THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
the Department of Primary Industry, Tasmania,
Mt. Pleasant Laboratories,
P.O.Box 46, Kings Meadows, TAS. 7249.

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DEADLINE FOR NEXT VET. PATH. REPORT IS DECEMBER 1

PRESIDENT'S PAGE.

The following paper on the A.S.V.P's attitude to registration of Veterinary Pathologists and Veterinary Pathology Laboratories was developed by the Executive of the A.S.V.P.

The New South Wales division of the Australian Veterinary Association requested the A.S.V.P.'s opinion before formulating its advice to the New South Wales Board of Veterinary Surgeons.

Time did not permit circulation of the paper to A.S.V.P. members before submitting it.

Members are invited to comment through the newsletter on the following paper. This will assist the A.S.V.P. executive to develop the A.S.V.P.'s position. Please send your comments to the Editor.

(Rod Oliver)
PRESIDENT
A.S.V.P.

ACCREDITATION OF VETERINARY PATHOLOGISTS AND/OR VETERINARY PATHOLOGY LABORATORIES

1. QUALIFICATIONS OF VETERINARY PATHOLOGISTS

Essential qualifications for recognition as Veterinary Pathologists should include:

(i) Registration as a Veterinary Surgeon
(ii) At least three years practising veterinary pathology and demonstrated ability.

The Veterinary Surgeons Board in each state is best placed to decide if a person is qualified as a Veterinary pathologist. The Board would need to take into account the needs of the community for veterinary pathology services and identify persons who the public and the veterinary profession can turn to for advice and service of veterinary pathology.

Membership of the Australian College of Veterinary Scientists (Pathology Chapter) should not be seen as the only acceptable qualification for registration as a Veterinary Pathologist, A number of M.A.C.V. Sc.s are involved in fields other than pathology and are not actively practising veterinary pathology.

The grandfather clause whereby experienced veterinary pathologists not registered by a stipulated date are excluded from future registration as a veterinary pathologist is not supported. With the relatively small number of people involved, registration can be handled on a case by case basis by Veterinary Surgeons' Boards in each state.

2. FACILITIES NECESSARY FOR VETERINARY PATHOLOGY

This is difficult to define, depending on the range of services offered. On the one hand, a veterinary pathologist could offer a highly competent histopathology service from his home by setting up a microscope bench in a room for reading slides.

Alternatively, a veterinary pathology laboratory may offer a comprehensive range of services for a number of disciplines. State veterinary pathology laboratories will likewise have strengths in some disciplines and not others.

Developing guidelines for veterinary pathology laboratories would be unproductive because

1. Only a handful of private veterinary pathology laboratories exist in Australia,

2. State laboratories are equipped to various standards and it would be impractical to expect massive reorganisation to meet specified guidelines.

3. Veterinarians operating clinical pathology and microbiology laboratories as part of their practices.

In terms of guidelines or accreditation for laboratories, the American Association of Veterinary Laboratory Diagnosticians has established an accreditation system for Veterinary laboratories. However, accreditation is based on competency in providing a broad range of disciplines, not just one discipline, e.g. pathology.

Registration of premises or facilities for veterinary laboratories is not supported because it is virtually impossible to come up with guidelines covering all situations. Veterinary pathology laboratories will be judged on their performance rather than their registration.

3. ROLE OF NON-VETERINARY PATHOLOGISTS

(i) Medical Pathologists and Medical Pathology laboratories: These have always offered a service to veterinarians who use the service for convenience, rapid turnaround and better service or are accessible to veterinarians, a major factor in rural centres. Human laboratories are traditionally orientated towards providing a rapid diagnostic service.

Recent developments have a direct bearing on medical pathologists doing veterinary pathology.

(a) The emergence of specialist veterinary pathology laboratories operated by qualified veterinary pathologists in Brisbane, Sydney, Melbourne and Adelaide. In Brisbane, Veterinary Pathology Services has captured the market for pathology services for Brisbane veterinary practices by offering an excellent service, rapid turnaround and interpretation.

A major reason why veterinarians prefer to use veterinary pathology laboratories is that veterinary pathology laboratories can provide interpretation of findings.

(b) Medical laboratories are becoming more automated and computerised. Laboratories are set up to detected abnormal findings and samples from animals, activate error systems on automated equipment, disrupting production runs. For this reason, medical pathology laboratories are becoming reluctant to process veterinary samples.

(c) Medical laboratories will become less enthusiastic as veterinary clients become litigation-minded. It will only take one claim for damages because an inappropriate technique was used to deter medical laboratories from encroaching into veterinary pathology.

(d) Costs. Medical laboratory fees are high, e.g. $52 per biopsy and deter veterinarians from routinely using these services.

(1) Species specialists: Specialists involved with one species may extend their expertise from areas like breeding, nutrition and husbandry into pathology. Species specialists who are non-veterinarians are not well placed to provide pathology services, though they may recognise some diseases. There is no substitute for pathology training in morphological recognition of disease.

The problem of unqualified persons performing acts of veterinary pathology is a long-standing one and should be addressed under the Veterinary Surgeon's Act, i.e. performing an act of veterinary science by an unqualified person.

SECRETARY'S REPORT

Sales Tax Exemption

Attention future busy ASVP executive members. The Australian Taxation Office has advised that costs for the Veterinary Pathology Report, if printed and collated by an outside commercial business on behalf of the ASVP, are exempt from tax.

ASVP CONFERENCE 28-29 January, 1990

Don't be intimidated by the "pilots' strike". BE POSITIVE get those bookings made. Planes are flying into Tasmania. You may even experience a little adventure on the trip! My daughter recently returned to the mainland and flew all the way, up front with the pilot and co-pilot. Who knows you may get to serve the drinks or haul out the luggage - be in it.

Roy Mason
Hon. Secretary

EDITORS REPORT

I hope that all Veterinary Pathologists out there are satisfied with the content and format of the **Vet. Path. Report**. It is encouraging to have so many contributions coming in from all over Australia. Remember that the **VPR** can also be a forum for discussion and debate. How about some contributions on Accreditation of Pathologists and Pathology Laboratories or personal feedback on the usefulness of the Pathology Registries?

Surely there are operational issues confronting Vet. Pathologists and Laboratory Diagnosticians in universities, departmental laboratories, and private diagnostic labs which could be aired in **VPR**. We have, in the past, concentrated on case reports, disease outbreaks and research briefings. Perhaps it's time to consider broader issues - like our role in "Sustainable Agriculture", Endangered Fauna Conservation, Aquaculture, Zoonoses, Animal Welfare issues, comments on the Slide of the Month etc., etc. Perhaps we should be raising more QUESTIONS (rhetorical or otherwise), rather than ANSWERS.

Quite apart from these uses for the VPR, we could think about including items on Personnel, New Diagnostic Techniques or Laboratory Innovations, correspondence from colleagues overseas, and information in reply to items published in earlier issues of **VPR**.

It is vital that we keep this newsletter as dynamic and useful as possible. The ASVP executive can keep you informed of issues as they become aware of them but the Society needs the input from the "rank and file" as well. We all have something to contribute, so over to you. Enjoy this issue.

David L. Obendorf
Hon. Editor

199O CONFERENCE UPDATE

AGM / SCIENTIFIC MEETING
LAUNCESTON, TASMANIA
28- 29 January, 1990.

Please refer to VPR No.25 page 3 for details and programming. **Papers are invited from members, either specifically on fish pathology or case reports on any species**. Presentations will be limited to 15 minutes. Presentations will take place after morning tea and after lunch on Monday 28 January. Abstracts should be prepared as follows:

. up to one A4 page
. typed single space using 12 point Letter Gothic, Prestige, or similar type with carbon ribbon. Main heading in the same type, upper-case, underlined and centred with author's name and address in lower case centred below.
. key references only, given in full.
. scientific names in the same type and underlined.
. margins 3 cm LHS, 1.5 cm other sides.
. to be in final form for reproduction by photocopying
. do not fold
. deadline (to Secretary, ASVP) - December 8, 1989.

PATHOLOGY TRAINING

Wayne Robinson has written to us concerning a National Training Programme for Veterinary Pathologists:

"At the 1989 meeting of the Australian Society for Veterinary Pathology, both the ASVP and the Pathobiology chapter of the Australian College of Veterinary Scientists decided that both groups should complement each other and work together as closely as possible. One of the initiatives from the May meeting was to examine the feasibility of a National Training Programme for Vet. Pathologists."

I have spoken to all concerned as it was agreed to report to the January meeting on the following:

1) Is there a need for a pathology training programme (both initial training and continuing education)? To answer this question we need to identify the numbers of veterinarians who work in the discipline of pathology.

2) If we agree that there is a need, we should then (a) approach the major employing bodies seeking their co-operation; and (b) decide on the bodies best suited to supervise training and continuing education.

3) Following (2) the scope of both initial training and continuing education should be defined.

Wayne would welcome any comments or ideas from ASVP members.

Please send correspondence to: Dr. Wayne Robinson,
School of Veterinary Studies,
Murdoch University,
MURDOCH, W. A. 6150

or phone (09) 3322418 or Fax (09) 3322507.

COMING EVENTS

1989

August 28-September 1 - Fifth Australian Arbovirus Symposium, Bardon Professional Development Centre, Brisbane. Contact Toby St. George, CSIRO Private Bag, PO Indooroopilly, QLD, 4068. Phone: (07) 377 0711. Fax: (07) 870 7034.

1990

January 26-28 - **FIN FISH WORKSHOP,** Tasmanian State Institute of Technology, Launceston. Contact Barry Honda, School of Applied Science, TSIT, PO Box 1214, Launceston, TAS, 7250. Phone: (003) 260 232.

January 28-29 - **ASVP ANNUAL GENERAL MEETING**, Mt Pleasant Laboratories, Launceston. Contact Roy Mason, Department of Primary Industry, Mt. Pleasant Labs., PO Box 46, Kings Meadows, Tas, 7249. Phone: (003) 415 217, Fax: (003) 444 961.

February 13-15 - **MECHANISMS OF DISEASE SYMPOSIUM ,** Wesley College, University of Sydney, Contact Mike Bryden , Post-graduate Committee in Veterinary Science. Phone: (02) 264 2122.

June 11-15 - **GOAT HEALTH & PRODUCTION,** Stephen Roberts Theatre, University of Sydney. Contact **PGCVS.**

July 6-8 - **SKIN DISEASES SYMPOSIUM,** Leura Resort, Leura, NSW. Contact **PGCVS**. Phone: (02) 264 2122.

July 16-20 - **SHEEP MEDICINE,** Stephen Roberts Theatre, University of Sydney. Contact **PGCVS**. Phone: (02) 264 2122.

August 3-5 - **PIG PRODUCTION SYMPOSIUM,** Leura Resort, Leura, NSW. Contact **PGCVS**.

September 24-28 - **RABBITS & RODENTS - LABORATORY ANIMAL SCIENCE** Stephen Roberts theatre, University of Sydney. Contact **PGCVS.**

MECHANISMS OF DISEASE

Course No 132 - 12, 13 & 14 February 1990

This symposium, which will be held immediately before the membership exams for the Australian College of Veterinary Scientists, will be a wonderful opportunity for practitioners and laboratory staff to consolidate their understanding of the diverse ways in which infectious agents cause disease. The symposium will cover two important areas, information and immunodeficiency diseases with an emphasis on mechanisms and how these mechanisms are reflected by clinicopathological correlations.

Modern concepts

The symposium will review modem concepts on how the various types of infectious agents, from viruses through to metazoan parasites, cause disease. It will update participants on both cellular and humoral responses to inflammation and will examine some interesting and unusual host parasite relationships that will illustrate how, in particular situations, the balance may be swayed either way, giving ultimate recovery or clinical disease and perhaps even death.

Immunodeficiency Disease

The symposium will also consider the most recent information on both congenital and acquired immunodeficiency diseases.

This symposium will be very much an interactive affair, as all the symposia have been, and in addition to a few lectures there will be considerable emphasis on group discussions and on the presentation of case material from which an indepth understanding will be gained.

The leaders for this symposium are Dr. Dick Sutton and Dr. Roger Kelly from the University of Queensland and Professor Reg Thomson from the Atlantic Veterinary College in the University of Prince Edward Island in Canada.

You should note that this symposium is planned to begin on Monday evening, 12th February, and will run through to Wednesday afternoon.

This will be a top course for the thinking practitioner and one which every veterinarian should look to attend.

Places will be limited and you should note that the venue for this symposium is Wesley College at the University of Sydney which will provide precisely the atmosphere we need for this symposium. Make sure you don't miss it.

TRAINING COURSE UPDATE

FIN FISH - Course No 128 - 26-28 January 1990

Venue: Tasmanian State Institute of Technology, Launceston
The fish industry and aquaculture generally has blossomed in importance in the Australian rural community within the last few years.

Uprising of Interest
At the time when the Post Graduate Committee was planning the course on fish diseases there was an upsurge of interest within the community in fish farming and all matters related to aquaculture generally.

As a profession we have often been slow to recognise the importance of industry developments and of the role which we as veterinarians should be playing in animal health, production and welfare except for those activities with which we have characteristically been involved for many years.

This has often led to a lack of experience within the veterinary profession in particular areas of veterinary activity and it is encouraging to recognise the tremendous amount of interest which there has been in the Fish Diseases Course and the course on Invertebrates in Aquaculture which has held in Brisbane earlier this year.

Fin Fish are Fun
The pressure for us to mount another course on fin fish diseases has been strong and I believe that it is likely that this course will overfill before the end of this year.

Certainly there is an enormous amount of work to be done within the fish farming and aquaculture industries and veterinarians wishing to carve out a niche for they could well look at a career in this industry which is crying out so badly for help and advice.

It is to be hoped that all veterinarians will continue to encourage the Post Graduate Committee to tackle new adventures which are developing on the periphery of what is commonly seen as the veterinary domain, for I believe that this is one important and vital role which we play within the profession.

Key Centre for Aquaculture
The Fin Fish Course will be a really top line course. It is being run at the Tasmanian State Institute of Technology and will be hosted by the Key Centre for Aquaculture. The course is to be a very practical one and will concentrate on the major diseases affecting fin fish today. Participants will engage in a considerable amount of practical laboratory work which will give them hands-on experience of the pathology and the diagnosis of these diseases.

We have been very fortunate indeed to have the support of the Australian Society for Veterinary Pathology and the Pathobiology Chapter of the Australian College of Veterinary Scientists who will be holding a joint meeting at the facility immediately after the workshop.

This scientific program will include sessions from Jeremy Langdon, Judith Handlinger and Barry Munday and will make the whole weekend a very valuable one for those interested and involved in fin fish work.

For those of you who have as yet only a marginal interest in fin fish but who would like to become more knowledgeable and more skilled in the procedures involved you should book in immediately to this workshop course which will be a really valuable one for you. Don't even bother to mark it down in your diary, just get your application in immediately so that you are not likely to miss out on a place. We are limiting the places and it will be filled on a first in best dressed basis. Don't miss out.

STATE REPORTS

from **David Williams**, Vic.

CSIRO AUSTRALIAN ANIMAL HEALTH LABORATORY GEELONG

AAHL'S first Training course (Peter Hooper)

We at AAHL were very pleased to host the annual general meeting of ASVP, then the week after, conduct our first training course for veterinarians. ASVP members will be interested to know that the first course was for pathologist and other laboratory veterinarians, most of whom were ASVP members. It is probably not accurate to say that it was strictly a course but largely a series of demonstrations in which the emphasis was hands-on experience.

The 15 participants were from regional laboratories from all over Australia and New Zealand. The laboratories were Rockhampton, Toowoomba and Yeerongpilly in Queensland; Orange, Wagga and Glenfield in New South Wales; Bairnsdale, Hamilton and Attwood/VRI in Victoria, and the principal laboratories of South Australia, Western Australia, Tasmania, Northern Territory and New Zealand. They were all senior experienced people so there were many benefits for AAHL as well.

The "course" was an intense 5 days starting Sunday evening 14 May to Friday 19 May. The mornings were spent in our Large Animal facility examining clinically and by post mortem a range of 12 exotic and endemic diseases. Fortunately, reproduction of all but one minor disease was satisfactory and the signs expected in an outbreak were evident. The diseases were "unknowns" to the participants but most were diagnosed by the experts within a couple of days. In a post course evaluation, all participants were pleased with the experience and they commented strongly on the value of the hands-on experience for training compared with audio-visual aids only.

The work with the animals was the reason for the course at the high security laboratory. However, in the afternoons, participants examined histological slides of cases of exotic disease, saw demonstrations of various tests, discussed some of the newer tests derived from molecular virology including polymerase chain reactions, diagnostic and research electron microscopy, and protein chemistry. At the end of the course, each participant was provided with a collection of histological slides. There is now consideration to send similar copies to the other laboratories that missed out, as well as a collection of Kodachrome slides of gross and microscopic pathology to all laboratories. Considering the amount of work involved, this won't be for a few more months. (We have a very small scientific staff, less than most regional laboratories).

In the evenings, there were informal discussions of the cases. As "unknowns", they were provided with theoretical histories and the results of laboratory tests when theoretically available and armed with this information they were expected to make reasonable differential diagnoses and not merely assume these were exotic diseases. There was one video-supported theoretical exercise of an "outbreak" near Geelong, some movies, and on one evening there was a discussion chaired by Dr. Ralph Salisbury, with people from the Bendigo and Benalla laboratories on the 1985 avian influenza outbreak and the relative roles of AAHL and the regional laboratories in general.

We at AAHL believe the course was a success, based partly on our own estimate and also on the participants' evaluations. Part of the reason for the success was the quality of the participants who were prepared to contribute so enthusiastically to all the sessions and activities.

REGIONAL VETERINARY LABORATORY HAMILTON

Malignant Catarrhal Fever in Chital Deer (Cor Lenghaus)

Three weaner and two adult Chital deer in a total mob of 120 were noticed to gradually lose condition, separate from herd mates, develop a stilted gait and die in 1-2 weeks. A unilateral or bilateral cloudiness of the cornea was noticed and there was terminal diarrhoea. We received fixed tissues from one animal and subsequently a moribund deer, 6 months old, which had a unilateral keratoconjunctivitis and associated hypopyon.

Histology of the fixed tissue showed there was a severe vasculitis of many vessels in all tissues submitted (liver, lung, heart, skeletal muscle, kidney and adrenal). The vascular lesion was clearly episodic and progressive; in some major vessels there were areas resolving by fibrosis, as well as areas of acute inflammation and fibrinoid necrosis. There was also a focal interstitial nephritis, and areas of infarction in the adrenals.

Autopsy of the whole animal revealed grossly abnormal mesenteric, coronary and perirenal blood vessels. These were irregularly thickened and corded and often contained thrombi. Lymph nodes were markedly enlarged, firm and wet. There were areas of haemorrhage in the wall of the rumen and colon, sometimes associated with ulceration of the luminal surface. There was prominent bruising of muscle masses. There were no ulcers in the oral or nasal cavities. Histologically, the vasculitis as noted earlier was the main lesion. Vascular pathology was most severe in the alimentary tract (where there was also associated infarction), brain, meninges, kidneys, liver, lymph nodes, adrenals, heart and spleen. Less severe lesions were present in the spinal cord, skeletal muscles, aorta, nasal turbinates, lung and eyes.

The above findings are typical of malignant catarrhal fever (MCF) in deer. The spectrum of clinical signs and post mortem findings for MCF in deer is well described in Proceedings No. 72, Deer Refresher Course, Post Graduate Committee in Veterinary Science.

REGIONAL VETERINARY LABORATORY BENALLA

Mucosal Disease (John Mackie)

So far this year, mucosal disease has been confirmed in 19 herds in the North-east region by isolation of pestivirus from 25 cattle. Affected cattle were from 3 months to 3 years of age. Illthrift was a constant clinical finding, with mild to severe diarrhoea and erosive lesions on the muzzle, buccal and nasal mucosa reported in most but not all cases. Interdigital dermatitis or coronitis, occasionally with ulceration, was also reported. A temperature rise was noted in less than half of the cases. Clinical pathology was carried out on 14 animals and was generally unremarkable. Only one animal had a leucopaenia while two animals had a leucocytosis. Three animals with diarrhoea had an elevated PCV consistent with dehydration and had a concurrent pre-renal azotaemia. Five animals had marginal elevations in some or all liver enzymes (GGT, GLDH, AST}.

Two cattle were presented for necropsy. Gross lesions included erosion and crusting of the muzzle, nares, buccal mucosa and conjunctival mucosa, abomasal ulceration, scurfiness of the skin above the coronary band and in one animal, cranio-ventral pulmonary consolidation.

Histological examination revealed necrosis and ulceration of the muzzle, nasal and buccal epithelium with ballooning degeneration of the occasional keratinocyte. Healing ulcers were present in the abomasal mucosa. In one animal, there was small intestinal villous atrophy, crypt dropout, dilation of some remaining crypts with attenuation of crypt epithelium and effusion of fibrin and neutrophils from the mucosal surface. There was a chronic bronchopneumonia in one animal, presumably due to secondary infection.

CENTRAL VETERINARY DIAGNOSTIC LABORATORY (Sue Friend, Peter Lording)

We have moved to 166 Union Road, Surrey Hills, 3127 and are now a wholly independent Veterinary Diagnostic Laboratory.

Phone: (03)888 5188
Fax: (03)888 5245

REGIONAL VETERINARY LABORATORY BA1RNSDALE

Bovine Chlamydial Abortion. (Ian Jerrett)

A foetus and maternal blood sample were received from a 2 year old heifer which aborted at 5½ months gestation. The foetus had firm mottled lungs and an enlarged liver. On histological examination there was severe pneumonia, meningoencephalitis and multifocal granulomatous hepatitis. No bacteria were grown from the foetal stomach contents but a Giemsa stain performed subsequently revealed numerous elementary bodies. Fluorescent antibody staining identified the infective organism as Chlamydia sp.

Blood samples taken from the heifer at the time of abortion and two weeks later showed a rise in titre from 1:16 to 1:64 in a Chlamydia CFT performed at ARVC Orange, using antigen prepared by the laboratory. Nine other unaffected heifers in the group showed titres ranging from 1:8 to 1:32. Both the aborting and the in-contact cattle were seronegative by the CSL Chlamydia CFT. CSL report that their antigen fails to produce a significant antibody response when inoculated into cattle, but appear to be unable or unwilling to improve the sensitivity of their test.

Toxoptasma Abortions (Ian Jerrett)

Four of 350 mature Elliotdale ewes aborted over a 1 week period. The ewes were 5-6 years old and aborted at 4 -4½ months gestation. Three placentas were received and all had small pale foci 1-2mm diameter throughout the cotyledons. Three foetuses examined were all normal.

Histological examination showed foci of necrosis and mineralisation in the cotyledons and immunoperoxidase staining demonstrated many Toxoplasma cysts in necrotic areas. In all three cases foetal brain and other tissues were normal and Toxoplasma gondii titres in foetal heart blood were <1:64 by the haemoglutination-inhibition test. In this series of abortions the diagnosis would not have been established if the placentas had not been received.

Coronavirus and Neonatal Diarrhoea in Calves (Ian Jerrett)

Coronavirus-like particles have been detected in a large proportion of diarrhoeic neonatal calves on four properties in a localised area of central Gippsland and in single samples from 3 other properties. While pathogenic strains of coronavirus have been identified in North America and Europe the pathogenicity of Australian strains has not been proven. In one of the above outbreaks of white scours, colonic lesions as described for coronavirus infection overseas were seen in 2 fatally affected calves. Coronavirus-like particles were detected in only one of the two calves but were also present in 11 of 18 samples from in-contact diarrhoeic calves on the property.

Cryptosporidia were also found in many of the faecal samples but were not present in the necropsied calves.

Rotavirus and Cryptosporidia continue to be the most common pathogens detected in cases of white scours. Testing for K99 E.coli has been performed by Bendigo laboratory on samples from a limited number of Gippsland properties but this pathogen has rarely been detected.

VETERINARY RESEARCH INSTITUTE PARKVILLE

Rainbow Ulcer Disease (Grant Rawlin)

Several fish breeders in the Melbourne area have noted a skin disease affecting colonies of New Guinea Rainbow Fish, Melanotaenia spp. The syndrome has been named 'Rainbow Ulcer Disease' by breeders. Owners have noted the disease is passed only between Rainbow fish in a tank containing several fish species. The adult fish are approximately 15 cm long, silver with multicoloured tones. Affected fish presented with multiple red, 1 cm ulcers in the skin, ventral to the gills and anterior to the tail.

A skin scraping was conducted during the post mortem examination which showed the presence of branched fungal hyphae. Secondary superficial fungal invasion is not unusual in skin diseases in fish and care must be taken when deciding on the importance of fungus in a skin scraping.

Histopathology was carried out and sections of skin and underlying muscle were stained with H & E, PAS and Z-N stains.

Focal granulomatous lesions with small necrotic areas were seen in the dermis and muscle. There was some acute inflammatory cell infiltration in these tissues. Around the edges of the necrotic foci were pleomorphic rounded organisms staining PAS positive.

Samples were taken for both fungal and bacterial culture. No fungus grew on Sabouraud Dextrose Agar with or without Actidone. There was a light growth of Aeromonas hydrophila. which is normally of little clinical importance.

It would appear that Rainbow Ulcer Disease is a deep mycosis, however the fungus involved is as yet unknown.

Lymphocystis Disease in a Gourami (Grant Rawlin)

Gouramis are an ornamental fish commonly kept in aquariums. This fish presented with small papilloma-like lesions on the posterior half of the body with the greatest concentration just on and anterior to the tail.

Sections of skin and muscle were taken for histopathology. Examination showed areas of epidermis interrupted by groups of very large (>200$_{\mu}$m) cells. These "lymphocysts" are basophilic with variable numbers of acidophilic inclusions. The cells have a thick PAS positive capsule. The capsulated cells are surrounded by acute inflammatory cells.

This disease is caused by a DNA virus which invades fibroblasts causing the enormous cell swelling. The disease has been reported both in wild and aquarium fish in Australia and overseas. It is thought there are several viruses involved which do not cross-infect families. Transmission is via skin abrasions. Lesions can also be seen in the viscera after haematogenous spread.

There is no treatment for this condition although lesions usually slough and recovery is complete.

Mycobacterial Infection of Fish (Grant Rawlin)

A formalin-fixed aquarium fish was presented for investigation of ulcerative lesions on the flanks. Ulcers were approximately 0.5 cm in diameter close to the pectoral fins.

Sections were cut of the affected area and were stained with H & E, PAS and Z-N. Histological examination showed granuloma formation in the dermis and muscle.

Acid-fast bacilli were common within the granulomas. Some dermal lesions also showed some PAS positive organisms.

The signs were interpreted as a Mycobacterial infection with secondary fungal involvement. These Mycobacteria species have been reported to be involved in the above syndrome, sometimes known as "Fish TB". M.tuberculosis can infect fish, however natural fish pathogens are M.marinum and M.fortuitum. Both the latter organisms can cause skin infection and delayed hypersensitivity reactions in humans.

VETERINARY CLINICAL CENTRE. WERRIBEE

Intestinal Mast Cell Tumour in a Cat (Jenny Charles)

A 15 year old castrated male domestic short haired cat presented with a 2 month history of vomiting after most meals. Abdominal enlargement due to ascites was observed and an exploratory laparotomy was performed after palpation of a mid-abdominal mass. The surgeon described envelopment of the anterior duodenum by coarsely lobulated firm homogeneous cream tumour tissue with marked diminution of the luminal diameter. Similar tumour masses were present in adjacent mesenteric lymph nodes and associated with the pancreas.

A scirrhous adenocarcinoma was the anticipated diagnosis, but biopsy material showed effacement of duodenal and lymph node architecture by diffuse sheets of pleomorphic mast cells. Tumour cells varied from round cells with distinct cytoplasmic boundaries, central oval muscles and clear or finely stippled cytoplasm to streaming spindle-shaped cells. Moderate numbers of accompanying eosinophils were irregularly distributed throughout the tumour.

Electron microscopic examination and metachromatic staining of sections are in progress to definitively eliminate a carcinoid tumour as a diagnosis.

Hypertropic Gastritis in a Snake with Cryptosporidium

Several tiger snakes from a reptile park at Ballarat showed persistent post-prandial regurgitation and variable emaciation.

The most severely affected was euthanased. Necropsy revealed marked dorsal muscle wasting, mild gastric mucosal thickening with accentuation of longitudinal rugae and a small volume of gastric luminal mucus.

Gastric sections revealed marked mucosal thickening, with hyperplasia of surface epithelium and often almost total replacement of glandular granular cells by mucous neck cells. Deeper glands showed frequent cystic distension. The lamina propria and submucosa showed moderate oedema and fibroplasia, with a mild diffuse infiltrate of plasma cells, with fewer lymphocytes and heterophils. Numerous spherical protozoal organisms were adherent to microvillus borders of the surface, pit and glandular epithelium, consistent with Cryptosporidium species.

An association of hypertrophic and mucous metaplastic gastric changes with Cryptosporidial infection is well-documented in reptiles, although much of the epidemiology of infection is unclear. Affected snakes tend to be mature, the clinical course is protracted and insidious and infection is presumed to be permanent. The longer clinical course in reptiles than in mammals probably accounts for the pronounced hyperplastic and regenerative changes in the former.

REGIONAL VETERINARY LABORATORY BENDIGO

Equine Herpesvirus Abortion (J F and R T Badman)

Equine viral abortion caused by equine herpesvirus was diagnosed at the Regional Veterinary Laboratory, Bendigo on the basis of histopathological and virological examination of specimens of 5 aborted horse foetuses from one Thoroughbred stud near Bendigo.

As at 22 August 1989, 3 healthy foals had been born and 10 mares are still in foal on the infected stud. Mares that aborted were healthy and cleaned normally - there were no discharges. The aborted foals appeared normal externally, but on post mortem examination the thoracic cavity contained an increased volume of straw coloured fluid and the lungs were firm in consistency. In some foals the livers were congested and in one, the liver was ruptured. Histopathology varied slightly, but was characterised by a diffuse pneumonitis with massive numbers of intranuclear inclusion bodies in macrophages and a bronchiolitis with intranuclear bodies in epithelial cells. In the liver there was occasional focal necrosis with hepatocytes on the periphery of the necrotic areas containing intranuclear inclusions.

Virological cultures were positive within 24-48 hours of inoculation.

The mares aborted in the last trimester of pregnancy, and serology was of no value in predicting which mares would abort.

Foetuses from 11 other properties have shown no indication of herpesvirus infection.

Isolation of an Atypical Actinomyces from a Dog (C J Lee, K McKechnie)

A two year old female Kelpie was presented with a history of depression, frothing at the mouth, tachypnoea and extensive fasciculation. The dog died before treatment could be administered. The dog was submitted for post mortem examination, with a provisional diagnosis of organophosphate poisoning.

Post Mortem Examination: The thorax was found to be completely filled with blood stained, purulent fluid containing many yellow sulphur-like granules Nocardia Sp. colonies. The lungs atelectatic with a large abscess in the right cardiac lobe. The parietal pleura mediastinum were extremely inflamed and thickened.

Histopathology: The spleen had a marked reduction in germinal follicles due to a lack of mature lymphocytes. Lungs were atelectatic with alveolar septa distended with proliferating alveolar macrophages and contained an abscess bordered with granulation tissue. There was a severe fibrinous pleuritis which contained multiple mixed colonies of Gram negative pleomorphic bacilli and Gram positive filamentous organisms. The provisional diagnosis was Nocardiosis.

Bacteriology: On anaerobic culture with CO 2 both Bacteroides melaninogenicus and an Actinomyces sp. was classified using API AN-Indent with the resulting Profile Number 4465573, which clearly identified the organism as Actinomyces odontolyticus.

Bergey (1986) stated that the natural habitat of A. odontolyticus is the oral cavity of man, that it rarely causes progressive actinomycotic infections and eye infections. More commonly, it is associated with periodontitis and formation of caries. The occurrence of A. odontolyticus is unknown in animals although experimental infection of laboratory animals has produced abscesses.

The pathogenesis of infection in this case is unknown, however, it appears that the organism once present is able to produce severe Nocardiosis-like pneumonia and pleuritis.

Reference: Bergey's Manual of Systemic Bacteriology. Volume 2:1415-1416 (1986).

A Drug related Anaphylactic reaction in a Horse (J M Lee)

History: A four year old Standardbred gelding was presented for post mortem examination with the following history.

The horse was suffering from an upper respiratory tract infection of three to four weeks duration. The private practitioner prescribed treatment with intravenous Norodine (Norbrook) a trimethoprim-sulphonamide. The drug was supplied to the trainer with instructions to give slowly intravenously.

When only 15mls had been injected the horse showed distress, pulled away and collapsed with severe gasping dyspnoea. The signs lasted several minutes before the horse asphyxiated and died .

Gross Pathology: All tissues were moderately autolysed. When exposed, the left jugular vein showed a puncture mark and associated haemorrhage and necrosis approximately 0.75m down from the angle jaw. The associated carotid artery showed no punctures or trauma.

The lungs were extremely congested with multiple superficial haemorrhages 2cm diameter under the visceral pleura. There were areas of emphysema of the lungs, especially dorso-cranially and of the mediastinum.

The pleura had multiple areas of thickening and fibrosis (3-4cm diameter) but no adhesions. There was a pericardial effusion and a serosanguinous pleural effusion.

Histopathology: Lungs showed marked congestion and bronchioles showed a reduction or collapse of their lumen.

Jugular vein showed perivascular necrosis unilaterally and extensive perivascular haemorrhage.

Bacteriology: The lung grew mixed coliforms and streptococci which were of doubtful significance.

Diagnosis: Probable anaphylactic shock.

Discussion: The above clinical and post mortem findings are typical of a Type I Hypersensitivity reaction (anaphylaxis) in horses.

The classical pathway for production of Anaphylaxis involves antigenic sensitisation of specific B lymphocytes which are transformed into IgE secreting plasma cells. The IgE binds to the plasma membranes of basophils and mast cells which are now sensitised to further antigenic stimulation.

Should the animal now be re-exposed to the same antigen, crosslinking of the attached IgE causes degranulation with release of vasoactive amines including histamine, prostaglandin, thromboxanes and leukotrienes.

The leukotrienes and histamine in particular are potent smooth muscle agonists.

This results in bronchoconstriction with severe dyspnoea and emphysema. Vasoconstriction of intestinal vasculature also leads to haemorrhage. If not arrested, Type I hypersensitivity may lead to death by asphyxiation.

An alternate pathway leading to mast cell and basophil degranulation due to the direct action of complement components has been recorded in the absence of IgE in previously unexposed animals. In this case activation of the complement cascade produces C'3a and C'5a portions (Anaphylotoxins). These react with specific receptors on the cell membranes to produce degranulation release of vasoactive amines.

Many drugs are known to induce hypersensitivity reactions, probably acting as happens which, if conjugated with large molecular weight compounds, eg. a protein, may act as an antigen. Antibiotics, especially penicillin, are frequently involved. Excess production of IgE is thought to be hereditary in some animals leading to atopy or the risk of anaphylaxis.

It is not known if this horse had been previously treated with Noradine or one of the other trimethoprim-su1phonamide drugs although it is highly likely.

Jim Rothwell, New South Wales.

REGIONAL VETERINARY LABORATORY, ARMIDALE

OUTBREAK OF EQUINE HERPESVIRUS ABORTIONS IN A NEW ENGLAND THOROUGHBRED STUD

(Stephen Love/Steven Hun)

An outbreak of equine herpesvirus (EHV)-l abortions is underway in a 100 mare thoroughbred stud in the New England area of NSW. The associated financial losses are considerable.

Foaling commenced uneventfully on the property early in August. The abortions commenced 2-3 weeks later, after a dozen or so normal parturitions. From that time until the present (mid September) there have been 10 abortions and 4 normal foalings.

All but one of the aborted foetuses have been examined at the RVL, Armidale. Necropsy findings, although varying in character and extent, have been consistent with EHV-1 abortion. Most foals have been near term or within a month or two of term. Some were presented still within the foetal membranes. All foetuses were quite fresh with no sign of autolysis, suggesting that death occurred at or around the time of expulsion.

Oedema and congestion of various tissues and organs, particularly the lungs, have been the most common findings at necropsy, A clear yellow pleural effusion, quite variable in volume, has also been a fairly consistent feature. Degrees of jaundice have been noted in a number of cases. Gross liver pathology has been somewhat more variable than some descriptions of the disease would suggest. A third of the foals so far have had congested livers with no obvious discrete lesions, another third have had scattered pale foci, while the rest have presented with livers peppered with obvious white to creamy focal lesions. Subcapsular oedema has been present in the livers of all foetuses. Changes in the lung have been similar in all foals and have consisted of congestion, focal haemorrhages and prominent interlobular oedema,

Histologic examinations to date have revealed scattered focal to locally extensive areas of necrosis, the extent and severity of the lesions correlating fairly well with necropsy findings. Bronchial and bronchiolar necrosis as well as oedema have been common findings in the lung. Necrotic foci usually have been encountered in the liver, and in a few foals there has been extensive necrosis of the splenic white pulp.

Virology has been completed on tissues from the first four aborted foetuses; the presence of EHV-1 has been confirmed in each case. Bacteriology as expected has been unremarkable.

The affected stud is an intensive operation run under close veterinary supervision. In Autumn pregnant mares are assigned to one of several paddocks. From then until after the completion of foaling, there is no direct contact between paddocks.

Each paddock is 7-10 hectares and contains irrigated pasture. Supplementary feeding is carried out daily (troughs). The stocking rate is 2-3 mares per hectare. The usual routine procedures are followed, including anthelmintic treatment of all horses by stomach tube every 8 weeks.

The practitioner in this case, an experienced equine veterinarian, has commented that normal stud operations may augment the transmission of pathogens such as EHV-1.

Also worthy of note is the fact that some affected foetuses may present with relatively subtle gross lesions. As a result a diagnosis of EHV-1 abortion may well be missed early in an outbreak or where abortions are sporadic, particularly if the investigation ends at necropsy.

COLLECTING URINE FOR LEPTOSPIRE DIAGNOSIS (S. Hun and Carol Quinn)

Leptospirosis is usually diagnosed serologically, but from time to time we receive other samples, usually urine samples, for the diagnosis of the disease. Some recent experience suggests that there may be ways of increasing the chances of success with such samples.

L. hardjo infection was diagnosed serologically on a dairy farm. MAT titres ranged from 100-900 and the disease was associated with milk drop syndrome, but abortions also occurred. Interestingly, the farmer was hospitalised for leptospire infection. We visited the farm in an attempt to isolate the organisms from sero positive animals. We sampled six animals from the herd taking milk and urine samples. To collect urine we injected the animals intravenously with diuretic and collected a mid-stream sample from the copious urine flow which resulted.

We succeeded in identifying leptospires from all the animals sampled. Dark ground examination of the urine revealed leptospires in two samples and culture of the urine in selective medium resulted in the growth of leptosopires in these two cases and in another three. Leptospira was grown from the milk but not in the urine of the remaining animal. The organisms seen in the urine by dark ground examination were present in large numbers and were seen much more easily than in most of the samples which we receive for this type of examination. They were all dead when examined the day after collection.

We drew the following conclusion from this exercise. Firstly, if the urine is submitted for Leptospira detection or culture then it is worthwhile using a diuretic to obtain the sample because it will contain larger numbers of the organisms than would usually be the case and contamination will be minimal. Secondly, culture of the urine results in isolation of the organism even in cases where it is not seen under dark ground examination of the sample. Thirdly, selective media should be used at the time of sampling for successful isolation. An improved selective median is now available.

Ref, B. Adler et al, "Development of an improved selective medium for isolation of Leptospires from clinical material (1986), Veterinary Microbiology, 12, 377-381.

REGIONAL VETERINARY LABORATORY, ARMIDALE

JOHNE'S DISEASE POSITIVE CALVES (S Hum)

Recently two 10 day old dairy calves underwent routine regulatory testing for Johne's disease prior to shipment to Queensland. One calf showed a CFT titre of 8, the second a titre of 32. The dams of these calves (first calf heifers) came originally from a Victorian farm of unknown status with regard to Johne's disease. Six weeks after the initial test the dams and calves were re-tested for Johne's disease and all appeared to be negative.

Since laboratory error in the first test has been excluded it is interesting why these young calves showed high CFT titres. In light of the second test it appears to be a false positive reaction. There may have been cross reacting antibodies concentrated in the colostrum which passively immunised these calves. An alternative hypothesis would be that subclinically infected CFT negative females may build up sufficient antibodies in the colostrum to be measurable in their young calves. Investigations are continuing.

REGIONAL VETERINARY LABORATORY WOLLONGBAR

DURIKAINENA SP. IN A KOALA **(PHASCOLARCTOS CINEREUS)** (PA Gill)

A young female koala was found, on the ground, in poor condition. She became progressively weaker despite supportive treatment (multivitamins, antibiotics and supplemented feed) and was euthanased 10 days later. Pathological findings included chronic suppurative metritis/cystitis due to Chlamydia sp. infection and severe, acute bronchopneumonia. Adult helminth parasites identified as Durikainema sp. (David Spratt CSIRO Division of Ecology and Rangelands, Canberra) were present in several b1ood vessels in histological section of lung. Larvae (which could be mistaken for microfilaria) were also present in blood vessels in the bladder and cerebellum. This is the second record of Durikainema sp. from the blood vessels in the lungs of a koala (the first record was also made at Woolongbar) but in this instance the parasite was found in tissues other than lung.

I am grateful to Dave Spratt for the following information:

There are three known species of helminth parasite recorded from the koala, a large anoplocephalid cestode, Bertiella obesa, a nematode lungworm, Marsupostrongvlus longilarvatus and a muspiceoid nematode, Durikainema sp. An intact worm is required in order to determine whether the Durikainema sp. in the koala is D. macropodi, a parasite which has been found in the mesenteric and portal vessels of kangaroos and wallabies.

REGIONAL VETERINARY LABORATORY WAGGA WAGGA

(John Glastonbury)

CATTLE

Abortion/Parturient Deaths.

In July and August 135 serums were received from 32 farms for investigations of isolated cases of abortion. Of the serums 17 and 6l yielded titres of <1:30 in the microscopic agglutination test for Leptospira Pomona and Leptospira hardjo, respectively. They all yielded negative results in the ELISA test for· Brucella abortus. On the basis of the GDPT involvement of pestivirus was indicated in one case.

Aerotolerant Campylobacter Isolated from Bulls. (Ian Links)

Since 1987 there has been a dramatic increase in submission of preputial scrapings for Campylobacter culture from bulls requiring accreditation to enter an Artificial Insemination Centre serviced by RV1, Wagga Wagga. At the same time there have been a number of submissions from cases of bovine infertility (Table 1).

Table 1. - Number of preputial scrapings submitted.

Year	Regulatory		Diagnostic	
	Beef	Dairy	Beef	Dairy
1987	30	30	13	2
1988	57	95	25	-
1989	153	142	23	9

Since May 1989 there have been a number of isolations of aerotolerant Campylobacter spp from preputial scrapings. These isolates can be differentiated into 2 groups:

Group A - biochemically consistent with isolates described in the literature as associated with reproductive problems (abortions) in cattle. These isolates do not grow at 43°C. Some isolates produce hydrogen sulphide after 5 days incubation and some grow in the presence of 1% glycine.

The source of these isolates is shown in Table 2.

Table 2. - Aerotolerant Campylobacter isolated from cattle.

Year	Regulatory		Diagnostic	
	Beef	Dairy	Beef	Dairy
1987	-	-	1A	-
1988	1A	-	-	-
1989	5B	1A, 11B	1B	-

A - No growth at 43 degrees C.
B - Grow at 43 degrees C.

* - number of isolates
** - includes isolation from one animal on 3 occasions.

The reason for the spate of aerotolerant Campylobacter isolations has not been determined.

SHEEP

Neonatal Mortality

A total of 115 neonatal lambs were received for post mortem examination during July and August. The majority either came from the "Hyfer trial" at the Agricultural Research Institute or from the flock at the Charles Sturt University. Post mortem examinations found evidence of the following:

Diagnosis	Number of lambs
Starvation	32
Predation	21
Foetal hypoxia	23
Dystocia	18
Exposure	15
Preparturient deaths	5 (1 litter)
Septicaemia (Erysipelothrix rhusiopathiae)	2
Goitre	1
Unknown	9

Abortion

Listeriosis abortion rates of 42.9% (63 ewes) and 0.6% (1,100) were attributable to listeriosis on 2 farms. Heavy growths of Listeria monocytogenes were isolated from material submitted from both farms. Histological examination found focal embolic necrotising inflammation in the liver, lung, kidney and spleen as wel1 as segmental necrotising placentitis.

Toxoplasmosis of the first 22 lambs to be born to 310 maiden ewes, 21 were born dead prematurely. Seven lambs were submitted for postmortem examination and the heart bloods of 2 yielded positive results in the latex test for Toxoplasma. gondii. Two brains were examined histologically and showed focal leucoencephalomalacia and cerebral mineralisation. Segmental necrotising placentitis was observed in the one placenta submitted. During pregnancy these ewes had been hand fed and the storage area was inhabited by a large population of cats.

Phytogenous Chronic Copper Poisoning

No doubt the lush stands of subterranean clover presently found throughout the Region have been responsible for this condition which was diagnosed in material submitted from 7 farms during August. Mortality rates varied from 2.8 to 9.7% and on 6 farms crossbred sheep were involved while Merinos were affected on one. The diagnosis was indicated by the histological findings of severe haemoglobinuric nephrosis and periacinar

hepatocyte necrosis with bile stasis. Mild evidence of the ingestion of pyrrolizidine alkaloids was detected in the livers submitted from 6 farms.

GOATS

Atropa belladonna. Poisoning

Three Cashmere bucks developed depression and blindness after gaining access to a stand of "deadly nightshade". Gross pathological findings were dominated by pulmonary oedema and numerous serosal haemorrhages. Polioencephalomalacia and a focal degenerative myocardiopathy were found microscopically. Polioencephalomalacia appears to be responsible for the previously reported blindness in this condition.

BIRDS

Exudative Diathesis

Two five-day-old ostrich chicks were examined at the laboratory. Post mortem findings were dominated by the presence of generalised severe gelatinous subcutaneous oedema and ascites. Skeletal muscles and brains appeared normal histologically. It would seem that these birds are particularly prone to deficiencies of vitamin E/selenium.

Lorna Melville, Northern Territory.

BERRIMAH AGRICULTURAL RESEARCH CENTRE

SUSPECT RUMENSIN POISONING (Morton Bell)

Heavy mortalities were observed in a buffalo feedlot south of Arnhem Land. All buffalo were male and between 2 and 4 years old. The total mortality was 27 out of 42 head. Deaths occur in all pens. The animals became weak and died suddenly. One month previously all animals had received botulism C & D and 5 in 1 vaccine and were drenched with Avomec.

It was very wet weather. The feeding management left much to be desired, a fine 5mm screen was being used on the hammer mill instead of the normal 15 mm one. This produced a very fine, powdery feed. Rumensin 10% had been started one week earlier at 1½ kg per 7 head. This was not mixed with the feed but sprinkled over the feed in the trough.

Two animals were necropsied. The animals were dehydrated with sunken eyes. Body condition was reasonable. The main findings were impaction of the rumen, oedema of renal pelvis, pleurisy and dust in bronchi.

The main histological features involved the liver with moderate centrilobular multifocal necrosis of small numbers of hepatocytes and nuclear swelling of scattered hepatocytes. There was a mild diffuse infiltration of mixed inflammatory cells. In the kidney the glomeruli were shrunken with proteinaceous material in Bowman's space, distension and disruption of some proximal tubules and nuclear swelling of some tubular cells. There was mild focal swelling of myofibril bundles in the heart of one animal.

The very fine, powdery, low quality feed had resulted in impacted rumens. The rumensin was being administered at 40-50 times recommended dosage. Much of the feed was not eaten, possibly due to rumen impaction or the high concentration of rumensin on the feed?

Monensin (rumensin) toxicity causes muscle necrosis. This was not evident at post mortem and no skeletal muscle was received. Only mild myofibril swelling was seen in one heart. The histological picture was suggestive of a cumulative toxicity, whether this was due to exposure to toxic plants prior to entry into the feedlot is uncertain. Rumensin at this concentration must have played a role in this high mortality.

LYMPHOCYTIS DISEASE IN THE NORTHERN TERRITORY

(M Pearce)

In May , 1989, lymphocystis disease was diagnosed in three barramundi (Lates calcarifer, Bloch) held at a local aquarium facility. The fish had been -held in quarantine for 3-4 weeks prior to introduction into the main aquarium with two bream (thought to be Acanthopagrus berda, Forskal). The curator of the aquarium stated that one of the bream had lesions very similar to the barramundi, but the bream was not sighted by the author.

Lymphocystis disease of fish is caused by infection with a virus of the family Iridoviridae. The disease has been reported throughout the world in a number of fish species, both marine and freshwater. To date, the disease has not been reported in native fish populations in Australia except an isolated incident in Sydney Harbour in 1985 (G. Reddacliff, per. comm.) The disease has, from time to time been seen in imported aquarium species in quarantine and, rarely, in captive exotic species that have passed through quarantine.

It is thought that there are different strains of the virus which may explain the apparent differences in host susceptibility to the disease. In experimental infections, Nigrelli (1952) showed the incubation period to be as little as 10 days, while Wolf (1962) found the range in incubation period to be 37-72 days at 12.5° C, but as short as 17 days at 25° C. Leibovitz (1980) describes the Incubation period as very long - usually many weeks. The mode of infection remains uncertain. Leibovitz (1980) states that the virus may be introduced in infected fish or fish products and the transmission of the virus is accelerated when water temperatures and stocking densities of fish are high. The disease usually runs a chronic course and is seldom fatal (Pilcher and Fryer, 1980). In aquaria, however, the disease may be more serious, with high mortality (Leibovitz, 1980).

The virus infects fibroblastic cells, allowing them to continue enlarging, but it prevents cell division. Consequently infected cells may reach sizes from 300-5000mm in diameter (Nigrelli, 1952; Wolf, 1962). In the affected barramundi, the affected cells appeared grossly as focal, pale nodules, 0.5-1mm in diameter on the skin surface, particularly on the fins and tail. The more advanced lesions had the appearance of tumours up to one centimetre across, with a cauliflower-like surface. Histologically, the lesions consisted of greatly hypertrophied cells, up to 750 mm across. The cytoplasm of infected cells was granular and slightly basophilic, and the nucleus hypertrophied; the cell was surrounded by a thick, hyaline capsule, 7mm wide. Hypertrophied cells were densely packed and the surrounding tissue had been invaded by numerous mononuclear inflammatory cells.

Material was forwarded to AAHL for electron microscopic examination. Numerous virus particles were found in hypertrophied cells. The virus was icosahedral and 225 ± 6 mm in diameter (vertex to vertex). A 46 ± 4 mm fringe surrounded a complex double-layered membrane which possessed sub-units of 3-5 mm in diameter (A. Hyatt, pers. comm.). These virus particles were consistent with descriptions given of lymphocystis virus (Pilcher and Fryer, 1980).

The source of the infection remains unknown. No fish were found with lymphocystis lesions following sampling of local creeks from which the affected fish and in- contact fish had been taken, Lymphocystis had not previously been seen in the Northern Territory and was tantamount to an exotic disease nationally. The affected aquarium was therefore destocked and disinfected. There have been no further reports of lymphocystis disease in the Northern Territory.

References

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Fraser Trueman, Queensland.

YEERONGPILLY VETERINARY LABORATORY

Swine Dysentery Mortality

A severe outbreak of Swine Dysentery was diagnosed when 270 of 600 grower pigs died over a ten day period. Clinical and necropsy findings were those of classical swine dysentery, and Treponema hyodysenteriae was readily isolated from large intestinal contents.

Unfortunately, most deaths occurred before the owner submitted pigs for necropsy, and correct treatment was provided. Two weeks before the outbreak, the owner had assisted with post mortems on a neighbouring property where a similar syndrome had caused 70 deaths in grower pigs.

Psittacosis - Fluorescent antibody Testing

Psittacosis has been commonly diagnosed in native birds and pet species. Necropsy examination in many cases revealed splenomegaly, hepatitis and air sacculitis. The fluorescent antibody test has been of great value in providing a rapid accurate diagnosis.

Over 60,000 pet fauna are introduced into Queensland each year from southern States, and the stress of capture and transport is a likely contributing factor to Psittacosis in pet shops and recently purchased birds.

Mycobacteriosis in rainbow fish

A breeder of the native rainbow fish, Melanotaenia splendida, submitted five individuals that had developed 0.5 to lcm diameter white skin ulcers from small red foci on the abdomen in three weeks. All fish were feeding well and active. Histologically, multiple soft tubicles containing acid-fast organisms were present in head and caudal kidney with a granulomatous inflammation surrounding the granulomas and, in one fish, extending to involve pericardium and gill arch connective tissues. Affected skin lesions showed a granulomatous inflammation which disrupted normal skin structure.

Mycobacteria sp. was isolated from skin lesions and kidney tissue confirming a diagnosis of Mycobacteriosis.

Edwardsiella tarda infection in an eel.

Edwardsiella tarda was isolated from a muscle abscess in an eel submitted from an exporter who held wild-caught eels in small ponds prior to export, it was reported that 0.5 to one percent of the eels would develop skin lesions during captivity. Histologically, a fibrino-purulent inf1ammation was present at the abscess site below the vertebral column in the midtail region. This isolation of Ed. tarda represents the first record of edwardsielliosis in an Australian native fish.

UNIVERSITY OF QUEENSLAND

Corynebacterium pseudodiphtheriticum endocarditis in a cow (Roger Kelly)

A 6-year old Limousin cow died of congestive heart failure after intermittent malaise and fever over several months, which was only partially responsive to antibiotics. As well as the expected necropsy findings of ascites, nutmeg liver, etc, there were bulky masses of pale, firm platelet thrombus on the cusps of both AV valves. The right atrio-ventricular orifice was almost obstructed, but there had been no cardiac murmurs on auscultation. There was septic infarction of lung and kidney. A heavy growth of C. pseudodiphtheriticum was cultured from one of the affected valves.

Verminous myelitis in a horse due to Angiostrongylus cantonensis (Roger Kelly)

An Appaloosa colt, 8 months old, showed rapidly progressing paresis for 14 days. It seemed to respond temporarily to steroid therapy, then became laterally recumbent and developed nystagmus and urinary incontinence and was killed. At necropsy there was moderately severe verminous arteritis of mesenteric arteries, but not the aorta, due to S.vulgaris. There were small haemorrhages in the cerebellar leptomeninges, and, at several levels in the spinal cord, some linear areas of poliomyelomalacia. Eosinophil infiltration in these areas was not obvious, but some sections contained slices of nematodes. Segments of formalin-fixed cord were dissociated and yielded segments of filarioid nematode which identified by Alan Waddell and Dave Spratt as Angiostrongylus cantonensis. How this parasite, the rat lungworm, whose intermediate host is a mollusc, came to infect the colt is a bit of a mystery.

Congenital cardiovascular anomaly in a koala (Roger Kelly)

A young koala failed to thrive and was about half its expected body weight at 10 months of age, with a rapid respiration rate and a loud heart murmur and increased pulse amplitude. At necropsy, the heart was greatly enlarged due to dilatation and hypertrophy of the right ventricle. There was a large patent foramen ovale, and pulmonary arteries were grossly distended, right down to the smaller branches. There was no significant ascites or passive venous congestion of the liver. Histologically, the pulmonary arteries were invested with a heavy fibrous sheath. The primary defect appeared to be at the level of the pulmonary microcirculation, causing pulmonary hypertension, persistent foramen ovale and cor pulmonale.

Familial epidermal epidermolysis in goats (red-foot) (Roger Kelly)

Several related Angora kids have shed one or both claws of their hooves, as well as developing oral mucosal ulcers. Some have been born with distorted ears, apparently because of intrauterine loss of epidermis followed by pre-natal healing. Histologically, skin and adnexa seem to separate from the dermis along the basement membrane. The condition has been provisionally named familial sub-basal epidermolysis.

Ruth Reuter, Western Australia.

ALBANY REGIONAL LABORATORY

PROGRESSIVE SPINAL MYELINOPATHTY IN CATTLE (R. Reuter)

A 14 month old Murray Grey bull was submitted to the laboratory with a history of rapid severe weight loss over the previous six weeks. A similar syndrome had resulted in the death of five other calves in the herd this year. In previous years one or two had been affected. The animals were all males. There was no evidence of scouring. On post mortem lesions were restricted to the nervous system where degenerative changes characteristic of progressive spinal myelinopathy were found. The lesions seen were identical to those described by Richards and Edwards In 1986. Although most of the animals seen by those authors were much younger, one of their cases was a 2 year old steer. On questioning the owner further, he reported that three or four calves each year were born "stiff" but appeared to "grow out of it". He could not say if this bull had been one of those.

Reference

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POST SHEARING DERMAL NECROSIS IN SHEEP (R. Reuter)

One hundred and fifty yearling wethers from a mob of 1200 Merinos developed swelling and scabby lesions on the tail one week after crutching by contract shearers. Some of the lesions were discharging purulent material. The mob had been crutched between rainstorms. Sections of the dry gangrenous areas on the tail showed necrosis of the epidermis with suppurative inflammation and large collections of bacteria in the dermis. Culture resulted in a heavy growth of Bacteroides in conjunction with small numbers of Staph. aureus, and Erysipelas. Since Bacteroides is a common inhabitant of the soil and intestinal tract, it was probably a contaminant of a shearing injury.

LAMENESS IN LAMBS AT WEANING (R. Reuter)

Four 6 week old lambs were submitted from a flock which had experienced a persistent problem with lameness around the time of weaning. The flock had been line bred for approximately 40 years. On post mortem, one lamb had a subaortic myocardial defect, one had spinal cord compression due to severe kyphosis, one had polyarthritis involving carpal and tarsal joints. The fourth lamb had no visible lesions. Histophilus ovis was isolated from the joints of the lamb affected with arthritis, however it is possible that inbreeding was a predisposing factor in this case.

Judith Handlinger, Tasmania.

MT. PLEASANT LABORATORIES - LAUNCESTON

WOMBATS and VITAMIN D DON'T MIX! (Dave Obendorf)
Abnormal soft tissue calcification, nephrocalcinosis and osteodystrophic boney changes (particularly of the sku1l and mandible) have been seen in several captive wombats (Vombatus ursinus). Over-zealous supplementation of milk replacer diets during orphan rearing and subsequent maintenance on dried dog foods is the usual accompanying history.

SNAKES AND LUNG PENTASTOMES (Dave Obendorf)
An adult male Tiger snake (Notechis ater) died as a result of a lung haemorrhage inflicted by several pentatomic (tentatively identified as Waddycephalus sp.) These large worm-like arthropods (a form of mite) produce deep ulcerative craters in the lung tissue and appear to ingest blood. Large specimens can severely damage the lung parenchyma, sometimes burrowing through the lung and emerging in the coelomic cavity. Adult patent pentatomic infection can be detected by faecal flotation and examination for characteristic ovoid eggs containing developed larvae. Any information on an effective treatment, short of open lung surgery, would be gratefully accepted.

CHRONIC PERITONITIS IN A PILOT WHALE (Dave Obendorf)
Occasionally one finds some interesting pathology in stranded pilot whales. In this case, a thick meshwork of fibrous adhesions had anchored a portion of the small intestine to the abdominal wall. The adhesions caused some narrowing of the effective gut lumen but did not appear to obstruct ingesta flow. The focus of the peritonitis was associated with a deep skin wound to the side of the abdominal wall, probably caused by the parasitic shark (Isistus sp.) The deep craterous wound produced by these sharks resolve with time to form depigmented skin scars. The wounds may extend into the blubber layer for 3-4 cm. Secondary bacterial infection and cellulitis of such wounds is sometimes seen.

POSSIBLE CEREBELLAR ABIOTROPHY IN PIGS (Dave Obendorf and Judith Handlinger)
An intensive piggery recently has had a worrying number of pigs displaying CNS signs. The condition is first noticed in weanling pigs and appears to become progressively worse with age. Animals display unco-ordination, swaying, propped wide legged stance, high stepping gait and bouts of shivering over the body. Older pigs display hind leg weakness and recumbency. Histologically, pathological lesions are restricted to the cerebellum, with Purkinge cell depletion and degeneration, a relative depletion of granular cell population, and the presence of numerous swollen axons in the granular layer.

It is highly likely that the condition is heritable and is expressed in the homozygous recessive form. The condition is seen in several piggeries with Canadian Yorkshire and Yorkshire cross pigs in Queensland. Spread to other piggeries in Australia via semen from heterozygous carrier boars is likely. Piggeries breeding from homebred sows may allow for the expression of the disease. In this instance, only one or two piglets/litter was affected from certain sow/boar matings. Further analysis of the breeding history is underway. Our thanks to John Gibson, Toowoomba Vet. Lab. for reviewing the cerebellar lesions.

YERSINIOSIS - NEW EXPRESSIONS? (J Handlinger & D Obendorf)

This year we have seen two expressions of Yersiniosis which we have not previously encountered.

Yersiniosis in pigs is described in other countries, but reports in Australia suggest that Yersinia can be detected in Australian pig faeces but without evidence of clinical illness. This year we found Yersinia pseudotuberculosis Type III associated with at least five piggeries reporting diarrhoea, poor growth rates and deaths. Isolation was mainly from faecal samples and to date only three chronically affected and one acutely affected pigs have been available for histopathology. The chronically affected pigs showed villous atrophy and a generalised mononuclear infiltrate plus focal polymorph distension of crypts. Focal Balantidium was also present. The acutely affected piglet showed multiple intestinal microabscesses identical to the yersiniosis lesions seen in ruminants.

Yersinia was generally the only enteric pathogen isolated and all responded well to in-feed terramycin treatment. However, other infections such as Pasteurella or Bordetella pneumonia were commonly present, and most outbreaks were associated with poor management, especially over the winter period. There was no common factor except in one case of pigs supplied by another affected piggery. We could not isolate Yersinia from feed or meatmeal.

Do others have experience of Yersiniosis in Australian pigs?

Yersinia pseudotuberculosis abortion was also seen in a sheep and a goat from separate properties. Y. pseudotuberculosis Type II was isolated from the kid. Y. pseudotuberculosis Type I isolated from the lamb. Both foetuses showed miliary hepatic lesions. Histopathology of the kid showed typical yersiniosis microabscesses throughout the placenta, liver and lungs.

CAMPYLOBACTER ABORTION IN GOATS (J Handlinger & S King)

When we sent out a diagnosis of Campylobacter abortion for a kid, we were initially surprised when the submitting vet responded with enthusiasm to this rare diagnosis in goats. However, subsequent searching of our records confirmed that although we routinely consider this as a differential diagnosis in goat abortions, and have diagnosed large numbers of goat abortions and large numbers of Campylobacter abortions, this was our first such diagnosis in goats.

This outbreak actually involved the loss of at least 40 of the 60 kids dropped in the affected mob of Angora goats. These goats had been run with sheep which had earlier shown a small abortion storm, suspected to be also due to Campylobacter though no samples were submitted. Initially most of the kids were born full term, but died without getting up. Later the kids were aborted before term after dying in utero. One doe which had been agisted on this property but removed about one week after the first diagnosis, aborted dead-in-utero kids four weeks later. The kids were suspected to have died a few days earlier.

Campylobacter was again recovered from the abomasums. No specific gross or histological lesions were seen in the kids. Placenta was only available from the later autolysing aborted foetuses. These were autolyzed but showed a diffuse hypercellularity.

JOBLINE

REGIONAL VETERINARY LABORATORY. BENALLA
VETERINARY PATHOLOGIST

An opportunity exists for a suitably qualified mate or female veterinary officer to join the diagnostic team at the Regional Veterinary Laboratory, Benalla for a period of 5 months.

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Benalla is a pleasant country town of 9,000 people within easy access to Melbourne and close to the mountains, bush, trout streams and vineyards.

Salary — Range - SCI(VET)1, $25,963 - SCI(VET)4, $47,005
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To supervise and undertake veterinary laboratory diagnosis, investigation, and advise on the treatment and control of diseases in animals.

To provide specialist veterinary advice to officers of this Department, other Departments, private veterinarians and the public.

QUALIFICATIONS:

Mandatory: A degree in Veterinary Science registrable in the State of Victoria.

Desirable: Post graduate qualifications in Veterinary Pathology, Biochemistry or Haematology.

Practical knowledge of livestock industries; experience in the diagnosis, epidemiology and control of significant livestock diseases; well developed interpersonal and communication skills and ability to work as part of a multidisciplinary team; ability in the management of people and/or resources.

Written applications are invited from men and women, quoting Position Number 00/00/5532/0 and giving details of qualifications and experience and a business hours contact telephone number.

Applications to Dr. R, Rubira, Department of Agriculture and Rural Affairs, P.O. Box 388, Benalla, Victoria, 3672.
Further information can be obtained from: Dr. R. Rubira (057) 62 2933.

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Department of Agriculture and Rural Affairs

POSITION ANNOUNCEMENT

Department of Veterinary Science
The Pennsylvania State University
University Park, PA 16802

Position: Veterinary Pathologist

Available: 1 January 1990

Salary and Rank: The appointment is a tenure-track position at the Assistant Professor/Associate Professor level depending on qualifications on a 48-week salary plan. Salary and precise rank will be commensurate with the qualifications of the applicant.

Duties and Responsibilities: The Veterinary Pathologist is responsible for the development and co-ordination of an independent and collaborative research program; for provision of pathology consultation to Departmental diagnostic programs and pathology support for the Laboratory Animal Resources Program; and for teaching at the graduate level.

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Application Procedure: All applications will be accepted until 1 December 1989 or until the position is filled. Interested persons should send a letter of application with detailed curriculum vitae and the names of three references to:

Dr. F. S. Ferguson, Chairman, Search Committee
207 Centralized Biological Laboratory
The Pennsylvania State University
University Park, PA 16802

Further information and a more complete position description is available from Dr. F. G. Ferguson.

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