VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
New South Wales Agriculture
Elizabeth Macarthur Agriculture Institute
Private Bag 8
Camden NSW 2570

Registered by Australia Post Publication No. VBG 6333

EDITOR: Gary Reddacliff

Number 33 February, 1992

PAGE CONTENTS

DEADLINE FOR MAY CONFERENCE PAPERS IS - MARCH 31, 1992
DEADLINE FOR NEXT VET. PATH REPORT IS - JUNE 15, 1992

FROM THE PRESIDENT

Greetings and best wishes for 1992 from your ASVP executive. VPR No 33 brings you a thick wad of developments from members Australia wide. Can I suggest that State Correspondents in future might consider including in the Report news of pathologists on the move, reports of group meetings or Registry training events and developments in their own organisations etc. for the interest and awareness of members interstate. This could alert us all to policy or industrial matters of (growing) concern.

It will be just 2 clear months until 9-10th May by the time you read this report.

PLAN NOW FOR ASVP ANNUAL CONFERENCE, ADELAIDE 1992!

We need to know your meeting/dinner attendance intentions, accommodation needs and proposed scientific contributions. Please use the back page tear-off slip to register your intention **NOW**. Full registration details will be available in the next mailing.

March 31st is the deadline for abstracts/papers to be included in the conference proceedings - one to two pages of original typewritten material on A4 paper, suitable for photo-reproduction. (Please note that this is also the standard preferred for articles for the VPR. Poor faxes, scribbled corrections etc. just make extra work for what is a voluntary group of State correspondents and the VPR editor!)

Your executive has allocated $100 as the peer reviewed "Best Member's Case Presentation Award" which will honour Bill Hartley's retirement from full-time duty with the Pathology Registry in June 1992. Be in it to win it in 92!

But what to present? Remember the theme is "Bone and Joint Pathology" with Professor Vernon Roberts, Professor of Pathology, University of Adelaide as the theme lecturer, supported by Dr Craig Riddel from Saskatchewan (author of "Avian Histopathology") and Dr Garry Cross (University of Sydney, speaking on pigs). Unfortunately Dr Rolfe Howlett will be overseas and unavailable. Local co-ordinator, Peter Philips has organised a guided tour of the specialist bone pathology unit at IMVS. You will be able to see the latest techniques in processing and examination of bones and joints. Members are invited to present material on this theme in any species, which will occupy most of Saturday. The AGM, leading on to dinner is planned for Saturday afternoon/evening and Sunday is free for members' presentations on any or all subjects. Please get your best, newest or most erudite discoveries down on paper and send in with your intentions for the conference as above.

I look forward to seeing you in Adelaide shortly.

Keith Walker
ASVP President 1991-93

FROM THE EDITOR AND TREASURER

Please note the requirements for abstracts/articles for the annual conference details in the president's message. These apply equally well to the VPR.

It is subscription time again, and as usual many of our number in their busy daily round of pathology have overlooked payment of last year's dues. Those forgetful souls are listed below. Please let us know if you have been unjustly listed!

A membership renewal form is found on the last page of this issue, along with the conference intention details. Please use it for your renewal so that our records can be kept up-to-date. The subscription for this year remains at $20, but will be re-assessed at the annual general meeting in May.

Unfinancial members for 1991:

ACLAND,HM	DANIELS, N	LEE, JYS	RAWLIN, G
ADAMS, NR	DUFF,BC	LEHANE, L	REECE, RL
ALLISON, J	ELLIS, TM	LENGHAUS, C	REUTER, RE
BADCOE, LM	FORSYTH, WM	MAIN, DC	RICHARDS, RB
BARKER, IK	GIESECKE, PR	MARSHALL, DJ	ROGERS, RJ
BARTON, M	GLAZEBROOK, JS	MELVILLE, L	SCOTT, PC
BAXENDELL, S	HARRIGAN, KE	MITCHELL, PJ	SLOCOMBE, RF
BELFORD, C	HOOPER, PT	MCKENZIE, RA	STEPHENS, L
CAMPBELL, RSF	HOPKINS, DL	MCORIST, S	STORIE, GJ
CARRIGAN, M	JACKSON, ARB	NICHOLLS, TJ	TAYLOR, JD
CHICK, B	JERRETT, I	OLIVER, RE	THAM, VL
CHOOI, KF	KABAY, M	PARSONS, J	THOMAS, J
CONDRON, R	LADDS, PW	PEET, R	VANSELOW, BA
COPLAND, MD	LANCASTER, MJ	RAHALEY, RS	WHITELEY, P
CORDES, DO	LANGDON, J	RAMSAY, G	WILLIAMS, DM

REGISTRY OF DOMESTIC ANIMAL PATHOLOGY
Successor to Bill Hartley

This issue carries a request for expressions of interest for the position of part-time Registrar of the Wildlife Pathology Registry at Taronga Zoo. The previous newsletter carried a similar request for the Domestic Animal Pathology Registry, owned and managed by the ASVP. Both positions are half-time, and combined or separate appointments may be made. The closing date for the Domestic Registry has been extended to the 31st of March. For further information contact:

Dr Tony Ross
NSW Agriculture
PMB 8 Camden 2570

FACVSc (Pathobiology) - TRAINING PROGRAMMMES

Les Sims (RVL Bendigo) put together a proposal for training modules in pathology as a course of study leading to the final examinations for fellowship of the college. His proposal and the comments of Dr. Phillip Ladds (Townsville), an examiner for the college are reproduced here for the interest of ASVP members. Comments from members are invited. These should be addressed directly to the authors or to Tony Ross (Camden), ASVP National Training Co-ordinator.

NOTES ON FELLOWSHIP TRAINING PROGRAMMES - Les Sims

1. There is a need to set up a system to provide advanced pathology training which takes into account the realities of the pathology scene in Australia. Unlike the US we do not have large pathology departments with multiple post graduate trainees enrolled. Most pathologists obtain the bulk of their experience outside the universities and often in regional laboratories.

2. It is therefore unlikely that any one of the Australian universities would have the resources to offer a complete external training package for preparation for fellowship examination. Collectively they could and this could be supplemented by input from other specialist pathologists outside the university system or from New Zealand.

3. It would be possible for the chapter and the ASVP to coordinate a training course based on a series of (say) 4 week intensive training modules designed to take college members from the membership to the fellowship level while meeting the training requirement for specialist registration.

4. Each module would be offered once every two years and (assuming a package of 24 modules) this could be completed on a “full time” basis in 2 years or on a part time basis in 4 years.

5. Each module would be designed in consultation with the college chapter/ASVP by one specialist/fellow from a university, the private sector or a government institute. This person would then prepare and deliver the programme at a predetermined time. This would ensure that a range of specialists would be used, improving the quality of the course, while spreading the workload across a broader number of people (it would be likely that no one person would need to prepare and deliver more than one module). The course programme would be on-going and therefore the preparation of the module would not be for just one course.

6. A fee for each module would be charged which would be paid directly to the person providing the module. The fee would need to be realistic - set at a level that would not discourage the trainees (who may be able to obtain assistance from their parent organisation) but which would provide incentive for the trainers. A total fee of $10000 for the complete package of modules (about $400 per module) would seem to me to be somewhere in the vicinity.

7. The training programme and the final examination (the fellowship examination) are completely independent in this scheme (see McGavins letter in Vet Path Report). It is likely that each module would have its own examination on completion but this would be for the benefit of trainees to assess their progress and provide information on where to concentrate their efforts.

8. The structure and delivery of individual modules would differ but the following suggestions are offered:

   i) each module would be divided into 4 weeks with each week requiring about 25 to 30 hours input.

   ii) the modules are advanced training packages to be aimed at people who have demonstrated a good level of knowledge of pathology which I will argue later is (or should be) equivalent to a course work masters plus 4 years hands on experience (I have pointed this out to answer criticisms that the 2 year time course is insufficient to cover the material properly).

   iii) modules might consist of sets of slides and transparencies (both training slides and "unknowns"), reference lists of expected reading, some course notes and course outlines detailing what is expected in each week of the module. Video taped "lectures" could be included. Each trainee would have an allotted time to contact the course provider each week and study group sessions (e.g. neighbouring trainees getting together) would be greatly encouraged. The possibility of telephone/teleconference link ups would need to be explored (cost prohibitive?). A flow of information back and forth from the trainees and the trainer would be expected (e.g. histo descriptions on "unknown" slides sent back to the trainer with comments on return, circulation of problem cases by the trainees to the group). Slide sets would be “on loan” to reduce the ongoing costs of running each module.

9. A maximum number of trainees for any one module would need to be imposed to allow sufficient contact with the trainer.

10. The part time (4 year) option has been designed for laboratory staff that need to complete other tasks dictated by their work (most of us).

11. Modules could be offered to other pathologist (or even members of other chapters - e.g. dermatology candidates) where appropriate on a one off basis. It is likely that some pathologists would enjoy a refresher course covering a specific system. The modules could also be taken by PhD students who wish to include some course work in their programme (the absence of which is seen by some as a weakness in our style of PhD's).

12. If this programme was adopted it would mean that fellowship examinations could be offered every two years, facilitating planning and budgeting by the college.

13. I see this as a step towards a genuine Australian system providing a definite "career plan" for graduates wishing to embark on a career in pathology. If adopted it would not be long before employers accepted the scheme and provided appropriate rewards (it is happening already in some states and in some job advertisements). Hence, one would spend a minimum of 4 to 6 years as a trainee pathologist, and a minimum of 2 to 4 years as a trainee specialist. (As an aside, I would like to see a system brought in whereby those with membership can call themselves "veterinary pathologists" and those with fellowships "specialist veterinary pathologists". Those in the first few years of their careers (before membership) would be "trainee pathologists". This would recognise the reality of the Australian situation. As stated above I believe that the membership examination should be at a standard equivalent to that expected of a student who has completed a course work masters and has 4 years practical pathology experience). Clearly there will be other ways of obtaining pathology training including the overseas option, but we need a system in place here.

14. A possible module series for pathology might look something like this (each of 4 weeks duration):

1. ADVANCES IN GENERAL PATHOLOGY
2. ORAL CAVITY, OESOPHAGUS AND STOMACH
3. SMALL INTESTINE
4. LARGE INTESTINE
5. LIVER
6. PANCREAS AND PERITONEAL CAVITY
7. RESPIRATORY 1
8. RESPIRATORY 2
9. HEART AND VESSELS
10. BLOOD AND BONE MARROW
11. SPLEEN, LYMPH NODES AND THYMUS
12. SKIN I
13. SKIN 2
14. URINARY TRACT
15. CNS 1
16. CNS 2
17. NERVE
18. BONES AND JOINTS
19. MUSCLE
20. EYE AND EAR
21. ENDOCRINE GLANDS
22. MALE REPRODUCTIVE TRACT
23. FEMALE REPRODUCTIVE TRACT AND MAMMARY GLAND
24. FOETAL PATHOLOGY

Other units might be needed in areas such as avian (or other species) pathology, microbiology, lab animals and fish pathology, however, these could be offered as options. There is probably a need for more general pathology but this could be dealt with under the various organ systems as appropriate. Note that this outline is only a suggestion for the pathology stream, not clinical pathology for which a separate but related programme could be developed.

JAMES COOK UNIVERSITY OF NORTH QUEENSLAND

POSTAL ADDRESS TELEPHONE: TELEX: FACSIMILE:
James Cook University
Townsville.
AUSTRALIA

GRADUATE SCHOOL OF
TROPICAL VETERINARY SCIENCE AND AGRICULTURE
Facsimile: (077) …….

Dear Les

Many thanks for your letter of 18 May, and enclosed information. In regard to these several matters I would comment as follows:

1. Fellowship Training Program

In general I am in agreement with most of the points you have raised and I ?????????????? ASVP and the Chapter will actively pursue this. My own feeling is that the Pathology Chapter is the more appropriate body to organise such a program and as you know Wayne Robinson began discussions on such a national initiative. I hope Chris Belford .will continue to work on this. I will encourage him to do so.

Some specific comments:

(a) Once appropriate titles for the different modules are decided upon (page 3 of your notes) the best qualified person in Australia should be approached to ascertain if he/she would be prepared to organise and teach the segment.

(b) Participating 'faculty' would then need to be briefed exactly on what is required (content of module, standard of instruction, expected quality and type of supportive practical material etc) and of deadlines for submission of material.

(c) One of the reasons why the University of Sydney post-graduate foundation courses have been so successful is that speakers have been adequately rewarded for their input. Therefore, I believe it would be useful, if some funding was available from the outset to provide some reimbursement for faculty towards course preparation.

(d) For this (largely 'pathology-by-post) system to work I am certain that it would be essential to have it coordinated by an enthusiastic person perhaps employed on a part time basis and attached to a University department, diagnostic laboratory or the registry. It is possible one of our retired colleagues might accept the challenge? Another possibility is that the University of Sydney post graduate foundation may (for a fee) coordinate the program once modules are prepared.

(e) Although most instruction will inevitably be in the form of assignments sent in kit form (and the AFIP in Washington has done this successfully for many years), direct contact between teacher and student must occur at some time. Cost is the obvious impediment so probably the best compromise would be module "workshops" organised to coincide with the annual ASVP - pathobiology Chapter gatherings. Perhaps three or four (or more?} modules could be handled annually in this way.

(f) Updating of modules may well be a problem. Most people preparing them will breathe a sigh of relief when the task is done and may be unavailable or unwilling to keep the contained information current. How can this be handled?

(g) Most important of all is to ascertain just to what extent would such an initiative be patronised. I believe the total fee of $10,000 you have mentioned is realistic. Clearly, much work is involved for all faculty members and their effort can only be justified if sufficient 'students' are likely to enrol in the program. A ten year prediction would be useful.

(h) In choice of modules (your Page 3) I would put a good deal more emphasis on general pathology as I feel this is an area that gets neglected by people handling the ‘case of the day’. Included in these modules would of course be inflammation, neoplasia, immunopathology, etc - topics which should have interest to people attempting fellowship examinations in many other areas.

I hope these comments are helpful.

JOBLINE

VPS PATHOLOGY SCHOLARSHIP

Veterinary Pathology Services recently advertised a 2 year training scholarship in pathology (AVJ Dec 1990).

The Scholarship, valued at approximately $36,000 per annum, will be jointly funded by VPS and the Ontario Veterinary College, University of Guelph. The recipient will be funded for a 2 year internship in pathology or clinical pathology at Guelph.

Conditions of the scholarship will include a requirement to attempt the American College of Veterinary Pathology Board examinations and undertake to return to Australia at the end of the internship. The scholar will also be encouraged to sit the ACVSc fellowship examinations on return to Australia.

8.

North Carolina State University

College of Veterinary Medicine

Department of
Microbiology, Pathology and Parasitology
919/829-4200 (Faculty)
919/829-4250 (Department Head)

4700 Hillsborough Street
at William Moore Drive
Raleigh, North Carolina 2706

December 3, 1991

Dr. J.R.W. Glastonbury
President, Australian Society for Veterinary Pathology
Regional Veterinary Laboratory
Wagga Wagga. New South Wales 2650
AUSTRALIA

Dear Dr. Glastonbury:

The Department of Microbiology, Parasitology, and Pathology, College of Veterinary Medicine, North Carolina State University in conjunction with the Chemical Industries Institute of Toxicology, Research Triangle Park is seeking applicants for a combined residency and PhD in Toxicologic Pathology.

I would appreciate your assistance in notifying suitably qualified individuals who may be interested in this position. Further information can be obtained by calling Dr. John M. Cullen at (919) 829-4350.

Yours sincerely,

John M. Cullen, VMD, PhD
Pathology Residency Coordinator

Combined Residency in Veterinary Pathology and PhD in Toxicology. The College of Veterinary Medicine North Carolina State University, and the Chemical Industries Institute of Toxicology (C.I.I.T,) announce a combined residency position in veterinary pathology and graduate training program in Toxicologic Pathology. The program will emphasize competence in veterinary pathology to prepare the trainee for certification by the ACVP. The program will consist of a residency in Veterinary Pathology directed by faculty of the College of Veterinary Medicine and research training. Graduate course work will be performed at NCSU and research studies at C.I.I.T., under the guidance of a C.I.I.T. scientist and Veterinary College faculty. Applicants must possess a DVM or equivalent degree and interest in toxicologic pathology; previous experience in pathology is desirable but not mandatory. Stipend is $19,496. Applicant should send a curriculum vitae, a statement of goals and interests, complete transcripts and 3 letters of recommendation. Closing date for applications for the position January 15, 1992 or until a suitable applicant is identified. Send communications and all application materials to the Office of Associate Dean for Services, Director of Internship and Residency Programs, North Carolina State University, College of Veterinary Medicine, 4700 Hillsborough St, Raleigh, NC 27606. For details about the program call Dr. John M. Cullen, Pathology Residency Coordinator at (919) 829-4350.

An affirmative action/equal opportunity employer.

National Veterinary Laboratory
Agriculture Protection Division
Department of Agriculture and Livestock
Papua New Guinea

Position: Chief Veterinary Pathologist (VO5)

Terms: Contract term is for 3 years, renewable.

Salary: K35,880 p.a. (KI = A$1.98), plus 25% gratuity of gross salary at 18 months and deferred gratuity of 15% on completion of contract (both taxed at 2%).

Duties: Be responsible for the Pathology Section, which at present comprises of a pathologist (VO2) and two technicians and also, as Officer-In-Charge, administer the running of the National Veterinary Laboratory. Will be expected to supply expertise in histopathology of multispecies material, with a bias to poultry; offer advice and information relating to livestock disease, when required, to the Divisional Head, as well as to field officers and clients; and to encourage and participate in relevant research and survey project.

Qualifications: Possession of a B.V.Sc. or a veterinary degree with equivalent to the MRCVS, with considerable experience in diagnostic pathology and, preferably, with some administrative experience.

Other Information: Secure housing provided at nominal rent; 6 weeks leave and leave fares every 18 months; airfares and school subsidies for children; settling in and settling out allowances.

The laboratory is well equipped, with a good library and, at present has 11 scientific staff.

Also vacant is the position of Senior Veterinary Microbiologist (VO4), which carries a salary of K29.745, with terms and conditions as above, and whose duties include supervision of the Microbiology Section which provides diagnostic services in bacteriology, mycology, serology and virology. Qualifications required are a B.V.Sc. degree or a veterinary degree with equivalent status to the MRCVS, with considerable experience in veterinary microbiology and, preferably, a bias to virology.

Enquiries and further information for both positions:

Director
Agriculture Protection Division
PO Box 2141
BOROKO
Papua New Guinea
Phone: 217405 Fax: 214630

Expressions of Interest - Registrar
Wildlife Pathology Register, Taronga Zoo, Sydney

The present Registrar, Dr. Bill Hartley has expressed the wish to retire from his combined position as Registrar of the National Domestic Animal Registry at Camden and the Wildlife Pathology Registry at Taronga Zoo.

Therefore, expressions of interest are invited from suitably qualified persons for the position of part-time (50%) Registrar of the Wildlife Pathology Registry or the position of Registrar of both the Wildlife and Domestic Animal Registries.

Duties of the Registrar of the Wildlife Pathology Registry:

- Examine all necropsy material from Taronga and Western Plains Zoos and selection of suitable cases for Incorporation in the Registry.
- Liaise with pathologists and veterinarians from relevant institutions throughout Australasia so as to obtain current case material from zoos, fauna parks, research establishments and from the wild.
- Expand the current material in the Register of relevant specimens, microscopic sections, and photographs of clinical signs, gross lesions and microscopic lesions.
- Provide advice as a consultant on native fauna cases to pathologists, veterinarians, zoological, research and academic institutions.
- Assist in training undergraduates and post-graduates in diseases of non-domestic animals.
- Prepare materials from within the Register for publication in national and international journals.
- Facilitate access to the materials and information held within the Register to relevant individuals and organizations.
- Maintain and improve the present computer data bases of the Register.

Salary: The equivalent of $48,800 per annum.
Conditions: NSW Public Service conditions of employment.

Term: Two years from June 1992, with review at June 1994 subject to satisfactory service.

Location: The Wildlife Pathology Registry is based at Taronga Zoo Mosman NSW

The Domestic Animal Registry is owned and managed by the Australian Society for Veterinary Pathology. It is currently located at the Elizabeth Macarthur Agricultural Institute at Menangle NSW.

Expression of Interest in the Registrar of Wildlife pathology should be directed to:

Dr John Kelly
Director and Chief Executive
Zoological Parks Board of NSW
PO Box 20
Mosman NSW 2088
Phone: (02) 969-2777
Attention: Dr J R Giles
Director, Scientific Policy and Research

QUEENSLAN D - Fraser Trueman

Laboratory News.

A fourth pathologist, Dr Neill Sullivan, has joined VPS in Brisbane. Neill was Senior Lecturer in Veterinary Pathology at Melbourne Vet School up to December 1991. Neill is a diplomate of the American College of Veterinary Pathologists and has a dual fellowship of the Australian College in Anatomical Pathology and Clinical Pathology.

VPS nationally advertised (AVJ Dec 1991) a 2 year training scholarship in pathology.
See JOBLINE this Issue.

Serological tests for FIP

Veterinary Pathology Services now offers a rapid ELISA for Feline Infectious Peritonitis, which detects circulating antibodies to the Coronavirus causing FIP in cats.

There is some conjecture over the use of serological assays for FIP. For example it has been recommended that ALL kittens should be sero tested at 10-12 weeks of age and only seronegative kittens sold[1]. Conversely, others[2] advise against the routine use of serology to screen for FIP.

The problem lies in the reported cross-reactivity of the assay with other Corona viruses, particularly Feline Enteric Coronavirus (FeECo). Some researchers are concerned that this results in an unacceptable level of false positive reactions.

The prevalence of FeECo in Australian cats is unknown but to date, we have not found a high level of seropositive cats using the test. However, until we have more data, we do NOT recommend the assay as a screening test for FIP in infected catteries. Its primary use is in clinical diagnosis of FIP in conjunction with clinical signs and other laboratory data.

[1]ADDIE AND JARRETT (1990) VET REC 126:164
[2] SPARKES ET AL (1990) VET REC 126:225

Atherosclerosis in a spider monkey

A mature female spider monkey with a discharging submandibular abscess was submitted for necropsy. The animal was grossly obese with accumulation of abundant peritoneal and retroperitoneal fat. The inflammation from the abscess had progressed along connective tissue planes in the neck, through the thoracic inlet and into the mediastinum and thoracic cavities. The monkey died as a result of terminal suppurative bronchopneumonia.

There were widespread yellow white plaques in the walls of all major arteries including the aorta, coronary arteries, carotid arteries, renal arteries and many others. Histologic examination revealed typical atherotic plaques characterized by subintimal accumulation of lipid frequently with lenticular cholesterol clefts and focal mineralisation.

The diet of the spider monkey would normally be regarded as adequate and suitable, consisting mainly of fruit with little access to foods containing high levels of lipid or cholesterol. The case is an illustration of "the tendency for captive primates with restricted exercise to develop atherosclerosis similar to the lesions seen in the human. The atherosclerosis was not directly involved in the death of the animal, death was due to the severe suppurative inflammation with septicaemia and bacterial toxaemia, multiple anaerobic organisms were isolated from the areas of inflammation.

Atherosclerosis is rare in most groups of mammals with the exception of captive primates. Atherosclerosis is also reasonably common in some groups of captive birds, particularly raptors fed on a high fat meat diet and kept with inadequate exercise.

***Rhodococcus equi* in an 8 week old foal**

A thoroughbred foal which was well grown for its age and in excellent external body condition was submitted for necropsy following sudden death. There were multiple pulmonary, mediastinal and mesenteric abscesses up to 5cm in diameter. Abscesses contained a tenacious yellow pus. There was generalized severe consolidation of the lungs, this change affected close to 100% of the lung leaving very few areas with inflated alveoli. The lung did not float in fixative. The mediastinal and mesenteric abscesses were present within respective lymph nodes, abscesses were also seen in the hepatic hilar lymph nodes. Examination of direct smears of the pus revealed abundant Gram positive coccobacilli. Histologically all lesions contained abundant, mainly intracellular, Gram positive coccobacilli. *Rhodococcus equi* was cultured from several sites.

Rhodococcus had been suspected on clinical grounds approximately one week before the death of the animal, the foal had been treated with high doses of both Rimycin and Erythromycin, antibiotics to which the organism was sensitive *in vitro*. The lack of response to antibiotic therapy and the rapid progression of the disease illustrate the difficulty of obtaining inhibitory concentrations of antibiotics within abscesses and foci of intense of inflammation. The death of the animal was due to respiratory failure following extension of the inflammation to a generalized suppurative bronchopneumonia.

Industrial glucose/alcohol toxicity in cattle

Eight beasts in a herd of 30-40 were found dead, several other animals in the herd were staggering, incoordinated, sometimes aggressive and showing other vague neurologic signs. One live but incapacitated animal was sacrificed. Histologically the rumen was characterized by severe hydropic degeneration of the squamous epithelium with generalized vesicle formation. The vesicles usually contained neutrophils and there was marked desquamation of the superficial keratinised layers. The subepithelial connective tissue of the rumenal papillae was hyperaemic and mildly oedematous.

The severe acute rumenitis is typical of rumenal acidosis, the pH of the rumen content, when measured at necropsy, was approximately 3.5 which is much lower than expected. The serum bicarbonate levels from affected surviving animals were also extremely low indicating severe systemic acidosis.

The history involved purchase of "out of date" industrial glucose by the farmer (on external advice) to use as an energy feed supplement in the cattle! Rumenal acidosis and production of ethanol, the latter responsible for behavioural changes.

Polyarthritis Nodosa in Brahman-cross steer

Multiple fixed tissues were submitted from an 18 month old Brahman cross steer which had been losing weight, developing a progressively stiff gait and "dropped" shoulder blades.

Histologically lesions were similar in all tissues and consisted of marked pathologic change in medium and small arteries and arterioles. The changes were characterized by adventitial, muscular and intimal hypercellularity with destruction of the muscle wall and formation of concentric laminations of fibroblasts. The lesions varied in age, the earliest lesions were characterized by fibrinoid necrosis of the vessel wall with hypertrophy and hyperplasia of the endothelial lining. Cellular infiltration consisted of mixture of neutrophils, histiocytes, multinucleate macrophages, lymphocytes, plasma cells and moderate numbers of eosinophils. In many there had been repeated occlusion of the vessel lumen with multiple recanalization. Secondary effects in all tissues were related to infarction and ischaemic necrosis.

The histologic appearance of the lesions suggests an immune mediated arteritis, however, no evidence of a specific aetiologic agent was found. Polyarthritis nodosa is sporadically seen in cattle and other species, the lesions differ from the acute vasculitis of *Salmonella* or the fibrinoid necrotising vasculitis of MCF. The lesions appear to be chronic and progressive.

The muscle wasting, stiff gait and "dropped" shoulder blades were clinically suggestive of a Selenium/Vitamin E myopathy, however, there was no histologic or biochemic evidence for this.

Multisystemic amyloidosis in a cat

A mature male DSH cat was submitted for necropsy. The cat was in good body condition with very pale mucous membranes. The abdomen was distended and contained approximately 300-400ml of free blood, haemorrhage originated from multiple foci in the liver and multiple foci of hepatic capsular rupture. Histologically the liver showed generalized severe portal hypercellularity accompanied by bile duct hyperplasia and early fibrosis. Infiltrating cells were a mixture of lymphocytes, plasma cells, neutrophils and eosinophils; there was piecemeal destruction of the limiting plate of the hepatic acini. There was widespread haemorrhage throughout the liver occurring as large blood filled cysts, some of which had ruptured through the capsule.

The spleen showed moderate extramedullary haemopoiesis.

Both liver and spleen showed extensive deposition of amorphous eosinophilic fibrillar material which showed typical amyloid apple-green birefringence with Congo red stain and polarized light.

The primary diagnosis in this cat was cholangiohepatitis, probably the so-called "feline chronic progressive cholangiohepatitis" which is thought to have an immune mediated pathogenesis.

Amyloidosis is an uncommon finding which usually occurs in disease states involving chronic inflammatory and immune responses. Two forms of amyloid are recognized, that formed from components of the immunoglobulin molecule and that formed from so-called "amyloid protein aa". Both have similar physical structure resulting in similar stain uptake and reaction to polarized light.

Generalized myositis and meningoencephalitis in a red kangaroo

An eight month old male Red Kangaroo was submitted for necropsy. The animal was in good body condition. The heart was characterized by extensive areas of pallor and necrosis, principally involving the left ventricular muscle. There was a necrotic diphtheritic membrane covering the tongue, oesophagus and the pars oesophagus of the complex stomach.

Histologically the cardiac muscle showed both acute coagulative myofibre necrosis and multifocal myocarditis with infiltration by histiocytes, lymphocytes and plasma cells. There were abundant intramuscular protozoan pseudocysts. Similar foci of inflammation and similar pseudocysts were seen in the striated muscle of the tongue and oesophageal wall. The brain and meninges showed multifocal mononuclear perivascular cuffing, parasite pseudocysts were not seen but free protozoal zoites were in one of the foci. The protozoal pseudocysts were thin-walled, unecapsulated and contained multiple zoites.

The diphtheritic inflammation of tongue, oesophagus and oesophageal portion of the stomach showed abundant pseudomycelial and yeast-like organisms consistent with *Candida*.

Traditionally the protozoa seen in acute myocarditis and myositis in macropods has been assumed to be toxoplasma, however, in some instances neither the affected animals nor in-contact animals show any evidence of seroconversion to *Toxoplasma gondii*. The organism may be a species of *Neospora*, this protozoal organism may cause similar lesions to toxoplasma and has been recorded in many different species in several countries.

Lead poisoning - parrots

6 Peach-faced parrots from an aviary of 24 birds showed vague neurological signs of muscle weakness, inability to stand or fly and 11 subsequently died.

Post mortem examination revealed small fragments of grey shining metallic material in the gizzard and there was a mild enteritis. Heavy metal toxicity was suspected.

The owner was an experienced bird breeder. When questioned by the referring veterinarian, he was adamant there was no possibility of lead toxicity; the galvanised cage wire was old and weathered and there was no lead paint available.

The practitioner carefully examined the aviary and eventually found two lead-headed roofing nails embedded in the dirt floor. Approximately 25% of each head had been recently consumed by the birds!

Fungal infections of companion animals diagnosed by histopathology in Queensland 1985-1990.

The following table gives a breakdown of the fungal infections diagnosed by VPS by histopathology in Queensland form 1985 to 1990. Note this does not include those numerous cases diagnosed by microbiology (wet mounts, KOH digests, culture, serology, cytology, Indian ink wet mounts).

	CANINE		FELINE		EQUINE	
ETIOLOGICAL DIAGNOSIS	N	%	N	%	N	%
ASPERGILLOSIS	7	9.3				
CANDIDIASIS	1	1.3	1	2.1		
CRYPTOCOCCOSIS	4	5.3	20	42.6		
DEMATIACEOUS HYPHOMYCETES	3	4.0	13	29.8	5	15.2
DERMATOPHYTOSIS	38	50.7	9	19.1	1	3.0
ENTOMOPHTHOROMYCOSIS	3	4.0	1	2.1	7	21.2
MALASSEZIA INFECTIONS	15	20.0	1	2.1		
MUCORMYCOSIS	1	1.3				
PROTOTHECOSIS	3	4.0				
PYTHIOSIS					20	60.6
SPOROTRICHOSIS			1	2.1		
TOTAL	75		47		33	

Aspergillosis: All cases have been systemic infections of German Shepherd Dogs associated with *Aspergillus terreus*,

Common lesions include multifocal pyogranulomatous necrotizing osteomyelitis, nephritis, myocarditis with other organs such as the spleen, liver, muscles and peritoneum occasionally involved. Diagnosis can usually be made by visualizing the fungal hyphae in urine sediment.

Candidiasis: This rare infection was secondary to parvovirus in 1 dog and primary in the oral cavity in the cat.

Candidiasis is usually diagnosed in immunocompromised mammals as it can be often found as part of the normal flora.

Cryptococcosis: A common primary disease of dogs and cats, often with skin, pulmonary, and/or CNS lesions.

Most cases are multiple skin nodules in which large numbers of organisms are present. Cytology is therefore useful in diagnosing these cases.

The organism is a common environmental saprophyte but not much is known of the habitat of the dog and cat strains. (The koala stain, for example, can be isolated from the soil surrounding river red gums).

Dematiaceous Hyphomycetes: This is a large group of organisms with naturally pigmented (brown) hyphae. There are several subgroups including phaeohyphomycosis and mycetomas.

Histologically, phaeohyphomycosis has large numbers of individual hyphae often with large dilated segments present within macrophages and neutrophils while in the latter subgroup, the fungus is found in distinctive colonies surrounded by both an immune complex reaction and pyogranulomatous inflammation (hence black grained mycetoma).

These pigmented fungi cause infection in all species, usually nodular dermatitis. The organisms are probably introduced into the skin through local trauma such as thorns and grow fairly slowly.

Dermatophytosis: Ringworm is common in both dogs and cats. Of all the fungal infections this one is the easiest to misdiagnose. Histological diagnosis relies on demonstration of the organism in tissue section; frequently we will diagnose folliculitis but will not be able to stain the fungus by cytochemical methods because it is there in such low numbers. Submission of multiple biopsies is therefore helpful in increasing the chances of specific diagnosis.

Entomophthoromycosis: This is one of the subgroups of "phycomycoses" and is characterized histologically by an eosinophilic precipitate around individual fungal hyphae. The most common organisms involved are *Basidiobolus* and *Conidiobolus*.

Frequently the fungi are not distributed uniformly throughout the affected tissue so a representative biopsy should include the edge of expanding growth/ulceration.

Malaseezia Infection: This is a common yeast infection of dogs which we are finding in association with generalized seborrhoea as well as in otitis externa. The budding yeasts are present in the superficial keratin.

DO NOT SURGICALLY PREP THE SITE OF SKIN BIOPSIES OR YOU WILL REMOVE ALL TRACES OF THESE ORGANISMS.

Mucormycosis: This is an uncommon opportunistic infection by one of the subgroups of "phycomycoses". Our case was secondary to enteritis or enteric torsion as the fungus grows well in devitalized tissue.

Protothecosis: Infections by achlorophilic algae are usually associated with systemic disease. All of our cases had renal, hepatic, splenic and ocular manifestations, some had enteric disease. Very large numbers of organisms present reflect a failed immune system and allow rapid diagnosis by cytology.

Pythiosis: This is the most common subcutaneous infection of horses in northern NSW and Queensland.

Infection by the aquatic comycete usually takes place following the wet season when horses spend considerable time in dams and lagoons. The fungus is probably a normal saprophyte of water lilies.

Branching yellow concretions or kunkers are needed for diagnosis as these are formed by masses of hyphae surrounded by degenerate eosinophils.

A vaccination program in conjunction with surgery is our recommendation for treatment.

Sporotrichosis: An uncommon disease of great importance as it has significant severe zoonotic potential. The disease commonly is manifest as skin nodules which follow the lymphatics. If you suspect this disease, a biopsy is safer than culture.

YEERONGPILLY VETERINARY LABORATORY

Atypical Marek's Disease with CNS Lesions in Meat Chickens (Peter Ketterer)

Samples were examined from 4 batches of meat chickens aged 21, 28, 31 and 34 days old. Mortalities ranged from 10 per day per shed in a flock of 80,000 birds to 50 per day in a flock of 100,000 birds. Affected birds showed signs of generalised flaccid paralysis of legs, wings and necks which were highly suggestive of botulism. However tests for botulinum toxin carried out on sera, livers and caecum contents of 4 birds were negative. Histology of sciatic nerves and plexuses demonstrated mild focal lymphocyte infiltrations. Spinal cords had severe lymphocytic myelitis; both grey and white matter were affected with large perivascular cuffs and lymphocytic inflammation of the spinal leptomeninges were present; focal gliosis was present in some cords. Histologically all regions of the brain showed a similar lymphocytic inflammation and a few brains had focal gliosis as was seen in the spinal cord. Other significant changes seen were moderate patchy hypoplasia of bursal lymphocyte follicles, abnormal focal lymphocyte nodules in some liver kidney and lung sections and focal myocarditis in some heart sections.

Attempts were made to isolate Marek's disease virus from direct kidney culture of 5 affected chickens and from buffy coats of 4 chickens by inoculation of kidney cell cultures. Although suspect foci were seen in several kidney explants the CPE could not be passaged.

The diagnosis of atypical Marek's disease was based on the histological lesions. Occurrence of severe CNS lesions and small peripheral nerve lesions is unusual with Marek's disease and a strain of very virulent Marek's disease virus with CNS tropism is suspected. Laying bird units on the same farm were considered to be the source of Marek's disease virus for the meat birds.

Botulism in fallow, red and chital deer (Ross McKenzie)

Botulism was diagnosed as the cause of death of 17 of 19 fallow deer in a herd at Tin Can Bay. Deaths occurred over a 2 week period beginning on 4 October 1991. Rusa, red and chital deer were also run on the property, but were unaffected initially. The fallow deer were in good condition and grazing ryegrass (endophyte-infected) and legumes including Wynn cassia. A mineral supplement mixed with molasses and cotton seed meal was supplied in troughs. The affected deer displayed progressive weakness, drooling of saliva and recumbency with death occurring in 1-4 days from onset. Necropsy and histopathology did not reveal significant lesions. A rat infestation had been controlled with a poisoning campaign and dead rats were numerous. Examination of the carcases of some of these revealed a strong source of botulinum toxin in 2 from the feed shed. Type D toxin was confirmed by mouse inoculation. No toxin could be demonstrated in sera or the feed supplement sampled. Botulinum toxin Type D was demonstrated in the rumen sample of one fallow deer after a concentration technique was applied.

On 21 October, advice was received that one chital deer had died and one red deer was clinically affected. The red deer was recumbent and had apparent semi-paralysis of the tongue. Hay was suspected as the most likely common factor involved. The rumen sample from the red deer and a hay sample were negative on both mouse inoculation and ELISA even after the concentration technique was applied.

Parvo virus infection in prawn post larvae (Peter Ketterer)

Sample of 4 batches of prawn postlarvae (PL 3-8} submitted from a north coast prawn hatchery were examined histologically for export health certification. Large basophilic intranuclear inclusion bodies were present in epithelial cells of the hepatopancreas in 2 batches. A single animal was affected in a sample of *Penaeus japonicus* postlarvae and numerous animals were affected in a sample of *Penaeus mondon* postlarvae. Unfortunately affected prawn batches were discarded at the hatchery because of high mortalities before samples could be collected for electron microscopy. Gross contamination of water had occurred between the 2 affected batches of postlarvae. The histology was diagnostic of infection with hepatopancreatic parvo-like virus which as been reported in Queensland in juvenile or adult prawns but not in postlarval prawns. It appears that this virus must be added to the list of those which can cause losses in the hatchery.

Deaths in Eels associated with Vibrio damsela infection (Peter Ketterer)

Heavy losses occurred in wild captured long finned eels (*Anguilla reinhardtii*) which were kept in a 3,000 litre recirculating freshwater tank prior to export. The first batch of eels to be held in the system consisted of about 1200 kg and of these 1000 kg were dispatched without incident. Four days later eels in the remaining 200 kg commenced dying and a total of 100 died over the next 6 days. High mortalities occurred in 2 further batches of eels which were introduced. Furabac (furaltodone) treatment was used for the first batch.

Eels died within 12 hours of showing inability to maintain their upright position in the water.

Some eels had white irregular shaped areas of depigmentation on up to 20% of the body surface. Some had red foci on the skin including the skin around the anus. Small numbers of eels had focal skin ulceration or raised soft areas of the body surface.

Eels in samples from batches 2 and 3 had linear white necrotic skin areas with a red border surrounding the anterior ventral portion of the median fin. In sample 3 eels had greatly swollen heads.

No gross abnormalities of internal organs were seen on post mortem examination. Swollen heads were due to subcutaneous gelatinous oedema, and focal body swellings consisted of oedema of muscle and hypodermis with fatty fluid exuding from the cut surface.

Histologically fish of samples 1 and 2 had severe gill pathology consisting of lamellar epithelial hyperplasia and hypertrophy associated with a heavy infestation of a protozoan with the morphology of *Ichthyoboda sp*.

Ulcers consisted of loss of epidermis and dermal stratum spongiosum including scales with scattered foci of dense bacterial flora in the surface tissue of the lesion. The necrotic skin lesions showed devitalised compact dermis and round cell infiltration in the deep hypodermis with necrosis and fragmentation of adjacent muscle fibres.

Muscle lesions consisted of necrosis and fragmentation of fibres and a protein rich exudate containing macrophages and large number of gram negative bacilli. A similar muscle lesion associated with marked hypodermal oedema was seen in eels with swollen heads.

Significant bacteria were not isolated from eels of Sample 1 probably because furabac had been used.

Vibrio damsela was isolated from the surface of the skin lesions from the hypodermis, from the skeletal muscle lesions and from the head kidney of the eels in samples 2 and 3. Pure cultures were obtained from skeletal muscle and kidney.

Stress and epithelial damage due to poor water quality, especially high ammonia level, and significant *Ichthyoboda* infestation are considered to be predisposing factors for the septicaemic *Vibrio damsela* infection in the eels. *Vibrio damsela* has been recorded as a pathogen in damsel fish and has been isolated from other marine animals but has not previously been reported as a cause of disease in freshwater fish. Virulence of the organism is associated with a potent extracellular haemolysin.

Human cases of infection have resulted in rapidly progressing extensive cellulitis and septicaemia, usually in patients who have been compromised by some other cause of poor health such as diabetes.

Disinfection of the tank and all equipment with chlorine was recommended to control the outbreak. Attention to water quality and cooling of the tank holding water were also considered important control measures.

OONOONBA VETERINARY LABORATORY

A Fatal Outbreak of *Dictyocalus viviparous* in Cattle In North Queensland (John Norton & Wendy Townsend)

In October 1991, 50 Santa Gertrudis cattle between 12-15 months of age were found dead on a property near Tully, North Queensland. Three hundred of the remaining 1150 animals in the herd were sick. Clinical signs included loss of condition and coughing. A post mortem on two animals revealed proteinaceous exudate and large numbers of round worms within the bronchi and bronchioles and, consolidation of extensive areas of the peripheral portions of the lungs. Histopathological examination of the affected lung showed changes varying in severity from thickening and increased cellularity of interstitial tissue to cuffing of bronchioles (the predominant cell types being neutrophils and eosinophils) and consolidation. The cuticles of parasitic larvae were occasionally seen in these consolidated areas, and were frequently associated with foreign body granulomas. One hundred and sixty-five worms were recovered from a 5cm section of bronchus. The appearance of the worms was consistent with *D. viviparous* Soulsby (1982).

The outbreak of verminous pneumonia was unusual because of

1. the age and origin of the cattle affected
2. the location of the outbreak.

We believe that lungworm larvae were introduced onto the Tully property with 150 head of cattle bought at a sale yard in the Rockhampton district. Pathologists at the Rockhampton Regional Laboratory have diagnosed isolated cases of lungworms in cattle grazing ponded pastures. All cattle introduced to the Tully property were placed in 2 small holding paddocks for 1 to 3 weeks. The animals brought from the Rockhampton district occupied the holding paddocks prior to the affected herd which came from properties in northern Australia. Examination of faecal samples taken at random from animals on the northern Australian properties failed to isolate lungworm larvae or eggs. It is highly unlikely that these animals would have been previously exposed to *D. viviparous* and would therefore be highly susceptible to infection, despite their age.

Soulsby E.J.L. (1982) Helminths, Arthropods and Protozoa of Domesticated Animals (7th edition) Lea and Febiger, Philadelphia).

Pseudomonas pseudomallei infection in camels (John Norton & Wendy Townsend)

Pseudomonas pseudomallei was isolated at this laboratory from the lungs of two North Queensland camels. The first case occurred near Cooktown in May 1988. The local vet examined a 3 year old camel and diagnosed pneumonia. The camel was one of a herd of seven recently brought from the Northern Territory. The second case occurred near Townsville in January 1990. An eight year old camel was found to be distressed, pyrexic, dehydrated and ataxic in its hind legs when examined by an attending veterinarian. The animal also had a heavy burden of *Haemonchus sp.* Necropsies revealed pneumonia in both animals and a concomitant pleurisy in the older camel. In the younger camel, multiple thick-walled abscesses were seen within the lung.

Histopathological examination on samples showed similar changes in the lungs of both camels. The interstitial tissue between the alveoli in acutely affected areas was markedly congested. Fluid and fibrin were present within the lumina of alveoli and accompanied by variable amounts of haemorrhage and cellular infiltrate. The majority of inflammatory cells were polymorphs. In more severely affected areas, the lumina of alveoli were distended by neutrophils. With progression the normal architecture of the lung was lost and replaced by masses of degenerating cells.

It is thought that stress associated with transportation in the three year old camel and the heavy worm burden in the eight year old camel may have made the animals more susceptible to infection.

VICTORIA - John Mackie

RVL BENALLA

HEPATIC AND RENAL FIBROSIS IN A CALF (John Mackie)

A 6 month old Murray Grey bull calf presented with a 3 month history of ill thrift, abdominal enlargement and diarrhoea. The body weight was 133 kg. At necropsy the liver was extremely enlarged, pale, very firm to cut and had an accentuated tabular pattern. The liver weighed 35 kg or 26% of body weight (the adult bovine liver normally weighs slightly more than 1% of body weight). The gall bladder was somewhat enlarged though not distended and the bile appeared normal. The wall of the extra-hepatic bile ducts and the gall bladder appeared slightly thickened. The kidneys were very firm to cut and there were large pale streaks on the subcapsular and cut surfaces.

Microscopic examination of liver revealed extreme fibrosis of portal areas. Within the mature fibrous tissue there were numerous bile ducts most of which had a distinct lumen and a well organised, columnar, cuboidal or squamous epithelium. Changes to some bile ducts included dilation, attenuation of the epithelium, necrosis of the epithelium and accumulation of bile, granular eosinophilic material or cellular debris in the lumen. Some bile ducts were surrounded by a mild inflammatory infiltrate of lymphocytes, plasma cells and macrophages. In the kidney, radially oriented sheets of mature fibrous tissue were present predominantly in the cortex. Within the fibrous tissue there was marked cystic dilation of tubules, attenuation of tubular epithelium and atrophy of some glomeruli.

The lesions would appear to be due to a developmental overgrowth of connective tissue. Similar hepatic lesions, presumably congenital, occur sporadically in young calves (Professor Ken Jubb, personal communication). In humans, congenital hepatic fibrosis has been described as a variant of poly cystic disease (Edmondson and Peters 1985).

Reference

Edmondson HA and Peters RL (1985) In *Anderson's Pathology*, Vol 2, 8th edn, edited by Kissane JM, The CV Mosby Company, St Louis, p 1163

RVL HAMILTON
Nutritional myopathy in rabbits - George Riffkin

RVL HAMILTON

Nutritional myopathy in rabbits - George Riffkin

A producer of 'meat rabbits' lost 75% of his young stock (20/26) before seeking veterinary help. Three 8-10 week old NZ white X Angora rabbits arrived at the RVL with a history that other rabbits had died suddenly over the previous 3 weeks. There had been no evidence of diarrhoea Coccidiosis was the disease suspected.

The rabbits all had distended abdomens. Two were reasonably bright end alert but disinclined to move. The third was severely depressed and moribund.

Common findings in all 3 rabbits were:

* A pale waxy appearance of skeletal muscles, particularly hind limb adductors
* Dark, brown coloured urine, with fibrin floccules in one
* Slight icterus
* Dehydration of all tissues

In addition, one rabbit had an acute fibrous peritonitis from a perforating jejunal ulcer.

Serum CK levels ranged from 8000-16000 u/L and Vitamin E levels on 2 samples were 500 and 680µg/dL

Histological changes in skeletal muscle were remarkably subtle given the severity of the clinical syndrome. Muscle fibres were generally swollen and undergoing hyaline degeneration. There were some small foci of necrosis of individual fibres but little evidence of mineralisation or inflammatory cell response.

In the kidneys of 2 rabbits there was occasional hyaline caste in collecting tubules. In the third rabbit (the one with peritonitis) there was also an acute glomerulonephritis, swelling of tubular epithelial cells and occlusion of Bowman's spaces with cell debris and inflammatory cells.

A diagnosis of nutritional myopathy was based on clinical, gross and microscopic changes as well as the high CK level. We are not sure what are normal serum Vitamin E levels in rabbits, however, levels around 500 µg/dL are generally regarded as being sufficient in ruminants.

The rabbits had been on a diet of dry proprietary rabbit pellets which may have been stored for several months. The addition of fresh vegetables to the diet appeared to solve the problem.

Reports of nutritional myopathy in rabbits are poorly represented in standard texts.

Seizures in a cat - Jonathan Webber

"Fluffy", an 18 month old neutered female cat was presented for examination at the Albury Veterinary Hospital. Over the previous month, Fluffy had a number of seizures, sometimes 3 in a day. Typically, she would be found lying flat on her side, panting and convulsing. She had also become less affectionate, cried out frequently, neglected her grooming and had a frightened look about her.

Questioning of the owners revealed that a month previously, the house stumps had been sprayed with "Aldrex 600" (aldrin 600 g/1) to prevent termite infestation.

The provisional diagnosis of organochlorine poisoning was confirmed by GC analysis of a fat biopsy for OC pesticides. The fat contained 370 mg/kg of dieldrin. (By way of comparison, the MRL for dieldrin in meat for human consumption is 0.20 mg/kg!) Aldrin is rapidly metabolised to dieldrin after absorption. The high levels in this cat were probably as a result of a combination of direct absorption through the skin and oral absorption following self-grooming.

Oral treatment with phenobarbitone controlled the convulsions and "Fluffy" is again back to her normal self!

Johne's Disease in a Sheep in Bhutan (Deborah Seward, Andrew Kelly)

An aged Merino/East Himalayan cross bred ewe was examined by Andrew Kelly during his recent visit to Bhutan.

The ewe was emaciated, showed no evidence of scour and had pelleted faeces. The intestinal lymph nodes were not grossly enlarged but appeared to be partially replaced by a fir whitish tissue. There was prominence of intestinal lymphatics. The wall of the last 50cm of the ileum was grossly thickened and had marked folding of the mucosa.

Histological examination of the ileal sections revealed the presence of clusters of epithelioid cells containing numerous acid fast bacilli within the lamina propria of the villous tips, between the crypts of the deep mucosa, amongst the connective tissue of the submucosa and within the lumens of several of the submucosal and serosal lymphatics. There was a moderate, diffuse mononuclear infiltration of the submucosa, serosa and surrounding the associated lymphatics.

Moderate medullary fibrosis was present in sections of the intestinal lymph nodes. A single focus of caseous necrosis, surrounded by epithelial cells including giant multinucleated cells, was present in one node, however, no acid fast bacilli were identified in any of the lymph node sections examined.

The clinical, gross and histological findings in this ewe are typical of Johne's disease.

Ovine Johne's disease is endemic in New Zealand and has been diagnosed on several properties in Central Western New South Wales. The epidemiology of the disease in Bhutan has not been extensively investigated.

Cor Lenghaus, CSIRO Animal Health Laboratory, Geelong.

Rabbit Haemorrhagic Disease: Assessing the potential of a new virus to control wild rabbits in Australia.

Wild rabbits (*Oryctolagus cuniculus*) remain Australia's most serious vertebrate pest, with damage done and control measures taken estimated to cost in the order o£ $100 million annually. Myxomatosis was spectacularly successful in controlling rabbits in the 1950's and 60's, however the emergence of less virulent field strains of myxoma virus and the increasing genetic resistance of rabbits, has limited its effectiveness during the past two decades. The spread of myxomatosis is dependent on biting arthropods, particularly mosquitoes, feeding on infected rabbits and re-inoculating other susceptible rabbits subsequently, so that spread in the drier, inland areas has always been much less predictable, related to periodic, heavy rainfall. It is in the more arid areas that rabbits are presently of most concern, due to soil erosion caused by their burrowing activities and their foraging, which removes so many of the smaller plants binding the soil. Any re-vegetation projects are essentially doomed unless they are surrounded by rabbit-proof fences because rabbits devour most of the seedlings which are planted. It is considered that some native plants could become extinct unless the grazing pressure of rabbits is significantly decreased. Insects, birds and small animals which rely on these plants for food and shelter could also disappear.

In 1984, an apparently new disease of farmed rabbits emerged suddenly in China. The disease spread rapidly via people trading in rabbits, or rabbit products such as meat, fur or skins. There was often a total depopulation of adult rabbits on any one property within days of the disease first appearing. Overall losses in China were estimated in the tens of millions of rabbits, and were only curtailed with the introduction of a formalin-inactivated vaccine, prepared from the livers of diseased rabbits which had died. An identical disease spread through rabbits in Europe from 1988, with losses in farmed rabbits estimated in excess of 60 million; a similar, short-lived epizootic occurred in Mexico, which was stamped out by vigorous control measures involving quarantine, complete de-stocking and clean-up of infected premises and preliminary restocking with sentinel rabbits.

The Chinese believe that they probably imported the disease with a shipment of Angora rabbits from Eastern Europe. The Europeans believe that the disease was initially established in hares where it was known as the European Brown Hare Syndrome {EBHS), before spreading to rabbits. In Italy, for example, hares are regularly imported from Central Europe or Argentina, and conceivably the disease was introduced in this way. Mexico imported the disease in a shipment of frozen rabbit meat from China. The virus of

Rabbit Haemorrhagic Disease (RHD) as the rabbit disease came to be known, is capable of infecting only rabbits and hares, and does not cause disease in a wide range of other domestic or laboratory animals and birds. The viruses which cause "classical RHD or EBHS are now considered to be separate but closely related entities. The distinctions are becoming a little blurred with the emergence/detection of further variants from the field; the whole issue is made immensely more complex by an inability to propagate the virus in cell culture. Only one group in China claims to be able to do so. At this time there is not even agreement as to whether RHD virus is a DNA virus related to parvoviruses, or an RNA virus similar to caliciviruses. And you thought the economy was in a mess?

RHD virus has been imported to AAHL, to assess its potential as a biological control agent for feral rabbits. This will also require studies in a range of domestic animals and wildlife. Ultimately, any progression to field studies will depend on the results of experiments done in a high security laboratory and decisions taken by the government of the day. Presently, the material which was imported has been passaged in outbred New Zealand white rabbits, after it was screened for any extraneous agents. RHD is an interesting disease in that young animals are reported to be refractory to infection - this alone would suggest that the aetiological agent is **not** a parvovirus, given their predilection for rapidly dividing cells. Unexpectedly at AAHL the virus did kill 8-week old rabbits ostensibly to be used for raising specific antibody after intramuscular inoculation. What follows is a composite of our findings in experimentally infected rabbits to date. These do not differ significantly from overseas reports. Direct electronmicroscopic examination of liver extracts have revealed only calicivirus-like particles present.

Most rabbits died within 48 hours after intramuscular inoculation of 0.5-1.0 ml of virus suspension obtained from a rabbit's liver. There were few premonitory signs of disease, some depression in the last few hours and terminally, an increased respiratory rate and effort with some bloodstained fluid at the external nares. Overseas reports mention signs of CNS irritation such as terminal paddling convulsions and involuntary crying not seen here. Diarrhoea was not a feature here but has been observed elsewhere. At autopsy there was some excess of bloodstained fluid in the peritoneal cavity. The liver was pale, swollen and friable. There were splash haemorrhages visible through the kidney capsule. The spleen was firm and swollen to several times its normal size. The lungs were very wet and congested, and in some rabbits there were strands of freshly clotted blood on the surface of the lungs particularly between the lung lobes. The trachea was filled with bloodstained frothy fluid, and the epithelium was discolored a dark plum red, due to extreme congestion of the thin-walled blood vessels in the subepithelial connective tissue; there was no acute tracheitis present as such.

Histologically the liver, lungs, kidneys and spleen were the organs most consistently and severely affected. The most severe liver lesion consisted of extensive coagulative-type necrosis affecting most of the hepatocytes in the liver but preferentially sparing the immediate centrilobular areas. There was almost total collapse of normal hepatic architecture in periportal areas. Sinusoids were distended and congested with erythrocytes, and contained numerous thrombi. In other rabbits, liver pathology consisted of more sporadic hepatocellular degeneration and death with a preponderance in periportal areas. In some rabbits there was no pattern discernible in the distribution of hepatocytes affected even when -50% appeared to have suffered some degree of histological damage. Intravascular coagulation was not a feature in less affected livers. An interesting and consistent finding in RHD infected rabbits was the presence of enlarged hepatocytes containing multiple, small, mineralised granules in their cytoplasm.

Lung lesions were centred on alveoli. There was a necrotising process centred on the vascular endothelium of alveolar capillaries. Presumably this was a direct effect of the virus; given the short course of the disease this necrosis is unlikely to have been antibody mediated. Alveolar walls were congested and there was haemorrhage into alveolar spaces. These spaces were often very distended; there seemed to have been a loss of low-protein fluid from alveolar spaces during tissue processing.

In the kidneys lesions graded from quite severe with parenchymal haemorrhage to moderately severe congestion only in some rabbits. In those with the more extensive changes, capillary loops in glomeruli were very congested and contained numerous thrombi. The vascular endothelium appeared to have

degenerated in some glomeruli as well as elsewhere in the parenchyma. At this time it is not known whether this is due to a direct effect of the virus or because of toxic/anoxic effects of coagulation products. There was leakage of protein into Bowman's space and considerably more protein in convoluted tubules and ultimately collecting tubules.

The spleen was extremely congested. There was apparent haemostasis and occlusion of vascular channels by thrombi. There was extensive destruction of the normal sinusoidal architecture, the fixed histiocytes and the lymphoid follicles. In some cases there were numerous necrotic cells present in vascular channels, possibly the remains of fixed macrophages. Occasionally there was a discrete vasculitis of penicillar arteries.

Lymph nodes were extremely congested. There was little evidence of the follicular necrosis as seen in the spleen but sporadic, necrotic lymphocytes were common. There was an apparent loss of heterophil precursors from the bone marrow.

There was extensive and rather diffuse haemorrhage in the heart, without obvious vasculitis or endothelial necrosis. The extensive myocardial necrosis reported in the Chinese literature has not been featured in the material examined at AAHL.

Other tissues such as the alimentary tract, pancreas, adrenals, thyroid, skeletal muscles, reproductive tracts and central nervous system were essentially normal.

V.R.I. ATTWOOD

Bonamiosis in Port Phillip Bay and Westernport Bay oysters - CT Rawlin and JC Parsons

Cultured flat oysters (Ostrae angasi) in Port Phillip Bay suffered losses up to 80% over the summer and autumn period of 1991. The affected oysters were usually in poor condition. Poor condition in this oyster species can be assessed at times without opening the oyster by the presence of algae on the lips of the shell where the shrunken mantle can no longer clean.

Histologically, a marked granulocytosis of the connective tissue involving ovary and digestive gland was seen. Relatively low numbers of 6 µm intra-cytoplasmic inclusions were seen in some ovarian cells and granulocytes. These inclusions were weakly eosinophilic and in some cases showed a faint "bulls-eye" structure.

These inclusions have been identified by electron microscopy as Bonamia spp. protozoa.

In some sections trophozoites of Perkinsus spp. protozoa were also identified although these parasites are unlikely to be causing the primary disease.

Further heart smears and electron microscopy have identified the Bonamia spp. involved as structurally identical to that found in New Zealand. The New Zealand Bonamia has decimated that country's natural flat oyster beds.

Sampling of oysters along the Victorian coast in December 1991 has shown Bonamia to be present in large numbers in Port Phillip Bay and in Westernport Bay. Mortalities have begun in Westernport Bay.

Bonamiosis has severely limited the profitability of flat oyster fisheries in France, Spain, England, Ireland, USA and New Zealand. From field observations, the organism has the same ability to damage the experimental oyster industry as it exists in Victoria.

Bonamiosis is an OIE List B disease and its presence has been reported to the OIE.

SOUTH AUSTRALIA - Viu Ling Tham

VETERINARY PATHOLOGY SERVICES PTY LTD., ADELAIDE

Bleeding disorders in the dog (Ruth Reuter)

Bleeding disorders in small animals seem to have gained prominence in this area in the last few months. The following cases illustrate some of the problems encountered by the clinicians and the laboratory involvement which has occurred.

I. Suspect Von Willebrand's Disease in an Irish Wolfhound.

An 11 week old female Irish wolfhound was submitted to a local clinic following sudden collapse. The mucous membranes were pale, the heart rate 200 with a systolic murmur. There was a history of possible access to Ratsak. A blood sample gave the following results.

	Patient	Reference range
RBC X 10/12	2.2	5.5-8.5
Hb (g/L)	38	120-180
PCV (L/L)	0.12	0.37-0.55
MCV (fl)	55	60-77
MCH (pg/L)	17.3	19-25
MCHC (L/L)	317	300-360
WCC X 10/9	18.1	6.0-17.0
Bands	1.6	0-0.2
Neuts	10.7	4.0-12.0
Lymphs	5. 2	0.9-5.0
Monos	0.4	0.1-0.6
Eosins	0.2	0-0.2
Total Protein (g/L)	37	55-81
Albumin (g/L)	19	23-40
Reticulocytes (%)	4.0	<0.5
Platelets	adequate	
Polychromasia	2+	
Anisocytosis	2+	
Hypochromasia	1+	
Activated lymphocytes	1+	

The hypoproteinaemia, hypoalbuminaemia and regenerative anaemia suggested blood loss. Prothrombin time and activated partial thromboplastin time were normal, tending to rule out warfarin poisoning. A Coombs test for auto-immune haemolytic anaemia was negative, and a faecal analysis showed no internal parasites such as Trichuris. A canine von Willebrand factor assay performed by Bruce Parry at the Veterinary Clinical Centre in Werribee gave conflicting results so a second sample was obtained. Although the dog had been given a transfusion prior to the second sample the Factor VIII-ra level was only 36 Cu/dl (Normal 60-180 Cu/dl; <50 consistent with von Willebrand's disease). Further samples could not be obtained as the dog was euthanased in extremis at this stage. However, samples from the mother and a half-sister were both low (50, 42 Cu/dl), and the father gave an equivocal result (66 Cu/dl).

Von Willebrand's disease is an inherited blood disorder due to a deficiency or abnormality of a glycoprotein associated with Factor VIII, and known as Factor VIII:related antigen. It has been identified in

a large number of breeds of dogs, particularly Dobermans, Scottish Terriers and Corgis. However, it has not, to our knowledge, been identified previously in Irish Wolfhounds.

II. Haemophilia A in a German Shepherd dog.

In August of 1991 a 5 year old male German shepherd dog was examined at a local clinic for suspect prostatitis and an enlarged popliteal lymph node. A blood sample submitted showed a mild elevation of blood urea, but no other significant abnormalities apart from a comment that platelets were sparse. Red blood cell parameters were normal. One month later the dog was resubmitted with severe pitting oedema and "erythema" of the left hind leg. A biopsy of the region showed marked dermal oedema and collections of mast cells and eosinophils suggesting a hypersensitivity reaction. There were also foci of haemorrhage and haemosiderin pigment in macrophages. A blood sample taken at surgery showed the following abnormalities:

	Patient	Reference range
RBC X 10/12	5.2	5.5-8.5
PCV	0.35	0.37-0.55
Eosins X 10/9	1.0	0.1-0.5

Over the next 5 days, blood was seen seeping from the biopsy wound and the scrotum became swollen. Prothrombin and Activated Prothrombin Times were marginally elevated. A repeat haemogram showed mild monocytosis and eosinophilia but no evidence of anaemia. On fine needle aspirate of the swollen area on the thigh, only erythrocytes and occasional immature blood cells were seen. When Factor VIII:C analysis was performed by Bruce Parry at Werribee the level in serum of this animal was 5 Cu/dl (normal 50-160 Cu/dl) consistent with a diagnosis of Haemophilia A.

This disease is due to a deficiency of Factor VIII:C, which is inherited as an X-linked autosomal recessive trait. The disease has been detected in several countries and most reports in the literature have incriminated a German dog named Canto von der Weinerau. In the case described here, Canto was also present in the pedigree.

NEW SOUTH WALES - Paul Gill

REGIONAL VETERINARY LABORATORY - ARMIDALE

Cryptosporidiosis in red deer fawns - (Stephen Lore)

Last month, fawns in a mob of 250 hinds and 80 fawns began dying. Fawns up to 2-3 weeks old developed a yellow, watery smelly diarrhoea, became depressed and died 12-24 hours later. Thirty five out of the 80 succumbed to the disease over a week.

The mob had been in the same paddock for 4 months. However, feed was short and animals were congregating on small areas because of hand feeding.

One dead fawn, and samples from another, were submitted to the laboratory for examination. There was little to see at necropsy apart from diffuse reddening of the small intestine. Autolysis precluded the demonstration of microvilli let alone cryptosporidia in histological sections. Faeces were positive for cryptosporidia by ELISA and FAT, but negative on ELISA for rotavirus and coronavirus. The protozoan was found in large numbers in ZN stained smears of faeces.

Hand feeding/concentration on a small area appeared to be an important risk factor in this case.

Equine epulis - (Stephen Love)

A bleeding, ulcerated tumour measuring 4 x 5 x 12cm from the left buccal cavity (adjacent to the lower premolars) of a female quarter horse was submitted for histopathology. The mass was first noticed 6 weeks previously and appeared to be growing. The age of the mare was not stated. Histologically, the stroma consisted of dense fibrillar collagen with regularly spaced stellate cells i.e. consistent with periodontal ligament stroma. Additionally, there were trabeculae of osteoid in the centre of the mass. Fibromatous ossifying epulis was diagnosed.

The epulides are fairly common in dogs and cats, but appear to be rare in horses. Comment on the nature and frequency of these tumours in horses would be welcomed.

Lymphoma in a horse - (Stephen Love)

In the last Vet Path Report (No. 32, Nov 1991, page 15), John Gibson described a generalised lymphadenopathy, possibly lympho-reticular neoplasia, in a 2 year old thoroughbred.

In July, 1990, we received a section from a subcutaneous tumour in a 15 year old horse (breed etc. not stated). The mass, a rapidly growing tumour (4cm diameter, 3cm high) below the eye, was being treated as a sarcoid.

The tumour consisted of sheets of lymphoid cells, possibly blast cells, and areas of necrosis. We suggested a diagnosis of lymphosarcoma. Bill Hartley agreed that it was a lymphoma, possibly malignant lymphoblastoma.

Lymphomas of course are relatively rare in horses.

Cattle deaths associated with brassicas - (Stephen Love, Shaun Slattery and Stephen Sinclair)

Shaun Slattery, District Veterinarian, Narrabri, investigated the death of 7 ex 19 heavily in calf cows in July. The owner reported that affected animals became sick suddenly, swayed a little, became recumbent and anorectic, scoured and then died. The passage of red-brown urine was also noted. At necropsy, the spleen was mottled, the abomasal mucosa was reddened and there were small paint brush haemorrhages on the intestinal serosa. Urinalysis was positive for blood/haemoglobin.

Serum from one sick cow, and urine and preserved tissues from a dead cow were submitted to the laboratory. At histopathology there was an acute periacinar necrosis in the liver (consistent for example with the hypoxia of anaemia) and a marked splenic haemosiderosis (consistent with IV haemolysis). There was also a rumenitis similar to that seen in grain poisoning. Serum biochemistry revealed elevations in AST and CPK, and a low serum phosphorus (1.04 mmol/L, normal 1.3-2.3).

The feed in the paddock in which these cattle grazed was lacking in quality and quantity. The 50 head of cattle were being supplemented with substantial amounts (10-15 200 litre drums a week, in 2 feeds each week) of discarded fruit and vegetables, mostly cabbages and cauliflowers. The level of cabbage leaf in the waste increased just prior to the first deaths occurring. Believing that an haemolytic anaemia associated with Brassica sp (Family Cruciferae) intake was the likely cause of death, follow-up blood samples were submitted several days later. Unfortunately all the sick animals had died. Samples were taken from a random selection (5) of those remaining. Two had a marginally depressed haemoglobin concentration and two had a slightly elevated bilirubin. In blood smears, macrotosis, occasional basophilic stippling of RBC's and higher than normal numbers of reticulocytes were noted. Conceivably these animals had been affected relatively mildly and were then in the recovery phase.

The species Brassica oleracea includes many well known vegetable and grazing crops. Everist states that broccoli, brussel sprouts, cabbage and common kale have been involved in cases of poisoning in man and livestock. The latter 3 have caused "Kale-anaemia" in ruminants. Parkinson and Sutherland (1954) reported haemoglobinuria in cattle in the Kingaroy district of Queensland. They suggested phosphate deficiency as a predisposing factor with the haemoglobinuria being precipitated by the consumption of cruciferous plants.

The findings in this case are consistent with a diagnosis of haemolytic anaemia associated with B.oleracea consumption.

References

Everist, SL (1981). Poisonous Plants of Australia.
Parkinson, B and Sutherland, A.K. (1954). AVJ 30. 232.
Seawright, A.A. (1982). Animal Health in Australia, Vol. 2: Chemical and Plant Poisons.

REGIONAL VETERINARY LABORATORY, WAGGA WAGGA - John Glastonbury

Cattle

St George disease A 1-year-old Hereford heifer from the western Riverina was destroyed because of a suspicion of this disease. Pathological findings included severe subcutaneous oedema, hydrothorax and peritoneum and an extremely prominent jugular vein. Strong indication of the diagnosis of St George disease was given by the histological detection of peliosis hepatis, medial hypertrophy of pulmonary arterioles and small arteries, myocardial fibrosis and pulmonary oedema. Any access this animal had to *Pimelia* sp is uncertain.

Hypovitaminosis A Two outbreaks were investigated in feedlots producing animals for the Japanese market. In the first, 18-month-old Simmental cross Hereford steers which had not received vitamin supplementation until August 1991 displayed swollen joints and a reluctance to move. The plasma vitamin A levels in 11 samples varied from 0.1 to 1.0 µmol/L.

On the second feedlot clinical signs included serous ocular discharge, cloudy cornea, blindness, ataxia and respiratory distress. The plasma Vitamin A levels in 4 samples varied from 0 to 0.08 µmol/L and squamous metaplasia was detected histologically in the salivary gland duct from 1 animal.

Sheep

Fusobacterium necrophorum polyarthritis Although relatively important in the north of New South Wales, this condition was diagnosed for the first time in the Riverina region during November. Three weeks after being mulesed and marked, 10-week-old Merino lambs developed multiple swollen pus-filled joints. The morbidity rate was 4.5% (550). Gram stained smears of the synovial exudate revealed numerous filamentous Gram negative rods and anaerobic culture yielded heavy growths of *Fusobacterium necrophorum*.

Salmonellosis Recently purchased 18-month-old Merino wethers were yarded for 2 days for various management procedures. A severe black scour and abdominal pain were observed in 20.0% of the animals and resulted in a final case fatality rate of 37.5%. Heavy growths of *Salmonella typhimurium* were recovered from the jejunum, ileum and caecum of 1 case submitted to the laboratory for examination. Pathological findings were dominated by severe diffuse congestion of the tunica mucosa of the abomasum, acute necrotic enteritis and acute haemorrhagic necrotic and segmentally erosive typhlitis.

Pigs

Isoimmune thrombocytopaenic purpura A litter of 10 piglets died at 5 days of age after showing weakness, anaemia and bleeding from the anus and iron injection as well as ear notching sites. The dam was on her sixth litter and had been mated once before to the sire. Spectacular postmortem findings included petechial to ecchymotic haemorrhages in the epicardium, mesenteric and peripheral lymph nodes, lungs, serosa of the alimentary tract, renal cortices and skeletal muscles.

The exotic disease Swine fever was part of the differential diagnosis. However, the antigen capture ELISA for pestivirus performed on the spleen and lung was negative and no viruses were recovered on culture from the spleen, lungs and heart.

Polyserositis Following on from Dick Sutton's item in the November edition of the Veterinary Pathology Report we have investigated an ongoing problem with polyserositis in a large intensive piggery. Initially, 7.5% of 2000 weaner pigs were noticed to be coughing and over one night, 8 died suddenly. Gross and histopathology findings were dominated by severe diffuse subacute fibrinopurulent pleuritis, pericarditis and peritonitis. No bacteria could be detected in smears or histological sections and culture on a variety of media yielded no growth. Subsequently sows became involved and developed anorexia and lethargy 5 days after lincospectin was removed from their diet. Pathological findings were similar to the above. Moderate growths of *Pasteurella multocida* were recovered from the lungs and *Mycoplasma hyorhinis* was demonstrated in fluorescent stained smears.

REGIONAL VETERINARY LABORATORY, WOLLONGBAR - Paul Gill

Mortalities in Green turtles during October, numerous Juvenile Green turtles were washed ashore, dead or moribund, onto beaches in southern Queensland and northern New South Wales. The three turtles postmortemed at RVL Wollongbar had severe necrotising enteritis involving up to 2/3rds of the small intestine. Microscopic examinations found a severe acute protozoan enteritis due to colonisation of the mucosa by schizonts, gamonts and oocysts of *Caryospora* spp. Multiple granulomas associated with spirochid fluke eggs were present in several tissues including lung, liver, kidney, spleen and brain. The population dynamics of this outbreak of chelonian coccidiosis are not resolved. Apparently, juvenile Green turtles are not usually found in the above coastal waters, they tend to be more in the mid-Pacific.

Verminous pneumonia in an infant Pygmy killer whale

A young Pygmy killer whale was found washed ashore on a beach near Coffs Harbour. It died three days later despite treatment. The lungs were described as congested and firm.

Histological examination found an eosinophilic interstitial pneumonia with haemorrhage and exudation of eosinophils associated with small nematode larvae.

Similar lesions are described in the lungs of pigs and cattle due to migrating *A. suis*.

Graeme Fraser

Flaccid paralysis in calves Two 10-week-old Brahman calves were investigated from a herd in which 4 calves had a recent history of diarrhoea, ataxia, flaccid paralysis, recumbency and death.

Microscopic examination found a degenerative neuropathy in the white matter tracts of the mid-brain and brain stem (including fasciculus tegmenti, trapezoid body and cerebellar peduncle) and spinal cord (especially the dorsal columns). The lesions consisted of myelin swelling with digestion vacuoles and central chromatolysis in neurones. Organophosphate poisoning was suggested as the most likely cause as the paddocks involved had previously been used for horticulture. Toxic residues were not found in either calf.

WESTERN AUSTRALIA - Ron Peet

ANIMAL HEALTH LABORATORIES - SOUTH PERTH

Suspect Vitamin A Deficiency in Emus - M.J. Kabay

Deaths in emu chicks with an upper respiratory tract infection syndrome were investigated. Birds developed a bilateral purulent conjunctivitis at about 10 days of age and mortalities continued until day 28. Mortalities up to 20% were experienced.

Seven birds were submitted for necropsy and all had a severe bilateral caseous conjunctivitis. The conjunctival sacs were grossly distended with cheesy, pus-like material, creating a bulging appearance of the eye. The nasal sinuses were distended by copious amounts of gelatinous creamy pus causing deviation of the nasal septum and hard palate. Other body organs appeared grossly normal although one bird had a fibrinous airsacculitis and peritonitis.

Histological lesions were restricted the conjunctival and nasal epithelium. The epithelium had a tall palisading pattern and was covered by a thick layer of degenerate cellular material. In one bird the tracheal epithelium appeared slightly hyperplastic and the occasional epithelial cell had undergone necrosis. However, the cilia appeared intact in all birds examined.

No recognised primary bacterial, mycoplasma or viral pathogens of the respiratory system were recovered. Two of the birds examined had liver Vitamin A levels (mg/kg) of 37 and 10. Levels below 100 mg/kg are considered significant in other species and analysis of 14 normal adult emus at slaughter showed that Vitamin A levels ranged between 115 and 258 mg/kg.

The gross and microscopic features of this syndrome are dissimilar to the lesions described for Vitamin A deficiency in domestic fowl. In the fowl squamous cell metaplasia of the pharyngeal and tracheal epithelium are a prominent feature. The pharynx of the emus examined appeared normal and tracheal lesions were only present in one of the birds examined. However, the accumulation of cheesy material in the conjunctival sac and nasal sinus is characteristic of Vitamin A deficiency in fowls.

Ulcerative enteritis in emus - M.J. Kabay

A syndrome of ulcerative enteritis and impaction of the jejunum and ileum was investigated in 3 to 6 week old emu chicks. Mortalities up to 30% were experienced. The pathology was consistent over three batches of birds examined but the causative agent was not identified.

Typically affected birds had multiple focal areas (2mm diameter) of ulceration of the intestinal mucosa. Ingesta was often tightly adhered to these lesions. Some mucosal lesions extended the full thickness of the intestinal wall producing red areas of necrosis on the serosal surface. These lesions were associated with a turbid peritoneal effusion. In about half the birds examined, the jejunum and ileum were grossly distended with sand or compacted faeces. Other body organs were normal.

Histologically, the ulcerative lesions were localised and sharply demarcated. Normal villous structure abutted foci of necrotic mucosa extending to the level of the lamina propria. The lesion was bounded by a palisading row of epithelioid macrophages. Within each necrotic focus, at the leading edge of the lesion, there were pockets of gram negative and some gram positive rods. The gram negative rod could not be recovered by aerobic and anaerobic culture but had the morphology of a *Bacteriodes* or *Fusobacterium sp*. in gram stains of tissue sections. Clostridium perfringens was recovered from some of the birds examined.

The gross and microscopic features of this syndrome are similar to ulcerative enteritis of quail. However, *Cl. colinum* was not recovered and no liver lesions were observed. The condition responded to treatment with furazolindone.

Lymphosarcoma in fallow deer - Ron Peet and Trevor Ellis

A 3 to 4 year old female fallow deer was presented to a practitioner (Dr S. Joubert) with a history of gradual weight loss over a period of some months. It had markedly enlarged lymph nodes which included prescapular, retropharyngeal, inguinal and popliteals. It was euthanased and post mortem exam revealed enlarged pleural and mesenteric lymph nodes with white raised areas (approximately 1-2 cms diameter) in the kidneys and smaller areas in the heart.

Histopathology revealed sheets of apparently neoplastic lymphoid cells invading these lymph nodes and the organs. These infiltrates consisted mostly of lymphoblastic cells which were pleomorphic with very little cytoplasm and they had a high mitotic index. Occasional mature lymphocytes were visible and the infiltrates had disrupted normal architecture of the organs and nodes with focal areas of haemorrhage and necrosis.

Fresh samples of lymph node submitted for bacteriology proved negative for Mycobacteria (TB had originally been suspected), Pseudomonas and other possible pathogenic bacteria.

The owner has lost 7 or 8 animals with similar symptoms over the last 5 years. Bloods were collected from 10 in contact deer and proved negative using the gel test for enzootic bovine leucosis.

To our knowledge, this is the first diagnosis of lymphosarcoma in deer in Western Australia and possibly in Australia.

Ergotism (nervous form) in weaner sheep - Ron Peet

After an unusually wet mild November, some sheep grazing a recently sprayed predominantly *Echuim plantagineum* ("Peterson's Curse or Salvation Jane") pasture became blind and showed nervous signs. The spray used was glyphosate (Roundup®- Monsanto) at the rate of 400 mls per hectare which has a relatively low toxicity for mammals (LD50 acute oral dose s 5000 mgm/kgm), and this was considered unlikely to be the cause of the problem. Forty six of 300 mixed age sheep died, only 4-6 month old sheep being affected - older (1.5 years) sheep were unaffected.

Five convulsing sheep were submitted to the local practitioner who diagnosed possible secondary brain damage form the pyrrolizidine alkaloids (P.A.) in the "Paterson's Curse", or polio encephalomalacia. He treated them empirically with thiamine, penicillin and fluid therapy but two died and the others recovered. Post mortem examination of the two dead sheep revealed nothing remarkable and brains from both plus liver and kidney from one were submitted to Animal Health Laboratories for histopathology. Apart from occasional random foci of PMN's in the liver there were no visible lesions and no evidence of megalocytosis indicative of P.A. poisoning.

The district government veterinary officer (Ms M. Glass) then visited the property and found seeded annual ryegrass (*Lolium rigidim*) growing along the fence lines. There was evidence of this having been eaten and she submitted seedheads to plant pathology suspecting A.R.G. toxicity. The plant pathologist (Mr. M. Barbetti) could find no toxic galls but reported the seedheads to contain many ergots (*Claviceps purpurea* infection). A diagnosis of the nervous form of ergotism was made on the basis of clinical findings, animal and plant pathology.

This is the second case of the nervous form of ergotism in sheep in Western Australia (Peet, R.L., 1989, Vet. Path. Report No. 24 p.6.) and the hyperthermia syndrome has been previously reported in cattle (Peet, R.L., McCarthy M.R. and Barbetti, M.J., 1991, Aust. Vet. J.68:21.)

The weaner sheep may have been selectively affected due to their grazing habit or having lower bodyweight making them more susceptible to the amount of toxin available. This disease should always be considered when investigating cases involving nervous signs, particularly after warm, mild weather favouring the production of ergots in seeded pasture and grain crops.

Ovine lymphosarcoma - Cleve Main

A rising 4 tooth merino wether from a flock with a history of lameness and ill thrift was submitted for necropsy.

With the exception of the mesenteries, all lymph nodes were markedly enlarged. In many cases they contained multiple red foci or were severely haemorrhagic. The spleen was enlarged and the liver contained a few small red foci beneath the capsule.

Histopathological examination of the lymph nodes revealed multiple haemorrhagic foci and replacement of the normal architecture by uniform population of lymphoblastic cells. Lymphoblastic cell infiltration was also present in the spleen and to a minor degree in the myocardium. Marked periportal infiltration by these cell types was also evident in the liver.

Lymphosarcoma in sheep is not common and is said to be caused by the bovine leukaemia virus. Blood samples from other animals in the flock have been sampled to rule out involvement of the enzootic bovine leucosis virus.

Chronic renal disease in a bull terrier - Cleve Main

A 4 year old white male bull terrier was euthanased following repeated episodes of renal failure.

Pertinent findings at necropsy were pale kidneys, reduced in size with an apparent reduction in the thickness of the cortices. A small (1-2 mm diam.) ulcer was present on the dorsal surface of the tip of the tongue.

Histopathological examination revealed marked fibrosis involving both renal cortex and medulla. It was particularly severe in the medulla where it was associated with dilatation of tubule segments. Lesions were also present in most glomeruli ranging from complete sclerosis to periglomerular fibrosis and adhesions of the tuft to the capsular endothelium. Occasional foci of mineralisation and oxalate crystals were evident in some tubules.

The lesions are consistent with the description of chronic renal failure in Bull Terriers and are thought to be another example of breed specific renal conditions in dogs (Robinson et. al 1989).

Reference: Robinson WF, Shaw SE, Stanley B, Huxtable CR, Watson ADJ, Friend SE and Mittens R. (1989) - Aust Vet J 66:193.

Chronic renal disease in a koala - Cleve Main

A young female koala was euthanased after a period of illness in which included azoturia and weight loss.

Lesions seen at necropsy were minimal but it was noted that the kidneys were of moist appearance and of unequal size.

Histopathologically the kidney lesions were characterised by diffuse fibrosis of the cortex and medulla. Some tubules were dilatated and, within some, structures consistent with oxalate crystals were present.

The aetiology of the condition is not known, but has been diagnosed in both mature and juvenile koalas. Investigation into the significance of the oxalate crystals is being carried out

MURDOCH UNIVERSITY

Pyrrolizidine Alkaloid Toxicosis with Secondary Encephalopathy in Steers - Brad Chadwick and Ron Peet (AHL)

A number of sudden deaths were seen in a mob of 370 steers grazing good spring pasture on a property at Gingin, 100km north of Perth. The unvaccinated steers had been recently transported from a station in north-west WA, but had failed to thrive.

An initial post-mortem examination of a very autolysed carcass found numerous brush-stroke haemorrhages, particularly on the epicardium and parietal pleura and peritoneum, which were suggestive of death due to enterotoxaemia. However, sporadic deaths continued following a vaccination programme.

Examination of two further deaths found severe hepatic fibrosis of a long standing nature with very little ongoing necrosis. Extensive brush-stroke haemorrhages were again evident throughout the carcass. In addition the brain in both cases showed a gross subtle laminar change throughout the cerebral cortex. Histologically there was a mild diffuse vacuolar change at the grey/white matter junction. Retrospective evidence from the property owner then included accounts of animals seen head-pressing or found dead against a fence post.

The changes seen were considered consistent with a primary hepatopathy and a secondary coagulopathy and encephalopathy. Epidemiological evidence from this and other properties subsequently found to be experiencing similar problems with steers transported form the same station source, linked the onset of deaths with the introduction of good quality feed, usually pasture.

Chronic poisoning by plants containing pyrrolizidine alkaloids was suspected, estimated to have occurred many months prior to the first deaths. Analysis of liver samples from the two steers at Gingin confirmed the presence of residual pyrrolizidine alkaloids in the tissue. Inspection of the original property found *Crotalaria crispata* which, due to unseasonal rain, had germinated along road and creek drainage areas, this had been eaten even though cattle supposedly find it unpalatable, and clinical/pathological cases are rare in our experience. However, cattle are apparently just as susceptible as horses if forced to eat pyrrolizidine alkaloid containing plants. (Hooper P.T. 1978 in "Effects of Poisonous Plants on Livestock" Academic Press, New York p.161-76).

ALBANY REGIONAL LABORATORY

Transmission of Virulent Footrot from Cattle to Sheep - David Forshaw

Virulent footrot was detected on a property at Mount Barker. Acting on departmental advice, the manager of the property de-stocked all contact sheep in two successive years. Virulent footrot developed in the two flocks of introduced sheep in the springtime. Sheep were absent for 50 days and 28 days in the first and second years respectively.

Protease stable *D. nodosus* was isolated from interdigital skin of cattle on the property. Deep ulcers were seen in the interdigital skin and the organism was regularly cultured from such lesions. The organism was also cultured from skin with no lesions. Minimal lameness was noted.

Artificial challenge of sheep with the organism isolated from the cattle feet produced severe under-running foot lesions. Sentinel sheep run with the cattle developed virulent footrot.

NORTHERN TERRITORY - Lorna Melville

BERRIMAH AGRICULTURAL RESEARCH CENTRE

Barramundi Picornavirus (Cor Lengnaus, Lorna Melville and Glenn Schipp)

A batch of approximately 800,000-1,000,000 Barramundi *Lates calcarifer*, were spawned on 15 December 1990, and hatched 16 December, at the department of Primary Industry and Fisheries' Stokes Hill fish hatchery, near Darwin. These fish were destined to be forwarded as small fry to commercial growers in the burgeoning Barramundi fish farming industry.

On 1 January 1991, some of this batch was noticed to be less active, inappetent and with a reduced fright response. The gills seemed more intensely reddened and their spleens were also prominently visible.

At this time there was a slightly increased rate of mortality, however, mortalities increased catastrophically and the whole batch had died by 5 January. During the course of this disease, all fish were noticed to change colour from black to a light shade of brown. Tissues forwarded to the Australian Animal Health Laboratory, Geelong, were confirmed by electron microscopy as infected with Barramundi picorna-like virus (BPV).

Histological lesions in fry examined at Berrimah, were confined to the developing central nervous system. The most striking lesion was a variable but usually severe cytoplasmic vacuolation of the neuronal layers of the retina, particularly in the broad, highly cellular, internal nuclear layer. In its most extreme form much of this layer was reduced to coarsely vacuolated foamy tissue held together by fine cytoplasmic strands. Similar spongiform vacuolation occurred in the dense layer of subependymal neurones in the brain and spinal cord. Individual large neurones throughout the neuropil were more sporadically affected. Basophilic intracytoplasmic inclusions which have been reported from other outbreaks of BPV, were not featured here.

The source of the outbreak is unclear, but the infection could have come from a contaminated food source (algae and copepods) obtained from a commercial grower, or imported with fish or fish products obtained from Queensland.

Sudden deaths in sacred ibises (Anton Janmaat)

Eight sacred ibises, out of a total of more than 50, died suddenly, over a period of 30-60 minutes, on a Sunday afternoon in front of startled visitors at the Howard Springs Nature Park. The ibises had just flown in and were waiting to be fed at the lagoon. The surviving birds took off but returned the following afternoon. No further deaths were observed within the park.

Six birds (four adult and two younger females) were presented for necropsy and all showed duodenal haemorrhage. In the adults, blood was present from the distal oesophagus to the cloaca with bloody staining of the vent in two birds. Five birds had small bones in the oesophagus, proventriculus and gizzard. Microscopic changes in the small intestine consisted of focal necrosis of the superificical epithelium, haemorrhage and moderate to marked hyperaemia. Incidental findings in one of the ibises were subcutaneous mites, endogenous stages of a protozoan parasite in cells of the collecting ducts and distal tubules, disseminated focal interstitial nephritis and a non-suppurative cholangiohepatitis.

We suspect chemical poisoning, possibly a bait, and will start with the analysis of stored material for diazinon after reading a Queensland report on sudden deaths in sacred ibises.

Disseminated fibrosarcoma in a Moluccan Rusa deer (Anton Janmaat)

"Tumours first seen on skin approximately 12 months ago. Since that time they have grown in size and spread to all parts of the skin covering the whole body with the majority of the lesions on the head". This picture suggests a viral aetiology possibly with insect transmission. Commenting on the close resemblance of the tumour to equine sarcoid, Associate Professor P.W. Ladds (JCU Townsville) queried the possibility of involvement of the same virus.

Vascular parasites in a Brushtail Possum (C Lenghaus & L Melville)

A brushtail possum found in a Darwin suburb in poor condition was taken to the Territory Wildlife Park for care. The possum died and tissues were submitted in formalin.

Histology showed massive numbers of filaroid-like parasites, occluding capillaries and other small blood vessels in the heart, lung and kidneys. Multifocal granulomas were present in affected tissues. Blood vessels showed endothelial proliferation, and smooth muscle hyperplasia.

Two other possums in the same area had been reported as being in poor condition. Unfortunately, despite alerting wildlife officers, no fresh specimens were obtained so the identity of the parasite remains unknown.

1991 Arbovirus Sentinel Program (L Melville)

Despite good rainfall during the wet season, virus activity was below average. 170 viruses were isolated, mainly from the EHD group. Bluetongue Type 3 was isolated over a very short period of time in late May/early June.

CPRS was the only site in the Northern Territory where bluetongue activity was recorded. EHD activity was restricted to the more northern monitoring sites while BEF, Akabane and Palyam group activity was widespread, being detected at all sites except Alice Springs.

Insect monitoring at bleeding sites has shown the major wet season culicoides is *C. peregrinus* (February-April), the major dry season culicoides is *C. brevitarsis* (July-September), while the transition months show a mixture of these two species with others such as *C. marksi* and *C. wadai*.

ANIMAL HEALTH LABORATORY, AZRI, ALICE SPRINGS (Denise McEwan)

This will be my last report as the laboratory is closing down at the end of December and all functions will be transferred to BARC, Darwin.

Ipomoea muelleri poisoning of cattle

This condition was diagnosed on a property where there were between 50-100 animals affected, with at least 18 known deaths including 3 bulls. Symptoms observed were head nodding, goose stepping with very straight front legs, inco-ordination and ataxia.

There were abundant green *Ipomoea* vines in the area and large numbers of seeds were found in the rumen of one animal. Post mortem and histopathology findings were unremarkable. Clinical pathology revealed an elevated CPU (1220 u/1), creatinine (207 u mol/L and GOT (177 u/1) .

Sera and urine are being tested for LSD derivatives.

Isotropis atropurpurea

Of some 660 two-year-old steers put in a small holding paddock 8-10 died and another 20-30 were noted to be sick within a week. Depression, weakness, scours, reluctance to move, apparent blindness, mild ataxia and death without struggling were features noted. On autopsy, parts of the gastro-intestinal tract particularly the abomasum were oedematous. Histopathology revealed acute renal proximal tubular necrosis; no oxalate crystals were present. No *Isotropis* was detected in the rumen contents, but a search of the holding paddock revealed a number of plants had been grazed. Incidentally the holding paddock had been pulled within the previous 2 years.

This station is in the area where some 1,300 travelling cattle died in 1923-24 due to this plant. Since then there have been a few reports of losses from it in the NT.

Rumex vesicarius

Oxalate nephrotoxicity due to this plant was observed when a mob of cattle were trapped into a small holding paddock with only topfeed and 'green pick' dominated by *Rumex sp*. available. Between 6-8 mostly pregnant heifers and 2 steers died.

Other conditions recorded in the past year have been *disseminated aspergillosis* in 2 German Shepherds, a subcutaneous fungal granuloma in a cat (not cultured) and recently several cases of psittacosis in aviary birds.

MEMBERSHIP RENEWAL

Please cut off and return with renewal to:

Honorary Secretary, ASVP
Regional Veterinary Lab
NSW Agriculture
PMB 8 Camden NSW 2570

Name: ……

Address: ……
(If not as listed in Nov VPR)

Phone: ……

Please indicate:

$20 (1992 subs) or $40 (1991 and 1992 subs)

INTENTION TO ATTEND ANNUAL CONFERENCE MAY 9-10 1992

I (intend / do not intend) to attend the Adelaide conference.

Please complete below if you plan to attend.

1 (will / will not) require accommodation to be booked at the University of Adelaide Residential Wing.

I (will / will not) be attending the ASVP dinner (separate from registration)

I (intend / do not intend) to present a paper at the conference.

The proposed title of my paper (if applicable) is: ……...

……

Dr L Sims
51 Carpenter Street
Bendigo Vic 3550