VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
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EDITOR: Gary Reddacliff

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DEADLINE FOR NEXT VET. PATH REPORT IS JANUARY 31, 1993

ASVP DIRECTORY

ASVP EXECUTIVE 1991-1993

President	Keith Walker	RVL Menangle, PMB 8 Camden NSW 2570	046293324
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	Richard Whittington	EMAI, PMB 8 Camden NSW 2570	046293343
	Tony Ross	RVL Menangle, PMB 8 Camden NSW 2570	946293312

APPOINTMENTS

Chairperson (Registry of Domestic Animal Pathology) Tony Ross

Newsletter Editor Gary Reddacliff (assisted by Patrick Staples)

CONVENOR - SLIDE OF THE MONTH

Peter Philips	C V L, Dept of Agriculture, GPO Box 1671, Adelaide SA 5001	08 2287537

STATE REPRESENTATIVES

Queensland	Jim Taylor. Toowoomba Vet Lab, PO Box Toowoomba 4350	07 6314365
Victoria	Deb Seward. PV1H, PO Box 406 Hamilton 3300	055 723722
South Australia	Vui Ling Tham. CVL, GPO Box 1671 Adelaide 5001	08 2287322
New South Wales	Paul Gill. RVL Wollongbar 2480	066 240298
Western Australia	David Forshaw, Regional Office, WA Dept Ag, Albany 6330	
Northern Territory	Lorna Melville. PO Box 79, Berrimah 0828	089 895511
Tasmania	Barry Munday. Uni Tasmania, PO Box 1214 Launceston 7250	003 260229

A TRIBUTE TO JEREMY LANGDON

All A.S. V.P members would be deeply saddened to know of the recent death of Jeremy. He was a brilliant scholar, scientist, pathologist and a pioneer in his dearly beloved field of marine pathobiology.

So extreme were the standards of excellence he set for himself that these eventually contributed to his severe illness and early death. His productive capacity for work was legendary and the number of scientific papers produced in such a short career bear witness to the energy.

He was forever willing to assist anyone and everyone with his vast knowledge and expertise in fish diseases and yet remained cheerful, friendly and a gentleman to all colleagues - both national and international.

We all deeply regret this tragic loss of a unique talent and a delightful person.

Ron Peet.

PRESIDENT'S MESSAGE

The McKinsey and Co report was published in early November in South Australia and its implications of re-organisation/staff reductions for VETLAB are far-reaching. The ASVP executive will study the report, liaise further with members in Adelaide and comment on the report to Hon. Terry Groom MP, Minister for Primary Industries before the end of the year. NSW Agriculture's activities have been under review for 15 months now and industrial changes have been forced into effect in Victoria. Clearly ASVP members in public employ face serious challenges to morale, working conditions and technical support concurrent with rising expectations by pathology service users in general. I believe we need to view these changes (problems) as an opportunity (challenge) and to seek positive outcomes for clients and providers alike.

The Brisbane May 1993 Conference will be held at the Queensland Veterinary School and Roger Kelly has agreed to act as local organiser. The provisional training theme is reproductive pathology in various species and don't forget a full day of members contributions - so start planning yours now!

In addition the ASVP Executive must be transferred in 1993 and overtures have been made in the sunny north. Nominations in writing will be accepted up to 7 days prior to the AGM but more of that later.

Finally 1 would like to wish members Australia wide a restful and joyful Christmas-New Year 1992 holiday. Enjoy your break, be it short or long and reflect perhaps that there is a time to sow and a time to reap, a time to rejoice and a time to cry, a time for every purpose under Christ the creator’s name.

Finally in 1993 if you feel the current Executive may be able to assist in any way do not hesitate to contact us at Menangle.

Yours faithfully,

KEITH WALKER
President
for the ASVP Executive 1992-93

8 December 1992

Timing of A.S.V.P. Conference

Philip Ladds raised the timing and venue of the A.S.V.P. annual conference in the last A.S.V.P. newsletter. The conference has traditionally been held on the Saturday and Sunday before the A.V.A. annual conference, with the venue at or near the AVA venue.

Each year this clashes with A.C.V.S Council meetings and sometimes chapter meetings.

The A.S.V.P. executive considered both timing and venue for the 1992 Adelaide meeting and the 1993 Gold Coast meeting. Participation within the A.V.A. conference program was explored.

The positives were:

i) not all members of A.S.V.P have to be members of A.V.A. to attend the conference.

ii) the A.V.A. will contribute $2000.00 per day towards running a pathology program within the A.V.A. conference.

iii) the A.S.V.P. program would be advertised as part of the AVA conference, resulting in wider recognition of our activities within the profession.

The negatives were:

i) all attendees must pay the A.V.A. weekly or daily conference fee (about $150.00 per day). This compares unfavourably to the present A.S.V.P. conference cost of $50.00 for the 2 days. Even if the ASVP used all of the AVA contribution to reimburse members, a 2 day program would cost $250 instead of $50.

ii) The Australian Veterinary Poultry Association tried this at Darling Harbour in 1991. They found that only three or four non-A.V.P.A. members were attracted to the program and the high attendance fee reduced attendance by A.V.P.A. members to half that of previous meetings.

The situation was discussed at the 1992 A.G.M. of A.S.V.P. in Adelaide. Due largely to the massive increase in daily attendance fees it was agreed that the status quo be maintained.

Recently the matter was discussed with Philip Greenwood, Chief Executive of the A.V.A. However, no way around the financial problem was found. The A.S.V.P. executive invites Members to express their view on this matter.

Tony Ross
A.S.V.P. COMMITTEE MEMBER.

MEMBERSHIP UPDATE

In September 1992 the ASVP had 159 "members" who received the Vet Path report. These comprised: **86 financial members** for 1992 (including 2 honorary or life members); **53 members in arrears for this year only**, but financial for 1991 (listed below [1]); and **21 members in arrears for both 1991 and 1992** (soon to be ex-members and also listed below [2]).

Please note that your executive is not infallible. We would hate to unjustifiably remove anyone from the mailing list, so please contact us if your records do not agree with the details presented below.

Gary Reddacliff
Honorary Secretary

---

[1] The following members have not yet paid for 1992. Please note that subscriptions are for a calendar year and have been traditionally paid at or around the annual conference in May each year. Late payers simply make a lot more work for your Executive. In the interests of communication amongst pathologists, these members will continue to receive the VPR through 1993 unless they advise the ASVP that they wish to resign. Subscribers in arrears, denoted , will not receive the VPR beyond this issue.

BEERS, P
BUTTON, C
CALLAHAN, JT
CHARLES, JA
CROSS, GM
CROWLEY, AM
DOUGHTY, FR
DOWLING, LA
FORSHAW, D
FRANCE, HP
FRIEND, S
GILL, J
GLEESON, LJ
GOGOLEWSKI, RP
GRAYDON, RJ
HANDLINGER, JH
HARPER, P
HASCHEK-HOCK, WM
HINDMARSH, M
HOWELL, JMC
HOWLETT, CR
HUM, S
HUMPHREY, J
HUXTABLE, CRR
JOHNSTONE, AC
JONES, R
LATTER, M
LEE, JM
LORDING, PM
LOVE, SC
MASON, RW
MCCAUSLAND, IP
MCCOLL, K
MILLER, RI
MITCHELL, G
MORRISON, J
MUNDAY, BL
NUNN, MJ
PASS DA
PRITCHARD, DH
RIFFKIN, GG
ROBINSON, WF
ROTHWELL, J
ROTHWELL, TLW
SEAWRIGHT, AA
SMALL, AC
SMITH, HV
SMITH, BL
SMITS, B
STEWARD, DJ
SUMMERS, BA
TIMMINS, BJ
TOWNSEND, W
WHITTINGTON, R
WICKHAM, N
WILLIAMS, OJ

[2] The following members have not paid for 1991 or 1992. This will be their last Veterinary Pathology Report unless payment is received before the next issue is printed.

ALLISON, JF
BARKER, IK
CARRIGAN, M
CHICK, B
CONDRON, R
FORSYTH, WM
GLAZEBROOK, JS
HARRIGAN, KE
JERRETT, I
LANCASTER, MJ
MAIN, DC
MCCORIST, S
OLIVER, RE
RAWLIN, G
RICHARDS, RB
ROGERS, RJ
SCOTT, PC
STEPHENS, L
THOMAS, J
WHITELY, P
WILLIAMS, DM

NEW SECRETARIAT FOR THE ASVP - UPDATE

As resolved at the last AGM, your Executive has investigated the costs of having the basic secretarial functions of the ASVP run on a commercial basis. Such a move is seen as having several benefits:

i) Considerably less unrewarding and time-consuming work for the incumbent members of the executive, allowing the executive to concentrate on the more important professional matters.

ii) A stable address for routine membership-related correspondence without the inevitable confusion caused when the executive shifts from state to state each few years.

iii) As a corollary of the above, better management of the membership lists with fewer unfinancial members, and more reliable and timely despatch of the VPR.

It is possible to implement such a system within the cost of $10 per member, and so we will be commencing with a new secretariat from January, 1993. As the cost is not yet finalised (but it will be less than $10 per member) we propose to increase the membership fees for 1993 by only $5 per member for 1993. Those (very few) members who have already paid in advance for 1993 have escaped a price rise!
Membership fees for 1993 will now be:

Australian members	$25
Overseas, VPR surface mail	$30
New Zealand, VPR airmail	$35
Other o'seas, VPR airmail	$40

From 1st January, 1993 all correspondence concerning membership (payment, change of address, contact of other members) or concerning receipt of the VPR should be directed to:

The Australian Society for Veterinary Pathology
PO Box 114
Walkerville
South Australia 5081

Phone 083446337
Fax 083449227

All correspondence with regard to scientific matters, VPR content etc. will, of course, continue to be directed to the incumbent executive, newsletter editor, committee member or state representative as appropriate. These will be listed in full inside the cover of each VPR.

Comparative Animal Pathology Registry
Taronga Zoo
P.O. Box 20
MOSMAN NSW 2088
02 969 2777 x 243 (Mon/Tue)

National Registry Domestic Animal Pathology
Elizabeth Macarthur Agricultural Institute
PMB 8
CAMDEN NSW 2570
046 293 309 or 361 (Thurs/Fri)

These registries are unique collections of histological slides, paraffin-embedded blocks and projection slides of normal and diseased tissues from a wide range of species. In total there are almost 11,000 cases represented by 50,000 slides. The material is catalogued and indexed and readily retrievable. For this work we thank Bill Hartley.

WHY NOT MAKE USE OF THESE RESOURCES? Yes, we (Bill has NOT retired) can provide a second opinion/referral service. Why not plan to spend a day or two at either or both registries on your next visit to Sydney. Cheap local accommodation is available at EMAI. The CAPR has a doubled headed microscope and the NRDAP has a 5 headed microscope plus facilities for good quality projection onto a video screen. Small group or individual sessions can be arranged to suit your requirements/interest.

PLANNED. The first pathology course is being prepared for Sydney, March 1993 and will comprise:
A guided tour of avian histopathology, new and emerging diseases, an update on veterinary immunology, and participant contributions - an emphasis on the respiratory system

HELP. THE REGISTRIES REQUIRE YOUR CONTRIBUTIONS:

Histological cases: (HE & special stained slides, blocks, tissues etc) of normal tissues, common diseases (classical cases, variants) & unusual conditions for <u>any</u> species.

Projection slides: Please take two (2) pictures rather than one and send the spare to the registry. It would be very useful to have an extensive collection of slides depicting clinical conditions, gross lesions and histological changes of all common diseases encountered in Australia, plus normals for comparison. Summaries of diseases which you prepare for talks etc. would also be useful. If you are concerned about plagiarism, write your name on the slide. My own collection contains slides given to me by other workers and their name/source is present on left hand side, species on the right hand side, and description/condition/diagnosis is underneath. The top is then free for indexing.

Cytology: Several veterinary pathologists have requested help in interpreting cytological specimens. Unfortunately, the registries are a little deficient in this area (except for blood smears). Please rectify that by submitting examples of paracentesis, impression smears, blood smears etc.

Books: There are no pathology textbooks in the **NRDAP** although there is access to the EMAI library. There are very few relevant textbooks in the **CAPR**. Therefore, if you have old/superseded/new books please send them in. In particular it would be nice to have copies of:

Comparative Histology (Leuke), tumours of the Domestic Fowl (Campbell, 1969) Evan's Histological Appearance of Tumours, Pathology of Domestic Animals (Jubb, Kennedy & Palmer), Veterinary Pathology (Smith, Jones & Hunt)

Reprints: Please send copies of these and **relevant slides of anything published** which is remotely linked to pathology (of any species).

Thank you for your attention. **ROD REECE, December, 1992.**

Notice of meeting of Australian members of the European Association of Fish Pathologists Hobart mid 1993

EUROPEAN ASSOCIATION OF FISH PATHOLOGISTS

An association dedicated to the promotion of the exchange of knowledge on, and assistance in the coordination of research related to fish and shellfish pathology, with over 800 members in 43 countries around the world.

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Bulletin of the European Association of Fish Pathologists

Members receive 6 issues per annum.

Meeting of Australian members and others interested in diseases of fish and shellfish to be held in Hobart mid 1993.

Contact: Dr. F. R. Roubal, Department of Parasitology, The University of Queensland, Queensland 4072. Phone: (07) 365 5750; Facsimile: (07) 365 5799 for membership application forms and further information.

10.

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 27606

October 2, 1992

We are seeking an individual for a visiting instructor's position in anatomical pathology. The position description is enclosed. If you are aware of anyone who might be interested in this position, please inform them of the opening.

Sincerely,

T.T. Brown, Jr., DVM PhD
Professor of Pathology

Visiting Instructor - Anatomical Pathology. Department of Microbiology, Pathology and Parasitology, College of Veterinary Medicine, North Carolina State University has an opening for a visiting instructor in Anatomical Pathology. This is a non-tenure-track position with an annual appointment. Candidates should possess a D.V.M. or equivalent degree and have completed formal training (residency or graduate program) in anatomical pathology. Candidates should have partially or completely met the eligibility requirements for certification by the American College of Veterinary Pathologists. This position is intended for an individual wishing time to prepare for board certification without having the full responsibility of an academic position. About 50% of time will be devoted to supervising anatomical pathology residents and participating in the necropsy and surgical pathology service program of the College of Veterinary Medicine. Remaining time may be devoted to preparation for board certification, pursuing research interest, training with industrial and government toxicologic pathologists in the Research Triangle area, etc. Salary range - $27,000 to $30,000 per year. Starting date, July 1, 1993. Applicants should send letter of application, curriculum vitae and names of 3 references to Dr. Talmage T. Brown, Jr., Professor of Pathology, Department of Microbiology, Pathology and Parasitology, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough Street, Raleigh, North Carolina 27606, (919) 829-4258. Application closing date is January 15, 1993 or until a suitable candidate is chosen.

North Carolina State University is an Equal Opportunity/Affirmative Action Employer.

11.

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 27606

October 27, 1992

We are currently recruiting applicants for 7 positions in anatomic pathology. These positions are 2-year training programs designed to prepare residents for certification by the American College of Veterinary Pathologists. Residents are encouraged to pursue graduate training towards a PhD degree following completion of their residency.

Enclosed, please find an announcement that we ask you to share with any interested individuals.

Sincerely,

John M. Cullen, VMD, PhD
Pathology Residency Coordinator

Residency in Veterinary Pathology. The College of Veterinary Medicine, North Carolina State university, announces the availability of two residency positions in veterinary pathology. The positions will begin July 1, 1993. An earlier start date for one of the positions is possible. The program will emphasize competence in veterinary pathology to prepare the trainee for certification by the ACVP. Individuals successfully completing the training program will be encouraged to continue their training in a research program leading to the Ph.D. degree. Applicants must possess a DVM or equivalent degree; previous experience in pathology is desirable but not mandatory. First year stipend is $19,496. Applicants should send a curriculum vitae, a statement of goals and interests, complete transcripts and 3 letters of recommendation. Closing date for applications are January 1, 1993 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, N.C. 27606. For details about the program call Dr. John M. Cullen, Pathology Residency Coordinator at (919) 829-4350.

North Carolina State University is an Affirmative Action/Equal Opportunity Employer.

12.

QUEENSLAND - Jim Taylor

QUEENSLAND DEPARTMENT OF PRIMARY INDUSTRIES

Leg Deformities in Emus - Greg Storie

The Queensland Government has recently permitted the establishment of commercial emu farming. As very little is known of the nutritional requirements of emus, a trial was undertaken at the Poultry Research Centre, Redlands, to assess the effect of three different diets on growth, feed intake and feed efficiency. Eggs were obtained from the Cherbourg Aboriginal Community Farm at Murgon and incubated and hatched at the Poultry Research Centre.

Lameness was first noticed at two to three weeks of age and, by four weeks, 20% of the 180 chicks were affected. Birds from all three groups were affected. Six four week old chicks were submitted for examination and all showed unilateral or bilateral swelling of the tarsus, clockwise rotation and lateral deviation of the tarsometatarsus and lateral luxation of the gastrocnemius tendon from its condyles. Bones seemed to be of normal strength. Liver manganese levels ranged from 10.70 to 19.20 mg/kg DM (mean 14.35 mg/kg, n = 6) which are comparable to those found in "normal" poultry. Investigations are continuing. Any suggestions would be appreciated.

***Adonis microcarpa* poisoning of pigs** - Jim Taylor and Ross McKenzie

Cardiac glycoside poisoning due to *Adonis microcarpa* (pheasant's eye) was diagnosed in grower pigs (45-70 kg live weight) in a herd near Bell in mid-September 1992. Lucerne hay contaminated with the plant was fed to the pigs. Vomiting, depression, ataxia and recumbency resulted. One pig developed diarrhoea. Out of about 30 pigs affected fifteen died. The first pig died 19 hours after access to the same feed. Necropsies revealed patchy cyanosis of the skin, moderately congested viscera, scattered fibrin tags in the peritoneum and no other lesions. Histopathology revealed small scattered foci of myocardial necrosis. Inspection of the cultivated lucerne used for the pigs' feed revealed a heavy infestation of *A. microcarpa* flowering and with immature fruit at the margin of the crop. A sample of the remaining hay selected by the producer as typical of that fed to the pigs contained about 15% *A. microcarpa* by weight. This hay had been fed to cattle subsequently without ill effect.

Lead poisoning of cattle exposed to sump oil-treated cattle yards - Ross McKenzie

Lead poisoning was diagnosed in a group of 30, two year old steers introduced to a property at Beaudesert 10 days before onset of illness in early October 1992. The group was fed in yards on a small amount of grain and sorghum silage and had access to dry pasture. Other cattle had fed on the silage without ill effect. Five steers died after becoming depressed, behaving as if blind and head-pressing. No response was obtained to intravenous thiamine. Liver concentrations of lead in two steers necropsied were 9.2 and 10.6 mg/kg. The leptomeninges of one had a few small foci of eosinophil infiltration. No malacic lesions or recognizable cerebral oedema were seen in the specimens available. The source of lead was thought to be the yard timber which had been painted with sump oil some years previously. Assay of samples revealed 1757.4 mg Pb/kg in wood splinters from the yards and 2.90 and 3.60 mg Pb/kg in two soil samples taken from the drip line beneath the rails. Previous unexplained deaths on this property may have been related to this source as well.

Reference:
Slatter, DH (I971). Aust Vet J. **47**:451.

***Corchorus olitorius* (jute) seed poisoning of feed lot cattle** - Ross McKenzie

Cardiac glycoside poisoning by seeds of the jute plant (*Corchorus olitorius*) was diagnosed in a group of 15 mixed breed, mixed age feedlot cattle at Rosewood in late April 1992 after four died and 11 others were ill. The group had been on grain for about two months. Illness was first seen 12 hours after a new batch of grain was started. The attending veterinarian first saw the cattle four days after the new grain was introduced. Severe diarrhoea was seen with the faeces containing blood and mucus. Necropsy of one animal revealed fluid rumen contents (pH 5), subepicardial petechiae, very congested and haemorrhagic intestines and haemorrhages in mesenteric lymph nodes. Small black angular seeds were present in the rumen and were confirmed as *Corchorus olitorius*. No salmonellae were isolated from faeces of two cattle. Histological examination of tissues from the necropsy revealed multi-focal myocardial degeneration and necrosis and pulmonary congestion and oedema. No further deaths occurred after the suspect batch of grain was replaced and the 11 affected cattle recovered within a week.

Reference:
McKenzie, RA et al. (1992) Aust Vet J **69**:117-118.

Dwarf Darling pea (*Swainsona luteola*) poisoning of sheep - Ross McKenzie

A field diagnosis of Darling pea (*Swainsona* sp.) poisoning was confirmed histologically in a sheep from a flock at Muckadilla, near Roma, in early October 1992. A small number (6-10) of sheep are affected yearly in the flock, but this season 300-400 (about 30%) were affected in a flock of 1100 three to five year old Merino wethers. Wethers were incoordinated and apparently blind (running into fences). About 20 had died from misadventure (drowned, caught up in trees). The wethers were in good condition and were three months off-shears. The pasture was dry with some green shoots available. No alternative pasture was available to which the wether flock could be shifted. Other paddocks had inadequate water supply or were reserved for the breeding flock. The manager was providing fodder shrubs as supplementary feed. He noted that the sheep were not seeking out the small green shoots in the pasture as was usually the case. There was a large amount of *Swainsona luteola* available to the wethers and ample evidence of its being grazed. Specimens were submitted from one affected wether to confirm the diagnosis. Histopathology revealed foamy vacuolation of neurones in several areas of the brain and spinal cord, pancreatic acinar cells, hepatocytes and renal tubular epithelium.

GRADUATE SCHOOL OF TROPICAL VETERINARY SCIENCE AND AGRICULTURE -

Philip Ladds and Leigh Owens

Interesting recent diagnostic cases have included a further case of disseminated pigmented mycotic encephalitis in a cat, non-suppurative encephalitis with extensive haemorrhage and malacia in a koala and hydrocephalus in a flying fox. Diseases in farmed crocodiles being investigated include mycotic glossitis, 'bubble foot' (in which there is subcutaneous emphysema and gangrene-like loss of digits), mycobacteriosis and giant cell enteritis.

Current research in aquaculture has continued to revolve around the Bohle Iridovirus. Experimentally, the water-borne route has been shown to be important in freshwater and seawater fishes and the virus survives equally well in both environments. Interestingly, mosquito fish are completely refractory to the virus whilst crayfish can carry it in the haemolymph for more than four weeks.

The most interesting diagnostic case in aquaculture was haemocytic baculo-like virus infection in penaeid prawns at a maturation facility. This is the first case in prawns and the first outside the Atlantic Ocean. The gill area was particularly targeted and grossly numerous, small melanised foci were present. It is believed there are no serious implications of this finding for the prawn aquaculture industry.

UNIVERSITY OF QUEENSLAND

Perforating gastric ulcer/hepatotoxicity in a 3 year old thoroughbred - Anita Gordon

A three year old thoroughbred gelding was spelled following a tendon injury. It had been in work for the prior six months. After three weeks at the spelling farm, the horse was referred to the University with a one week history of inappetance. At presentation, it was also jaundiced and depressed, and died that night prior to any diagnostic work-up. At necropsy, the gelding appeared to have exsanguinated via a perforating ulcer of the gastric fundus located along the greater curvature. There were several deep ulcers of the fundic mucosa, up to 3cm in length. The liver was small, orange in colour and of normal texture. Microscopically there was severe periacinar necrosis, with megalocytosis and syncytia formation of surviving hepatocytes. Pyrrolizidine alkaloid toxicosis is a likely cause of this liver lesion. None of the other horses at either the spelling farm or racing stables have shown signs of a similar illness. The gastric ulceration in this horse was thought to be related to a protracted course of phenylbutazone, administered eight months ago for a carpal injury.

Generalised Toxoplasma (or Neospora) myositis in a bull terrier - Anita Gordon

A seven month old de-sexed female bull terrier presented with a swallowing disorder, then gradual hind limb weakness. A diagnosis of myaesthenia gravis was made, and the dog initially responded to treatment with pyridostigmine and corticosteroids. However, by 12 months of age the dog had developed muscle wasting, an inability to open the mouth and generalised weakness. At necropsy, there was pronounced streaky pallor of all skeletal muscles examined. Histology revealed widespread but patchy myositis with infiltrates of plasma cells as well as frankly suppurative foci. Numerous Toxoplasma (or Neospora) tachyzoites were visualized. In addition, there were a few foci of hepatic necrosis. Generalised Toxoplasma myositis usually occurs in young pups and kittens; possibly the prolonged steroid therapy contributed to the generalised nature of the myositis in this dog.

Pericardial and Cardiac defects in Keeshond littermates - Dick Sutton

Two, five month old male Keeshonds, part of a litter of eight, were submitted for elective necropsy following detection of variable abnormal cardiac signs which included systolic and diastolic murmurs, pulse amplitude variation and palpable 'thrills' on the chest wall. Exercise tolerance and general alertness was still good. Both pups had ventricular septal defects in the heart. One pup had a large defect (0.6 cm diameter) which was very high in the septum at the base of the aortic valve. The aortic valve showed nodularity and the proximal aorta was dilated. The other pup had a smaller defect (0.3 cm) with a fibrous nodule on the edge providing partial obstruction to the aortic outflow tract. The hearts of both pups showed mild left ventricular dilatation. Both pups showed ventro-caudal bifurcation of the pericardial sac with bilateral attachment on each side of the xiphoid cartilage leaving a 'window' allowing direct contact between the heart, diaphragm and sternum. There was also a defect in the ventral musculature associated with the diaphragm attachments allowing some direct communication between the thoracic and abdominal cavities. Herniation of omental fat had occurred in one pup. A third littermate, a male, was also diagnosed with a similar disorder but was not submitted for necropsy. A genetic basis for this disorder is a strong possibility; a repeat of the mating would be of interest.

Hepatitis in a Doberman - Dick Sutton

A six year old male Doberman with a history of progressive loss of exercise tolerance, anaemia development, hypoproteinaemia with pitting oedema of the legs, jaundice and terminal vomiting of bloody mucous and general depression, showed at necropsy, a shrunken green-coloured cirrhotic liver characterised by diffuse nodularity; the nodules varied from 1-2cm up to about 2cm in diameter. Histologically there was extensive communicating portal fibrosis with the hepatocytes being isolated into

islands. Some hepatocellular regeneration was apparent. There was a considerable amount of retained bile pigment and scattered foci of non-suppurative inflammation. Chronic active hepatitis, which this case is presumed to be, is well documented in the Doberman. It is of unknown aetiology although the very high incidence in females has lead to a genetic-based cause being hypothesised. This case is of interest because it is in a male. Liver copper levels are usually high, not because of a specific storage disease such as in the Bedlington, but rather a failure of biliary excretion which can occur in a number of chronic liver diseases. Liver copper was not measured in this case.

VETERINARY PATHOLOGY SERVICES

Angiostrongylus cantonensis Infection in a Bennetts Wallaby

In a one year old Bennetts Wallaby there was a four week history of illness which began with the sudden onset of hind limb paresis which progressed to posterior paralysis, fore limb spasticity, unilateral deviation of the upper body, nystagmus and convulsions. The animal was treated with vitamin E, levamisole, prednisolone and several doses of Ivermectin with the Ivermectin treatment appearing to provide temporary clinical improvement. Euthanasia was performed as the progressive nature of the condition became apparent. On post mortem examination, the cerebellar leptomeninges had a red granular appearance, and a focal linear area of haemorrhage was detected in the midbrain.

Histopathologic examination of the cerebellum and brainstem confirmed a severe, disseminated, focally necrotising meningoencephalitis associated with the presence of numerous nematode larvae with morphological characteristics of *Angiostrongylus cantonensis*. There was a heavy mixed leukocytic infiltrate in which eosinophils were prominent, and macrophages were present in areas of malacia. The extensive cavitating nature of the lesions, possibly associated with a surprisingly large number of parasites, was thought to be the reason for the lack of satisfactory response to treatment.

Reaction to Hip Replacement Implant

An adult male German Shepherd had a marked 'granulation tissue' reaction around the loose femoral head implant of a total hip replacement. Histopathology revealed a severe foreign body reaction with reactive granulation tissue, histiocytes and multinucleated giant cells. The severity of the reaction and dysplasia of the cells present suggested that neoplasia should be considered as a possible differential diagnosis.

This was one of the few histopathology cases where we asked the opinion of a human pathology lab. Their comment was that the reaction was typical of that seen in humans following removal of failed total hip replacements. The reaction is in response to the glue Methacrylate and possibly to titanium from the implant.

Thyroid Carcinoma In an 18 year old Arab Stallion

Histopathology of a perilaryngeal mass showed a relatively poorly differentiated solid type carcinoma with focal evidence of attempted follicle and colloid formation. Venules draining the lesion were lined by neoplastic thyroid cells. T4 assays did not show any functional abnormality. Thyroid carcinoma, particularly the poorly differentiated forms, is often non-secretory. Metastasis is fairly frequent, usually haematogenous via the venous drainage and the jugular vein, with secondaries being most common in the lung. Large tumours may cause respiratory problems.

Leishmaniasis seen in Sydney

A nine year old dog presented to the veterinarian with haemorrhage from the oral cavity. On physical exam major findings were absence of an oral lesion to explain the haemorrhage and a palpable, likely splenic mass. Salient findings on blood submitted to the lab included severe microcytic, poorly responsive anaemia, mild thrombocytopenia, severe azotaemia (Creatinine 0.77mM) and hyperphosphataemia (5-

7mM). Additionally, there was hyperglobulinaemia (64g/L). Fine needle aspiration of the spleen revealed plasma cell and macrophage hyperplasia and numerous characteristic *Leishmania* organisms in occasional splenic macrophages. Renal failure secondary to glomerulonephritis is a common scenario with visceral leishmaniasis.

Leishmaniasis is an **exotic disease** caused by a protozoal organism. It is quite small, approximately 3/µm long by 1.5µm wide and is found within macrophages. After talking to the veterinarian, it was revealed that the dog had been imported from the Mediterranean basin, an endemic area for *Leishmania spp*.

Melioidosis in two Pinipeds

The first animal was presented in an agonal state on 16 February 1992 and died shortly after blood was taken. Haematology revealed marked leucopenia with neutrophils showing severe toxic changes indicating an overwhelming infection. The liver had multiple pinpoint necrotic white foci in all lobes. Histologically there was severe necrotising hepatitis, splenitis and enteritis.

The second animal died on 27 February 1992 with blood from nostrils. It had severe, grossly haemorrhagic pneumonia. Histopathologically there was severe necrotising suppurative pneumonia, lymphadenitis and splenitis.

Pseudomonas pseudomallei were isolated from multiple organs from each animal. Pseudomonas pseudomallei are a soil frequenting organism. Clinical cases become more prevalent during the wet season in northern Australia and infection is thought to be by ingestion.

The animals' concrete environment was contaminated by water run off following flooding of a reptile display (including earth and plants) in a geographically higher location in the marine park

VICTORIA - Deb Seward

RVL BAIRNSDALE

Bovine abortions - Ian Jerrett

Tissues were received from 150 bovine abortions this season. Most sporadic abortions remained undiagnosed but a small proportion was due to infection with *Aspergillus famigatus, Bacillis lichenifomis, Campylobacter fetus, Salmonella* spp and *Actinomyces pyogenes*.

Infection with *Mortierella wolfii* was diagnosed as the cause of an abortion storm involving more than 15 cows in a Maffra dairy herd. Dermatitis and thromboembolic encephalitis were present in one foetus, suppurative pneumonia in a second and necrotising placentitis with vasculitis in tissues from a third abortion. *M.wolfii* was isolated and all of 7 maternal blood samples tested positive to the *M. wolfii* CFT. Mouldy maize silage was the suspected source of infection but overgrowth of silage cultures with *Aspergillus fumigatus* prevented confirmation. A similar abortion storm in the Bendigo area was confirmed to be due to *Mortierella* infection when 8 of 10 maternal bloods forwarded from RVL Bendigo tested positive to the CFT.

Protozoal infection was diagnosed as the cause of two outbreaks of abortion and a number of sporadic abortions throughout Gippsland. Focal leucomalacia and gliosis were consistent features, and hepatic necrosis and non-suppurative myocarditis were often present. The identity of the causative protozoa in this syndrome is still uncertain. Protozoal structures when visible have stained positive by immunoperoxidase for *Sarcocystis cruzi* in cases encountered at this laboratory. We have not yet checked these protozoa with immunoperoxidase for *Neospora caninum*, and we are keenly awaiting information on the life cycle of *Neospora caninum*.

Presumptive food poisoning in a dog - Kit Button, Ken Slee

An owner cooked up a copper full of bovine livers and kidneys one Saturday. The resulting "stew" cooled overnight and was fed to 8 of 9 foxhounds on Sunday morning. By evening, all 8 which had eaten the "stew" were showing signs of muscle tremor, a hunched appearance, apparent colic and vomiting. Several dogs later developed bloody diarrhoea. One dog died and was submitted for autopsy. This dog had a necrotising haemorrhagic enteritis - numerous bacilli were seen in the lumen and *lamina propria* of the SI and *Clostridium perfringens* was cultured in heavy growth from both the SI and from samples of the cooked meat.

"Pig Bel" is a syndrome of haemorrhagic enteritis which is recorded amongst the people of Papua New Guinea following ritual feasting on large volumes of inadequately cooked pork. Slow cooling of a large volume of poorly cooked meat is a typical feature of clostridial food poisoning in humans. This appears to have been the case in the above outbreak in dogs.

Inherited abnormalities in progeny of an AB sire - Peter Mitchell

Arthrogryposis and a bleeding disorder have occurred in progeny of cows inseminated with semen from a bull at an artificial breeding centre. Fifteen of the progeny were examined at the laboratory - seven still-born calves with arthrogryposis and eight live-born and apparently normal calves. The still-born calves were all full term but were born dead with no evidence of dystocia (although local veterinarians reported some dystocias in other calves from the same sire). Alt had fixation of limb joints that could only be relieved by cutting the joint capsule. Some bones were severely distorted, particularly joint surfaces, but there was no joint fusion. Some calves had flaps of skin between their hind legs extending down to the

stifle. Skeletal muscles, brains, spinal cords and peripheral nerves appeared normal, so the cause of the arthrogryposis is unknown.

The live-bom calves were examined at <48h old. All had haemorrhages in the subcutis and underlying muscle, particularly in areas prone to trauma, petechiae notably throughout the lungs and abomasal mucosa, and haemorrhages in the AV valves of the heart. As there was no history or signs of dystocia or foetal hypoxia, the haemorrhages were regarded as abnormal. Tests showed prolonged bleeding times compared with normal calves - two had bleeding times > 15 min (tested by using Surgicutt devices on shaved ears). However, clotting times, as well as prothrombin times and activated partial thromboplastin times tested at Werribee, were similar to the normal calves. The cause of the haemorrhages and prolonged bleeding times is not yet known.

Occasional deaths have been reported among the live-born calves, but these deaths were probably due to the range of infectious agents that commonly afflict neonatal calves (none of these dead calves have been submitted to the laboratory). At this stage in the investigation, there is no evidence that the calves with bleeding tendencies have any reduced chances of survival.

There is no history of comparable problems in the ancestry of the bull. The bull probably has some chromosome damage but this has not been uncovered by karyotyping to date. So there are many unanswered questions, and opportunities for further research.

Toxoplasmosis in a canary - Janeen Samuel

A canary breeder lost 11 out of 70 birds over one week. The owner reported that the birds became sick and died over 2 to 3 days: their breathing became “heavy” but there was no change in their droppings. Deaths occurred in all ages of bird.

We received one dead bird for examination. No lesions were seen grossly, and no bacteria were isolated from the liver or lung. Histologically, there was focal non-suppurative myocarditis, focal myositis in the wall of gizzard, focal hepatitis, and a non-suppurative interstitial pneumonia. In several areas of the brain, blood vessels showed thrombosis and/or perivascular cuffing by lymphocytes and heterophils, and there was accompanying malacia. Adjacent to these areas there were several pseudocysts consistent in appearance with *Toxoplasma gondii*.

We were unable to discover whether the canaries’ feed had been exposed to cats. However, the history included the observation that new hay had been placed in the nesting boxes two weeks before the deaths began.

REGIONAL VETERINARY LABORATORY, BENALLA

1. PULLORUM DISEASE IN JAPANESE QUAIL

A high mortality (30%) of Japanese quail (*Coturnix coturnix*), was reported from the Cheshunt area. Samples were submitted for necropsy. The striking lesions were white spots (2mm in diameter) throughout the heart, small intestines and pancreas, with red spots in the liver and lungs. Histopathological examination revealed the ‘spots’ to be areas of necrosis with a lymphocytic infiltrate. Salmonella were cultured from heart and liver samples.

The organisms isolated were biochemically and serologically indistinguishable from *Salmonella pullorum*. This was confirmed by the Microbiological Diagnostic Unit, Department of Microbiology, University of Melbourne, and by The Australian Salmonella Reference Laboratory in Adelaide. Other quail flocks have been exposed by the transfer of fertile eggs and an eradication program is underway. *Salmonella pullorum* is a notifiable disease and has not been reported in domestic poultry in Victoria for many years.

(Michael Hindmarsh)

2. YERSINIOSIS AND GIZZARD WORM IN A DIAMOND FIRETAIL

An aviary kept diamond firetail finch died after a period of depression and diarrhoea lasting for 36 hours. At necropsy there were yellow granulomas throughout the liver and one in the lung. There was extensive haemorrhage beneath the koalin of the gizzard and there were whole millet seeds present in the lower intestine. Microscopically the granulomas contained necrotic centres containing numerous bacterial colonies consistent with Yersinia and a marked mixed cellular reaction. There was extensive degeneration of, and bacterial colony growth in, the koalin layer of the gizzard which had lifted. There were inflammatory cells and nematode eggs in the koalin and numerous adult nematodes beneath it The underlying glands are inflamed and atropied. The malfunctioning of the gizzard was attested to by the undigested seeds in the intestines. Yersinia pseudotuberculosis was cultured from the liver lesions. The nematodes are most likely Acuaria skrjabini. The debilitation of the bird and inflammation of the gizzard likely contributed to allowing the yersiniosis to develop.
(Judith S. Nimmo Wilkie)

3. BLUE GREEN ALGAL POISONING IN CATTLE AND SHEEP

Blooms of Microcystis aeruginosa were associated with deaths in both cattle and sheep. Two mature Friesian cattle died after having access to water containing *M. aeruginosa*. Post mortem findings included ascites, pericardial effusion and pulmonary oedema. Histopathological lesions included the massive, haemorrhagic periacinar necrosis classically associated with blue-green algal toxicity.

The affected sheep, even those that died, did not show this lesion. Despite the biochemically confirmed jaundice and hyperbileacidaemia, the vast majority of hepatocytes were still intact in most sheep, with only an occasional necrotic cell present. Enzymes of hepatocyte origin were only mildly elevated in circulating blood. Hepatocytes were generally swollen with some vacuolation and bile thrombi were sometimes evident in canaliculi. Perisinusoidal macrophages were more prominent than normal. Jaundice, photosensitisation and occasionally gastroenteritis can occur following repeated administration of smaller doses of the toxin.
(Malcolm Lancaster, Michael Hindmarsh)

4. AN OUTBREAK OF TICK PARALYSIS IN FOWLS

An outbreak of leg-paralysis occurred recently in a free-range poultry flock. Affected birds were 6 months of age and just coming into lay. Affected birds were unable to move and died within 1-2 days from starvation or cannibalism or secondary infections. They would eat, drink and remain alert if hand-fed. The birds had been vaccinated for Marek's disease.

Four birds submitted to the RVL were heavily infected with larval *Argus persicus* ticks. No significant gross or histological lesions, including any suggestion of Marek's disease, were found in any of the birds. The birds were not significantly anaemic and no *Borrelia* organisms were seen on blood smears or in tissue sections. Treating the birds with an insecticide produced rapid and complete regression of the clinical signs. These findings are confirmatory of tick paralysis to the toxin produced by engorging nymphal ticks. Birds do mount a mild immunity to the toxin but only after repeated exposures.
(Judith S. Nimmo Wilkie)

SOUTH AUSTRALIA - Vui Ling Tham

VETLAB, ADELIADE

CAPRINE JOHNE'S DISEASE - (VUI LING THAM)

In June, this year a 6-year-old Anglo-Nubian doe from a very well managed small property in the Adelaide Hills was euthanased as she had lost over 50% of her body weight and had been scouring (initially intermittently but subsequently persistently) over a year, and not responding to treatment with sulpha drugs or to treatment for Giardia and helminth.

Post mortem examination carried out soon after the euthanasia revealed thickened and roughened mucosa of the ileum and colon with multifocal mucosal erosions. The remaining tissues were normal. Faeces and portions of the ileum, colon and mesenteric lymph node for microbiology and duplicate samples of ileum, colon and mesenteric lymph node in 10% formalin for histopathology were submitted to this laboratory.

Histologically, the ileum showed heavy infiltration of epithelioid cells in the mucosa resulting in thickening and elongation of the villi, multifocal polymorphonuclear leucocyte infiltrate in the mucosa, and moderate multifocal epithelioid cell infiltrate in the submucosa. Numerous acid-fast bacteria were present in the epithelioid cells.

Aggregates of epithelioid cells were also noted in the colonic mucosa and submucosa accompanied by inflammatory cell (including polymorphonuclear leucocyte) infiltrate and multifocal mucosal ulceration. Numerous acid-fast bacteria were present in the epithelioid cells.

The mesenteric lymph node showed moderately heavy multifocal infiltration of epithelioid cells in the cortex and medulla accompanied by areas of caseous necrosis and calcification. Numerous acid-fast bacteria were present in the epithelioid cells and areas of necrosis.

Smears prepared from the faeces, the ileal and colonic mucosa, and the mesenteric lymph node showed large numbers of acid-fast bacteria, *Mycobacterium paratuberculosis* was isolated from the faeces and the tissues.

Foci of necrosis and calcification have been reported in Johne's disease in goats and sheep, though these changes are rarely seen in bovine Johne's disease.

This appears to be the first report of caprine Johne's disease in South Australia. Interestingly, some two years ago this doe returned from lease at another Adelaide Hills property where 2 goats from NSW had died after scouring profusely and becoming emaciated but unfortunately the cause of the diarrhoea was not investigated.

(Acknowledgment - Dr Elizabeth Aikenhead from the Strathalbyn Veterinary Clinic, Strathalbyn, SA supplied the history, performed the necropsy and submitted the specimens to VETLAB for examinations.)

HEPATIC NECROSIS AND HAEMORRHAGES IN A YELLOW FOOTED ROCK WALLABY (*PETROGALE XANTHOPUS*) - (Vui Ling Tham)

In June, this year a one year-old male yellow footed rock wallaby from the Adelaide Zoo was found dead. It had several episodes of non-specific illness over the previous three months. Post-mortem examination revealed a large quantity of blood in the abdominal cavity and areas of haemorrhage and necrosis in the liver. Portion of the liver, kidney and spleen were submitted to VETLAB for histopathology.

The changes in the liver ranged from mild to moderate multifocal sinusoidal dilation and hepatocellular dissociation and necrosis to severe multifocal hepatocellular dissociation and necrosis with replacement haemorrhage. Part of the Glisson's capsule was markedly thickened containing areas of necrosis and haemorrhage, foci of calcification and some haemosiderin laden macrophages. There was also mild to moderate biliary hyperplasia.

The splenic changes consisted of multifocal thickening of the trabeculae, mainly involving the connective tissue component. Areas of necrosis, haemorrhages and some haemosiderin containing macrophages and an aggregate of giant cells were present in the thickened trabeculae.

There were no significant findings in the kidney.

Internal haemorrhage (very likely from the liver) is considered to be the cause of death of this wallaby. We do not know the aetiology of the above changes. The hepatic changes somewhat resemble peliosis hepatis. Query defect in collagen and/or reticulin. We welcome comments from anyone who knows the aetiology or likely aetiology of the above changes.

NECROTTC ENTERITIS IN A RAINBOW LORIKEET (*TRICHOGLOSSUS HAEMATODUS*) - (Vui Ling Tham)

Towards the end of October, this year a dead rainbow lorikeet from an aviary in the Murray Bridge area was submitted to this laboratory for necropsy. This bird was depressed for a few days and then died. The sickness and deaths started in the aviary about a week after the introduction of a scaly breasted lorikeet (*Trichoglossus chlorolepidotus*) from the Barossa area in mid September, this year. Since then two scaly breasted lorikeets and five rainbow lorikeets (including the one submitted for necropsy) have died after being depressed for a few days.

Post mortem examination revealed a fair body condition but no soiling of the vent; multiple discrete yellowish diphtheritic lesions in the wall of the middle third of the intestine easily visible from the serosal surface, and slight cloudiness of the air sacs. Smears prepared from the intestinal lesions showed numerous *Clostridium*-like bacteria mixed with other bacteria, particularly gram negative rod-shaped bacteria.

Histologically, there was severe multifocal necrotising and ulcerative enteritis associated with *Clostridium*-like bacteria and other bacteria including gram negative rod-shaped bacteria.

Culture from the intestinal lesions yielded a heavy growth of non-haemolytic *Escherichia coli* and a mixture of anaerobes including *Clostridium perfrigens*.

This disease appears similar if not identical to the one in Queensland reported by Janeen Samuel and Christine Cannon (See PGF Control and Therapy No 953 (1979)). A similar disease was reported in NSW (See Veterinary Pathology Report No 15, Jan 1987, p.9)

NEW SOUTH WALES - Paul Gill

RVL WOLLONGBAR

Craniofacial tumour in a koala - Paul Gill

A young male adult wild koala presented with a moderate mucohaemorrhagic unilateral nasal discharge. Computerised tomographic scans of its head revealed a tumour extending from the right retrobulbar area into the ethmoidal and maxillary sinuses. Postmortem examination found an ovoid tumour approximately 1.5 x 0.5cm x 0.5cm firmly attached to the frontal bone about 1cm caudal to the right eye. The tumour extended through the skull to the caudal ethmoids. On cut-section, the retrobulbar mass was firm, white, shiny and arranged in 2-5mm nodules. Microscopic examination found variably-sized groups of hypertrophied chondrocytes with sparse to relatively abundant chondrial matrix, separated by dense bands of fibrous tissue. In many areas, particularly in the intracranial portion of the tumour, bony trabeculae were produced. This tumour is very similar to the craniofacial tumors of mixed cartilage and bone described in koalas by Paul Canfield (Aust Vet J (1987) **64**:20-22). These tumors normally produce facial deformity, but no deformity was evident in this koala despite the invasion of the skull and sinuses.

Haemoglobinuria in cows grazing peas - Graeme Fraser

Haemoglobinuria occurred in 5/14 beef cows grazing the residue of a pea (*Pisum sativum*) crop. Laboratory examinations excluded diagnoses of babesiosis and leptospirosis. McBarran (in 'Medical and Veterinary Aspects of Plant Poisons in New South Wales', Department of Agriculture, New South Wales, 1976) reports *P. sativum* seeds may cause haemoglobinuria and jaundice in humans. It would appear *P. sativum* can cause a similar condition in cattle.

Ulcerative dermatitis in silver perch - Dick Callinan

Over 50% of silver perch at a large fish farm had dermal ulcers. The ulcers were present most often on scaled areas of the body but also on the fins and head. They ranged from about 3mm in diameter with focal scale loss, pallor, petechiation and softness of the underlying tissue, to 1cm in diameter, with slightly raised reddened edges and depressed pale centres with exposed stratum compactum. On cut surface, there was often reddening and swelling beneath the stratum compactum. Histological examinations revealed the following stages of ulceration:

a) early ulcer - focal to locally extensive epidermal loss with moderate to severe subacute inflammation and necrosis in the stratum spongiosum associated with scale loss and waterlogging;

b) dermal ulcer - locally extensive necrosis and sloughing of the stratum spongiosum and necrosis of the underlying stratum compactum which was associated with the presence of numerous bacilli between lamellae and beneath the stratum;

c) *Aeromonas salmonicida* was cultured from several ulcers. This is the first confirmed natural infection of Australian native fish by *A. salmonicida.*

RVL WAGGA WAGGA

Staff - During early October 1992 an Enterprise Agreement was completed for the Professional Officers employed by NSW Agriculture. The agreement draws together Veterinary Officers, Scientific Officers, Engineers and Librarians into a common salary scale. The scale is composed of 7 Grades but promotion on personal merit is not possible beyond the 3rd Year of Grade 4.

Promotion within the first 4 Grades will be based upon the result of performance assessment and the meeting of certain relatively stringent but reasonable criteria. Interestingly Membership and Fellowship of the Australian College of Veterinary Scientists are included in the criteria.

The starting salary for Veterinarians is 4th Year of Grade 1 or $30,191/annum. The many Veterinarians in NSW Agriculture, who in the past have been caught on Grade 2 in the old Veterinary Officers' salary scale, will be transferred to Year 3 Grade 4 in the new scale plus paid an allowance of approximately $1400/annum. Officers-in-Charge of Regional Veterinary Laboratories will continue to be paid by way of an allowance which will be $3000/annum. This is double the previous allowance.

Frothy bloat in cattle - Sudden death amongst 6-month-old Hereford and Hereford-cross heifers resulted in a mortality rate of 3.4% amongst 291 animals at risk. The pasture was lush and mixed including a moderate amount of clover.

The diagnosis was based upon a process of elimination and the gross pathological findings which included congestion and haemorrhagic lymph nodes of the upper respiratory tract, haemorrhages in the tunica mucosa of the trachea, interstitially in the thymus and in the epicardium as well as parietal pleura and in some cases frothy ruminal contents. However, untoward delays in performing necropsies did confuse this picture. Enterotoxaemia and hypomagnesaemia were eliminated as diagnostic possibilities.

Pasteurellosis in sheep - Soon after shearing, 10-week-old Merino lambs developed extensive subcutaneous oedema and dermal necrosis. Morbidity and case fatality rates were 10.0 (200) and 25.9% respectively. Heavy pure growths of *Pasteurella haemolytica* Type A were recovered from the subcutaneous tissues and histologically there was severe fibrinoid necrotising vasculitis in dermal blood vessels.

Ethyl fenthion toxicity in birds - Twenty sick and 10 dead Sulphur-crested cockatoos were found upon inspection of the Tidbinbilla Nature Reserve in the ACT. Examination of liver, gizzard contents and grain from the crop detected 1.5, 20.0 and 150.0 mg/kg of ethyl fenthion. Non-specific pathological findings included pulmonary congestion and haemorrhage as well as mild haemorrhagic enteritis.

RVL MENANGLE

Clinical signs and lesions associated with Pestivirus infection in cattle - Keith Walker and Gary Reddacliff

The following is not intended to be an exhaustive treatment, but rather a perspective gained from recent routine diagnostic submissions at RVL Menangle. The epidemiology of pestivirus infections in cattle is complex, and the excellent Sydney University Post Graduate Committee in Veterinary Science review article "Mucosal Disease of Cattle" by IR Littlejohns should be consulted for more background information.

Probably the commonest presentation of pestivirus infection in a herd is infertility due to early embryonic loss and abortion, or the birth of calves with varying degrees of central nervous system damage, depending on the stage of gestation when the dams were infected. Maternal infection before immunocompetence of the foetus (about 120 days), if it does not cause embryonic loss, may result in the birth of persistently infected, carrier animals, which may later develop clinical signs. Infection later in gestation will induce an immune response in the foetus, but will not give rise to persistent infection. The detection of pestivirus antibody in pre-suckle serum of calves or pericardial or thoracic fluids of an aborted foetus is diagnostic of intrauterine infection in these animals.

The clinical breakdown of a pestivirus carrier animal causes signs which include ill-thrift diarrhoea, oculonasal discharge, skin scurfiness and lameness. Lesions include shallow ulcerations of the muzzle, mouth, pharynx and oesophagus, and similar lesions at the skin-horn junction on the feet. Histologically, these mucosal lesions are non-specific, but the association with severe necrosis of Peyer's patches in the intestine is typical of persistent pestivirus infection. When these signs appear acutely and progress rapidly to death, the disease has been called "acute (or classical) mucosal disease", and may be associated with superinfection of the carrier animal with another strain of pestivirus. Such acute presentations are not particularly common. The more common, chronic manifestions are often associated with intercurrent disease, and may not be recognised as pestivirus infections unless virological examination is undertaken.

With the sensitive and cost-effective antigen-capture ELISA for pestivirus developed by Dr Tony Shannon, EMAI Virology, we are recognizing a wide variety of conditions in cattle associated with persistent pestivirus infection, including of late:

***** Three clinically normal carrier steers were detected at yearling age in a herd with a history of early embryonic loss and poor perinatal survival from heifers joined the previous year (MN92/2779).

***** A steer with ill-thrift, lameness, oral ulceration, ventral lung consolidation and severe abomasal ostertagiasis (MN92/2765).

***** A dairy heifer, half the size of her age peers with unusual hoof growth in all four feet requiring regular paring (MN92/3379).

***** A bull being fattened for showing, with profuse diarrhoea over ten days, and bilateral oculonasal discharges (MN92/3183).

***** Scouring and respiratory disease unresponsive to treatment in an AIS cow (MN92/3135).

***** *Klebsiella* infection of the placenta in a case of stillbirth/dytstocia. The calf was detected as persistently pestivirus infected by antigen-capture ELISA on the spleen (MN/2567).

***** Acute interstitial pneumonia associated with *E. coli* infection in a cow with chronic diarrhoea and weight loss (MN92/4036).

***** Severe chronic bronchopneumonia, associated with lungworm larvae and heavy pure growth of *Moraxella nonliquefaciens*, in an illthrifty, heavily louse infested 10 month old steer. The animal also had severe scurfiness and some erosions around the feet, with a very heavy infestation with *Chorioptes bovis* (MN92/2562).

* Severe miliary pulmonary abscessation associated with lungworm larvae in a 9 month old heifer (MN92/2381).

Presumably many of the observed intercurrent diseases are associated with immunosuppresion due to the persistent pestivirus infection.

WESTERN AUSTRALIA - David Forshaw

ALBANY REGIONAL LABORATORY

Probable superphosphate poisoning in rams - David Forshaw

2 rams from a mob of 100 died five days after shearing. 50% of the mob appeared depressed and three refused to be driven.

Necropsy of the two dead animals revealed marked sub-cutaneous congestion and severe pulmonary congestion and oedema. There was mild sub-cutaneous oedema of the head and neck and excess clear pericardial, pleural and peritoneal fluid in one. The mucosa of the abomasum was congested with petechial haemorrhages. Both had equivocally swollen, pale kidneys. Histologically, there was severe acute tubular necrosis.

The rams had been held in a superphosphate shed prior to shearing and the farmer thought he could see where they had licked material from the floor.

Coronavirus infection in calves - David Forshaw

20 of 36 calves purchased 11 days previously from another property had been scouring for two days. The veterinarian also noted fever (39.7-41C) and conjunctivitis. The calves were still quite bright. Six faeces samples were submitted and a heavy growth of E.coli was cultured from three. An unusual mixture of coliforms, bacillus and fungi was cultured from the other three. Four animals had particles consistent with coronavirus in their faeces.

This is only the second time corona virus-1ike particles have been seen in cattle faeces in W.A.

Cardiac myopathy in steers - David Forshaw

Two 18 month old steers "dropped down dead" while being moved from a clover/grass paddock. Two others staggered and were left in the paddock. Others appeared sluggish when moved.

Post mortem descriptions of one were of pulmonary oedema, petechial haemorrhages on the heart and fibrosis of the heart muscle.

Histopathological findings were multifocal extensive areas of acute myodegeneration with mild mononuclear cellular response.

Trace element analyses on fixed liver samples - Cu 81mg/kg, Se 0.13 mg/kg. Further blood testing of the herd revealed plasma seleniums of between 5 and 7 ng/ml which are considered marginal.

This appears to be a case of selenium deficiency in adult cattle.

Enzootic ataxia in red deer *Cervus elaphus* in Western Australia by Ron Peet and Kevin Hepworth (Dongara Veterinary Hospital)

Enzootic ataxia occurs in feral, park and farmed deer of different breeds in New Zealand, the United Kingdom and Europe (Wilson 1984). It has long been considered to be associated with copper deficiency and resembles swayback in sheep with a progressive bilateral demyelination of the tracts of the spinal cord {English 1984). However, enzootic ataxia differs from the condition in sheep in that it is not found in neonates or young deer but is found in young adult or even mature deer (Wilson 1984). Also there is some uncertainty as to the cause or causes of enzootic ataxia in deer apart from copper deficiency since some animals with low blood and liver copper levels do not develop the disease (English, Wilson 1984). Other factors involved may be a hereditary pre-disposition to the disease which can appear after introduction of a new stag and the in-breeding which is likely to occur on a deer farm. Other trace element deficiencies and toxins have also been suggested as aetiological factors (English, Wilson 1984).

One hundred and ten male and female deer 1 to 2 years old were pastured on a coastal property near Geraldton in Western Australia. The owner had purchased the property and converted it from a wheat and sheep enterprise to a deer and emu farm. As trace elements had been added for some years, and when hind limb weakness was noticed in some of the deer, nutritional myopathy was suspected. This hind limb ataxia became more pronounced with exercise with some animals going down and being unable to rise. If left alone for some time they regained their feet and walked reasonably normally. However, deaths occurred in four recumbent animals and heparinised blood was then collected from 7 animals. Glutathione peroxidase levels were 143± 7 lUg Hb (Mean ± SEM) and Vitamin E values were 1.14 ±. 11 mg/1. These were not considered deficient but plasma Vitamin B12 levels were 0.07 ± .02 µg/l and these were considered low. Sampling was done in January 1992 (summertime) on dry pasture when cobalt deficiency does not usually occur in Western Australia. Unfortunately, there was insufficient blood remaining for blood copper estimations.

The owner agreed to sacrifice a clinically affected animal and a post mortem examination was performed. Marked haemorrhagic areas were seen in many muscles including quadriceps, biceps, triceps and psoas groups. There were no other visible lesions, and a range of tissues including skeletal muscles, heart, lung, liver, kidney, spleen, abomasum and the entire spinal cord and brain collected in buffered 10 percent formalin for histopathology. Fresh liver was also collected for trace element analysis, fresh brain for bacteriology and a faecal sample submitted for parasitology.

Examination of these samples at Animal Health Laboratories (AHL) revealed no larvae of *Elaphostrongylus cervi* in the faeces and *Staphylococcus aureus* was cultured from the brain. Histopathology revealed evidence of acute and chronic skeletal myopathy characterised by areas of haemorrhage, myonecrosis, regeneration attempts with myoblast formation and fibrosis.

Microscopic examination of the spinal cord revealed widespread demyelination of tracts at all levels sampled which included cervical, thoracic and lumbar. Foci of demyelination were also seen in the medulla oblongata, but there were no visible lesions in the brain and on this result, the *S. aureus* isolate cultured from the fresh brain was considered to be a contaminant.

There was marked haemosiderin deposition in some macrophages and Kuppfer cells in the liver and in the spleen, but no significant findings in the other tissues. Fresh liver analysis revealed apparently adequate levels of selenium, 0.50 mg/kg and Vitamin B12, 0.73 mg/kg but the liver copper was 4 mg/kg (62.8 µmol/kg). Sixty µmol/kg is considered to be the critical liver copper level below which enzootic ataxia may occur in some deer and general copper deficiency is indicated by liver copper levels less than 100 µmol/kg (Mackintosh et al 1986).

The animals were supplemented with copper and no further clinical signs or deaths reported until August 1992 when one animal became recumbent and was submitted for post mortem examination. Markedly swollen kidneys were visible at necropsy, but there were no other visible lesions.

Histopathology revealed a severe, chronic nephrosis characterised by swollen tubules containing protein casts and areas of marked interstitial fibrosis. There was some status spongiosis in the brainstem but no significant lesions in the spinal cord, or any other tissues submitted. Fresh liver analysis of this animal showed selenium levels of 1.39 mg/kg and copper of 117 mg/kg (1837 µmol/kg) indicating that supplementation had been successful. The cause of death in this animal was considered to be severe kidney failure of unknown aetiology.

Enzootic ataxia was diagnosed on the basis of histopathology of the first animal and clinical symptoms of its cohort group. The apparently low plasma Vitamin B12 levels are of interest but may or may not be related to the enzootic ataxia. The low liver copper in the first animal is probably significant in that there have been no further cases or symptoms on the property since copper supplementation were implemented.

To our knowledge this is the first record of this disease in deer in Australia.

References

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TASMANIA - Barry Munday

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ITEM FOR THE VETERINARY PATHOLOGY REPORT

During some recent bioassays undertaken with eucalypt-based pulp mill effluent it was observed that common jollytails (*Galaxias maculatus*) kept in town water supply had equivalent hepatic, nuclear pleomorphism to those maintained in seawater containing 5% pulp mill effluent. It was presumed that trihalomethanes formed during chlorination of the town water supply were responsible for the lesions seen in the fish kept in freshwater.

Two conclusions were drawn from these observations:

1. It is no more dangerous to swim in 5% pulp mill effluent than to drink chlorinated town water.

2. It is always preferable to drink wine rather than water.