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VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
University of Melbourne
Veterinary Clinical Centre
Werribee Vic 3030
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DEADLINE FOR NEXT VET. PATH REPORT May 31, 1996

SECRETARIAT

PO Box 114 Walkerville SA 5081
Phone: 08 3446337 (Pat Bosence) Fax: 08 3449227

ASVP EXECUTIVE 1995-1997

President	Ron Slocombe	University of Melbourne, Veterinary Clinical Centre Werribee Vic 3030	03 9741 3500
Secretary	Karl Harrigan	University of Melbourne, Veterinary Clinical Centre Werribee Vic 3030	03 9741 3500
Treasurer	Mark Williamson	Australian Animal Health Laboratory, Ryrie Street, Geelong Vic 3220	052 27 5000

Committee Members

Ian Jerrett	PO Box 1271 Bairnsdale Vic 3875	
Alison Havadjia	Centaur International PO Box 1284 Bairnsdale Vic 3875	051 52 0800

APPOINTMENTS

Chairperson (Registry of Domestic Animal Pathology)	Tony Ross
Newsletter Editor	Karl Harrigan
Coordinator (Training Committee)	Vacant

CONVENOR - SLIDE OF THE MONTH

Rod Reece — National Registry of Animal Pathology, EMAI,
Private Mail Bag 8, CAMDEN NSW 2570

STATE REPRESENTATIVES

Queensland	Bruce Hill, Rockhampton Vet Lab, QDPI, Box 6014 Rockhampton MC Q 4702	079 360211
Victoria	Malcolm Lancaster, Dept Agric PO Box 388, Benalla Vic 3672	057 611100
South Australia	Ruth Reuter, VPS, PO Box 96, Plympton SA 5038	08 3623544
New South Wales	Paul Gill, RVL Wollongbar 2480	066 261261
Western Australia	David Forshaw, Regional Office, WA Dept Ag, Albany 6330	09 8420500
Northern Territory	Anton Janmaat, PO Box 79, Berrimah 0828	089 992240
Tasmania	Barry Munday, Uni Tasmania, PO Box 1214 Launceston 7250	003 243812

Welcome from the ASVP Executive

After some minor hitches in transferring files accumulated under the outgoing executive, the transition to establishing the new executive in Victoria has progressed.

The executive has recently received preliminary notices regarding the annual meeting to be held in combination with the NZSVCP in Christchurch. The emphasis of the meeting is on wildlife diseases, and members are invited to submit cases, scheduled for the second day of the meeting, for discussion. The Executive encourages members to attend and hopefully the ASVP will be well represented. Additional information regarding the conference is included elsewhere in this publication.

Since taking office the Executive has been engaged in two major issues of interest to the membership.

The first relates to a continued and deliberate government policy affecting most states, where contraction of State diagnostic laboratory services continues. The decline in real positions for veterinary pathologists as well as bleak long-term prospects for those interested in either specialised training or advancement in a career in veterinary pathology remain serious problems. The incoming executive was charged with the task of ensuring the interests of the membership were known to the Australian Animal Health Council. Through personal discussions with some of the members of this newly formed body, it became clear that the ASVP was not in a position to directly influence the constituency of the Animal Health Committee. It is only very recently that the Council constitution and terms of reference have been finalised, and it is now timely that the ASVP make representation to the Council with regard to these matters. The ASVP Executive has made a submission to the NSW State Government in response to a request from the membership, in regard to mooted closures in two of the NSW State Diagnostic Laboratories. Surprisingly, no similar requests for submissions came from the Victorian membership, despite similar closures and contractions in the laboratories of this state.

A second emerging issue involving the ASVP Executive is that precipitated by legislation which came into effect in 1996 in Victoria, changing the reporting requirements for notifiable diseases within the state and requiring registration of all laboratories that render diagnoses of livestock diseases. Associated with this process of registration will come some means of quality assurance. Clearly, through legislative requirements in Victoria, some form of quality assurance program will be imposed on the membership in this state. Independently, some of the larger commercial veterinary diagnostic laboratories both in Victoria and elsewhere are currently in the process of either seeking accreditation or certification as a means to demonstrate specialised expertise with the obvious intent to use this in promoting their place in a competitive market. Given these developments, the ASVP can perhaps become an active influence in the establishment of appropriate criteria for quality assurance programs. What is also clear is that if veterinary pathologists do not develop standards for self-regulation, it is likely that regulations will be imposed on veterinary diagnosticians, either by legislation or by expectation from the public. Development of quality assurance programs also has significant implications for more formal educational programs, in mandatory reporting of diseases, in mandatory maintenance of archives of records and specimens and in participation in national disease surveillance schemes. It is the view of this Executive that the ASVP must be actively involved and promote the development and participation of quality assurance programs, education and career development for veterinary pathologists. How the ASVP should proceed in regard to these matters has yet to be resolved.

On behalf of the executive, I encourage your input on these matters and look forward to meeting as many of you as possible in New Zealand.

Professor Ron Slocombe
President ASVP

ASVP Members who are now crossed off for non-payment since 1993:

CONDRON R.	CREEPER J.
GODWIN J.	HUMPHREY J.
MORRISON J.	ROGERS R.J.
STEWARD DJ.	

ASVP Members still outstanding for 1995:

BELFORD C.	BOULTON J.C.
CHOOI K.F.	DUFF B.C.
GILL J.	GLAZEBROOK J.S.
GLEESON LJ.	HAESCHEK-HOCK W.M.
HANDLINGERJ.H.	HOOPER P.T.
HOWLETT C.R.	McCAUSLAND I.P.
MELVILLE L.	SEARSON J.
TRUEMAN K.F.	VANSELOW B.A.

SUBSCRIPTIONS ARE NOW DUE FOR 1996

(See Renewal below)

MEMBERSHIP RENEWAL

1996 - NOW DUE

Please send cheques with updated details to:

ASVP Secretariat
PO Box 114, Walkerville 5081

(Enquiries concerning membership should be directed in the first instance to the above address. For phone enquiries ring Pat Bosence on 08 344 6337 or Fax 08 344 9227. Cheques should be made payable to **Australian Society for Veterinary Pathology)**

NAME: …….

ADDRESS: ……...

……. P.C. ……..

PHONE: …… FAX: ……..

1995 Subscription	[ ] Outstanding
1996 Subscription	[ ]
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Conference Information

Notice of Joint Meeting of New Zealand Society for Veterinary and Comparative Pathology and Australian Society for Veterinary Pathology.

The NZSVCP committee extends a warm invitation to ASVP members and others interested in diseases of wildlife to attend the combined meeting of the Societies.

Theme of Conference: Disease of wildlife species and impact on environment,

Venue: Christchurch, 20-21 June, 1996

On the first day the programme will cover aspects of Rabbit Calicivirus disease, diseases of marine mammals, diseases of marsupials, and diseases of native avian species. The topics covered in these segments will be augmented by a set of histological slides which will be made available to attendees prior to the meeting. The second day is given over to members' papers on any appropriate subject, although presentations relating to the general theme of the conference will be encouraged.

Society AGM's will be held at the conclusion of the technical programme on June 20th.

The conference is timed to enable members of both Societies to extend their visit to include the NZVA/AVA and Wildlife Branch Conferences which are held in Christchurch the week of June 23-29.

The technical committee is keen to include Australian material in the programme and calls for submissions of proposed papers and microscope cases urgently. For the latter please include the slide and a brief history.

Contact for the technical programme is:

Alistair Johnstone
NZ Registry of Animal Pathology
Batchelar Animal Health Laboratory
PO BOX 536
Palmerston North, New Zealand
Phone: (06) 351 7950, Fax: (06) 351 7910

Enquiries regarding accommodation and general matters should be directed to:

Karen Bailey
Lincoln Animal Health Laboratory
PO BOX 24
Lincoln, New Zealand
Phone: (03) 325 3900, Fax: (03) 325 3918

Karen reports that the joint meeting will be held at The George Hotel which is 10-15 minutes walk from the Christchurch Town Hall where the NZVA/AVA 2nd Pan Pacific Veterinary Conference will be held during the subsequent week, 23-28 June.

Accommodation is available at "The George", room rates Deluxe NZ$129 + G.S.T., Executive NZ$140 + G.S.T.. This is a special rate for the ‘pathology group’. You will have to point out that you are with the ‘Veterinary Pathology Conference’ to get these rates.

Those wishing to make bookings will have to make their own arrangements, though Karen has indicated a willingness to assist. Address for "The George": 50 Park Terrace, Christchurch, N.Z., PO BOX 13.063, Christchurch N.Z., Telephone (03) 379 4560, Fax (03) 366 6747.

The location is overlooking Hagley Park and the Avon River adjacent to the Botanical Gardens and Museum, 5 minutes to central city, C.B.D. and casino! It is 12 km from Christchurch International Airport. The brochure indicates 54 rooms, 4 suites, total of 80 beds. I have some other information re "The George" should you wish to know more.

Other accommodation, outlined in NZVA/AVA 2nd Pan Pacific Veterinary Conference Handbook, is as follows.

Park Royal Hotel Cnr Durham & Kilmore Sts.		AUD$210/night	NZ$231/night
Centra Cnr Cashel & High Sts.		AUD$180/night	NZ$197/night
Noahs Hotel Cnr Worcester St & Oxford Tce.		AUD$154/night	NZ$169/night
Quality Durham Cnr Durham & Kilmore Sts.		AUD$144/night	NZ$158/night
Quality Central 776 Colombo St.		AUD$138/night	NZ$152/night
Cotswold Hotel 88-96 Papanui Rd.		AUD$108/night	NZ$119/night
City Travel Lodge 356 Oxford Tce.		AUD$98/night	NZ$107/night
Camelot Court Hotel 28 Papanui Rd.		AUD$90/night	NZ$99/night
YMCA 12 Hereford St.	Standard Ensuite twin	AUD$50/night AUD$64/night	NZ$54/night NZ$70/night

Personal experience in telephoning Christchurch from Melbourne eg. Karen Bailey, is that you dial 0011-64-3-325-3900. The 0 of the 03 prefix is omitted.

The NZVA/AVA 2nd Pan Pacific Veterinary Conference will be held at the Christchurch Town hall during the week of 23rd-28th June. A conference handbook has been published outlining the programs. It notes that participants should "be prepared for mid-winter temperatures throughout the week. Woollen clothing, overcoats, knitted jumpers and jackets"! Average day temperature of 10°C.

I shall endeavour to obtain other "appropriate" information for anyone who wishes to ask for it. Meanwhile I wish all a successful and enjoyable conference.

Members please note that the technical committee via Alistair Johnstone, would like to obtain indications of participation as soon as possible.

Karl Harrigan

LETTER FROM NEW ZEALAND

Dear ASVP/NZSVCP members

Once again it is time to start thinking about the annual conference. The theme this year is Wildlife and it will be held rather earlier than usual - June 20 and 21. (This is so it can be held in conjunction with the Second Pan Pacific Veterinary Conference, which is being held June 23-28, so that as many as possible of our Australian colleagues from the Australian Society for Veterinary Pathology can attend), it will be held at the George Hotel, Park Terrace, Christchurch.

This letter is to call for cases for the conference and titles of papers to be given. As well as case slides, history and other relevant data, block/s and/or wet tissue to be used for the slide sets would be appreciated, so the NZ Registry for Animal Pathology can get on with set assembly as soon as possible.

Please send material as soon as convenient and no later than April 15 to: Brenda Batchelar, Animal Pathology Registry, B.A.H.L., P.O. Box 536 (or Tennant Drive, if sending by courier), Palmerston North, New Zealand. Your submissions are much appreciated.

Also enclosed is a rough draft of the intended program and more information on accommodation. We shall look forward to seeing you in June.

Regards.

Karen Bailey for The Organising Committee.

Preliminary Programme - NZSVCP/ASVP combined meeting 20 and 21 June. 1996

Thursday - 20 June. 1996

8.00am to 10.00am	Rabbit Calicivius: Review of disease. The Australian Escape. Ecological impact. MAF/DOC strategies to manage release. Discussion time on slides.
Morning Tea	
10.30 am to 12.00 noon	Cetacean/marine Mammal Diseases including discussion time on slides.
Lunch	
1.00 pm to 3.00 pm	Marsupial Diseases: Wobbly possum syndrome (history, pathology, transmission experiments). Other recent developments in marsupial disease. Discussion time on slides.
Afternoon Tea	
3.30 pm to 5.30 pm	Avian Diseases, with emphasis on native birds: Aspergillosis. Others. Discussion time on slides.
5.30 pm	Annual General Meeting of NZSVCP and ASVP
8.00pm	ANNUAL DINNER.

Friday 21 June, 1996

Cases from contributors with preferred emphasis on wildlife.

Accommodation and Travel Information - NASVCP/ASVP meeting 1996.

The meeting will be held at the George Hotel, Park Terrace, Christchurch.

Telephone: 64 3 379 4560
Fax: 64 3 366 6747

Reservations: 0800 100220 (NZ)
1-800 121 980 (Australia)

A block booking of 30 rooms has been made for attendees for the nights of 19, 20, 21 June at a special room rate of $129 + GST (deluxe room) or $145 + GST (executive room) per night.

In order to take advantage of this rate you **MUST**:

1. Book before May 20.
2. Mention you are with the "Veterinary Pathology Conference".

Bookings can be made directly with the Hotel. If you encounter problems, contact Brian Cox, C/- the Organising Committee at Lincoln Animal Health Laboratory, Box 24, Lincoln, New Zealand.

Telephone: 64 3 325 3900
Fax: 64 3 325 3918

There are alternative accommodations of various styles within a reasonable distance, including a YMCA hostel and a YHA hostel within walking distance. Information on Christchurch hotels and a city map is included in the Pan Pacific Veterinary Conference Handbook, distributed at the end of March. This handbook also gives general information on Christchurch, including getting around, transport from the airport etc.

Registration fees for the meeting: We do not aim to make a profit, just to cover our costs. On this basis, the fee for the meeting is likely to be in the vicinity of $75 (NZ), per person, payable at the meeting. The meeting dinner on the Thursday night will be extra, expected to be approximately $50 per person.

If you intend to attend please complete the slip below and return by May 20 to:

Brian Cox,
Box 24,
Lincoln,
New Zealand
Fax: 64 3 325 3918
Email: coxb@lincoln.mqm.govt.nz

I will be attending the NZSVCP meeting on 20 and 21 June, 1996.

Name: ……
Address: .....
……..
Contact: Phone: .....
Fax: …….
Email: …….

Number attending technical sessions: 20 June = …….
21 June = …….
Number attending dinner, Thursday evening 21 June = …….
I will be accompanied by: .....
……

Jobline

POSTGRADUATE EDUCATIONAL OPPORTUNITY

Internship in Veterinary Pathology (half-time)
in the Department of Veterinary Pathology
The University of Queensland

The internship is a traineeship designed to give the appointee a broad training in the practice of veterinary diagnostic pathology in an academic environment. The appointment will be for two years (non-renewable), during which period the appointee will participate in the routine activity of the busy necropsy, surgical pathology, cytological and clinical pathology diagnostic services run by the department. This internship will necessarily involve work mostly with companion animals, since most of the practices from which the department draws its material are urban.

The presence of experienced mentors, archival case material and excellent library facilities makes this position an ideal preparation for examination for membership of the Pathobiology Chapter of the Australasian College of Veterinary Scientists.

Additionally, the appointee is expected to enrol in a part-time research program at the Masters level. To this end, the appointment is half-time to allow time for study. Conversion to a full-time research project at the PhD level at the end of the internship is possible should appropriate research funding be available.

Applicants must possess a veterinary degree registerable in Queensland. The annual remuneration for this half-time position will be $17,069.

The expected commencement date is 1st February 1996.

Interested persons please contact:

Professor W.F. Robinson
Department of Veterinary Pathology
The University of Queensland
St. Lucia, QLD, 4072

Ph (07) 3365 2565, Fax (07) 3365 1355

POSITION VACANT

VETERINARY PATHOLOGIST/CLINICAL PATHOLOGIST

Alpha Scientific is a rapidly expanding diagnostic laboratory in New Zealand's north island seeking a qualified clinical and/or anatomical pathologist.

The present team includes two specialist veterinary pathologists, both with North American training and 13 technicians. The laboratory currently receives 26 000 cases annually and supplies a full range of diagnostic services to small and large animal veterinarians and two zoological gardens.

Essential to the position are a veterinary degree registerable in New Zealand and recognised post graduate training and expertise in clinical and/or anatomical pathology. Board eligibility with the American College of Veterinary Pathologists is also required.

The position involves substantial client interaction, so excellent communication skills are essential.

Board certification by the ACVP or Fellowship of the Australian College of Veterinary Scientists is highly desirable.

Hamilton is located 1.5 hrs drive from Auckland, 2 hrs from the nearest ski fields and close to the beaches of the west and east coasts. The city has a population of 100 000, and offers cultural, sport and recreational activities. It is in the centre of the Waikato district, site of the country's top dairying and thoroughbred industries.

Salary is commensurate with qualifications and experience, starting at NZ $60 000.

Applicants should submit letter of interest, curriculum vitae and names of three referees to:

The Laboratory Manager
Alpha Scientific Ltd
141 Ellis Street
Hamilton, New Zealand
Fax: 64-7-8462346, Ph: 64-7-8462266

Queensland - Bruce Hill

Malignant catarrhal fever in a Rusa deer herd. Anita Gordon, Department of Veterinary Pathology, University of Queensland.

A herd of eight 18-month-old male Rusa deer *(Cervus timorensis*) were housed in experimental animal accommodation which included some sheep at the opposite end of the building. Following the introduction of new sheep (mostly ewes but not recently lambed), seven deer developed clinical signs of fever, depression, inappetence, diarrhoea, corneal opacities and hypopyon. Following a variable clinical course all died or were killed over a 5 week period. Six of these were examined at necropsy. The last deer died suddenly some three months later.

Gross pathological findings were highly variable between animals, bilateral ocular lesions being the most consistent. Other findings included cutaneous and gastrointestinal erosions, particularly of the oral cavity and muzzle, sparse haemorrhages of the intestinal or urinary bladder mucosa, tracheal hyperaemia, and miliary pale subcapsular renal nodules. In addition many animals showed poorly defined foci of myocardial pallor and one animal had severe multifocal myocardial necrosis. Lymphadenopathy was not prominent.

Histologically there was a severe patchy vasculitis characterised by adventitial and frequently subintimal infiltrates of mononuclear cells mostly blastic lymphocytes. There was infrequent fibrinoid medial necrosis. Changes were best demonstrated in the carotid rete, lungs, arcuate arteries of the kidney and submucosal vessels of the intestine.

Widespread lymphoid infiltrates were present in a few animals, particularly in the kidney where they tended to be periglomerular. Myocardial changes were present in many deer and ranged from focal interstitial lymphoid infiltrates to extensive myocardial necrosis with mineralisation.

Pathological findings confirmed the clinical diagnosis of malignant catarrhal fever.

Victoria - Malcolm Lancaster

Centaur, Bairnsdale

Theileriosis in a Gippsland Cow. (Alison Havadjia)

A central Gippsland practitioner (who just happens to be the husband of the writer!) was called to a "sick" cow. The patient was a ten-year-old Friesian who had been induced to calve with a corticosteroid approximately six weeks earlier. Clinical examination revealed pale mucous membranes and lethargy, but little else. Blood was taken for haematological and biochemical evaluation. Results were as follows:

HAEMATOLOGY

	Results	Reference
Hbg/dl	3.3	8.0-15.0
PVC/l/l	0.10	0.24-0.46
RBC x 10^{12}/1	1.5	5.0-10.0
	2-10	
MCV fl	61	40-60
MCH pg	22	11-17
MCHC g/dl	36	30-36
WBC x 1**O**^{9}/l	17.4	4.0-12.0
Seg.Neutrophils	6.8	0.6-4.0
Band Neutrophils	3.5	0-0.1
Lymphocytes	4.4	2.5-7.5
Monocytes	2.1	0-0.8
	0.60-1.64	
Eosinophils	0.5	0-2.4
Basophils	0.1	**O**-01
Platelets x 10^{9}/l	340	100-800

BIOCHEMISTRY

	Results	Reference
Bili - Total umol/l	23	2-12
Bili - Conj umol/l	5	0-2
Bili - Unconj umol/l		18
GLDH IU/l	106	0-45
GGT IU/l	100	**O**-60
CPK IU/l	225	0-400
Urea mmol/l	14.4	2.1-10.7
Creatinine umol/l	61	90-180
Phosphate mmol/l	2.37	0.80-2.80
Calcium mmol/l	2.11	1.90-2.60
Magnesium mmol/l		0.67
Plasma Protein g/l	86	60-85
Albumin g/l	23	27-40
Globulin g/l	63	30-45

Smear examination revealed pleomorphic piroplasms of a blood protozoan within 25% of erythrocytes. The majority was rod shaped, but some tear drop and ring forms were also present. The organisms measured approximately 1.5-2.0µm) in length and 0.5-1.0µm in width. There was moderate anisocytosis, mild polychromasia and occasional nucleated red cells in the smear. The organisms were presumptively identified as *Theileria sp.*. *Babesia sp*. was considered a less likely possibility. Yeerongpilly Veterinary Laboratory confirmed the organism as *T.buffeli*, with negative direct fluorescent antibody tests to both *B.bovis* and *B.bigemina*.

The significant features of the above profile were: a regenerative haemolytic anaemia; elevated GLDH and GGT, presumably due to secondary anaemic/hypoxic liver damage; a mild prerenal azotaemia; and hyperproteinaemia/hyperglobulinaemia/hypoalbuminaemia, due to dehydration, liver damage or immune stimulation (the albumin may have been partly artificially lowered because of the hyperbilirubinaemia).

The reported tick vectors for *T.buffeli* are *Haemophysalis longicornis* (bush/scrub tick) and *H.bancrofti*. Only the former species inhabits (south-eastern) Victoria. It is found mostly on cattle but also on other species such as horses and a variety of marsupials.

The affected cow had only been on the property in question for approximately three months but she had lived her life in central Gippsland. No ticks were noticed at the time of examination, but the farmer had previously found bush ticks on his cattle. No specific treatment was given apart from segregation into the "chopper" paddock and she died 10 days later. No post mortem samples were available. A week after the initial visit blood specimens from 10 in-contact cows were collected and examined for the presence of parasitaemia. All were negative and no further cases have been subsequently diagnosed.

T.buffeli is rarely encountered "south of the border" and is reported to be only occasionally pathogenic.

Presumably the recent corticosteroid injection interfered with this cow's immune system sufficiently to enable the parasite to replicate and cause clinical disease. The possibility of the presence of another concurrent disease contributing to her death cannot be excluded.

Centaur, Benalla

INTUSSUSCEPTION IN A BORZOI PUP - Peter Phillips.

A 5 weeks old male Borzoi pup was submitted 4 days after first being presented to the clinician because it was "not eating".

The pup had not responded to intravenous fluids and antibiotics and after one bout of bloody diarrhoea had died with "froth from its nose". Another pup from the same litter was presented to the clinician at the same time and died within 24 hours with froth and fluid coming from its nose. The pups were not vaccinated and the mother's vaccination history was not known.

At necropsy the pup had a small clot of frank blood at the anus and the belly and inner thighs were stained with bright yellow urine. All tissues were pale from anaemia. The abdomen had excess blood-tinged otherwise-clear fluid. An ileal intussusception extended well into the colon, which contained no faeces. Anterior to the intussusception the ileum was distended and contained bloody fluid. One adult *Toxocara canis* was present. The jejunal mucosa was sloughing. The liver was pale with a slight yellow tinge. The lungs were wet and heavy and the airways contained white froth. Excess clear fluid was present in the thoracic cavity and pericardial sac.

Histopathology demonstrated normal heart and kidney and a uniform mild hepatic fatty change. The intussuscepted ileum was congested and haemorrhagic, however, the anterior ileum and jejunum displayed moderate to numerous *Cryptosporidium sp.* at the lumenal surface of remaining enterocytes. A diagnosis of intussusception with a possible roundworm/cryptosporidiosis initiation was made.

ERYSIPELAS IN EMUS - Peter Phillips.

From a group of 800 emus of mixed age in a 20 hectare paddock with a 400 metre frontage to a 120 hectare lake, 5 were found dead one morning and another 5 the next. The dead birds were less than 1 year old. None had been observed to be ill. However, 7 more were dead on the third morning and close observation revealed 8 sick birds.

Clinical signs included varying degrees of flaccid paralysis, paresis, sagging necks, wings held out but "floppy", dyspnoea, injected conjunctival and oral mucosae and salivation.

The practitioner's necropsy report read "Petechial haemorrhages of conjunctivae, abdominal fat, heart (external surface), kidney, serosal and mucosal surfaces of small and large intestines. Fibrin tags on liver and corresponding surface of abdominal air sac. Red oral mucosa. Large yellow blotches on liver. 5-10 ml straw-coloured pericardial fluid. Pale spleen. Large amount of saliva in mouth. Pink-yellow abdominal fat."

Three 8 month old affected birds were referred to the Centaur Bendigo Laboratory. One was DOA, one was moribund, whilst one was ambulatory but weak and "depressed". The live birds were euthanased using 1/V Lethabarb.

Gross pathology was essentially dehydration, marked pink discolouration of fat, widespread petechial haemorrhage but especially of the small intestinal serosal surface and blotchiness of the liver. The DOA bird had a markedly reddened small intestinal mucosa with apparent haemorrhagic diathesis.

Histopathology revealed large numbers of small gram-positive bacilli in small vessels of all tissues.

The livers had wide-spread perivascular mixed-cell inflammatory infiltrate. There was necrosis within splenic white pulp and oedema and hyperaemia of villous lamina propria of the small intestines with some mixed inflammatory infiltrate. *Erysipelothrix. rhusiopathiae* was cultured from livers. Bloods from 9 birds were submitted later for serology. One returned a positive SAT for E. *rhusiopathiae.*

The reason for the original outbreak is not known, however, it is thought that the emu's propensity for cannibalising dead birds may have contributed to its rapid spread through the group. Early provisional diagnosis of botulism was understandable given the paresis, "floppy" necks and sudden deaths.

Centaur. Hamilton

Actinomycosis in a ferret - Janeen Samuel

Material was received from a ferret with a request for examination to rule out the possibility of tuberculosis. The only history provided was that the animal was a female, with pleural effusion and mediastinal abscesses.

The fixed material (presumably mediastinal although this was not specified) consisted entirely of granulomatous inflammatory tissue. The inflammatory cells were predominantly mononuclear - lymphocytes and macrophages with small numbers of neutrophils and there was vigorous proliferation of fibroblasts and capillaries. No giant cells or epithelioid cells were seen. There were numerous "club colonies", each surrounded by a broad zone of inflammatory cells. Gram staining demonstrated gram-positive branching filaments within the colonies.

Culture of the pleural effusion yielded a mixed growth of an aerotolerant actinomycete and an organism of the Haemophilus-Actinobacillus group which did not fall into any of the common veterinary species. No acid-fast bacteria were detected by ZN staining of either smears or sections.

The combination of an actinomycete and an actinobacillus (typically *Actinobacillus actinomycetemcomitans*) is not uncommon in human actinomycosis, which also is frequently restricted to the soft tissues. I have not been able to find a reference to actinomycosis in ferrets.

Porcine epicarditis associated with *Vibrio cholerae* - Janeen Samuel

This case involved piglets from a single, one-month-old litter in a back-yard piggery. Two other litters in the same piggery were unaffected. Initially two piglets died suddenly after being apparently healthy. Two others in the same litter were noted to be scouring. Fixed material received from one of the dead piglets revealed a fibrinous, proliferative epicarditis, affecting particularly the atrium and the roots of the great vessels but also involving the ventricles. The inflammatory cells were predominantly mononuclear with small numbers of neutrophils. The myocardium and endocardium were normal. The liver showed periacinar necrosis and haemorrhage while in the lung there were macrophages and occasional neutrophils in the alveoli; these changes were attributed to circulatory failure. Occasional megakaryocytes were present in the spleen. Culture of the heart blood yielded a pure growth of an organism which was identified biochemically as *Vibrio cholerae*. We postulated that this organism was a terminal invader and that the initial cause of the epicarditis had been an *Actinobacillus* or *Haemophilus* species.

Six days later another pig from the same litter died and was submitted for autopsy. The findings were: fibrinous pericarditis and marked hydropericardium; mild hydrothorax and atelectasis of the ventral parts of the lungs; ascites and fibrinous peritonitis, especially pronounced on the visceral peritoneum overlying the liver and spleen. The joints appeared normal. Histological findings were as in the first piglet with the addition of a fibrinous peritonitis overlying the liver and spleen. Both gross and histological findings were suggestive of Glasser's disease or *Actinobacillus pleuropneumoniae* infection. However, the heart tissue, liver and spleen yielded pure cultures of an organism identical to that isolated from the abdominal fluid, in mixed culture with a *Clostridium* species.

The isolates from both cases were identified by the Microbiological Diagnostic Unit, University of Melbourne, as *Vibrio cholerae* non-01. These strains do not produce cholera toxin although they may cause diarrhoea. (Note that two litter mates were scouring.). We can find no reference to them causing epicarditis in any species.

Our thanks are due both to the MDU and to Dr Pat Blackall of ARI, Yeerongpilly, who also examined the isolates for us.

South Australia - Ruth Reuter

Granulomatous sebaceous adenitis in an Akita. R. Reuter, Veterinary Pathology Services, Adelaide.

Multiple skin biopsies were submitted in formalin from a 2-year-old male Akita with a chronic skin problem. The presenting sign was recurrent corneal oedema and de-pigmentation around the eyes and muzzle. The animal was also suffering from a heavy flea burden. The sections taken were from the nasal philtrum, the lower left lip and the left thorax.

Although the lesions varied in severity, all sections showed essentially similar changes. There was orthokeratotic hyperkeratosis with multiple granulomas forming in the periadnexal areas, replacing sebaceous glands. Rudimentary sebaceous glands were still seen in the section from the nasal area, however, they were absent from the thoracic section, which showed relatively milder inflammatory changes.

Granulomatous sebaceous adenitis has been described in young to middle aged dogs, particularly Standard Poodles, Akitas and Samoyeds. The aetiology of the condition is not definitely known, however, there is strong support for a genetic disorder in the Standard Poodle. Immune mediated destruction of the sebaceous glands or leakage of irritant material from the glands stimulating a foreign body response have been suggested as the pathogenesis.

The disease is confirmed by the typical pattern of localized inflammation, accompanied by the absence of sebaceous glands in multiple biopsy specimens. The lesions are usually sterile, and most often involve the head and trunk. However, the Akita is prone to develop secondary bacterial folliculitis and furunculosis. In the later stages of the disease there may be follicular atrophy, associated with alopecia.

A Registry for Sebaceous Adenitis in Standard Poodles has been established in California. Examination of multiple biopsies from clinically normal individuals is being done there to attempt identification of animals at risk.

Reference

Dunstan RW and Hargis AM (1995) Kirk's Current Veterinary Therapy XII, Small Animal Practice, WB Saunders Co, Philadelphia, Pp 619-622.

Tasmania - Barry Monday

Report on fish histology/histopathology workshop 23-25 January 1996 at the Department of Aquaculture, University of Tasmania. - B. Munday.

A very successful interactive workshop was held at the Fish Health laboratory of the University of Tasmania. Principal tutors were Barry Munday and Barbara Nowak of the University of Tasmania and Judith Handlinger from the Tasmanian Department of Primary Industry and Fisheries. Participants consisted of four veterinarians (two servicing aquaculture), one fish health technician and seven graduate students from the Departments of Aquaculture and Plant Science (interested in the toxic effects of microalgae).

The course was based mainly on the body systems and focused on slides provided by the tutors and participants.

The feed-back from participants was very positive and more such workshops may be organised if there is sufficient demand.

Other News:

Debra Seward has joined the veterinary pathology group at Mt. Pleasant.

The new extensions/renovated laboratories at Mt. Pleasant were opened by the Minister for Police on 1/12/95.

New South Wales - Paul Gill

Lab Closures in NSW.

The NSW Minister for Agriculture, Mr. Richard Amery, has announced that the RVLs at Armidale and Wagga Wagga are to close by 1 February, 1997. The closures are linked to a $28 million cut in Treasury funding for NSW Agriculture, to be achieved over the next 3½ years. The cost saving will be made by not replacing staff that die, retire or resign, by voluntary redundancies and by asset sales.

All the staff and workload at Armidale and Wagga are to be transferred to the Department's EMAI laboratory at Menangle near Sydney. Nutrition researchers will then be transferred from Menangle to Armidale and cereal researchers will be transferred from the Biological and Chemical Research Institute, Rydalmere (Sydney) to Wagga. BCRI is to close.

According to local media, Mr. Amery has said that RVL Wollongbar will stay open for the next 3 years. RVL Orange will also stay open. However, no staff or work is to be transferred to these RVLs, as had been proposed when RVL Armidale was to close in 1988. Wollongbar and Orange RVLs are/were in federal Labor held electorates.

Farmers, veterinarians and community bodies have campaigned to stop the closures, with submissions to Mr. Amery and the Premier, Mr. Carr arguing the relative inaccessibility of Menangle, the value of local expertise and the necessity of disease surveillance for trade purposes and for exotic disease detection. As a result the above proposal has recently been referred to the Standing Committee on State Development (which consists of 7 members of the NSW Legislative Council) for review.

The proposed closure of RVLs Wagga and Armidale is a further step in the destruction of the regional laboratory system built up in Australia over the past 30 years. It comes at a time of increasing demand for services by the rural community as well as increasing requirements for export market certification due to non-tariff trade restrictions. The closures may have implications for the laboratory system in other states. They will affect veterinary pathology in Australia and membership of the ASVP as Wagga and Armidale have been great nurseries for the profession.

Members are asked lo support the retention of the laboratories by writing to:

The Ron Richard Amery MP
Minister for Agriculture
PO Box 57
MOUNT DRUITT NSW 2770
Fax 02 625 9965

The Hon Bob Carr MP
Premier & Minister for the Arts
1 Farrer Place
SYDNEY NSW 2000
Fax 02 349 4594

Regional Veterinary Laboratory, Wollongbar

Necrotizing dermatitis in a neonatal calf - Paul Gill

A neonatal crossbred beef calf was sparsely haired and had randomly distributed 1cm ovate pigmented plaques over her face. The skin of the dorsum, axilla and groin had extensive areas of parakeratotic hyperkeratosis with parakeratotic scale. The skin of the limbs had irregular patches of parakeratotic scale. From hoof to carpus/tarsus, the skin was thin and eroded and there was marked subcutaneous oedema.

Histological examinations found that large segments of epidermis were markedly infiltrated by degenerate leucocytes, the inflammation extended through the full thickness of the epidermis and often involved the superficial dermis, sebaceous glands and hair follicles. The affected epidermis was necrotic and often contained multiple lacunae of proteinaceous exudate. Aerobic culture of the skin yielded dense mixed bacterial flora overgrown by Proteus sp. This severe generalised necrosuppurative epidermitis/dermatitis is very unusual, more so in a neonate. If anyone has seen anything similar or has any useful comments, please contact Paul Gill (066 261 261).

Multiple drug resistant *Salmonella typhimurium*

In September and October, S. *typhimurium* phage type 135a, was isolated from several calves on 2 dairy farms at Johns River, near Taree, NSW. The isolates were of one biotype. They were resistant to 6 commonly used antibiotics and sensitive to trimethoprim and gentamycin.

Subsequently 46 calves on 9 properties at Johns River were examined. 8 properties were within 3km of each other and the other was 7km distant. 8 herds were involved.

S. *typhimurium* was recovered from 5 of 6 calves and from rat-infested hay on one of the index properties. Two of those calves had *S. typhimurium SA* titre $\geq$ 10. The isolates were of the same biotype and antibiotic sensitivity as the initial isolates.

All other calves, including 8 on the other index property, were serologically and culturally negative for S. *typhimurium*. Salmonellae were not recovered from calves' drinking water on any of the properties, even though some drinking water had excessive faecal contamination (>1000 faecal coliforms per 100 ml).

Equine Morbillivirus

In view of the recent concerns raised by the death of the horse trainer at Mackay who had contact with a morbillivirus infected horse 15 months previously, we recently reviewed sections of all horse lungs and kidneys sent to RVL Wollongbar since 1991 and found no evidence of the characteristic endothelial hyperplasia in lung and kidney associated with EMV. This negative finding which was communicated through the region in local newspapers and by 2 radio interviews, helped allay concerns about EMV by the horse owning public of northern NSW.

Lead Poisoning in an Eclectus Parrot - John Boulton

A 2 year old male eclectus parrot died after 2 days lethargy. Blood contained 12.4 µmol lead/L. At necropsy, it had ecchymotic subcutaneous haemorrhages - trauma had been suspected. Kidney contained 503 µmol lead/kg (wet) - greater than 120 is consistent with lead poisoning. There was no endothelial swelling in the brain.

The nuclei of many epithelial cells of convoluted tubules in the kidney contained acid-fast intranuclear inclusions. The inclusions were round, ovoid or elliptical and no more than 0.5µm diameter (no bigger than the nucleoli). They were very much smaller than the renal epithelial intranuclear inclusions that occur in lead poisoning in fruit bats (see MFC case, August 1988), primates or dogs. The intranuclear inclusions could not be confidently identified in HE-stained sections. There were no acid-fast inclusions in the bird's liver, spleen, pancreas, adrenal gland or testes. There were no acid-fast inclusions in the kidneys of other species of parrot with other diseases from other properties. The source of lead was not determined.

NORSEQ meeting, Toowoomba Vet Lab, December 1995

The NORSEQ Veterinary Pathology Group met at Toowoomba Vet Lab on 2 December. The following cases were presented and discussed:

- Friesian axonopathy - Paul Gill, RVL Wollongbar
- Canine granulomatous meningo-encephalitis - Dick Sutton, University of Queensland
- Oligodendrocytoma - Wayne Robinson, University of Queensland
- Protozoan encephalitis in a dog - Jim Taylor, Toowoomba Vet Lab
- Herpetic encephalitis in a calf - Jim Taylor, Toowoomba Vet Lab
- Limousin encephalopathy - John Boulton, RVL Wollongbar
- Classic swine fever - brain lesion - Roger Cook RVL Wollongbar
- Polioencephalomalacia - lead poisoning - John Mackie, VPS Brisbane
- Rabies-like inclusions in an English Pointer Dog - John Mackie, VPS Brisbane
- Leptomeningitis in a Cotton Top Tamorin - (*Angiostrongylus contonensus*) Melissa Novak. VPS Brisbane

- Suppurative encephalitis (*Burkholderis pseudomallei*) in sheep - Bruce Hill Rockhampton Vet Lab
- Myelomalacia in pigs associated with hypovitaminosis A - Steve Hum, RVL Armidale
- Lymphoplasmacytic meningoencephalitis (*Mycoplasma hyorhinus*) in a pig Jim Taylor, Toowoomba Vet Lab

Roger Kelly gave an overview of neuropathology and Francisco Uzol from Patagonia, a graduate student at Queensland Veterinary School, gave a well illustrated talk on 'mal seco'-a grass sickness-like condition in Patagonian horses.

Regional Veterinary Laboratory, Wagga Wagga

Reproductive failure in cattle - John Glastonbury

From 27 September to 16 October 1995 a Shorthorn stud located on the banks of the Murrumbidgee River experienced problems with calves dying during or soon after premature parturition. The progeny of 19 (13.3%) out of 143 females, 2 to 12 years old, judged to be pregnant at rectal examination in June 1995 were affected. Satisfactory vaccination programs for venereal campylobacteriosis and leptospirosis were in place. No cattle had been introduced onto the property for 7 years.

Three dead calves, judged to have progressed 8 to 9 months through gestation, were submitted to the laboratory. They appeared to be small for their ages and gross pathological findings included variable aeration of the lungs, ecchymotic haemorrhages on serosal surfaces and excessive volumes of blood-stained cerebrospinal fluid.

Serous fluids from two of the calves yielded titres of 50 in microscopic agglutination tests for *Leptospira interrogans* serovars *pomona* and *hardjo*. Bacteriological examinations were performed on the three calves with negative results. Pestiviral antigen was detected in the lung or spleen from each of the calves by means of the antigen capture ELISA

Histologically, each of the animals had mild, chronic, multifocal, non-suppurative cholangiohepatitis, one had mild, chronic, non-suppurative epicarditis and another, non-suppurative adrenalitis. The first calf to be examined had severe, acute, multifocal leukoencephalomalacia, without any obvious tachyzoites or cellular response, in the deep white matter of the cerebral cortex.

Serum samples from four of the offending dams yielded 3+ reactions in the GDPT for pestivirus.

The evidence overall strongly incriminates pestivirus as the cause of this reproductive failure, especially as the owner has subsequently commented that some of the "survivors have affected vision and some are undersized". The leukoencephalomalacia was similar to what we often observe in small ruminants (see below) which have been aborted due to infection with *Toxoplasma gondii* and suggestive of *Neospora caninum* involvement.

However, the latter organism generally causes the abortion of autolysed foetuses earlier in gestation, non-suppurative epicarditis is almost a universal microscopic lesion and the areas of leukoencephalomalacia are usually associated with a granulomatous response. The results of FATs for *N. caninum* are awaited with interest.

Toxoplasmosis in sheep and goats - John Glastonbury

Five of 450 Merino ewes aborted towards the end of gestation. In a foetus submitted to the laboratory, mild degrees of non-suppurative inflammation were detected Histologically in the portal triads of the liver, kidney, adrenal glands, myocardium, leptomeninges and the choroid plexuses. Many foci of leucoencephalomalacia attended by a granulomatous response were found throughout the white matter in the brain. Pleural fluid from the foetus yielded a Titre of 1:16 for IgG + IgM in the latex agglutination test for *Toxoplasma gondii.*

Four sets of twins from 80 Angoras were aborted near term. The only significant microscopical lesion in one animal submitted to the laboratory was severe, acute, multifocal leucoencephalomalacia throughout the brain's white matter (comparable to the above bovine case). Its pleural fluid gave a reaction of 1:4 in the latex agglutination test for T. *gondii.*

Permanent clover-induced subfertility - John Glastonbury

Ten reproductive tracts were received from infertile ewes, which had been grazing subterranean clover. Histological sections were prepared from mid-cervices, uterine horns and Fallopian tubes. Cystic changes were observed in the tunica mucosa of four cervices and two uterine horns. A diagnosis of phyto-oestrogenic induced subfertility was proposed. I am always relieved to find cysts as I am not overly confident of correctly assessing the "uterine" appearance of the cervical mucosa and the morphometric changes in this part of the female reproductive tract attributed to the ingestion of phyto-oestrogens.

Acute Chromium Toxicity in cattle - Barbara Moloney

History: A mob of 41 cows aged 6 to 8 years suffered severe mortalities, with 11 deaths over 3 days, following accidental exposure to tanning fluid. A drum (a legacy from the previous deceased owner) had been sitting in the paddock for a number of years and was noticed to have been leaking onto the pasture at the time that the deaths commenced. Local enquiries indicated that the drum probably contained a tanning concoction but there was no recorded evidence of its composition.

Clinical signs: Prior to death the animals were observed to be ataxic and some showed "stringhalt" type gait with blindness, then recumbency and rapid progression to death. The ground was observed to be "polished" where the animals had been licking at the leaking fluid. The pattern of deaths progressed within the first 12 to 24 hours of exposure, six in the next 24 hours and another 4 on the third day.

Biochemistry/Toxicology:
Tanning fluid:
* chromium 9g/L
* Reinsch test for heavy metal - negative
* Picric acid test for cyanide - negative

Cows: Indications of severe muscle and/or nervous tissue damage:
* elevated aspartate aminotransferase (323 U/L)
* elevated creatinine kinase (25,620 U/L)
* elevated liver chromium - 0.013 mg/g (13 ppm)
* negative Reinsch test and picric acid test on gut contents and tissue.

Harrison and Staples (1955) New Zealand Veterinary Journal 3:63-73 indicated that liver chromium levels of >30 ppm were needed to confirm chronic chromium poisoning but marked rises did not occur in acute poisoning. "Normal" livers were found to have chromium levels of <0.5 ppm. All values are expressed on a wet-tissue basis.

Gross pathology: Serosal congestion of intestinal tract and subcutaneous congestion.

Histopathology: Moderate congestion of the intestine; mild, diffuse myocardial haemorrhage; moderate renal haemorrhage; severe pulmonary emphysema. Unfortunately brain was not submitted.

Bovine Abortion associated with Yersinia pseudotuberculosis - Barbara Maloney

History: a single calf aborted at approximately 7 months gestation.
Serology: Foetal IgG <80ug/ml.
Gross pathology: The liver was enlarged with yellowish mottling and oedema around the gall bladder. The kidneys were oedematous on the cut surface.
Histopathology: There was a mild, sub-acute focal necrotic hepatitis, a moderate, acute foetal bronchopneumonia and vasculitis.
Bacteriology: Culture of liver,lung, spleen and foetal stomach contents yielded Yersinia pseudotuberculosis.

Regional Veterinary Laboratory, Armidale

***Yersinia pseudotuberculosis* enteritis in a buffalo. - Steven Hum and Shaun Slattery**

A week after transport to a new property a four month old buffalo calf developed diarrhoea. Its condition deteriorated over two weeks and died one night. Excess serous fluid was present in the peritoneum with fibrin tags over the surface of the liver. The mucosa of the small intestine contained numerous focal haemorrhages up to 5mm in diameter and the serosa was thickened. The mesenteric lymph nodes were enlarged and oedematous.

There was vascular congestion in the small intestine progressing to focal haemorrhages in the mucosa. There were numerous superficial micro abscesses in the lamina propria centred around bacterial colonies and a fibrinopurulent exudate within the lumen. The serosa was congested with prominent oedema and, in areas, fibrin with a low number of infiltrating mixed inflammatory cells. In the mesenteric lymph nodes there were medullary and subcapsular congestion, lymphoid atrophy and abundant proteinaceous fluid in sinusoids. The serosa was thickened, oedematous and infiltrated by mononuclear cells, mainly lymphocytes and plasma cells.

Yersinia pseudotuberculosis was isolated on selective media from the small intestine and the mesenteric lymph node.

No eggs or oocysts were found in the faeces. Selective culture was negative for Salmonella. The Tetra ELISA for Rotavirus, Coronavirus, E. Coli K99 and Cryptosporidium was also negative. No pestiviral antibodies were found in the serum and the tissues were negative for pestiviral antigen.

Yersinia pseudotuberculosis is a well known cause of enteritis in cattle and other species but it appears that it has not been reported in buffaloes in Australia.

Ostrich deaths associated with an acute fibrinohaemorrhagic tracheitis - Stephen Love, Steven Hum, Bob Coverdale and Barbara Vanselow

In the last two weeks of November there was an outbreak of respiratory disease and deaths on an ostrich farm in northern NSW. Five out of a total of 28 mixed age and sex birds died over approximately a one week period, this period coinciding with steady to heavy rain.

Sporadic deaths preceded the outbreak. One bird from a group of five 10 month old birds (pen CP1) died 2 weeks prior to the cluster of deaths, this bird having an aspergillar air sacculitis. Another bird from this group died approximately two months previously with aspergillar air sacculitis being suspected but not confirmed. This group had been agisted on another farm until August.

The first case (AN95.4055.BV) in the outbreak was also from pen CP1. After a short period of respiratory distress, the bird died. A tracheitis and air sacculitis was found. The airsacs were negative for fungus but *Aeromonas hydrophila* was cultured.

Cases 2 (AN95.4056.0C) and 3 (AN95.4058.SH) were adult birds from the same pen (QP2), and had also returned from agistment (albeit from a different property) in August. These two birds died on successive days after a day or so of respiratory distress and lethargy. A few other birds in various pens were coughing. The two dead birds had a severe fibrinohaemorrhagic tracheitis, particularly affecting the proximal two-thirds of the trachea. Apart from excess clear pericardial fluid, other organs and tissues, including the airsacs, appeared normal. About this time prophylactic treatment of birds with Lincospectin, particularly of those appearing to be at all unwell, was instituted.

Case 4 (AN95.4096.SL), an 8 month old female from pen CP1, succumbed the next day. This bird was seen to be sluggish two days previously and was treated with Lincospectin . Necropsy revealed a fungal air sacculitis (with conidiating *Aspergillus fumigatits-like* colonies). Trachea and lungs were normal.

The last case, case 5 (AN95.4120.SL), died two days later, at the end of the period of wet weather. It was the last of the group of juveniles in pen CP1. This bird also had severe fibrinohaemorrhagic tracheitis. The airsacs, sinuses, pharynx etc. appeared normal.

Findings in each bird with tracheitis were similar. Apart from Case 1, which also had an air sacculitis, changes appeared to be confined to the proximal two-thirds of the trachea. Histologically the mucosa and submucosa were congested and haemorrhagic, with fibrin exudation and a heavy inflammatory cell infiltration dominated by heterophils. The casts in the lumen consisted of fibrin, cellular debris and masses of bacteria. In each case, culture of the trachea yielded a mixed growth dominated by *Aeromonas hydrophila*.

Western Australia -David Forshaw

Chronic interstitial nephritis in brush tailed possums - John Creeper, Agriculture Western Australia, South Perth

Over a period of 8 weeks 5 Brush Tailed Possums aged between 12 months and 2-3 years were found moribund. On post mortem examination the kidneys were seen to be pale and the capsular contour distorted. Histologically all possums showed a chronic, diffuse, fibrosing and lymphoplasmacytic interstitial nephritis. No leptospires were seen in silver stains or on EM. Livers of 3 of the 5 possums showed unusual multinucleate hepatocytes. All the possums were found within the confines of the Perth Zoo precinct but were not exhibits. Similar renal changes have been subsequently seen in a Ring Tailed Possum from the south west of WA.

Toxoplasmosis in a sea lion. - Marc Kabay, Agriculture Western Australia, South Perth

An adult male sea lion was found moribund on Penguin Island (offshore from Perth W.A.) and died several hours later. At necropsy both lungs were congested and the peritoneal cavity contained a slightly turbid effusion. There was minimal body fat and the stomach and intestines were void of ingesta.

Microscopic lesions were present in the brain, lung and liver. There was a mild non-suppurative meningitis with focal gliosis in cerebral cortex.

Glial foci contained 20µm diameter stippled bodies resembling toxoplasma cysts. Pulmonary alveoli contained abundant numbers of exfoliated macrophages and protein rich fluid. Occasional toxoplasma-like cysts were seen adhering to alveolar walls.

Non-suppurative necrotic foci were present in the liver. Sometimes these lesions also contained toxoplasma-like cysts. There were no significant findings in the kidney, spleen, muscle, gut or heart. The cysts stained positive using a toxoplasma specific immunoperoxidase method. Clarified cell free extract from homogenised lung tissue was positive for antibodies to toxoplasma using the latex agglutination test.

A brief search of current scientific literature suggests toxoplasmosis in sea lions is uncommon and may not have previously been reported. As in other species, this infection was probably secondary to physiological stress depressing immunity and allowing reactivation of dormant cysts. The animal had just completed a southern migration from a breeding rookery in North West WA. Feral cats are present on this northern island.

Multiple fibropapillomas in a Green Sea Turtle. - Shane Raidal, Murdoch University

A juvenile green sea turtle weighing approximately 10 kg was found stranded at Denham, Shark Bay, Western Australia. There were multiple small (1-3cm in diameter) and several relatively large (5-10cm in diameter) round, pedunculated tumours attached to the skin of the axillary and inguinal regions. The tumours were relatively soft and were covered with small papillary projections. One relatively large tumour protruding from the ventral inguinal region had an ulcerated surface and a necrotic central core. There was a 1-2cm diameter lobulated tumour attached to the conjunctiva on the ventral eyelid of the right eye.

The animal was euthanased by intravenous injection of sodium pentobarbitone into the vertebral venous sinus by a midline approach along the ventral surface of the cranial edge of the carapace. A necropsy examination was performed. In each kidney there were several, well-demarcated, spherical, smooth, white tumours measuring 0.5-1cm in diameter.

Some were raised from the surface of the kidney and their cut surface was white. Other visceral organs appeared grossly normal. Several skin and kidney tumours and randomly selected 1 cm^3 segments of the visceral organs were removed, fixed in buffered formalin and processed routinely for histological examination. The skin tumours were non-encapsulated and composed predominantly of well differentiated, fibroblastic tissue covered by a diffusely, slightly thickened epidermis. The superficial tissue was basophilic, highly cellular and occasional mitoses were present in this layer. Deeper fibroblastic tissue was less cellular, disorganised and vascularised. Throughout some neoplasms were moderate accumulations of free melanin granules and perivascular aggregations of lymphocytes and occasional plasma cells. The tumours in the kidneys were composed of non-encapsulated, dense, well differentiated but disorganized fibroblasts within a dense collagenous ground substance. There was infiltration around renal tubules and collecting ducts at the margins of the neoplasms. Other organs appeared histologically normal.

Fibropapillomatous neoplasms such as those described have been recorded in wild green sea turtles throughout the Pacific and western Atlantic oceans and, in some marine habitats such as the Hawaiian Islands and Florida; there is a high incidence and high public awareness of the condition. In Australia there have been several recordings of similar disease in green sea turtles in the waters off the Queensland coast.

In the present case the neoplasms described were morphologically benign and the turtle appeared relatively strong and in good condition when captured. However, according to the literature, if left to progress naturally the neoplasms can grow to relatively large sizes (>30cm) and depending on their location can interfere with swimming, vision, respiration and feeding. Affected turtles often have internal fibromas, such as described in the lungs, kidneys, heart, gastrointestinal tract and liver. Growth of visceral neoplasms may compress adjacent normal tissue and organs. Consequently affected turtles have a reduced ability to survive in the wild. The gross and histological lesions described are similar to neoplasms in other species caused by papilloma viruses. However a recent experimental transmission study provides evidence that the disease may be caused by a herpes virus.

Congenital goitre in triplet kids. - Shane Raidal, Murdoch University

Two of triplets born to a dairy doe were born dead. The one kid that was born alive died a short time after birth. All three had marked bilateral swellings on either side of the neck below the jaw. One kid was presented for post mortem. The thyroid glands were markedly enlarged (5 x 3cm), firm and dark red. A remnant median connection was present between the two glands. The cut surface was dark red and firm. Histologically there was marked hyperplasia of cuboidal epithelial cells and in some areas there were follicles containing colloid. A diagnosis of congenital goitre was made on the basis of gross and histological findings.

Hematomyelia in a horse - a post anaesthesia surgery complication. - Shane Raidal, Murdoch University

Post anaesthetic and surgical haemorrhagic myelopathy (haematomyelia) is a rarely encountered condition associated with general anaesthesia and surgery in the horse.

A healthy two-year old colt developed flaccid paralysis of the hind limbs, tail and anus 2 hours after routine general anaesthesia and castration. Aortic and iliac pulses were present. The horse was euthanased and submitted for post mortem examination. Differential diagnoses were myopathy, peripheral neuropathy, fracture and a central neuropathy. There was marked congestion, areas of atelectasis and sub-pleural bullae affecting the cranioventral right lung. The abdominal visceral organs appeared normal. The vertebral column and external surface of the spinal cord appeared grossly normal. However extensive areas of dark grey tissue were evident on cross-sectioning of the spinal cord through the last thoracic and first several lumbar segments. Histological examination revealed acute haemorrhages throughout the grey matter, mainly on the right side, of the spinal cord in these areas. Some neurones appeared acutely swollen.

Systemic Cryptococcosis in 2 Horses. - Mandy O'Hara, Murdoch University

On 2 occasions this year we have seen horses with systemic cryptococcosis. Both horses were from the same geographic area and were under 2 years of age.

The first, a 6 month old Connemara pony, had a diffuse, miliary, interstitial pneumonia, with a marked, nodular, mesenteric and mediastinal lymphadenopathy. *Cryptococcus neoformans var. gattii* was cultured from lung. Histologically there was an extensive, multifocal, coalescing, pyogranulomatous interstitial pneumonia containing cryptococcal organisms. The mesenteric lymph node was diffusely oedematous with capsular fibrosis and cryptococcal organisms within the stroma.

Similar lesions were found in an 18 m.o. Arab colt, although the pulmonary reaction was restricted to the caudal lung lobes. These lesions were fibrosed and cavitating, filled with yellow, viscid, mucoid material. Cryptococcal organisms were identified histologically but serotyping is not yet complete.

An association between elevated environmental levels of *C. neoformans var gattii* and flowering river red gums (*Eucalyptus camaldulensis*) has been established in South Australia (Ellis, D.H. & Pfeiffer, T.J. 1990). Whether excessively high environmental contamination with *C. neoformans var gattii* is responsible for the disease in these horses is yet to be determined.

Ellis, D.H. & Pfeiffer, T.J. 1990. "Natural Habitat of *Cryptococcus neoformans var. gatlii*". J. Clin. Microbiol. 28. p1642-1644.

New Albany Regional Veterinary Laboratory - David Forshaw, Agriculture Western Australia, Albany.

Building of the New Albany Agriculture Centre is just about complete. The centre is the home for the new Albany Regional Veterinary Laboratory which will house the State Footrot Reference Laboratory plus Parasitology and Pathology sections. The laboratories have been designed with biosecurity in mind and the post mortem room and adjacent specimen preparation area have facilities to enable HEPA filtering of air and treatment of effluent before disposal into the sewage system. An animal house and holding yards are situated immediately adjacent to the lab. The animal house has a number of pens with heated floors to enable footrot research to continue even in our mild winters.

The office will ultimately house 120 staff when Agriculture Western Australia's regionalization program is complete but the RVL must be the smallest veterinary laboratory in Australia with a permanent staff of seven.

Despite a few hiccups such as the animal house being built 0.5m out of position and the resultant need to bend the post mortem room loading rail to compensate, the building will be in use by early March 1996. The total cost of the project is more than $9 million. All staff are looking forward to the move to the new facility which features such unheard of staff luxuries such as air conditioning (of the labs only - it doesn't get that hot in Albany...), new furniture, carpeted offices and even a shower for people working in the post mortem room.

Northern Territory - Anton Janmaat

Moderately severe clinical disease associated with bluetongue serotype 20 inoculation in sheep

Bluetongue serotype 20 was first isolated in Australia from a pool of *Culicoides spp* collected at Beatrice Hill near Darwin in 1975. Early pathogenicity work with this virus was of necessity carried out with cell culture passaged virus. Mild clinical disease was demonstrated when inoculated to sheep.

In 1992 BLU 20 reappeared in the NT when two isolations were made from sentinel cattle. Serology confirmed these were the only two animals infected.

In 1995 BLU 20 was again isolated, this time from 54 cattle and 7 buffaloes. The large number of isolations meant that a quantity of cattle blood was available to test the pathogenicity of "wild" BLU 20 in sheep. Two sheep were inoculated with cattle blood from which BLU 20 had been isolated. A third sheep was inoculated with BLU 23 for comparison, as this serotype is generally considered the most pathogenic Australian serotype.

The first sheep inoculated with serotype 20 was recumbent by day 6 but without a significant rise in temperature. Haematology on Day 7 showed a lymphopaenia and elevated creatine phosphokinase and bilirubin. By day 9 it showed severe coronitis, congestion of the nasal and buccal mucosa and facial oedema. The sheep remained recumbent until Day 11 when it was euthanased. Its temperature at this time had dropped to 39.3; there was severe coronitis and congestion of the nasal and buccal mucosa with ecchymotic haemorrhages at the mucosal junctions. Haematology at this time showed continuing lymphopaenia and elevated creatine phosphokinase and bilirubin. At necropsy there was haemorrhage at the base of the pulmonary artery, myocardial haemorrhage, congested lungs and ecchymotic haemorrhages on the abomasum and liver. Histopathology showed interstitial pneumonia and focal myocarditis.

The disease in the second sheep was less severe and the duration of its illness more prolonged. By day 6 it was reluctant to move but there was no significant temperature rise. Haematology on day 7 showed a lymphopaenia. Serum enzymes were normal. The sheep showed moderate facial oedema, coronitis and congested mucous membranes and intermittent recumbency over the next two weeks. It died on Day 22. Necropsy findings included pulmonary artery haemorrhage and congested lungs. The major histological finding was purulent bronchopneumonia.

By comparison, the sheep inoculated with serotype 23 became reluctant to move by Day 7 with a temperature of 41°C. By Day 12 it had developed severe coronitis, congested mucous membranes and facial oedema. Haematology at this time showed a lymphopaenia and raised creatine phosphokinase and bilirubin. By day 14 it had a copious nasal discharge with haemorrhage and erosion of both the nasal and buccal mucosa and was euthanased. Post mortem findings included consolidation of the left apical lobe of the lungs, haemorrhages at the base of the pulmonary artery and the apex of the heart and pale areas in the cardiac muscle.

Histopathology showed generalised interstitial pneumonia, with purulent bronchopneumonia of the apical lobe. There was focal myocarditis.

These preliminary observations suggest that the current strain of BLU 20 may be as pathogenic as serotype 23. Molecular analysis of the 1995 isolate of serotype 20 has shown it is more closely related to Indonesian and Malaysian isolates of bluetongue virus than to the Australian prototype strains of BLU 1, 3, 9, 16, 20, 21 and 23. This suggests that the current BLU 20 isolates are incursions from elsewhere.