couple of Friesian heifers with laminitis. One showed the typical crossing of the front legs and the other was recumbent and would only rise after much "encouragement". The recumbent heifer was shot and the feet removed and autoclaved. There was evidence of haemorrhage/congestion in the sensitive laminae as shown by discolouration and loss of architecture (this was presumptive because, obviously, cooking does nothing for the architecture). All third phalanges in the fore feet showed rarefaction/absorption and one had fractured transversely (Barry Munday).

Avian Mycobacteriosis in captive wallabies. A small enclosure was home for 21 Bennett's wallabies. Six animals died in the late winter when no remaining pasture remained and hand feeding was essentially the only source of nutrition. Animals became very emaciated, weak and reluctant to move. On post mortem two submitted wallabies had enlarged retropharyngeal and mesenteric lymph nodes containing pus. Numerous acid fast bacilli were seen in Ziehl-Neelsen smears of the lymph node pus. Mycobacterium avium was isolated from these nodes. Malnutrition and inadequate shelter were obvious contributory factors to this problem. On questioning the owner about the prior use of the enclosure, she remembered buying some 'old layer hens' two years previously. They were 'very light' and 'all died over the next few months'. (David Obendorf).

**NEWS FROM VICTORIA** - Sue Friend

Bairnsdale Regional Veterinary Laboratory - Kit Button

Renal Dysplasia in Simmental Calves

Two purebred Simmental calves, approximately 3 months old were presented for necropsy. The calves were in poor body condition and lame as a result of a quadrilateral hoof wall defect. Distal horn was normal and clearly demarcated by a line separation from proximal horn which was irregular and furrowed. Both calves had small, firm, pile, shrunken kidneys and biochemical evidence of renal dysfunction (markedly elevated serum urea and creatinine concentrations). Histologically, renal tubules were disorganised and dilated with an excess of interstitial tissue resembling granulation tissue. Crystals resembling oxalate crystals and cellular debris had accumulated in some tubules. A diagnosis of congenital renal dysplasia was made. Early in-utero infection by BVD virus has been suggested as a cause of renal dysplasia in-utero or post-nata1 exposure to the virus. One of two calves was cultured for BVD virus with negative results.

Onion Poisoning

Two animals out of a herd of 100 (15-20 month old Red Polls) were noticed lagging behind the rest of the mob. Both were lethargic, appeared to be losing condition and had pale mucous membranes. The owner had reported seeing dark urine in one of the animals. The diet of the herd included vegetable waste comprising up to 50% raw onions. A provisional diagnosis of onion poisoning was made and subsequently confirmed by laboratory findings of a regenerative anaemia (3.4 and 5.5g/dl Hb) and the presence of considerable numbers of Heinz-bodies. One of these animals died. The farmer had been feeding this type of diet for the past few years with no apparent ill effects.

Possible Antidote to 'Avomec' Overdose?

Avomec acts by enhancing release and uptake of the neurotransmitter, gamma-amino butyric acid (GABA). The classical inhibitor of GABA is picrotoxin. In years gone by picrotoxin has been used in man and animals as an antidote to barbiturate overdose. Its affect, when given alone, is similar to strychnine, causing excitation and convulsions.

Picrotoxin has been shown to neutralise ivermectin in vitro and there is one report in the Veterinary Record of successful treatment of a dog that had been overdosed with ivermectin.

Treatment of cattle with picrotoxin is likely to be a hazardous procedure, and may only be warranted in extreme cases.

We have some picrotoxin on hand and will try to evaluate it if the opportunity arises.

Blindness in Kids associated with Closantel (Seponver) Overdosage

Fifty, 6 to 12 week old Angora kids varying from 3 to 10kg body weight were inadvertently overdosed with Closantel. Dosage was approximately 8ml per kid (4 to 13 times the recommended dose of 1ml per 5kg). Oxfendazole (Synanthic) was dosed at the same time at no less than twice the recommended dose of 1 ml per 3.6kg.

Two days later approximately 30 of the kids were apparently night blind (unable to locate or follow their dams at dusk). Six of the 30 affected kids became completely blind whereas those less severely affected gradually recovered over 3 to 4 weeks. Of the six blind kids, one died on Day 4 after over dosage occurred, three recovered fully over 3 to 4 weeks and two remained blind up to Days 39 and 41 respectively when they were killed. Histopathological examination at the RVL revealed severe spongiform change of optic fasciculi, chiasm and tracts.

Changes similar to the above have been noted after overdosage of rafoxanide. Closantel and refoxanide are both members of the halogenated salicylanilide group of anthelmentics. Oxfendazole overdosage is unlikely to have been involved. Goat owners should be warned against overdosing with this group of anthelmintics.

Hamilton Regional Veterinary Laboratory - David Willams

The weather has remained generally damp and warm throughout spring, allowing for an abundant growth of grass. The lush feed apparently 'diluted out' copper, cobalt and selenium in the pasture, because deficiencies of these trace elements were commonly found in weaner sheep during the past 2-3 months. Cobalt deficiency was manifest by illthrift, mild photosensitisation of ears and muzzle and profuse lachrimation, with extension tear-staining of facial wool. While previously we had recognised this disease along the coast and in a restricted area around Melville Forest, this year cases were recorded in a more extensive area, bounded by Balmoral, Poolaijelo, Merino and Glenthompson. Copper deficiency was associated with bone fragility, especially of limb bones, rather than as nervous system disease (enzootic ataxia) or steely wool. Selenium deficiency white muscle disease was comparatively common. Further cases of hypocalcaemia, weakness and collapse were reported in lambs in the Willaura and Warrnambool areas. Phosphorus deficiency (low serum phosphate levels) was associated with 'no visible oestrus' and cystic ovaries in about 20% of 150 dairy Friesians. There was also a suggestion of inadequate total nutrition.

Death of Piglets

Thirty of the 45 piglets born to sows which farrowed outside died when 10-20 days old. Piglets became weak and listless and died in 12 hours. There was low-grade mastitis in some of the sows and considerable cold stress for the piglets. The provisional cause of death was hypothermia and hypoglycaemia. Gross post mortem findings on 4 piglets were unremarkable except for dehydration and an absence of food in the digestive tract. There were no significant bacteriological findings.

Histologically there was a moderately severe, non-suppurative meningoencephalitis. The most likely cause was Haemagglutinating Encephalitis Virus, first recognised in Australia in the mid 1970's, but exotic diseases such as Hog Cholera or Pseudorabies could not be excluded without follow up serology and virology. Investigations are continuing.

Hepatitis in a Monitor Lizard

A mature monitor lizard died and its liver was submitted for examination. There were numerous 2-4mm dry, yellow foci scattered throughout the organ. Histologically these were seen as areas of focal necrosis, heavily infiltrated with fungal hyphae. Beauveria bassiana, a common soil fungus, was isolated. This has been reported as a pulmonary pathogen in reptiles. It is interesting to note that this same organism was show a by Bassi in 1835 to cause disease of silkworms. At that time it represented the first clear demonstration that specific micro-organisms were responsible for animal disease. As such it pre-dates the classic work of Robert Koch on anthrax, by 40 years.

Red Foot in Lambs

Further to the report on red-foot in lambs in the last issue, a non-exotic pestivirus, Mucosal Disease Virus, has been isolated from tissues from affected animals.

Bendigo Regional Veterinary Laboratory - Rod Badman, Tony Fahy

Myelin Degeneration in a Friesian Calf

A neonatal Friesian calf, born one week early, was inco-ordinated and could only rise by itself onto its hind legs, but could stand when assisted. Two similarly affected calves were born on the property last year to the same sire. The calf was submitted to the laboratory because of the possibility of an inherited defect. No gross lesions were found but widespread myelin degeneration was present in brain and spinal cord. No neuronal lesions were present.

Glenfield Veterinary Research Laboratory conducted tests for citrullinaemia with negative results. They have had several Friesian calves with similar histories and neurological lesions in the past year.

Ascites and Heart Failure in Broilers

A recent problem in a broiler shed of 20,000 birds created a few heart flutters amongst staff at the RVL remembering the Fowl Plague outbreak of 1985. Above normal mortalities in 6-week-old birds were the reason for the initial submission. Autopsies on 6 birds revealed congested musculature, enlarged hearts, fibrinous exudate in pericardial sacs of some birds and ascites in one. Bacteriology was unrewarding and as further submissions were made, the progression of the disease from cardiac enlargement to ascites became more pronounced.

Histological changes in the many birds examined showed consistent changes in the myocardium of oedematous separation of muscle fibres, and moderate mononuclear cell infiltration. Lymphoid tissue in many birds was undergoing lysis and in several birds amphophilic inclusions were present in nuclei of lymphoid cells, consistent with adenovirus Type II infection. Its role in the cardiomegaly and ascites syndrome is unknown.

Vomiting and Wasting Disease (HEV infection)

A disease with many of the features of vomiting and wasting disease has been seen on two intensive piggeries during the last 12 months. The first sign of a problem was vomiting in sows and 10-day-old pigs. Although scouring was also seen in up to 30% of suckers it was not a major clinical sign. Piglets affected with vomiting subsequently became lethargic, did not suck from the sow (but did drink water) and rapidly lost condition. The pre-weaning mortality rate rose from an average of 12% to 30%. However, affected surviving piglets continued to fade away over a 7-14 day period and were subsequently destroyed. A combination of death during the acute stage and destruction of chronically affected suckers led to a 90% loss of piglets each week for a 3 week period.

Concurrently with the problem in the farrowing house, approximately 20% of newly weaned pigs became anorexic two days post weaning, drank little water, and by 4 days post weaning had markedly lost condition. These animals were not seen to vomit. All affected weaners were subsequently destroyed because of cachexia.

On one property, CHS lesions consistent with HEV infection were seen in clinically affected piglets, whilst on the second properly no such lesions were observed in those animals chosen for autopsy.

Virological and transmission studies were inconclusive.

The disease ran a course of approximately 3 weeks on each property and disappeared with no subsequent problems. Vomiting in sows and wasting in the weaners were usual clinical manifestation of the disease.

Benalla Regional Veterinary Laboratory - Jeremy Langdon, Jim Rothwell, Malcolm Lancaster.

An Iridoviral Disease of Fish

Epizootic haematopoietic necrosis (EHN) is a disease or redfin perch caused by an iridovirus first isolated in 1984. Outbreaks occur recurrently in the juveniles of the year, with massive mortalities in late spring and summer in Lake Mokoan, Nillahcootie, Hume. Eildon, Blowering and other waterways in Victoria and New South Wales. Adults appear to have acquired immunity in these waterways but sporadic EHN outbreaks have occurred in adult perch in more isolated waterways. The disease in perch is characterised by high mortalities, erythema of the nostrils and brain, splenomegaly and multiple pale l-3mm foci in the liver. Histopathologically there are necrotic foci in the renal haematopoietic tissue, liver and spleen. Confirmation is by viral isolation in RTG-2, $BF_2$, Redfin Embryo or other fish cell lines.

The disease has been reproduced experimentally in redfin perch, and the virus also affects cod, Atlantic salmon, mosquito fish and galaxiids in transmission trials. A recent alarming development was the diagnosis of the EHN virus as the cause of a disease in cultured rainbow trout on two fish farms. Mortalities were low but the presence of the virus in trout has serious implications for the movement and export of salmonids and salmonid products.

Fatal Viraemia in a Wombat

A 7 month old hand-reared pet wombat that became hypothermic, lethargic and died over 2 days was submitted. On necropsy the liver contained multiple white foci with a diameter of 1-3mm. There were widespread petechial and ecchymotic haemorrhages particularly of the intestines. Histology revealed multifocal necrotising hepatitis, enteritis, colitis, splenitis, pneumonitis and lymphadenitis. In the liver and around some enteric foci there were numerous basophilic to amphophilic intranuclear inclusion bodies. This case appears to be a viraemia probably due to Herpes virus or Adenovirus infection. Virus isolation and electron microscopy are being undertaken. This disease has not been reported previously.

Foot and Mouth Lesions in Pigs

A large white plaque with irregular edges was found on the dorsal surface of the tongue on one pig from Myrtleford. There were erosions along the margins of the tongue and on the coronary bands of a second pig. Exotic disease was discounted after detailed examination of the gross lesions by veterinary field staff experts who happened to be visiting the laboratory that day.

Specimens were submitted to Attwood for virus culture as similar lesions hive been reported in parvovirus infection in piglets. Subsequent virus isolates were sent to Australian Animal Health Laboratory and a rapidly cyto-pathogenic virus, most probably an enterovirus, was isolated. Piglet inoculation trials failed to reproduce foot or oral lesions although scouring did occur. Virus particles were seen in the faeces.

Haemolytic Anaemias

A Murray Gray cross calf presented with severe jaundice, a PCV of 0.06, and haemoglobinuria. Numerous nucleated erythrocytes and spherocytes were present in the peripheral blood. There was extramedullary haemopoiesis in a range of organs.

Four stud Southdown lambs were very jaundiced and had regenerative anaemia. In addition there was renal tubular degeneration and/or regeneration, with marked interstitial fibrosis and lymphoid cell accumulation but no significant haemosiderosis.

The lambs and their mothers were pastured on almost pure Yarloop subclover, and the serum copper in the one lamb assayed was high. However, the corresponding liver and kidney levels were not elevated. Leptospires were not seen, and anti-leptospiral antibodies were not detected.

No aetiological diagnosis was reached in either case, but a number of cases of haemolytic anaemia have been seen in Murray Gray calves. The probable sire of the dam of this calf originated from a stud herd that has yielded other cases.

**SOUTH AUSTRALIA** - Prepared by Peter Phillips

LEAD POISONING IN WATERFOWL - (Mike Hindmarsh, South East Regional Veterinary Laboratory)

Australia's first major lead toxicosis of waterfowl occurred at Bool Lagoon Conservation Park, South Australia where 26% of the Magpie Geese (Anseranus semipalmata) died in the late winter of 1986 (84 died out of a flock of 320).

The geese showed emaciation, distended gall bladders and eroded gizzard linings on post mortem.

Lead shot was found in nearly all of gizzards examined while liver and blood lead assays snowed significantly elevated results.

Black Duck (Anas superciliosa) were also studied after the mortalities in the Magpie Geese and 14.5% were found to be carrying lead shot in their gizzards (14 out of 97 ducks caught) and 25% had significantly elevated blood lead levels (22 out of 83 samples obtained).

The mud of the lagoon was sampled for shot and quantities were found equal or exceeding densities of many of the waterfowl areas in the United States of America. As a result of the above findings Winchester (Aust.) Pty Ltd will be importing steel shot into Australia and will be manufacturing locally within three years.

FESCUE FOOT IN SHEEP - (Mike Hindmarsh)

A group of wethers showed extensive raw areas of the lower limbs. They had been shedded prior to shearing and then run on a pasture containing William's Grass = Tall Fescue (Fescue arundinacea). This occurred prior to the late onset, of winter rains which allowed the sheep access to the normally swampy area.

This ergot-like condition has an unknown toxin cause, but the toxin appears to arise in or on the leaves of the fescue either as an ergotoid alkaloid or a mycotoxin.

ARSENIC TOXICITY IN POULTRY - (Peter Phillips, Vui Ling Tham)

Depression, regurgitation of feed, watery diarrhoea and deaths in a breeder flock of chickens led to pathology examination of several birds. The only gross findings were haemorrhage of the thin muscle of the gizzard, oedema of the ventral surface and sub-koilin layer of the gizzard.

Histopathology was severe oedema and degeneration of the koilin (keratin) layer and sub-koilin, marked necrosis and purulent inflammation of the mucosa of the gizzard. In some affected birds, a series of columns of calcification extending from the lower koilin layer into the mucosa were observed. Many basophilic, calcareous granules were observed at the luminal surface of tubular epithelial cells of the kidneys of some birds.

In three affected but surviving birds submitted three days later, gizzards and intestines were normal. One of these birds had had severe, multifocal coagulative hepatocellular necrosis with replacement haemorrhage and heterophil infiltrate. Another had similar hepatic pathology with splenic necrosis and heterophil infiltrate.

Analysis of a feed sample from the day of the first death revealed an arsenic level of 3,500 p.p.m. How the feed came to contain 0.35% arsenic remains unknown.

**WESTERN AUSTRALIA** - Clive Huxtable

Murdoch University (David Forshaw)

Cryptococcosis

A number of cases of cryptococcosis have been diagnosed at the Murdoch University Veterinary Hospital. Cryptococcosis is a disease that seems to have a high prevalence in Western Australia and recently, the impression is that the number of cases is rising.

Most of the casts seen are in cats, many of which are ultimately necropsied. The lesions are usually confined to the oropharynx, nasal skin and perhaps, regional nodes or salivary glands, but in a recent case, skin lesions were seen over the lateral thorax and caudal aspect of the thigh. This cat presented with a profuse nose-bleed and swelling over the left frontal. sinus.

On post mortem, lesions were seen in submandibular lymph nodes, conjunctiva, sinuses and turbinates as well as the disseminated skin lesions. A case was also seen recently in a goat with progressive dyspnoea. Post mortem examination of this goat revealed very solid proliferations within the lung parenchyma and the provisional diagnosis was neoplasia. Histologically the lesion was typical of cyptococcus with many organisms and minimal inflammatory response.

An unusual feature of the infections seen here is the very small capsule around the organisms, and "pseudo hyphae" formation of organisms on impression smears. Culture results, however, have confirmed the diagnosis of cryptococcosis.

Necrotic enteritis in a Musk Lorikeet

Necrotic enteritis was recently diagnosed in a captive Musk Lorikeet. Grossly there was a severe diphtheritic enteritis. Histologically there was a severe necrotizing enteritis associated with the presence of many large Gram positive rods.

Unfortunately the organism was not cultured but undoubtedly this was a clostridial enterotoxic problem.

Salmonellosis in a Galah

Gross lesions included pale and swollen kidneys, swollen congested liver, congestion of other viscera but not spleen which appeared normal. Histologically, there were focal areas of acute necrosis scattered through the liver. Increased numbers of mononuclear cells in sinusoids and occasional fibrin thrombi in sinusoids. Colonies of bacteria were seen both free in the sinusoids and within Kuppfer cells. There was extensive tubular degeneration and interstitial inflammation in the kidney and bacterial emboli were seen in some glomeruli and interstitial vessels.

Salmonella were isolated from the liver only, in pure heavy growth. Identification of the Salmonella is proceeding.

Animal Health Laboratory Perth (Ron Peet)

Pasteurellosis in a Gibbon

A 10-year-old female white-handed gibbon in good body condition found dead in night quarters. Previously eating well, no illness noticed and vaccinated against Type C botulism since it lived on an island exhibit surrounded by a lake.

Post mortem examination revealed an empty stomach and no visible lesions.

Histopathology showed occasional foci of necrosis in a random pattern with numerous apparent bacteria in these areas and in sinusoids. Similar organisms were visible in blood vessels of other organs and a focal endocarditis was present in the heart. Pasteurella multocida was isolated in heavy pure growth from the liver and Gram stains of the tissues were consistent with this diagnosis of P. multocida septicaemia.

Bunbury Regional Laboratory (Clive Main)

Mannosidosis

This disease was diagnosed in a 4-month-old Aberdeen Angus calf which came from a small closed herd. The same bull had been used in the herd for 10 years.

Clinically the calf displayed tremors of the head and muscle, ataxia and aggression.

Histopathological lesions consisted of varying degrees of vacuolation of the Purkinje cells as well as neurons in other parts of the brain and spinal cord. In addition there was vacuolation of epithelial cells of the lacrimal gland, pancreas and to a lesser extent tubular epithelial cell of the renal cortex. Lymph node sinuses contained numerous large foamy cells.

Mannosidase activity tests on plasma samples from the rest of the herd revealed that in addition to the calf's mother and the bull, 4 other animals could be regarded as suspects. Granulocyte tests were not carried out.

Johne's Disease

Two cases, additional to the one reported previously (October 86 No. 14), have been diagnosed. As before, the animals involved were imported into the State, this time from Victoria. One of the animals came from a property on which Johne's Disease had never been diagnosed.

Case 1 - A five year old cow that had been imported into Western Australia 2.5 years earlier, commenced scouring one month after calving. After conventional treatment had failed faecal samples' were submitted to the laboratories for examination. Numerous acid fast bacilli were present in a smear prepared from that sample and the animal was subsequently slaughtered. Post mortem examination- revealed marked thickening of the terminal ileum and caecum and the characteristic coarse transverse corrugations of the mucosa. Diagnostic lesions characterised by the presence of large numbers of epithelioid cells containing acid fast bacilli in the lamina propria and submucosa were seen histologically.

Case 2 - An 8 year old cow imported into the State in 1980 started intermittent scouring after aborting her calf in February. In contrast to the previous case this animal remained in good body condition until the time of its slaughter at an abattoir. Macroscopic examination of its intestinal tract revealed reddening and some swelling of the mucosa of the terminal ileum. Numerous mildly haemorrhagic foci were also present on the caecal mucosa and the first part of the colon. The characteristic lesions so evident in Case 1, were not present.

Histopathological examination revealed an absence of the large aggregations of epithelioid cells. Instead there was a diffuse infiltration of mononuclear cells into the lamina propria some of which were epithelioid in appearance. Very small groups of similar cells were also present just below the muscularis mucosa. However, numerous acid fast bacilli were evident in the lamina propria and to a lesser extent the submucosa.

Albany Regional Laboratory (Ruth Reuter)

CIover Infertilitv in Sheep

A decline in lamb marking percentage in a Merino stud flock from 95% to 45-50% over of a period of three to four years prompted the owner to seek assistance from the Department. The property on which the sheep were run consisted of ten paddocks which had been cleared at various times over the past 50 years. From a mob of 3000 breeding ewes 650 six tooth ewes were sent for slaughter and 30 reproductive tracts obtained from the abattoir for examination. Fifteen of these had moderate to severe lesions of plover disease, which included blunting of the cervical villi, cyst formation in the cervix and endometrium and squamous metaplasia of cervical glands. Subsequent examination of the property revealed that eight of the ten paddocks were composed of subterranean clovers of which 95% were species known to be high in oestrogens, primarily Dinninup and Dwalganup.

Bovine Lymphosarcoma

A five month old Poll Hereford bull calf was presented live to the laboratory with a history of sudden onset of clinical signs. Six days previously he had been seen lying by himself in the paddock, but was able to get up and move around without difficulty when approached. The following day he began knuckling on the left hind foot. The knuckling affected both hind legs by 24 hours later; however, the calf was able to walk one mile into a yard. Three days later it was unable to rise.

On arrival at the laboratory the calf was bright and alert, with control of the head and neck but otherwise paralysed. The most striking feature at post mortem was generalised enlargement of the spleen and all body lymph nodes. Small white foci were present in the lung and liver. The spinal meninges were thickened and grey in colour, particularly around the dorsal nerve roots. A blood smear taken at the time of necropsy showed very large numbers of immature and bizarre lymphocytes present. Histology confirmed the diagnosis of lymphosarcoma with extensive meningeal invasion. Serology performed for Bovine Leukaemia/Leucosis Virus was negative.

Cobalt Deficiency in Fallow Deer

A group of young fallow deer were brought from Victoria to Western Australia in July 1986 to join a herd already present in the Bow Bridge area. The introductions included young does and bucks as well as several pregnant animals. By November around 20 of the deer were found to be in poor body condition with rough coats and evidence of hind leg inco-ordination.

None of the pregnant does were affected. A post mortem performed on one 11-month-old maiden doe which collapsed and died while being yarded showed serous atrophy of body fat stores and congestion of the liver and kidney. Three weeks later a young male was brought to a local veterinary clinic for examination and treatment. He appeared bright and alert but was weak and ataxic. He was eating well but by the next day was unable to stand and died four days later. On post mortem there was emaciation, serous atrophy and submandibular oedema. Cultural and microscopic examinations were unrewarding; however, biochemical analysis revealed a very low Serum B12 level. Other parameters were within the normal range. Information received subsequently revealed that the pregnant does had bean given cobalt and selenium pellets prior to leaving Victoria. The rest of the animals had not been treated. The area to which the deer were introduced is historically deficient in cobalt.

Polioencephalomalacia in Calves on Pasture

Within the space of two months, two six month old Friesian calves from a group of 50 were found staggering, blundering into fences and exhibiting opisthotonus. The herd was on green pasture consisting primarily of kikuyu, clover and other grasses. One of the calves died on the pasture, the second walked to a holding yard and collapsed. Treatment with Thiamine IV in conjunction with antibiotics and corticosteroids had no effect, and the calf was euthanased four days later. Characteristic lesions of polioencephalomalacia were found on microscopic examination of the brain. This condition has been described in postured animals, usually associated with a change from poor to good pasture. In this case the pasture had been consistently good and the cattle had not been shifted. Predisposing factors could not be found.

Anasarca and Nephropathy in Lambs

A mob of 1600 Merino wethers were shorn and returned to their paddock with no further handling or treatment. Four days later there were five animals dead and 40 showing signs of abdominal distension and subcutaneous oedema. One animal from each of two other mobs shorn around the same time showed similar signs. The oedema appeared to be associated mainly with shearing cuts of varying degrees. Post mortem examinations on several animals showed anasarca, hydrothorax, hydropericardium, ascites, congested liver and enlarged pale, mottled kidneys.

Microscopic examination of the kidneys revealed acute coagulation necrosis of tubular epithelium with extensive leakage of proteinaceous fluid into Bowman's space and hyaline casts in the tubular lumens. Impression smears of liver, spleen and kidney of one sheep contained large numbers of Gram positive rods resembling Clostridia. Smears of the oedematous area surrounding a shearing cut on another animal showed similar bacteria, and B haemolytic colonies grew anaerobically on blood agar 48 hours after inoculation. However, the isolate seems reluctant to grow in the various media for identification so has not as yet been typed. It does not appear to be one of the species commonly associated with "Malignant Oedema" or wound infection.

Other conditions occupying the veterinary staff at present include cobalt deficiency in sheep exhibiting classic signs of anaemia, weeping eyes, ill-thrift and pale livers on post mortem; Annual Ryegrass Toxicity with characteristic clinical signs but a paucity of histologic lesions; and larval resistance tests on faecal samples.

THE ROLE OF VETERINARY LABORATORIES INTO THE 1990's.

Provision of a Professional Service for Professionals - A Report prepared by Terry Nicholls, RVL Benalla.

A two day seminar was held at the Central Veterinary Laboratory Adelaide (I.M.V.S.) in November 1986. The seminar was designed primarily to clarify the future directions of the Central Veterinary Laboratory and a number of speakers from around Australia were invited to present their views on the role of Veterinary Laboratories into the 1990's. The seminar was organised by Dr. Martin Copland and proceedings are available from him on request.

This report provides a short personal view of the issues and conclusions reached.

There is an inescapable trend throughout Australia, both in the Conservative and Labor states, to reduce public sector spending and to pursue the user pays principal in the provision of services. The declining importance of rural commodities in both the internal and export markets and the difficulty in financing a wide variety of social and educational and other reforms has led to a reduction in agricultural sector spending in some states.

Government veterinary diagnostic laboratories are seen as having primary responsibility for endemic animal disease diagnosis investigation and research, as well as a pivotal role as a source of trained veterinary expertise in exotic disease outbreak crises. However, government veterinary laboratories in many states may be faced with progressive dismantling and starvation of funds until they are unable to provide even these basic services.

It is also apparent that the basic services provided by many government veterinary laboratories can be augmented by entrepreneurial management. In essence the government veterinary laboratory would provide the basic services required by the community, with guaranteed minimal funding, in exchange for entrepreneurial freedoms which would allow the laboratory to develop new revenue earning services. If this approach works the government contains expenditure to C.P.I. limits, or even makes money. The laboratory expands its services to the community efficiently and improves the work environment of its staff. This sounds too good to be possible.

To achieve the impossible the following ten points are considered essential in the entrepreneurial management of government veterinary diagnostic laboratories:

1. The ability to deliver a fast, efficient and accurate veterinary diagnostic service.

2. A one-line budget is needed with complete interchange ability of wages and operating money.

3. There is a need for a SIGNIFICANT RESEARCH and INVESTIGATORY capability.

4. Staff selection needs to be oriented to the commercial environment in commercial areas.

5. There needs to be an agreed scale and base level of self-sufficiency negotiated with the government or department.

6. There needs to be significant freedom from Public Service Board regulations and government constraints in the areas of employment, where excellence and performance are essential, in hours of employment and flexibility in those hours, and in purchasing and marketing.

7. Laboratories need to pursue what they do well, that is interpretative veterinary diagnostic services, as well as research and investigation. Diversification should proceed slowly and carefully.

8. There needs to be realistic pricing and recognition of real costs. For example the labour component of an individual test needs to be costed at approximately 350% of the labour rate to be realistic.

9. It is counter productive for the government to retain funds received for commercial services as overall services decline and costs escalate.

10. There needs to be a turnaround from the introverted peer group motivation of most government scientific laboratories to an extroverted commercial approach centred on service to the client.

Many of you will be able to relate your laboratory to a number of these points. Some laboratories, for example, are not encouraged to do research and have a limited investigatory capability. None have a one line budget and none would be free from Public Service Board regulations and government restraints in employment. I would suggest that many of our laboratories are introverted and staff are not as motivated towards provision of services as they might be. The professional training and subsequent acquisition of further tertiary qualifications and experience generally mitigates against extroverted and market oriented pathologists.

The highlight of the two days was a lecture by Dr. John Hellstrom from New Zealand. He clarified and organised many of my thoughts on the entrepreneurial management of government veterinary laboratories. The veterinary laboratory section of the New Zealand Ministry of Agriculture and Fisheries appears to have negotiated a set of conditions which includes most of the points 1-10 listed above. Victorian laboratories are part-way down this track but are becoming increasing entangled in bureaucratic and government constraints.

Our future as veterinary pathologists depends on being able to negotiate, in a rational way, the above 10 points with governments. Failure to follow the New Zealand model may well lead to a severe withering of our ability to deliver basic services and a diminution of our potential to respond to future crisis such as the recent outbreak of Fowl Plague at Bendigo. I feel strongly that the veterinary profession and our laboratories are under siege from a number of quarters. The issues discussed and the points distilled from this conference show clearly the direction we should be heading. Many of us are more conservative in the assessment of our role and the provision of our services; however, I think any temptation to sit and let things happen is fraught with danger. We must PROMOTE, EXTEND, and IMPROVE our services and performance, particularly in the areas where we excel. We must demonstrate clearly to the community at large our value and potential. I hope this report provokes thought and discussion within our group.

EDITOR'S COMMENT:

I feel that the Veterinary Diagnostic Laboratories are at the crossroads. We need to address the problems now. Any contributions from members on the general philosophy espoused by Terry Nicholls or comments on the situation in their own state would be welcomed. These can be addressed to the Executive of the A.S.V.P. (c/- The Secretary) and, if the submitter desires, be published in the next A.S.V.P. report.

IMPORTATION OF LIVE ANIMALS INTO AUSTRALIA - Ian Links

With the resumption of the import of live animals in recent years, albeit under strict quarantine controls, there has been an unspoken fear that exotic diseases or new strains of currently endemic diseases could be introduced into Australia. This applies particularly to diseases of a more chronic or insidious nature particularly where a persistent carrier state can occur. Such diseases once present in Australia could prove difficult or impossible to eradicate.

Recent events suggest that such fears are real. The emergence of overt clinical atrophic rhinitis in pigs in the past 18 months is one such example. Until proven otherwise it appears likely that a toxigenic strain of Pasteurella multocida was introduced via imported pigs. Another example involves an imported bull released from quarantine which was recently shown to be a carrier of Mucosal Disease virus. Our current knowledge of the pathogenesis of this disease would indicate that the animal had been infected in-utero. Other incidents may have occurred of which we are unaware.

Specific pathogens which could involve the carrier state include herpesviruses and pestiviruses (swine-fever) while potential pathogens among the flora of the intestinal tract (e.g. E.coli), respiratory tract (e.g. mycoplasmas) and uro-genital tract (e.g. leptospires) are also a potential risk.

Although screening procedures are undertaken for some of these agents freedom from infection cannot always be guaranteed. There is also the risk of previously unrecognised pathogens being introduced, as the emergence of AIDS in humans and contagious equine metritis in horses demonstrates. Does the post-mating vaginal discharge syndrome in sows recently recognised in Australia and previously described in Britain (Muirhead, 1984, 1986) fit into this category?

This is an appropriate time to air our views on this matter. Once again comments can be submitted to the executive of the ASVP (c/- The Secretary) and if the submitter approves they will be published in the next issue of the ASVP report.

Reference

GIBBS, E P J (1981) Adv Vet Sci. Comp Med Vol 25: 71-100 "Persistent viral infectious of food animals: their relevance to the international movement of livestock and germ plasm."

MUIRHEAD, M R 1984) Vet Ann 24: 118-126 "The investigation of a production problem in pigs: low litter size, vaginitis and endometritis".

MUIRHEAD, M R (1986) Vet Rec 119: 232-235 – “Epidemiology and control of vaginal discharges in the sow after service".

## DIARY OF COMING EVENTS

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

23rd May, 1987 - Annual Conference, A.S.V.P., {University of Sydney)

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

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

1st September. 1987 - Close of applications to sit membership examinations, Australian College of Veterinary Scientists.

## VET. PATH. REPORT PUBLICATION DATES

JULY, OCTOBER, JANUARY, APRIL.