VETERINARY PATHOLOGY REPORT

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

Registered by Australia Post Publication No. VBG 6333

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Number 31 August, 1991

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DEADLINE FOR NEXT VET. PATH. REPORT IS OCTOBER 1, 1991

FROM THE EDITOR

Welcome to VPR issue No. 31!

This issue has been a little delayed in the changeover of the executive to NSW. However, a wealth of Interesting material has been forthcoming and we hope to continue the high standard set by previous executives.

Please submit material for the next issue (expected in October/November) to your state representatives as soon as possible to allow it to be produced on time. Deadline for receipt of material will be October 1.

A reminder to all members - Please keep us advised of any change of address so that you will continue to receive your copy of **VPR!**

The roles of secretary and newsletter editor are now somewhat combined and although all correspondence should still be addressed to the secretary, many of the functions will necessarily be undertaken by other members of the executive. All membership enquiries will be bandied by our capable new treasurer, Edla Arzey. Specialist registration and training matters will be the province of Tony Ross and Keith Walker, while matters concerning the Pathology Registry will be fielded by the ongoing management committee.

Gary Reddacliff
Honorary Editor

ASVP EXECUTIVE 1991/1992

President:	Keith Walker	(046) 293309
Secretary:	Garry Reddacliff	(046) 293314
Treasurer:	Edla Arzey	(046) 293332
Committee Members:	Patrick Staples	(046) 293343
	Richard Whittington	(046) 293343
	Ron Gogolewski	(046) 293360
Co-opted member: (Registry/Specialist registration)	Tony Ross	(046) 293312
Fax number (all Executive members)		(046) 293400

MINUTES OF ASVP - AGM 11 MAY 1991

APOLOGIES: Peter Phillips, Dick Sutton, Fraser Trueman, John Searson, Jim Rothwell, Ray Webb, Bob Coverdale, Robin Giesecke, Mary Barton, Malcolm Lancaster, Rod Oliver, Phil Ladds.

MINUTES OF PREVIOUS AGM: (Accepted as valid - Philbey/Ross)

Minutes of the 1990 meeting held at Tasmanian State Institute of Technology 28 January 1990 were circulated in the Veterinary Pathology Report, Number 27, March 1990 (pages 4-7) Correction to minutes - 28 January 1990, not 28 March 1990.

CORRESPONDENCE: (Accepted - Walker/Glastonbury)

GENERAL CORRESPONDENCE - INWARD

2 May 1991	Keith Walker - nominations for 1991-1992 ASVP executive
17 April 1991	From Peter Phillips - apology
16 April 1991	From John Callahan re membership of ASVP
	From George Reppas re membership of ASVP
3 April 1991	Australian Post re Registration of Vet Path Report
31 March 1991	From Bill Hartley requesting time at AGM to give an account of the Domestic Animal Pathology Collection
10 September 1990	Robin Vandegraaf - re joint AVA, ASVP meeting at AVA AGM Adelaide 1991
13 August 1990	Jeremy Allen, letter of thanks for support from ASVP in supporting his nomination for 1990 R J Moir Medal
May 1990	Australia Post - regarding surface mail indicator on registered publications.
23 April 1990	John Holmden re possibility of uniting ASVP with AVPH to produce an expanded Veterinary Public Health and Pathology group.
11 April 1990	Wayne Robinson - requesting address labels of ASVP members in Australia and NZ.
26 February 1990	Adrian Philbey ASVP logo contributions
19 February 1990	Jeff Marshall - change of address

OUTWARD

24 April 1991	To John Callahan re membership of ASVP
23 April 1991	To George Reppas re membership
12 April 1991	To all State representatives seeking agenda items for forthcoming AGM
22 November 1990	To Peter Phillips re slide of month
July 1990	To Alistair Johnson AHL Palmerston North NZ re ASVP meeting in May 1991 - invitation to NZ pathologists
21 June 1990	To John Holmden from REO re joint ASVP/AVPH extended group putting forward his opinion on the matter.
April 1990	To Wayne Robinson - address labels

OUTWARD CORRESPONDENCE SPECIALIST REGISTRATION

5 December 1990	R Oliver to all State/Territory Veterinary Boards. Outlining concern that some members of ASVP with long term experience in Veterinary Pathology unable to be granted Specialist Registration through Grandfather Clause or because of our restrictive guidelines.

Requesting Boards to appraise each application on each its merits.

16 October 1990 — A D Ross for R Oliver to Martin Copeland
Summary of situation in Australia with regard to Specialist Veterinary Pathologists

* Clear differences between states
* Power to register and set policy is with each state's Board of Veterinary Surgeons
* Advisory Committee has no statutory power. (Its advice may or may not be taken by each board).

13 March 1990 — A D Ross (for R Oliver) to Secretary Australian Veterinary Board Conference

INWARD CORRESPONDENCE - SPECIALIST REGISTRATION

9 January 1991 — To R Oliver from Veterinary Surgeons' Board of South Australia. "South Australia is in the somewhat unique position of having a legally prescribed requirement through the regulations to the Veterinary Surgeons Act of 1985.

6(b)

(a) is a certificate of specialisation in that branch of veterinary surgery given by the Advisory Committee on Registration of Veterinary Specialists."

4 January 1991 — To R Oliver from Vet Surgeons' Board of ACT advising next scheduled meeting 5 February 1991. Will consider his letter then. (Refer to letter sent to all Vet Surgeons' Boards by REO dated 5 December 1990).

3 January 1991 — To D Obendorf from John Glastonbury.

In response to letter by Russell Rogers in Vet Path Report. John believes that ACVSc Pathobiology qualifications should be the basis for specialist registration.

19 December 1990 — To R Oliver from J Craig Registrar WA Vet Surgeons Board. Nineteen registered specialists. Eight in Pathobiology. None registered contrary to the advice of the Advisory Committee.

Suggest the concept of attempting to achieve a nationally recognised standard through overview work of the Advisory Committee on Registration of Veterinary Specialists.

Thrust for Register of Specialists arose because of concern for an authenticated list of persons with clearly recognised expertise for the <u>purpose of practice</u> as opposed to research.

11 December 1990 — To R Oliver from Ruth Reuter

Responding to an article in Vet Path Report (December 1990 issue). Her definition of qualification includes certification by American College of Veterinary Pathologists which requires a broad knowledge of all species.

Her strong belief is that "in selecting people to evaluate applicants for specialist registration, the background and individual qualities of the evaluators should be considered, not the institution for which they work!"

18 October 1990 — To D Obendorf from M D McGavin, Dept of Pathobiology, College of Vet Medicine, Knoxville, Tennessee, USA

Discussing and presenting views on Registration of Specialist Pathologist:

* Need for economic reward to justify sacrifice of individuals.
* American College of Vet Path - does not license competent pathologist; but identifies lop quality pathologists.

  Careful not to imply that failure to pass the ACVP examination should imply incompetence. This is certainly not so.

* Area of concern with examination is breadth as well as depth of examination, e.g. with advent of marine animal pathology, the breadth and depth of examination becomes even more of a problem.

* Real advantages to have licensing authority completely separate from those promoting and/or authorising training.

* Suggests ASVP be the "working" organisation for persons involved in veterinary pathology and specialist status be evaluated by another body, e.g. ACVSc?

* Suggest establish a group (as in North America) classified as “Veterinary Laboratory diagnosticians” which better recognises the wide expertise of many veterinarians in Diagnostic Laboratories rather than to try and include them under the category of “Veterinary Pathologists”.

29 June 1990 — To R Oliver from Australian Veterinary Board’s Conference Secretary

Reply to correspondence from REO of 13 March 1990. Advised added item 19.9 to Appendix 1 of the General Criteria which can be applied “as a Suitable Specialist Qualification” 18 May 1990.

1 May — To R Oliver from Jim Sutherland, Secretary Treasurer Pathobiology Chapter ACVSc.

Draft submission of ASVP to Vet Boards Conference (13 March 1990).

* Effective pathway to specialist qualification in Vet Pathology within Australia via Fellowship examination through ACVSc.

* Fellowship examination has four discipline areas emphasising diagnostic laboratory medicine. These are morphologic pathology (option in avian pathology), clinical pathology, microbiology and microbial diseases and parasitology and parasitic diseases.

* Confirms that recipients have broad experience and in-depth knowledge in applied diagnostic laboratory medicine.

* Do not suggest registration on basis of typical narrowly focused research based higher degree.

* Chapter recognises need for a training program leading to specialist registration in veterinary pathobiology.

19April 1990 — To R Oliver from Susan Friend - concern that all of her letter was not printed in Vet Path Report. Requested letter to be printed in entirety.

23 February 1990 — To R Oliver from A D Ross. Requesting comments on ASVP submission to NSW Board of Vet Surgeons.

9 February 1990 — To R Oliver from Ruth Reuter

Accreditation of Veterinary Laboratories and the status of diagnosticians vis a vis specialisation. Need to consider the definition of "pathologist" or "laboratory diagnostician".

Ruth believes there are legal as well as sociological issues.

OUTWARD CORRESPONDENCE - TRAINING

29 June 1990 — R Oliver to Wayne Robinson

* Letter of concern regarding what was considered by ASVP as a draft questionnaire on a national training programme in Veterinary Pathology.

* ASVP members of joint working party on national training programme were Tony Ross and Keith Walker.

* Tony and Keith concerned that questionnaire as circulated did not incorporate many of their suggestions.

9 February — R Oliver to Tony Ross
Concerning possible survey of ASVP members to assess attitudes on training.

INWARD CORRESPONDENCE - TRAINING

3 January 1991 — To D Obendorf from John Glastonbury

* Responding to paper by Russell Rogers in December 1990, Vet Path Report

* John endorses the activities of the Pathobiology Chapter of the ACVc. College qualifications should be the basis for specialist registration.

6 December 1990 — To R Oliver from Wayne Robinson

Acknowledging letter of 11 October 1990 (received 20 November 1990). Advised will consult with Pathobiology Chapter members for their opinions.

14 December 1990 — To R Oliver from Keith Walker

Background notes for discussion on ASVP and training. Discussion suggested to take place at EMAI preferably Monday, 17 September 1990.

9 July 1990 — To R Oliver from Wayne Robinson

Reply to letter from REO, Secretary not sure which one but could be 29 June 1990.

* Surprised at comments in letter.
* Background explanation given by Wayne about his activities in preparing questionnaire.

28 May 1990 — To R Oliver from A D Ross.

* Concern over bias in proposed draft questionnaire drawn up by Wayne Robinson.
* ASVP representatives on Joint Working Party consider these suggestions largely ignored and they are not happy with questionnaire as prepared and circulated.
* Suggested ASVP not accept responsibility for costs of Wayne Robinson's questionnaire .

8 May 1990 — To ASVP members from Wayne Robinson.

Questionnaire on National Training Programme.

5 March 1990 — To A D Ross from Wayne Robinson

* First draft of questionnaire
* Estimated cost of $500-$600 to prepare and process. Seeking ASVP to contribute towards cost.

REPORTS

PRESIDENTS REPORT

The Australian Society for Veterinary Pathology is fortunate to be able to hold its 1991 Scientific Meeting and Annual General Meeting at the Elizabeth Macarthur Agricultural Institute at Camden.

The meeting also brings to a close two years of devoted service by my fellow Tasmanian veterinary pathologists. Secretary, Ron Mason has demonstrated a high level of commitment to the Secretary's position, including the organisation of this meeting. Judith Handlinger has had the onerous task of keeping track of ASVP finances. The services provided by the Tasmanian Department of Primary Industry, e.g. word processing have been invaluable. The ASVP cannot function without a committed executive.

The Veterinary Pathology Report under the guidance of Dr David Obendorf has gone from strength to strength. It has diversified into dealing with areas of topical interest for veterinary pathologists, e.g. specialist registration. The newsletter is unique to Australia in that it provides the most recent and extensive documentation of diseases of all animal species in Australia.

The ASVP has taken an active role in addressing problems with specialist registration of veterinary pathologists. Representations were made to the Veterinary Surgeons Board Conference in Townsville in May 1990 at Townsville on Specialist Registration. The Board agreed to consider broadly based PhDs and coursework Masters Degrees as acceptable for registration as veterinary specialists.

The Veterinary Surgeons' Boards in each state was written to about ASVP's concerns about specialist registration, its applicability to veterinarian pathologists and the exclusion of some eminent veterinary pathologists from recognition.

Progress on Specialist Registration has been difficult to achieve due to the number of organisations involved i.e. State Veterinary Boards, Veterinary Surgeons' Board Conference, Advisory Committee for Registration of Veterinary Specialists and the Australian College of Veterinary scientists (Pathobiology Chapter). Current restrictive requirements for registration of specialists in veterinary pathology, i.e. Fellow in Pathobiology or Diplomate ACVP could have the unintended effect of having few registered specialists in veterinary pathology in Australia.

The National Registry of Domestic Animal Pathology has been placed in a temporarily stable financial position to January 1992 by the Commonwealth and States concerning its funding operations for two years from January 1990. The funding situation has enabled Dr Bill Hartley to run training courses at Camden or interstate, with each state government veterinary laboratory being able to hold a training course annually. Up to 3000 cases are now filed in the National Registry of Domestic Animal Pathology, making it a very valuable training facility.

ASVP members should lobby their laboratory directors to ensure that funding to maintain the Registry is forthcoming for 1992 onwards.

The ASVP is urged to take an active interest in the area of charging for animal disease investigations. Charging for the diagnosis and investigation of animal diseases is progressively being introduced in most states in Australia. Charging has the potential to deter veterinarians and farmers from submitting animals for diagnosis and erode the ability to maintain and monitor surveillance of animal diseases. This is of concern to AQIS who is required to maintain surveillance of the animal disease status in Australia, the Animal Health Committee and Chief Veterinary Officers. The ASVP should make the Animal Health Committee aware of its position on the matter.

The executive now passes from Tasmania to New South Wales. I would like to wish the incoming executive all the best for the next two years.

TREASURER'S REPORT

This is the final report from the Tasmanian executive. Income for 1990 was considerably higher as many members paid their part year 1989 subscriptions and 1990 subscriptions together. It is expected that income fluctuations will stabilise as the new financial year is established.

Vet Path. Report expenses were slightly lower as there were only three editions during 1990. This caused delays which reduced the expected income from Jobline, though some accounts are still outstanding.

The 1990 Conference Report shows -only a slight loss to be subsidised by subscriptions, even though the number of registrants was lower than usual due to Tasmania's relative isolation. This is largely due to the use of Tasmanian Department of Primary Industry and Tasmanian State Institute of Technology facilities, plus the preparation of the conference proceedings "in house". This decision to produce the proceedings without commercial printing stretched to the capacity of the executive and the goodwill of the Department. Commercial printing is to be recommended in future.

To cover costs for commercial services for this type of printing and production as well as possibly other secretarial and treasury duties the subscriptions would ultimately need to be raised. This would leave the executive greater time to pursue the other aims of the society. As issues of professional politics appear to be of increasing concern this may be necessary. Subscriptions should therefore be reviewed in the context of the current aims of the society.

As well as the question of general subscriptions, overseas membership dues need to be addressed. There is no record that the 1989 review of subscriptions addressed the issue of overseas fees rising in line with increased local fees. Insufficient members were present to address this adequately at the last meeting. The fees for the past two years have not , therefore, been raised for overseas members, though the costs for postage were higher.

Additional postage varies with zone and with article weight variations. At current rates the expected additional postage for airmail postage of four Vet Path Reports and one Conference Proceedings per year to overseas members are $7 for Australasian zones, and $12 for other areas.

I therefore move that the overseas membership subscriptions be raised a margin above the general subscription and that in the first instance this margin be $7 above general subscriptions for Australian overseas members, and $12 above general subscriptions for other countries. This margin will need periodic review, but not necessarily each time general subscriptions are raised.

The conference report also records a discrepancy in the amount charged by T.S.I.T for conference accommodation. We have attempted to rectify this with T.S.I.T, but the situation is as yet unclarified.

Judith Handlinger
Hon. Treasurer

STATEMENT OF INCOME AND EXPENDITURE OF ASVP FOR 1990

INCOME

Opening balance	5544.87
Subscriptions	3442.81
Jobline	100.00
Interest, cheque account	152.57
Interest, fixed deposit	291.55
Conference - registrations	1270.00
- dinner payments	1000.00
- accommodation payments	616.00
	12417.80

EXPENDITURE

Corporate affairs	25.50
Govt. duties, taxes on bank accounts	17.06
Vet path report - stationary, printing	402.00
- postage	515.71
- registration	50.00
Conference - invited speaker, airfares	986.00
- dinner + other catering	1268.69
- accommodation as charged	280.00
- proceedings expenses	272.32
- incidental expenses, taxis	17.00
- conference refunds	206.00
Incidental postage and stationary	53.55
	4093.84
Closing Balance	8323.97
	12417.80

1990 CONFERENCE ACCOUNT

INCOME

Net registration fees (paid 1989, 1990)	1340.00
Net conference dinner payments	1080.00
Net accommodation payments	672.00
	3092.00

EXPENDITURE

Airfares for invited speaker	986.00
Proceedings stationary and postage	272.32
Dinner + taxis for dinner	1084.20
Other catering	201.49
Accommodation as invoiced	280.00
	2824.01
Accommodation discrepancy	392.00
	32116.01

Number of participants - 32 inc students	
Operating loss 1990 conference	$124.01

MEMBERSHIP REPORT

It was suggested that a list of financial members with addresses be included in this ASVP report (Ross/Kelly). This was not possible and it will be included in the October issue.

MEMBERSHIP REPORT FINANCIAL

Northern Territory	4
New South Wales	37
Victoria	28
Queensland	17
South Australia	8
Western Australia	16
Tasmania	5
	115

OVERSEAS

Africa	1	
Canada	2	
Indonesia	1	
Singapore	1	
Switzerland	1	
United Kingdom	1	
USA	8	
New Zealand	1	
	15	Total 130

UNFINANCIAL - NOT PAID SINCE 19S8/89

AUSTRALIA

Northern Territory	1
New South Wales	11
Victoria	5
Queensland	5
South Australia	2
Western Australia	2
	26

OVERSEAS

Fiji	1	
France	1	
United Kingdom	2	
USA	5	
New Zealand	2	
	11	Total 37

NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY ANNUAL REPORT

Funding

The Registry received funding for a two year period beginning January 1990 for a half time Registrar and travel expenses. Funds were provided by each state and territory through the Standing Committee on Agriculture.

Dr Bill Hartley was appointed to the position.

Activities

Since the annual general meeting of the ASVP in February 1990 the Registry has been busy and has achieved the objectives set for it by the management committee.

Features of the period February 1990 to May 1991 were:

* Dr Hartley gave seminars and training courses in Tasmania, Victoria, New South Wales and South Australia. Visits to Queensland and Western Australia are planned for June 1991.

* An additional 450 cases have been selected for inclusion in the histological collection which now stands at 2376 indexed cases.

* The collection was used by individual pathologists from several states visiting the Registry for supervised training and personal study.

* All cases are now on the computerised index system which can be searched by species, organ or tissue and pathological change.

* Whilst on leave during 1990 the Registrar visited the Armed Forces Institute of Pathology in Washington D.C and the Beltsville USDA lab to make contact and discuss matters of mutual interest. Case material was obtained for the Registry from there and from two diagnostic laboratories in California and Utah.

* A second opinion service has continued to operate through the period.

* Colour transparencies have been sent to speakers for training presentations.

Support

Members of the ASVP can support the Registry in at least 3 ways

* Send case material for inclusion in the Registry.

* Use the Registrar for refresher courses and training programs.

* Lobby Chief Veterinary Officers to support continued funding during September 1991 in the lead up to the Animal Health Committee meeting on 1 & 2 October 1991 in Sydney where the submission will be considered.

Thank you to ASVP members for your support and positive feedback during the year.

On your behalf the other member of the Management Committee would like to thank Bill Hartley for his enthusiastic hard work and for sharing his expertise with so many of us.

Tony Ross (Chairperson) Keith Walker
John Glastonbury Bill Hartley

Motions to AGM

* The meeting congratulate Bill Hartley for his efforts which resulted in a very successful year for the Registry of Domestic Pathology.
(Ross/all)

* The meeting instructs the executive of ASVP to prepare a submission for funding for a further two year period for submission to the October 1991 meeting of the Animal Health Committee. (Ross/all)

VETERINARY PATHOLOGY REPORT

Dave Obendorf provided a verbal report in which he asks us to note:

- generally scientific articles in the VPR are not critically reviewed
- letters to the editor have increased
- state services are the largest contributors - would like more variety
- the Report is achieving good standing both here and overseas, as a vehicle for training and awareness.

There was a vote of thanks to Dave for his efforts in maintaining the high standard of the Report.

ELECTION OF OFFICE BEARERS

Following candidates have been nominated as 1991-1992 executive of ASVP.

All nominations have been accepted by nominees.

President:	Keith Walker
Secretary:	Gary Reddacliff
Treasurer:	Edla Arzey

Committee Members: Patrick Staples, Richard Whittington, Ron Gogolewski.

State Representatives

STATE REPRESENTATIVES		
STATE	**CURRENT**	**ELECTED**
Victoria	Grant Rawlins	John Mackie (RVL Bairnsdale)
NSW	Tony Ross	Paul Gill (RVL Wollongbar)
Queensland	Fraser Trueman	Fraser Trueman (ARI, Yeerongpilly)
Western Australia	Ruth Reuter	Ron Peet (AHL South Perth)
Northern Territory	Lorna Melville	Lorna Melville (Berrimah Ag.Lab.)
South Australia	Vui Ling Tham	Vui Ling Tham (CVL Adelaide)
Tasmania	Judith Handlinger	To be Advised

GENERAL BUSINESS

ASVP logo: The design by Malcolm France was chosen and appears on the cover of this issue.

Veterinary Pathology Training: Refer to correspondence (above). In summary, the membership considered that the ideal training scheme would have as its end point Specialist Registration, it would be cheap, and would not be controlled by employers. Thus a scheme was wanted in principle, but would be difficult to achieve in practice, and unlikely to be resolved at this meeting.

Specialist Registration: Refer to correspondence above. Lengthy discussion led to the following motions being carried:

1. That the ASVP Committees on Specialist Registration and Training be retained, and continue to help formulate training programmes leading to undertaking ACVS Fellowship exams which are currently (one of the) criteria for specialist registration.

2. That the committee be entrusted to provide advice to pathologists seeking specialist registration, whether by examination as above or via the "grandfather clauses" in the various states.

3. That the ASVP write to the next meeting of the Australian Veterinary Boards, seeking that a Specialist Veterinary Pathologist be co-opted to the Advisory Committee (given that 2/3 of the applications for specialist registration have been from veterinary pathologists).

PAYMENT FOR SOME SECRETARIAL TYPE DUTIES

(For example preparation of Veterinary Pathology Report or Proceedings) If quality of Society publications is to improve, commercial assistance will be required to produce publications or arrange meetings (e.g. AGM's). This costs money.

REVIEW OF LOCAL AND OVERSEAS MEMBERSHIP FEES

Cover increased costs of running ASVP.

Cover cost of postage - e.g. overseas for airmail delivery

	Vet Path Report	Proceedings
New Zealand	A$1.70	2.50
USA	A$2.50	4.50
Africa	A$3.00	5.00

Suggest overseas members choose whether they receive **VPP** and Proceedings by surface or airmail. Indicate this on their renewal notice.

Surcharge subscription for airmail by single article amount x 4 for VPR (i.e. NZ surcharge for Airmail AS6.80 For VPR) plus $2.50 Proceedings = 410.00. Surcharge on subscription for USA would be A$15.00 surcharge on subscription.

Motion carried: That Executive investigates the real cost of running the Society and advise the membership accordingly.

MEMBERSHIP

John Callahan Medical Scientist, 20 years in human pathology. Currently self employed performing equine haematology and biochemistry

(Peninsula Equine Laboratory Services)

Rules of ASVP Membership - re application J Callahan

(a) and (b) Hold a veterinary degree

(c) Distinguished scientists who do not hold a veterinary degree may become an Associate Member after a motion recommending them as such is passed at a general meeting and on payment of annual subscription.

Mr. Callahan might be eligible under (c). He would be advised that he would need a proposer and a seconder and his application would be considered at the next meeting.

OTHER BUSINESS

1. **Next year's AGM**

   To be held around the time of AVA in Adelaide. A proposal to have it as a two day specialist program within the AVA was rejected on financial grounds.

2. **Charging for Government Laboratory Services**

   Motion (Ross/Beers) carried: That the ASVP views with concern the effect of charging for government laboratory services or surveillance of disease in animals, and that the executive explore the development of a joint policy with the AVA on the matter.

QUEENSLAND - Fraser Trueman

VETERINARY PATHOLOGY SERVICES PTY.LTD. (G. Mitchell)

FEMORAL HEAD AVASCULAR NECROSIS

A 12 month old female Silky Terrier was presented with a history of hind leg lameness and coxofemoral radiographic changes. Grossly the femoral head was flattened and showed extensive pitting of the articular cartilage. Histologically there was necrosis of the articular cartilage and the subchondral bone with partial replacement of necrotic bone by fibrovascular tissue. Fragments of articular fibrocartilage were present deep in the trabecular bone of the femoral head. The accepted pathogenesis involves repeated episodes of ischaemia, affecting individuals in which the venous drainage of the femoral head is mainly subsynovial rather than intraosseous. Experimentally, increased intra-articular pressure occludes these superficial veins, producing lesions similar to the natural disease. The disease occurs mainly in small and toy breeds. The lesion is often incorrectly termed Legg-Calve-Perthes disease, which assumes an identical pathogenesis to the human condition.

ALPHA-FUCOSIDOSIS IN CATS (First Report)

A 20 month old female DLH cat with progressively worsening neurological deficits and abnormalities was euthanased. Histologically there was generalised accumulation of foamy macrophages around blood vessels, vacuolation of glial cells and vacuolation of neurones throughout the CNS. Special stains were inconclusive, though accumulation of PAS positive granules in cerebellar Purkinje cells was noted. The changes are typical of a range of inborn errors of metabolism leading to specific enzyme deletion and lysosomal "storage" of intermediate metabolic products. The female litter mate of this cat was showing similar clinical signs. Blood and urine were collected. A wide range of enzyme tests were performed, there was no detectable alpha-fucosidase in peripheral blood leukocytes - this is pathognomonic of Fucosidosis. Although the condition is reported in English Springer Spaniels, it has never been recorded in cats before. The cats came from a relatively closed population in a NSW country town, unfortunately the father was an unknown feral tom and the mother was euthanased. Both kittens were speyed at 6 months and the male litter mate accidentally killed so no breeding is possible! The blood tests were performed by Dr. Bill Carey's group at the Adelaide Children's Hospital, the group is intensely interested in animal models of "storage" diseases to aid in research on the equivalent conditions in humans. They have extensive experience with other metabolic error diseases in cats. We would be interested in any similar conditions you may come across in cats or dogs. Please contact us for further details.
Ref: Canine Fucosidosis: Clinical Findings. RM Taylor, BRH Farrow, PJ Healey, J.Sm.Anim.Pract (1987) 28:291-300

GLOMERULONEPHRITIS IN A HORSE

An 8 year old Arab mare with a clinical history of severe colic, collapse, shock, abdominal distension and anaemia was euthanased. Premortem serum biochemistry showed elevated Urea (24.2mmol/L), high normal Creatinine (0.16mmol/L), Low albumin (10g/L), normal Ca++ (2.4mmol/L), low normal Phosphate (0.8mmol/L), Low Na+ (133mmol/L), High K+ (5.5mmol/L) and a PCV of 0.29L/L with 8g/L Fibrinogen.

Enteric or renal protein loss were suggested, however, the serum changes were not considered particularly indicative of renal failure, particularly as a re-test 6 days later showed a fall in serum Urea to 10mol/L.

Histopathology of the kidney showed generalised glomerular abnormalities characterised by capsular fibrosis, fibroepithelial crescent formation, glomerular adhesions, obliteration of capillary lumina, increased

mesangial cellularity, increased mesangial matrix and glomerular obliteration - all changes typical of immune complex glomerulonephritis. Most tubules contained proteinaceious casts, indicating extensive renal protein loss. Anaemia in chronic renal disease is often associated with decreased production of erythropoietin by the kidney. The disease is due to chronic immune complex formation; antigens incriminated include EIA retrovirus, Salmonella and other chronic infections.

ACUTE RUMENITIS ASSOCIATED WITH PRESUMPTIVE KIKUYU TOXICITY

9 of a 30 cow herd died overnight with signs including incoordination and abdominal pain. Gross PM changes were unremarkable. Histologically there was a severe acute rumenitis characterised by epithelial necrosis, infiltration of neutrophils between the cornified layer and the spinous/basal layer of the epithelium and into the lamina propria and submucosa. This is consistent with a non-specific "chemical" rumenitis. Similar lesions are seen in Urea/Ammonium toxicity (high pH and ammoniacal smell to rumen contents), carbohydrate overload (low pH, lactic acidosis and characteristic odour) and in the presumed Mycotoxicosis of Kikuyu poisoning. Outbreaks usually follow damage to the pasture by insect larvae, e.g. army worm. An army worm outbreak was seen in this pasture 2 weeks prior to the cattle deaths. The lesions differ from those of mycotic, bacterial or viral rumenitis.

GASTRITIS ASSOCIATED WITH SPIRAL BACTERIA IN A DACHSHUND, A WHIPPET AND A CAT

Stomach mucosa was examined histologically from a seven year old male Dachshund with a history of chronic anterior abdominal pain unresponsive to treatment, a six week old Whippet pup with "coffee grounds" vomiting and an 11 year old Siamese cat with persistent vomiting for 10 days. There was moderate mucosal atrophy in all three animals with patchy lymphoplasmacytic infiltration of the mucus and submucosa by a lymphoblastic lymphosarcoma in the cat. Abundant Helicobacter and Gastrospirillium-like organisms were seen in the crypts and superficial mucus. Similar organisms have been associated with gastritis in a range of species including humans. Diagnosis can only be made on gastric biopsy and histopathology - another consideration for those anterior abdominal pain cases where no other diagnosis is made. The recommended treatment for the condition in humans requires combined therapy with Bismuth sulphate and suitable antibiotics such as Amoxycillin or Metronidazole.

TOXOPLASMOSIS IN A RED-NECK WALLABY

A six month old Red-neck Wallaby was presented for necropsy after a sudden onset of diarrhoea and depression. There was patchy haemorrhagic necrosis in the small intestine, severe pulmonary consolidation and multifocal necrotic white foci in the cardiac muscle. Histologic examination showed abundant Toxoplasma tachyzooites in all affected tissues.

The definitive host for Toxoplasma is the cat, intermediate hosts are infected by ingestion of oocysts from cat faeces. Young macropods appear to be particularly susceptible to infection and it is important to minimise contact between the risk animals and cat faeces. Unfortunately this Red-neck Wallaby lived at a cattery?

(R. Miller)

SYSTEMIC HERPESVIRUS & TRICHOMONIASIS INFECTION IN A PIGEON

A freshly euthanased adult pigeon was submitted for autopsy with suppurative conjunctivitis and yellow caseous peel lesions in the mouth and syrinx. Grossly there were multifocal yellow foci of necrosis in the liver, diffuse enteritis, airsacculitis and splenomegaly. Wet mount studies of the oral lesions revealed very large numbers of motile flagellated protozoa typical of Trichomonas. No bacteria were isolated from the liver but histopathology revealed necrotising hepatitis and splenitis with typical herpesvirus intranuclear inclusions.

Trichomoniasis is common in pigeons with lesions being common in the oral cavity rather than in the oesophagus or crop as seen in budgies. Treatment of the flock with Flagyl or Emtryl is needed to reduce the levels of infection.

Little is known of pigeon herpesvirus in Australia. It is a species specific infectious disease with low mortality. However, deaths may occur in combination with other debilitating diseases as shown here.

(C. Belford)

HYPOKALAEMIA IN BURMESE CATS

We have recently seen three cases of hypokalaemic polymyopathy in three Burmese cats. This syndrome has been identified in USA, Britain and New Zealand. The clinical signs of this syndrome are ventral flexion of the neck, fore and hind limb lameness or ataxia and sometimes a hunched sitting posture. There appears to be muscle weakness, sluggish reflexes, particularly righting reflexes and neurologic problems may be suspected. In two of the cases presented organophosphate poisoning or thiamine deficiency was suspected.

Poor response to thiamine therapy was observed and the serum cholinesterase in the suspect OP poisoning case was 2400u/L where the normal range is >1200n/L. Electrolyte data are as in the table below. In each case response to therapy was dramatic and rapid with improvement of clinical signs overnight. Therapy has been oral 'Slow K' of one 600mg tabs every second day - one per day resulting in vomiting in one case. Care should be taken with fluid therapy and oral administration of potassium is likely the safest treatment. Continued ongoing dietary potassium intake is essential and periodic determinations of serum potassium concentration are advisable.

The purpose of this article is to alert practitioners to the clinical presentation of this syndrome, the laboratory diagnosis and the form of therapy.

	Case 1	**Case 2**	**Case 3**	**Normal**
CPK	757	959	ND	10-250
Na+	161	158	154	145-155
K+	3.0	3.2	3.2	3.8-5.0
Cl-	117	115	113	110-120
HCO_3-	23.8	19.6	25.6	17-24

Yeerongpilly Veterinary Laboratory

Kikuyu poisoning of cattle (R. McKenzie)

Kikuyu poisoning was diagnosed in a herd of beef cattle of mixed breeds. In the first 3 days of the incident, 12 deaths occurred in a total of about 20 head affected in the herd of 45. The herd had been shifted to a small paddock containing an almost pure sward of kikuyu (*Pennisetum clandestinum*) 3 days before clinical signs were first noted. This paddock had been heavily infested with "cut-worm" (grass-eating caterpillars) 3-4 weeks previously and was recovering, having been subsequently irrigated with piggery effluent. Despite this, the owner thought that the grass was not responding as well as expected. Several substantial periods of rain had fallen in the week before the poisoning incident, after a long dry period. Clinical signs seen were lethargy, continuous drooling of clear saliva, aimless licking movements of the tongue, mild abdominal distension, kicking at the abdomen suggestive of abdominal pain, a staggering gait, periods of recumbency, ruminal atony sunken eyes (dehydration) and episodes of "sham drinking" during which water was sought out but very little if any was drunk. No diarrhoea was seen. Fatally affected cattle became permanently recumbent before death, had more noticeable abdominal distension and some regurgitated rumen fluid. Necropsy revealed sloppy green rumen contents and congestion of forestomach and abomasal mucosae. Focal superficial omasitis and focal shallow ulceration of the abomasal mucosa were seen histologically in one animal sampled. These findings are consistent with the literature description of the disease, for example by Martinovich & Smith (1973) *NZ Vet J* ***21***:55-63 and Smith & Martinovich (1973) *NZ Vet J* ***21***:85-89.

Myoporum sp. poisoning of sheep (R. McKenzie)

Myoporum sp. poisoning was diagnosed when 180 mixed age Merino wethers died after a flock of 1500 healthy wethers were released into a paddock near Blackall. The paddock had been spelled for 4 months previously. The wethers had been in yards for the 2 previous days and were hungry. Deaths occurred over an area of half a square kilometre within 2 days of the flock entering the paddock. Necropsy of one affected sheep taken to Blackall DPI by the owner revealed jaundice and a congested liver. Histopathology revealed severe periacinar necrosis of hepatocytes with haemorrhage. Surviving hepatocytes were swollen and vacuolated. Some mild proliferation of bile ductule epithelium was present and a mild nephrosis was seen in the kidney. Plants collected from the paddock by the owner and identified at the Arid Zone Institute, Longreach, included *Myoporum acuminatum. Myoporum deserti* was also known to exist in the area.

Enzootic ataxia (swayback) in Kids (R. McKenzie)

Enzootic ataxia was diagnosed in a Cashmere goat flock after necropsy of one affected 5-week-old kid. Four were affected in a group of 50 kids. Clinical signs were progressive paresis and ataxia, particularly of the hind limbs. Wallerian degeneration of the spinal cord white matter and sciatic nerves was seen histologically. The liver copper concentration was 3mg/kg d.m.

Malignant catarrhal fever in rusa deer (R. McKenzie)

Malignant catarrhal fever was diagnosed in a yearling rusa stag in a herd at M. Kilcoy in early March 1991. Three deer died in a herd of 45. The affected animal examined lost condition over 3 weeks and was very weak. It carried a heavy *Boophilus microplus* burden. Necropsy by the submitting veterinarian revealed serous atrophy of fat reserves, very congested mesenteric blood vessels, congestion of the liver and lungs and swollen mesenteric lymph nodes. Histopathology revealed extensive vasculitis in the mesenteric vessels, mesenteric lymph node, kidney and lung tissues submitted. This disease has been recorded previously in rusa deer in Victoria (*Aust. Vet. J* ***58***:81 1982) and in Queensland (A.R. Mackenzie, personal communication).

VICTORIA - John Mackie

VICTORIA - RVL HAMILTON

NECROBACILLOSIS & POX VIRUS INFECTION IN A JUVENILE EASTERN GREY KANGAROO (DebSeward)

A captive, 15 month old eastern grey kangaroo which had been losing condition over the previous month was found dead. The field post mortem report described the presence of numerous small nodules containing a yellow, cheesy material over the serosal surface of the forestomachs and throughout the liver. A 2cm diameter wart-like growth on the hind limb was also noted.

Fusobacteria, morphologically resembling *F. necrophorum* were cultured from the liver lesions. The epithelium from the skin lesion contained the typical, large, eosinophilic, intracytoplasmic inclusions of pox virus infection.

Both necrobacillosis and pox virus infections are recognised conditions in macropods.

Pox virus lesions occurring as solitary or multiple skin lesions have been reported in eastern and western grey kangaroos and the quokka. There is one report of a pox virus infection of the tongue of a brush-tailed possum. The skin condition in macropods has been suggested to grossly and histologically resemble the papilloma-like lesions of human molluscum contagiosum.

Reference: Rothwell, T.L.W. (*et al*, 1984) Poxvirus in marsupial skin lesions AVJ **61**:409

SOUTH AUSTRALIA - Vui Ling Tham

Tumour diagnosis in dogs using immunohistochemistry (Petra Hajduk)

Immunohistochemistry is useful in tumour diagnosis to identify the tissue origin of the tumour cell population. Detection of tissue-specific or cell-specific antigens assists in the classification of tumour cell populations.

Antisera directed against 4 different human proteins were tested on normal dog tissue sections. These were: LCA (leucocyte common antigen) specific to haematolymphoid proliferations; vimentin, a protein present in mesenchymal cells; AE1/3, a cytoteratin marker specific for epidermal cells; and Factor 8, specific for endothelial cells.

Tissue sections were fixed in 10% formalin and embedded in paraffin. The avidin-biotin peroxidase method of staining was employed. Sections were deparaffinized and predigested with trypsin. Undigested sections were also tested for staining. The sections were then incubated in a humid chamber with primary antibody (mouse anti-human) directed against each of the proteins. Ibis was followed by incubation with the biotinylated secondary horse anti-mouse immunoglobulin. Streptavidin-horseradish peroxidase was then added followed by development with DAB (diaminobenzidine) and counterstained with Lillie-Mayers haematoxylin.

The results were:

LCA	No positive staining on dog tissue.
Vimentin	Positive staining on dog tissue, does not require trypsin predigestion.
AE1/3	Positive staining on dog tissue, requires trypsin predigestion.
Factor 8	Positive staining on dog tissue, requires trypsin predigestion.

The cross-reactivity of the 3 positive staining human antisera with corresponding dog antigens is valuable for tumour identification in this species.

For further reading:

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2 MOORE AS, MADWELL BR, LUND JK: Immunohistochemical evaluation of intermediate filament expression in canine and feline neoplasms. Am J Vet Res **50**:88-92, 1989.

3 SANDUSKY GE, CARLTON WW, WIGHTMAN KA: diagnostic immunohistochemistry of canine round cell tumours. Vet Pathol **24**:495-499, 1987.

4 VON BEUST BR, SUTER MM, SUMMERS BA: Factor VIII-related antigen in canine endothelial neoplasms: an immunohistochemical study. Vet Pathol **25**:251-255, 1988.

Dirofilaria immitis in a Cat. (Peter Phillips - Vet Lab)

An eleven years old de-sexed female domestic short-haired cat was presented to a client practice in an extremely dehydrated condition and with enlarged kidneys on 24 June 1991. The cat was treated for renal failure, but was presented again on 28 June after disappearing for 24 hours. The cat was hospitalized but died overnight.

A post-mortem examination revealed two heartworm-like nematodes in the right atrium/vena cava junction. Tissues and the nematodes were submitted.

The nematodes were confirmed as Dirofilaria immitis while the kidney was affected by an adenocarcinoma apparently arising from tubular epithelium. The lung had multiple small metastases.

The chronology of the heartworm infection as related to the adenocarcinoma is unknown.

NEW SOUTH WALES - Paul Gill

REGIONAL VETERINARY LABORATORY, WOLLONGBAR

Kikuyu poisoning in cattle (Paul Gill)

We have had a number of plant poisonings associated with feed shortages due to prolonged dry conditions.

Kikuyu poisoning was diagnosed as the cause of deaths in several beef herds grazing drought damaged pasture which had regrown after recent rain. Serum samples from several sick animals had elevated urea (11-26 mmol/L, creatinine (237-705 µmol/L) and potassium (5.9-8.7 mmol/L) concentrations. It appears urea, creatinine and potassium estimations may have diagnostic value in investigations of kikuyu poisoning.

Sporozoa in pigeons (Paul Gill)

A year old female racing pigeon was submitted with a history of poor exercise tolerance. Histological examinations found zoites and meronts of a sporozoan parasite in blood vessels throughout the lung. There was no associated inflammation and blood cells were not parasitised.

REGIONAL VETERINARY LABORATORY, WAGGA WAGGA (Jocelyn Godwin)

From Tuesday 7 to Thursday 9 May, 1991, a highly successful Footrot Technical Workshop was held at the laboratory. The subject of the workshop was "Standardisation of Diagnostic Tests for Ovine Footrot" and it was attended by 40 representatives from all States which suffer from this scourge. Existing laboratory tests were standardised, procedures for interlaboratory quality control were instigated and plans for more detailed research into gene probes and the protease ELISA were formulated.

Multifocal symmetrical encephalopathy in Angus calves

A further 3 cases of this condition have been examined from 165 animals at risk on the original farm. There were 2 males and 1 female and they varied in age from 1 to 4 weeks. Clinical signs included lateral recumbency, opisthotonus, head and body tremors, medial lateral nystagmus, mild strabismus, intermittent tetanic spasms and proprioceptive deficits. Histological examination has consistently revealed multifocal symmetrical malacia in the medulla oblongata, cerebellar peduncles and spinal grey matter. This lesion is characterised by neuronal necrosis, rarefaction of neuropil, formation of spheroids and infiltration of myelin macrophages. Peter Healy's work at EMAI hopefully will assist in delineating the biochemical pathogenesis and establishing similarities to Leigh's Disease in humans.

Chondrodystrophy in calves

Material was submitted from 3 farms. However, discussions with practitioners indicate that the ‘acorn’ calf syndrome has been quite prevalent during the past months of June and July.

A female calf was submitted to the laboratory from the farm on which 7 of 15 calves had died during parturition. This calf had a crown-rump length of 73cm, a domed cranium and femoral and humeral lengths of 15 and 13 cm respectively. No antibody to akabane, palyam, EHD and pestiviruses was detected in a serum sample. The liver contained lµg/g of manganese. This suggests a deficiency of manganese as recently reported form North America and New Zealand. Normal levels of hepatic manganese vary from 2.5 to 8.0µg/g.

Mucosal disease in Weaner calves

Soon after having been given access to self-feeders containing oats the majority of 26 mixed sex Angus weaners, 9 months of age, developed mucopurulent ocular and nasal discharges, diarrhoea, dyspnoea and weight loss. Pestivirus was recovered from 4 nasal swabs. Gross observations in 1 animal submitted to the laboratory included mild interdigital dermatitis, exudative dermatitis around the external nares and focal plaques over the dorsum of the tongue. Segmental ulcerative cheilitis, parakeratotic glossitis, necrotic enterocolitis and lymphoid depletion were detected histologically. The capture ELISA detected the presence of pestivirus antigen in the mesenteric lymph nodes, lung, spleen and gastrointestinal tract. Sixty-six blood samples, including samples from "all possible carriers" were submitted from the outbreak herd. Five were antibody negative in the GDPT and they were also negative in the antigen capture ELISA and virus isolation for pestivirus.

"Stinkwort"*, Dittrichia graveolens*, enteritis in sheep

A mob of 800 12-month-old Merino sheep were being supplementary fed oats on a paddock which was bare except for a prolific growth of "Stinkwort". Resulting loss of condition and diarrhoea led to morbidity and case mortality rates of 75.0% and 11.7%, respectively. Histological examination detected multifocal pyogranulomatous enteritis with awns being visible at the centres of the granulomas and extending to the tunica submucosa. Following removal from the paddock the clinical signs abated.

Osteodystrophy in Ostrich chicks

Valgus deformity was investigated in two 2-month-old Ostrich chicks. The birds had been over supplemented with calcium giving an extremely high calcium phosphorus ration in the diet. The physes of the proximal tibias reveal apparently severe thickening of the zone of hypertrophic chondrocytes.

Spironucleus sp. in King Parrots

King parrots, *Alisterus scapularis*, in the parks of Yarralumla, ACT, have been developing illthrift and diarrhoea for variable periods prior to death. To date 9 birds have been examined pathologically and gross observations include emaciation, dehydration of subcutaneous tissue and moderate quantities of ingesta in the alimentary tract. Wet preparations of the intestinal mucosa from 1 bird submitted alive to the laboratory revealed numerous flagellated protozoa with a morphology consistent with *Spironucleus* sp. They appeared to be most numerous in the duodenum. Subsequent histological examination of material from this bird revealed catarrhal enteritis with numerous ovoid protozoa being present in the crypts of Lieberkühn. No *Spironucleus* sp. were detected in wet preparations of the intestinal tract of the remaining birds but a moderately severe chronic enteritis was detected histologically in 1 bird. The 3 diagnostic possibilities appear to be infection with *Spironucleus*, tannic acid poisoning from the acorns produced by the Oak trees in which the birds are perching, or another unidentified toxicoses.

REGIONAL VETERINARY LABORATORY. ARMIDALE

MYSTERY MYOPATHY IN FALLOW DEER (DAMA DAMA) (Stephen Love)

On 21 February this year, several adults (yearlings and older) in a mob of 50 mixed age and sex fallow deer developed severe stiffness and lameness, especially in the forelimbs, several days after being put into a ploughed, fallow (no pun intended) paddock. Pasture in the paddock was grass dominant, similar to other paddocks on the 40 acre holding, and had been grazed by a group of stags without ill effects for the month previously.

Affected animals soon became recumbent. Some were treated initially with corticosteroids, without apparent response, as tendonitis associated with the rough surface of the paddock was considered a possibility. The first animal died on 24 February. From 28 February to 10 March, seven more animals died. Each of these was examined at this laboratory.

At necropsy there was obvious pallor of forelimb and hindlimb muscles, particularly thigh and shoulder muscles. Large haemorrhagic areas often joined areas of pallor. In some cases muscles (e.g. triceps) had ruptured. With later mortalities there was pale streaking of some muscles and these were gritty when cut. In every case the distribution of muscle lesions was clearly bilaterally symmetrical. Muscle such as the tongue, diaphragm, psoas etc were not affected.

Histologically there was massive necrosis of skeletal musculature with sparing of the tongue. Two had focal cardiac myonecrosis, most had varying degrees of non-zonal individual cell hepatic necrosis and also pulmonary congestion and oedema (with one having acute focal pneumonitis). The majority, particularly those surviving longest, had a terminal bacteraemia. (Unidentified Streptococcus spp were isolated from the liver, kidneys and lungs of cases 6 and 8, and Salmonella typhimurium was isolated from the liver, kidney and lung of case 7). In each successive case the muscle lesion appeared to be of increasing duration suggesting a sudden episode with some deer surviving longer than others. (Only one clinically affected animal survived; it subsequently recovered, apparently completely).

Dr Bill Hartley, who kindly reviewed the histological sections for me, also commented that in most cases there was a focal infarctive type myonecrosis associated in at least some cases with vascular thrombosis. Whether this is a cause or a result of the necrosis is unclear.

After the necropsy of the second deer, blood was collected from 5 of the affected animals; each of these then received a selenium/vitamin E injection (without apparent response). Plasma Vitamin A levels (mean 0.6, range 0.4-0.9 umol/1) were low by sheep standards while plasma Vitamin E (mean 8.3, range 5.9-10.3 umol/1) and glutathione peroxidase activity (mean 256, range 87-341 U/g Hb), (each animal had received a sheep selenium pellet several months before) were considered acceptable. Liver selenium, Vitamin A and Vitamin E were assessed in 4 of the necropsied deer and were within (sheep/cattle) reference ranges.

Biochemical tests appear to preclude a nutritional myopathy associated with Vitamin E and/or selenium deficiency. Likewise the history (despite careful collection and re-evaluation) is not supportive of a diagnosis of exertional rhabdomyolysis. The proximal causes remain unclear.

REGIONAL VETERINARY LABORATORY, ARMIDALE

VITAMIN A DEFICIENCY IN FEEDLOT CATTLE (Barbara Vanselow)

Vitamin A deficiency was diagnosed in finisher cattle in a 12,000 head feedlot producing beef for the Japanese market. It was estimated that approximately 2,000 cattle were affected at any one time (i.e., cattle between 220 and 300 days in the feedlot).

The clinical condition became obvious in the summer months over the last two years. Animals were noticed to be suffering from clear ocular and nasal discharges, swollen hind legs, varying degrees of lameness and incoordination, exophthalmus, 'glazed' bluish appearance to the eyes, poor adaptation to dull light with pupils more dilated than normal and occasional cases of complete blindness. In advanced cases the swelling or oedema in the hind legs progressed to include oedema of all four legs, and extending to the shoulder and under the jaw. A number of affected animals were also seen to have corneal ulcerations and trauma, possibly as a result of injuries incurred through poor vision.

Affected animals also showed a poor tolerance of hot weather. Their body temperatures were above normal in the middle of the day when compared to unaffected cattle (cattle that had been in the feedlot approx. 220 days). This was associated with increased respiration, drooling of saliva and occasionally panting. During prolonged transportation in hot weather a number of these animals died from heat stress.

Serum vitamin A levels were subnormal in the finishing cattle i.e., between 220 and 300 days in the feedlot. In a group of 5 clinically affected cattle the mean (±sd) serum Vitamin A level was 0.2±0.1 uM/1 (0·9 uM/1 is the accepted normal level). Even randomly selected cattle in the finishing shed had sub-normal levels (0.4±0.4). The liver from a clinically affected animal showed no detectable Vitamin A!

Serum Vitamin A levels from 5 animals at 220 days were marginally adequate. CPK values were elevated in all clinically affected cattle.

Grossly and histopathologically oedema occurred in subcutaneous tissue, within muscle bundles, and within nerve bundles. The aetiology of the oedema has yet to be determined. Other patholgical findings included squamous metaplasia of the parotid salivary duct and papilloedema of the optic nerve.

At this feedlot the observed clinical syndrome was summer associated, perhaps because of the reported higher requirement for Vitamin A in hot weather. The feed was based on a grain and silage diet with no added vitamin supplement or fresh green feed. Although all animals were injected with Vitamins A, D and E upon entry into the feedlot no further supplementation was given and presumably the liver stores were depleted to a critical level between days 220 and 300. Vitamin A requirements are high for rapidly growing animals and may be particularly high when the preference is for 'marbled' meat with a high fat deposition.

Since this diagnosis was made the condition has been recognised in a feedlot at Wagga, also producing beef for the Japanese market.

This outbreak poses the following questions:-

1. What is the Vitamin A status in Australian feedlots?
2. What are the requirements in Australian climatic conditions?
3. What is the best way to supplement the diet to suit the Japanese requirements for white fat and no "artificial" additives?
4. What is the role of vitamin A in heat regulation?
5. What is the pathogenesis of the oedema? Oedema is not a consistent feature of Vitamin A deficiency in cattle but was recorded in Indiana in the 1904's (madsen and Earle, 1947) and recently in Japan (Okuda et al, 1983,and Tojo et al. 1988).

References

Madsen, L.L. and Earle, I.P. (1947). j. OF Nutrition 34: 603.
Okuda M, et al (1983), J. Jap. Vet. Med. Ass. 36: 528-533.
Tojo H, et al (1988), J. Jap. Vet. Med. Ass. 2: 108-113

REGIONAL VETERINARY LABORATORY. ORANGE

Ovine nasal zygomycosis (Mark Carrigan)

A fungal disease causing asymmetric swelling of the nose and disseminated granulomatous lesions has been diagnosed in Merino sheep in the Walgett-Collarenebri-Goodooga area of north-western New South Wales and in the Dirranbandi-Hebel-Thallon districts of southern Queensland. The condition was first diagnosed in March and has now occurred on approximately 50 properties. In all cases only very low numbers of animals are affected, with the average incidence being 0.2%.

Affected sheep have a marked swelling of the tissues of the face from the nostrils to just anterior to the eyes. The swelling is nearly always asymmetric, with one nostril becoming markedly distorted. In advanced cases there is a bloody discharge from the affected nostril. Necrosis of the hard palate is often observed and flystrike of the mouth and nose is common. Sheep lose condition rapidly and die within 10-14 days of the appearance of the swelling.

At necropsy, longitudinal sectioning of the head reveals a severe necrotising rhinitis, usually involving one nasal fossa. The affected fossa is filled by a foul smelling mass of greenish brown necrotic material, with inflammation extending dorsolaterally into the soft tissues of the face ventrally, to involve the hard palate and medially through the nasal septum. The palatine involvement ranges from focal, necrotic lesions 0.5 - 1cm in diameter, to extensive with necrosis and erosion of the palate extending from the posterior border of the dental pad to the level of the last molar. Subcutaneous tissues are thickened with granulation tissue which contains large necrotic areas. Granulomatous lesions are often found in the regional lymph nodes, particularly the parotid and retropharyngeal nodes. The lungs have multiple, randomly distributed areas of consolidation, ranging in size from 0.5 cm to 5.0 cm. Multiple small areas of pleural congestion, thickening and oedema are present on the parietal and visceral pleura, particularly on that pleura covering or in contact with superficial pulmonary lesions. The pericardium is similarly involved.

Histological abnormalities are present in the subcutaneous tissues of the nose, nasal turbinates, nasal septum, hard palate, lymph nodes of the head, lungs and pleura. The changes were similar in all tissues with a heavy cut pouring of eosinophilic proteinaceous exudate, congestion and haemorrhage, with numerous eosinophilic foci of necrosis scattered throughout. There is a heavy diffuse infiltration with macrophages, plasma cells, lymphoid cells, eosinophils, neutrophils and some foreign body giant cells. Many vessels throughout the tissues are thrombosed and have focal necrosis of their walls, and there are large areas of degeneration and necrosis often associated with the thrombosed vessels. In Gomori methenamine silver stained sections large numbers of fungal hyphae are present in the tissues, predominantly within or associated with foci of necrosis. The hyphae are thin-walled, occasionally septate with irregular branching and appear as tubular structures 5 to 10µ in thickness. It is difficult to identity fungal hyphae in H and E stained sections and they are poorly stained by PAS.

Conidiobolus incongruus has been isolated from nasal tissue, parotid and retropharyngeal lymph nodes, and pulmonary lesions. The fungus was readily cultured from these tissues on Sabourauds dextrose agar, Mycosel and 10% sheep blood agar at 30°C and 37°C.

WESTERN AUSTRALIA - Ron Peet

ANIMAL RESOURCE CENTRE - PERTH W.A (Dr. D. A. Pass)

THIAMINE DEFICIENCY IN CHICKENS

The animal resources centre runs a flock of SPF layer chickens which are housed in cages containing 20 hens and 2 roosters. The birds are fed a standard high quality layer diet to which thiamine was added at a rate of 2gm/tonne in a form of a vitamin mineral premix.

Over the preceding 3 years occasional cases of suspected thiamine deficiency occurred in mature roosters and chicks in the first week of life. Affected roosters were thin, depressed, hung their heads down to the floor or had ventroflexion of the neck, and were weak. Affected chicks were weak and ataxic or had opisthotonos, tremors and convulsions. Roosters and chicks responded to injection of thiamine hydrochloride although roosters did not fully recover neurologically.

Histological lesions in chicks were not obvious in most cases but some birds exhibited necrotic Purkinje cells in the cerebellum. This lesion was consistently seen in roosters and in one case was associated with thrombosis of small vessel in the cerebellum and surrounding haemorrhage. Wallerian degeneration of peripheral nerve axons was found in some birds. Analysis of autoclaved grower diet revealed apparently adequate levels of thiamine (1.6 mg/kg) and it was assumed that the layer diet would also be adequate, but the layers were supplemented weekly in the water with a multivitamin preparation as it appeared that the "adequate" level was inadequate.

In September 1989, due to a slip up, the water supplementation was discontinued. Over the next 4 months egg production declined and more roosters and some hens developed clinical signs. Because of the gradual nature of the problem we did not reach a diagnosis until mid-January 1990. From that time the level of thiamine in feed was increased by the addition of twice the level of premix and 4g thiamine mononitrate per tonne and a B vitamin complex was given twice weekly in the water. There have been no subsequent problems although it took many months for production to return to normal.

It is not understood why the birds developed thiamine deficiency when the feed apparently contained adequate levels, but it is now apparent that with the husbandry system that is in operation, and where autoclaved feed is used, a much higher level of thiamine is required than that needed for conventional systems.

INDOMETHACIN TOXICITY IN THE RAT

Ex-breeder female Wistar Furth inbred rats on experiment developed an illness in which the major clinical signs were depression, hunching of the back, diarrhoea, rapid weight loss and pain on palpation of the abdomen. The rats had been anesthetised either with chloral hydrate administered intraperitoneally or halothane administered via a chamber. Affected animals had received the non-steroidal drug indomethacin (12.5 mg/kg) following anesthesia to reduce the inflammatory effects of the experimental procedure.

Post mortem examination revealed peritonitis, ballooned necrotic segments of small intestine and multifocal ulceration of the intestinal wall. Ulcers often extended through all layers of the intestinal wall. Microscopic examination of the wall revealed sharply demarcated mucosal ulceration and necrosis of adjacent muscularis. Although bacteria were present in the necrotic and inflammatory debris within the ulcer, specific organisms that could cause such ulceration were not detectable using a variety of stains.

The lesions described are typical of those caused by indomethacin and other non-steroidal anti-inflammatory agents (Satah *et al* (1981) Gastroenterology **81**:719-725). Indomethacin is regarded as one of the most "toxic" non-steroidal anti-inflammatory drugs.

ANIMAL HEALTH LABORATORIES - SOUTH PERTH (Ron Peet)

PROBABLE ANNUAL RYEGRASS TOXICITY ABORTIONS IN SHEEP

Four hundred heavily pregnant merino ewes were moved to an ungrazed stubble paddock in preparation for lambing in the northern wheatbelt district of Western Australia. Abortions of near full term lambs commenced within a week and 150 lambs were eventually lost despite moving the ewes to another paddock. Two ewes went down during the droving but subsequently recovered and appeared normal. Four fresh foetuses with membranes were submitted to AHL for postmortem examination. All lambs appeared to have died "in utero" having the characteristic partially "cooked" appearance. Histological examination of tissues was therefore unrewarding, but group C *Streptococcus equisimilis* was cultured from 3 of the 4 foetal livers. No Campylobacter, Salmonella, Listeria or other known abortigenic bacteria were cultured and complement fixation tests performed on 10 ewe bloods were considered negative for Chlamydia. Histological examination of livers of 2 ewes subsequently necropsied revealed large eosinophilic intracytoplasmic inclusion bodies in hepatocytes. Grass material collected from the contour banks in the paddocks revealed many rye grass seed heads infected with bacterial and nematode galls. No

ergots were found. The abortions were considered to be due to ARGT even though the ewes did not show characteristic nervous signs. The isolation of *Streptococcus equisimilis* from 3 of 4 foetal livers is perplexing but possibly explained by ascending infection through a compromised placenta? Abortion due to ARGT has been previously reported, but usually associated with nervous signs and deaths in the ewes. None of these ewes died and the abortion syndrome was also reported on two neighbouring farms where ARGT had been previously diagnosed.

Reference
Voge P. (1987) - Proceedings No. 103 Post Grad Ctee Vet. Sci P145.

RUMENITIS ASSOCIATED WITH INGESTION OF AFGHAN MELON (CITRILLUS LINATUS) (David Forshaw)

15 of 750 weaners died 10 days after being moved from a paddock of 'green summer feed' onto wheat stubble. Numerous Afghan melons were growing along the fence lines. The attending veterinarian described affected animals as suffering obvious abdominal pain and dehydration. One animal was necropsied. Post mortem descriptions were of red patches on the serosa of the rumen and reddened abomasal folds. Very little grain was observed in the rumen but a considerable amount of melon including seeds were seen. Histological examination of the rumen revealed widespread necrosis of the mucosa with lifting of the epithelium from the intact basal layers. There was a severe acute inflammatory reaction in the submucosa associated with the necrotic areas.

It was considered unlikely that the lesions observed were due to acidosis secondary to grain ingestion because of the small amount of wheat in the rumen, however, no measurement of rumen pH was made. Melon poisoning was the presumptive diagnosis.

Mortalities in cattle following ingestion of the ripe fruit of prickly paddy melon (Cucumis myriocarpus) have been reported in Queensland and NSW. Clinical signs included severe abdominal discomfort. Although Afghan melons are of a different genus, they are reported to contain cucurbatacins, the compounds claimed to be responsible for the condition associated with cattle eating prickly paddy melon.

It is puzzling why this condition is not recognised re commonly if indeed it is due to ingestion of Afghan melon. The plant is widespread in the agricultural areas of WA and often eaten. Factors conducive to the accumulation of cucurbatacins in cucumbers include strong illumination, dry air, a sudden rise in day temperature and a high level of nutrition. All but the last factor were present in this case.

Carter GI (1990) AV**J 67**:276
McKenzie RA *et al* (1988) AV**J 65**:167
Everist SL (1981) Poisonous Plants of Australia, Revised edition, Angus and Robertson, Sydney.

MASTITIS - BOTTLENOSE DOLPHIN (Tursiops truncatus) (Clive Main)

An adult female dolphin was submitted for necropsy. The animal had been found in the shallows during a holiday period and put in deep freeze pending an opportunity for an examination.

No external lesions were found. The mammary slits were slightly pink and tumorous. The gastrointestinal tract was empty and the gastric lymph node markedly enlarged. The left mammary gland was fibrotic and contained a large amount of necrotic and purulent material.

Histopathological examination was limited due to the animal having been frozen. Sections were taken of gastric lymph node and both mammary glands. Chronic lymphadenitis was evident in the lymph node. In one area there was a core of necrosis accompanied by a granulomatous inflammatory reaction. A multi nucleated giant cell was also evident but no acid fast bodies were found. Fibrosis and pockets of necrotic debris and polymorph neutrophils was evident in the left mammary gland. Similar but less severe changes without fibrosis were present in the right gland.

Bacterial culture of the left gland yielded a mixed growth of *Salmonella meuschen, Vibrio cholera* and *Streptococcus* sp. Serotyping of the *Vibrio* sp. isolate is not yet complete.

Mastitis in dolphins is said to occur frequently, and due to parasitism by worms of the genera *Placetonema* and *Crossicauda* (Griffiths, 1983). No parasites were found in the mammary tissues but the changes in the gastric lymph node were considered to be consistent with damage due to wandering parasite larvae.

Reference: Griffiths, D.J. (1983) Aust. vet Practit 13(1)

POLLUTION? OR JUST A RED HERRING! (Jeremy Langdon)

On 14th February a large ship, the "Sanko Harvest", ran aground about 25 km from Esperance and broke up, spilling 30,000 tonnes of superphosphate and diammonium phosphate and about 600 tonnes of oil into the pristine and productive waters of the Recherche Achipelago. Naturally there were concerns about pollution and health of the marine life. In May, an angler caught three herring (yes, Tommy Ruff to crow eaters) from the yacht club jetty in Esperance with "bleeding sores"; the press went beserk and if the Fisheries people did not want to make an appearance at the royal commission they had better investigate! After retrieving the victims from somebody's freezer we set to work, noting bilaterally symmetrical arrow-like wounds running vertically downwards from the dorsal midline of the body, just behind the dorsal fin. Hmmm, bird strike? Smears of the wounds revealed numerous Gram positive cocci, very uncommon on the skin of fish but not so in the beaks of birds, at least dirty ones such as inhabit rubbish dumps. Like terns, common in the area on beach and tip, and which fish by striking downwards, often unsuccessfully. The other pathologic findings being unrewarding to us and the press, the aetiology was deduced to be a bad tern, and as for pollution, it was just a red herring.

MURDOCH UNIVERSITY

SQUAMOUS CELL CARCINOMA OF THE TONSIL (Brad Chadwick)

Reportedly one of the more common tumours of dogs in the United Kingdom, squamous cell carcinomas of the tonsil appear to be rare in dogs in Australia and, as far as we are aware, not yet reported here in cats.

In the space of two months, three cases were observed, two of which involved aged dogs: the first in a 13 year old Kelpie, consisted of a proliferative squamous cell carcinoma (considerably less common than the invasive SCC) involving the tonsil and oral cavity; the second in another aged dog showed invasive and destructive tendencies more conventional of an SCC. The third case was in a six year old cat and consisted of a very invasive lesion which was also slightly unusual histologically, showing very little keratinisation of squamous cells.

Information on any similar cases would be appreciated.

(Clive Huxtable)

29.

1. **Fibroadanomatous mammary hyperplasia in a bitch**

This is something we have not seen before, has anyone else? The lesion was a single rapidly-enlarging nodule removed from the mammary area of a young female Australian cattle dog. The histology was typical of the lesion as seen in cats, with extensive intralobular ductal hyperplasia within a loose and highly mitotic fibrous stroma. In cats it is considered to be a hormone-induced lesion, occurring mainly in young, non-pregnant, entire females. Usually, all or most glands are involved, but the condition is benign. The endocrine status of the bitch in this report is as yet undefined.

2. **Nephroblastoma in a female Hare wallaby**

This 3 yo animal was one of an experimental group and, although in good condition, was killed after developing nervous signs (described as "uncoordination"); it was presumed to be due to cryptococcal meningitis. No gross or histologic lesions could be found to account for the nervous signs, but there was a firm, white, 5*3*2 cm nodular mass projecting from the left kidney and replacing about half of its volume. Histologically the mass was composed of sheets and cords of poorly differentiated cells amongst which there were attempts to form embryonic nephron elements,

3. **Enigmatic porto-systemic shunting in a dog**.

The subject was a one year old male German Shepherd dog, in which clinical and laboratory evidence for shunting was unequivocal. The dog came to necropsy; the liver was grossly unremarkable, but there were numerous varicose mesenteric veins of the type expected when severe hepatic fibrosis and atrophy lead to secondary shunting. No typical anomalous porto-caval channels were evident. There was no ascites. Histologically, the liver had severe periacinar congestion, mild focal sinusoidal ectasia, canalicular bile plugs and dysplastic portal tracts. In the smaller triads there were arteriolar (and some ? venous) proliferations, as expected in congenital shunts. There was status spongiosus in the reticular formation of the hindbrain. The puzzling feature in this case was the evidence for portal/sinusoidal hypertension (collateral mesenteric channels, sinusoidal congestion) without ascites and without gross atrophy and distortion of the liver. Typical congenital shunts should not give rise to portal hypertension. Any comments would be welcome.

LETTERS TO THE EDITOR

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3 January 1991

Dear Sir

The paper from Russel Rogers, published in the last edition of The Veterinary Pathology Report, December 1990, demands a reply. It contains many inaccuracies that cannot go unchallenged.

I was an examiner at the membership level in Pathobiology for the Australian College of Veterinary Scientists for the period 1985 to 1990. In 1985 and 1986 my co-examiner was Professor R S F Campbell from James Cook University, while for the remaining years, Associate Professor P W Ladds from the same institution filled this role. For a period of 2 years, May 1985 to May 1987, I was President of the Australian Society for Veterinary Pathology. As well, I have been employed by NSW Agriculture & Fisheries as a veterinary laboratory diagnostician for 18 years and for the past 7 years have been Officer-in-Charge of one of their Regional Veterinary Laboratories.

My decisions to sit for the membership examination and later, to become involved with the College as an examiner, were based on a number of factors. To me the College provides the ideal vehicle for independently assessing a veterinarian's competency in Veterinary Pathology. Since 1980, the examiners have come from both academia and state diagnostic laboratories, with their backgrounds providing a good balance between General Pathology and Systemic Pathology, particularly of economic livestock. Offering oneself for examination provides a challenge to master all aspects of Veterinary Pathology, not just the areas one is exposed to on a regular basis. Having been a graduate for a number of years, when I sat for the examination, I found the study of General Pathology extremely valuable in subsequent investigations of diagnostic dilemmas. In contrast, a research based postgraduate degree did not appear to me to provide as solid a foundation in Veterinary Pathology.

Before specifically answering some of Russel's points, I would like to briefly outline the examination procedure in Pathobiology at the membership level. The examination is divided into 3 segments. In the written examination candidates have to answer 6 questions, with limited choice within some of the questions. Where possible, the questions are framed so as to enable the candidate to use his practical and theoretical knowledge to explain animal disease entities of practical diagnostic relevance. The following table demonstrates the composition of the written paper for the period 1985 to 1990.

TABLE

The percentage of questions devoted to General Pathology and types of animal in membership examinations for the Australian College of Veterinary Scientists

YEAR	GENERAL PATHOLOGY	TYPE OF ANIMAL		
		Avian	Economic Livestock	Companion and Native
1985	12.5	12.5	57.5	17.5
1986	16.7	0	75.0	8.3
1987	46.7	3.3	29.2	20.8
1988	50.0	3.3	28.3	18.3
1989	30.8	6.7	41.7	20.8
1990	50.0	0	23.3	26.7

The practical segment is divided into 2 parts. In the first, candidates have to view 40 transparencies depicting specimens of gross pathology and provide a brief description and possible aetiologies. Over recent years 54% of cases came from economic livestock, 38% from companion and native animals and 8% from avian species. In the second, histological slides from 6 to 8 cases, 1 blood film and 1 electron micrograph are examined. Candidates have to describe their observations and offer morphological and aetiological diagnoses. Similarly, the origins of these cases were economic livestock 48%, companion and native animals 37% and avian species 15%. .

The final segment is the oral examination of 45 minutes duration. The basic aims have always been to ascertain what candidates know and not what they don't know and to assess their ability to think and solve diagnostic problems.

In the period 1980 to 1990, 39 candidates sat for the membership examination and 34 or 87.2% were successful. Twenty seven or 69.2% of the candidates were employed in state diagnostic laboratories, 9 or 23.1% in universities and 3 or 7.7% in private laboratories. Of the 5 unsuccessful candidates, 4 were employed in state diagnostic laboratories and 1 in a university.

My specific comments on Russel's points are as follows:

1. The existing system with its emphasis on examination assures the public and the profession of the person's competency in Veterinary Pathology. The background of the candidates show that laboratory diagnosticians are accepting the challenge. A Specialist must be an expert in all facets, not just in 1 species in 1 geographical location. This approach also allows mobility of specialists between government, university and private laboratories.

2. Specialist Registration is for Veterinary Pathologists not research scientists.

3. In view of the 87.2% pass-rate over the past 10 years, the statement that "the Pathobiology Chapter of the College has a reputation for imposing unreasonable standards" is patently not true. From my experience from both sides the examinations have always been conducted in a fair and honest manner with the advancement of Veterinary Pathology to the forefront.

4. With 69.3% of candidates working in state diagnostic laboratories, it is obvious that universities and private laboratories are not being unduly favoured.

7. The backgrounds of examiners used by the College is consistent with the concept that "Evaluation should be done by proven performers in the field of diagnosis of diseases of commercial livestock".

8. In my experience state diagnostic services are highly valued by the community and the veterinary profession.

1b. Three members of my staff have successfully undertaken the Membership Examination and have found it highly appropriate for laboratory diagnosticians.

3b "Diagnosis and Control of Livestock Diseases" is far too broad a specialist category. The age of specialisation is upon us and we in NSW Agriculture & Fisheries are proud of our multi-disciplinary approach, using pathologists, microbiologists, parasitologists, epidemiologists and species specialists to solve complex or new disease problems.

In conclusion, I fully endorse the activities of the Pathobiology Chapter of the Australian College of Veterinary Scientists. College qualifications should be the basis for Specialist Registration and could be used advantageously by State Departments for promotional purposes. I encourage all young laboratory diagnosticians to accept the challenge, study conscientiously, gain the necessary experience, pass the examinations and reap the rewards. We are too small a group to become fragmented in the pursuit of excellence in Veterinary Pathology.

J R W Glastonbury MVSc MVS MACVs
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31 July 1991

Dear Gary,

ASVP Slide-of-the-Month April 1991, RN90/3113

We have had correspondence from 2 institutions regarding our ASVP Slide-of-the-Month April 1991, RN90/3113, which we presented as a case of anaphylaxis or Type 1 hypersensitivity in rabbits following administration of formalised whole cell *Streptococcus suis* Type 2 bacterin. The correspondents expressed concern over the nature of the inflammatory infiltrate and the time of onset of the condition in relation to the initial administration of the bacterin. He would like to provide some additional material on this case for the benefit of ASVP members.

The first question related to whether the inflammatory infiltrate in the lungs comprised eosinophils or neutrophils. Sections of lung were sent to the Murdoch University Veterinary School in Western Australia for staining by the solachrome cyanin method. This demonstrated that the predominant inflammatory cells in the lungs were eosinophils, which would be consistent with an immune-mediated eosinophil-chemotactic reaction, perhaps anaphylaxis or Type 1 hypersensitivity. Alternative hypotheses as to the aetiology of the condition, which included contamination of the bacterin with bacteria or endotoxin, or Pasteurella multocida septicaemia, probably would have resulted in sequestration of neutrophils rather than eosinophils in the alveolar capillaries.

We were also questioned about the time of onset of the condition in relation to the time of initial administration of bacterin. Four days would probably be insufficient time for a hypersensitivity response to develop. However, it is possible that the rabbit may have been sensitised to antigens from a previous subclinical bacterial infection, perhaps with a Streptococcus sp, containing antigenic determinants in common with S suis Type 2. This could explain the brief period of time between the onset of the condition and the initial administration of the bacterin,

We hope that these comments have clarified the interpretation of the findings.

John R W Glastonbury
Officer-in-Charge
Regional Veterinary Laboratory
Wagga Wagga

Adrian W Philbey
Veterinary Research Officer
Regional Veterinary Laboratory
Wagga Wagga

34.

In a letter to John Glastonbury from Professor Jerry Stevens of North Carolina State University:

RESIDENCY POSITION IN VETERINARY CLINICAL PATHOLOGY

The College of Veterinary Medicine, North Carolina State University, announces a residency position in veterinary clinical pathology beginning August 1, 1991, or as soon thereafter as possible. The program will emphasize competence in clinical 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 D.V.M. or equivalent degree; previous experience in clinical pathology desirable but not mandatory. Stipend is $19,496. Applicants should send a curriculum vitae, a statement of goals and interests, complete transcripts and three letters of recommendation. Closing date for applications is January 15, 1992, or until a suitable candidate is found. 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 Street, Raleigh, NC 27606.

For details about the program call Drs. J. B. Stevens/Carol Grindem, Clinical Pathology Residency Coordinators at (919) 829-4488/829-4277.

An equal opportunity/affirmative action employer.

NEW ZEALAND SOCIETY FOR VETERINARY AND COMPARATIVE PATHOLOGY

CONFERENCE 1991

21 June 1991

Dr Rod E Oliver
Mt Pleasant Laboratory
PO Box 46
King Meadows
Tasmania 7249
AUSTRALIA

Dear Rod

I am pleased to inform you that organisation for this year's conference is proceeding smoothly and we hope that you will reserve these dates for coming to Hamilton :

9TH AND 10TH DECEMBER 1991

The accommodation, meals and conference will all be in the one complex. The costs should be about $90-$100 per day including meals. This depends on numbers attending. The venue is:

MANAGEMENT LODGE
UNIVERSITY OF WAIKATO

All cases have been selected for the slide sets and we aim to have them distributed in August. There will be about 75 cases in the set. Not all of these will be presented at the conference. We will be approaching individuals to present cases to spread the workload between participants. Just a reminder the topic this year is:

DISEASES OF PIGS

This is also the time to consider preparing papers for the general pathology session.

Thank you for your quick response in supplying Rob Fairley with cases to select for this conference.

More details and registration forms will be sent later.

A F Julian
Secretary