Australian Society for Veterinary Pathology

VETERINARY PATHOLOGY REPORT

Brought to you by:

University of Melbourne
Veterinary Clinical Centre
WERRIBEE VIC 3030
Ph (03) 9741 3500 Fax (03) 9741 0401

PUBLICATION NO. PP544059/0003

EDITOR: Karl Harrigan

Number 46 **JULY 1997**

CONTENTS

DEADLINE FOR NEXT VET. PATH REPORT IS:
30 September 1997

SECRETARIAT

PO Box 114 Walkerville SA 5081

Phone: 08 8344 6337 (Amanda Whyte) Fax: 08 8344 9227

ASVP EXECUTIVE 1997-98

President: Clive Huxtable, School of Veterinary Studies
Murdock University, MURDOCH WA 6130
Phone: 08 9360 2297

Secretary: Cleve Main, Animal Health Laboratories
3 Baron Hay Court, SOUTH PERTH WA 6151
Phone: 08 9368 3351

Treasurer: Jeremy Allen, Animal Health Laboratories
3 Baron Hay Court, SOUTH PERTH WA 6151
Phone: 08 9368 3466

COMMITTEE MEMBERS:

Barry Richards Animal Health Laboratories
3 Baron Hay Court, SOUTH PERTH WA 6151
Phone: 08 9368 3424

Other position vacant

APPOINTMENTS:

Chairman - Registry of Domestic Animal Pathology:	Tony Ross
Newsletter Editor:	Cleve Main
Coordinator (Training Committee):	Judith Wilkie

CONVENOR - SLIDE OF MONTH:

Rod Reece,
National Registry of Domestic Animal Pathology, EMAI,
Private Mail Bag 8, CAMDEN NSW 2570
Phone: 02 4640 6333

STATE REPRESENTATIVES

Queensland: Bruce Hill,
Rockhampton Vet Lab, QDPI
Box 6014, Rockhampton, QLD, 4702
Phone: 079 36 0211

Victoria: Malcolm Lancaster, VIAS
475 Mickleham Road
ATTWOOD VIC 3049
Phone: 03 9217 4200

South Australia: Ruth Reuter
VPS
PO Box 445
GLENSIDE SA 5065
Phone: 08 8372 3700

New South Wales: Paul Gill,
RVL
WOLLONGBAR NSW 2480
Phone: 02 6626 1261

Western Australia: David Forshaw
Regional Office, WA Dept Ag
ALBANY WA 6330
Phone: 08 9892 8444

Northern Territory: Anton Janmaat
PO Box 990
Darwin NT 0801
Phone: 08 8999 2240

Tasmania: Roy Mason
TAWQDS
PO Box 46
KINGS MEADOWS TAS 7249
Phone: 03 6336 5216

MINUTES - ANNUAL GENERAL MEETING

Australian Society for Veterinary Pathology Inc.

University of Melbourne, Veterinary Clinical Centre

Bellevue Hotel, Brisbane

3 May 1997

1. **Opening:**

The meeting opened at 4:05pm, 31 members in attendance.

2. **Apologies:**

P. Phillips, W. Hartley, P. Hooper, M. Williamson, D. Middleton, R. Miller, W. Townsend, M. Copland, R. Reuter, D. Forshaw, T. Rothwell, M. France, J. Gisbon.

3. **Minutes of 1996 AGM:**

Published in Veterinary Pathology Report Number 45, March 1997, copies available at the meeting.

Acceptance moved P. Gill, seconded W. Robinson. Carried, none opposed.

4. **Business arising from the Minutes:**

(a) Motion by Prof Slocombe/Dr Wilkie that "*Consideration of ASVP/AVA affiliation be made an Agenda Item for the next Annual General Meeting"* and

(b) Motion by Prof Robinson/Dr Friend that *"The ASVP Executive correspond with the Australian Veterinary Association regarding special interest group affiliation with the AVA especially in relation to ASVP veterinary members who are not also members of the AVA"*.

In accordance with the consensus of the 1996 AGM, a paper outlining the probable advantages/disadvantages of an ASVP/AVA affiliation together with a draft outline of possible by-laws etc of an AVA affiliated Australian Society for Veterinary Pathology had been prepared by the President and circulated to members by mail with the Notice of Meeting/Draft Agenda. Copies of these papers are appended to these Minutes.

The advantages/disadvantages of an ASVP/AVA affiliation were discussed at length. Various members emphasised the advantages of association with the AVA would be to expand the profile of Veterinary pathology within the entire Veterinary profession.

Considerable concern was expressed as to the cost of AVA membership together with the cost of attendance at an AVA Conference, which, following affiliation, would be the probable venue for the Society's Annual Conference. Most concern centred on the possible/probable requirement that all veterinarian members of ASVP be members of the AVA. It was further pointed out that the proposition had recently been voted on by the membership in postal ballot, the motion for affiliation being defeated by 37 votes to 33 (Minutes, ASVP Annual General Meeting, May 1995, published Vet. Path Report 42, September 1995).

Moved J. Mackie, seconded P. Ladds that "*The proposition that the ASVP become a Special Interest Group of the AVA again be put up for determination by the membership by postal ballot".*

Motion carried, 23 for, 8 against.

Consensus of the meeting was that the new ASVP Executive would pursue this issue including provision of a statement from the AVA concerning membership of Special Interest Groups together with a detailed assessment of the total costs to members of affiliation with the AVA.

(c) In reference to correspondence with the Australian Animal Health Council, it was pointed out that a document drawing the attention of the Council to current deficiencies in veterinary surveillance services in Australia had been sent and that a report from the Council had been scheduled for March 1997 but that none had been received to date.

(d) In reference to the issue involving Dr D Obendorf and the Tasmanian Department of Primary Industry and Fisheries, it was pointed that that this matter had been resolved and that both parties had agreed upon a joint statement to be published in the DPIF newsletter "Prime News" in June 1997.

The ASVP Executive had already received a request from Dr Obendorf to publish the joint statement in the Society's newsletter and was awaiting agreement from the Minister DPIF, Tasmania, before proceeding.

Moved R. Whittington, seconded J. Mackie that "*The joint statement between Dr Obendorf and the Tasmanian DPIF be published in the Society's newsletter, The Veterinary Pathology Report, as soon as practicable*". Motion carried, none opposed.

5. **Correspondence:**

Routine correspondence was handled by the Executive.

Notable items brought to the attention of members were:

(a) Letter from Dr R. Giesecke thanking the Society for its acknowledgement of her contribution to the Training Committee and condolences following the death of her husband. Dr Giesecke requested that, in recognising her contribution to the Training

Committee, the Society also acknowledge the input of other members, in particular J. Mackie, J. Glastonbury, R. Kelly and W. Robinson.

(b) Letter from Dr W. Hartley expressing his thanks to members who have supported his efforts to build up pathology collections of both domestic and non-domestic animal diseases over many years.

Acceptance of inward correspondence; moved C. Main, seconded B. Richards. Carried, none opposed.

Acceptance of outward correspondence; moved C. Main, seconded A. Begg. Carried, none opposed.

6. Business arising from Correspondence:

Nil.

7. **Annual Reports:**

(a) President's Report

In reflecting on the last two years, the outgoing Executive of this organisation has seen dramatic changes in the way diagnostic services are delivered within the Eastern States in particular, and in the continued erosion of base support for our educational institutions. Thankfully, few members have left the organisation, but regrettably, few have joined. We must all be concerned about these trends if they continue, and somehow must attract new talented individuals into a field of uncertain future. The new Executive will have some interesting issues to resolve.

- Should our membership list be available to all who seek information about Australia's veterinary pathologists, and what information should be kept confidential?

- Should the ASVP become actively involved in developing and administering quality assurance testing programs and if so, under what umbrella?

- How can we best encourage the development of educational modules and overcome intellectual property and copyright hurdles?

- Can we incorporate a web site and multimedia in such activities? In fact, should we have a web site for the ASVP in general?

- Does the ASVP newsletter and "Slide of the Month" best serve the educational needs of the group or can we better utilise information technologies?

- How long should we keep non-paying members on the books and should they continue to receive mailings?

Obviously, the outgoing Executive has some ideas on these matters but it will be left to the incoming Executive to work these through.

The topic for this Annual Meeting was a timely one, since new technologies to disease diagnosis may serve as either an adjunct or eventually a replacement for diagnosis based on routine anatomic and clinical pathology. As new tests come on line, a registry indicating where in the nation tests can be done might be a useful function the ASVP could provide for its members. On behalf of the outgoing Executive, I thank the local organisers for their efforts in providing an interesting insight into these emerging diagnostic technologies and for their careful planning in the choice of venue.

In closing, I wish to thank the other members of the Executive, in particular Karl Harrigan, who shouldered the bulk of the Executive's activities as Secretary.

Ron Slocombe
PRESIDENT

(b) **Secretary's Report**

Following the retirement of Mrs Pat Bosence, the Secretariat in Adelaide has been conducted by Ms Julietta Carin and now by Ms Amanda Whyte. While there have been some minor problems arising from the transfer of duties, the Secretariat arrangements are working very well. Amanda Whyte is to be congratulated for the efficiency with which she has coped with the recent rush to make papers etc available prior to this Annual General Meeting.

Members are reminded that all subscription and membership matters including changes of address are handled through the Secretariat. Notification of change of address etc is the responsibility of members. Amanda currently has the mail of several members being returned to her as they are unknown at the address she has on record. Recent laboratory rearrangements may well be the reason for a number of our current membership addresses being out of date. This may be the reason that some members may not have received their copy of the recent Vet. Path. Report and papers for this AGM. Alternatively, some members names may have been deleted from the current membership list if they have failed to forward subscriptions over the last few years. As of midday, 2 May 1997, there were 124 names on the ASVP membership list. Most had not yet paid their 1997 subscription.

I have continued to utilise the Society's State Representatives to obtain and disseminate information on certain issues. I thank them for their help and cooperation. It would in future be useful, if any State Representative is to be absent for any prolonged period, to inform the Secretary so that alternative arrangements to obtain/disseminate information can be made.

Professor Robinson and the local organising committee have arranged an excellent program and venue for the 1997 ASVP Conference and Annual General Meeting. I thank them for making all these arrangements and for the publication of the conference proceedings.

Karl Harrigan
HONORARY SECRETARY

(c) **Treasurer's Report**

This report is based on bank statements issued up to the close of business on 1 April 1997. The closing balance is similar to 1995. With no conference in 1996, income and expenditure were both lower than in previous years. The secretariat has received only a small number of subscriptions for 1997.

Statement of Income and Expenditure of ASVP in 1996

INCOME	
Opening Balance	$9,098.86
Subscriptions	$1,310.00
Credit Interest	$167.69
SUB TOTAL	$10,576.55
EXPENDITURE	
Vet Path Report/Secretarial Services	-$894.91
Bank fees and taxes	-$84.02
SUB TOTAL	-$978.93
CLOSING BALANCE	**$9,597.62**

Mark Williamson
HONORARY TREASURERS

Attention was drawn to the significant amount expended in bank charges and taxes. The meeting recommended that the incoming Executive review means of minimising these charges.

Moved R. Rahaley, seconded J. Glastonbury, that '*The reports be accepted*'. Carried, none opposed.

8. Committee Reports:

(a) **Training Committee Report**

At the Annual General Meeting held at the University of Melbourne, Veterinary Clinical Centre, Werribee on 26 November 1996, the training committee of the ASVP was asked to consider the problems of ownership of intellectual property rights with respect to production of training modules.

Copyright:

The Australian Copyright Council was contacted and they have sent an information sheet covering some issues with respect to licensing and assigning rights (copy enclosed). The copy initially resides with the author but can be assigned exclusively or non-exclusively to another body. The body (in this case the ASVP?) who will be in control of the modules and their use, should probably have the copyright assigned to them.

Payment:

The options are:

1. Solicit ASVP members to contribute their work without payment.
2. An up-front fee
3. Royalties (percentage of profits)
4. A combination of the above.

The Copyright Council provides recommendations and guidelines if required. A further issue to be decided would be what percentage (if any) of any profits would go to the author, the editor, the institute(s) producing the module and the ASVP.

Other Issues for consideration:

- Ongoing revision of material (authors to do this?).
- Support for authors in terms of assistance, financial and practical, with planning/writing modules (ASVP/Uni of Qld?)
- Support for trainees using modules (authors to do this?)
- The marketing role (ASVP to do this?)

Pilot Module:

- Which system or topic would be the most appropriate one to commence with, in terms of a) usefulness, and b) ease of preparation.
- How is the author to be selected? The committee could approach specific appropriately qualified persons (restricted to ASVP members?) or volunteers could be called for.

Collaboration with existing organisations:

The Post-Graduate Foundation was contacted for information on how they handle some of the above issues with respect to the authors of their distance education modules, which Dr Doug Bryden willingly supplied to me. He expressed an interest in collaboration in this project if it was deemed appropriate by the members of the ASVP, and would be willing to meet with us if requested. He indicated that the ASVP could act as a body in any such collaboration and could be the recipient of the payments for, and co-ordinators of, the modules if desired. The Post-Graduate Foundation would be willing to supply technical help, tutors to assist authors, marketing expertise, financial backing for production etc. Is the ASVP interested in considering this offer?

The opinion of ASVP members is solicited in regard to these issues.

Judith S Wilkie
TRAINING COMMITTEE CO-ORDINATOR

The information provided by the Australian Copyright Council appended to this report will be included in the Veterinary Pathology Report, July 1997. Discussion centred on how the Committee might proceed.

Moved J. Wilkie, seconded W. Robinson, that "*The ASVP authorise the Training Committee to contact the Post-Graduate Foundation in Veterinary Science, University of Sydney, for discussion of the proposal for development of education programs*".

Motion carried, none opposed.

(b) **National Registry of Domestic Animal Pathology, Registrar's Report**

The NRDAP continues to function well and contains histological slides of most common diseases of farm animals which occur in Australia and some examples of exotic and foreign diseases. Currently, there are selected indexed histological slides and reports of 978 cases from cattle, 532 sheep, 286 pigs, 156 goats, 315 horses, 30 deer, 503 dogs, 186 cats, 687 poultry and 163 other species (total - 3,386). By way of explanation, when the NRDAP and the Taronga Zoo pathology collection were initiated and staffed by one registrar in two part-time positions, the decision was taken that deer and fish be included in the latter, hence the low representation of these species in the DRDAP. This arrangement needs reassessment.

In general, the NRDAP has not been well supported by contributions from Universities or private veterinary pathology laboratories and more cases from companion animals are needed. The material added to the registry derives from cases submitted as good examples of particular conditions, cases solicited by the registrar after reading reports and suitable cases from the regular supply of material for second opinion.

The shortage of transparencies of gross lesions is still a deficiency which needs to be seriously addressed. Funds were provided by ASVP and in 1996 used to purchase a transparency duplicator attached to an EMAI camera; a suitable camera for the registry will be purchased shortly. Ideally, it would be better if veterinary pathologists took an extra photograph and submitted the surplus to the registry on a routine basis.

Resignation:

Rod Reece has been part-time Registrar of NRDAP for 4.5 years and has resigned to take effect from Friday 9 May 1997. He will be commencing duties as a diagnostic pathologist within the Regional Veterinary Laboratory at EMAI.

"The position as Registrar has been most interesting and challenging and I wish to thank you, my colleagues, for allowing me to be of service to you in this way. I also wish to extend my

appreciation to the management committee for their assistance and encouragement. I wish the new registrar, whoever that may be, all the best."

Until a new registrar is appointed, Rod will act as caretaker.

Bovine Spongiform Encephalopathy etc

In September-October 1996, Rod visited the UK on a study tour of bovine spongiform encephalopathy and related diseases funded by Australian Animal Health Committee and coordinated through CSIRO AAHL. This led to his involvement with Gerald Wells, Peter Hooper and others in the National Workshop on Diagnosis of Spongiform Encephalopathies of Domestic Animals held at Werribee in late November. Arising from that the NRDAP agreed to run short training courses on BSE and related diseases within each of the states for veterinary pathologists who were not able to attend the Workshop. The timing for these had to be put off due to conflict with priorities from Rod's other half-time position and personal commitments. Rod has obtained release from his new position to deliver these in June-July.

Rod Reece
REGISTRAR NRDAP

Following presentation of the Registrar's Report, Dr Ross, Chairperson of the Registry Management Committee, thanked Dr Reece for his contribution of the Registry and indicated that, as the Registry would not necessarily be housed in its current location in perpetuity, now might be a convenient time for expressions of interest not only for a new Registrar but also for a future location for the Registry.

Dr Ross drew attention to the future possibility of the Registry providing an ongoing Quality Assurance (QA) program in veterinary histopathology for Australian diagnostic laboratories.

A vote of thanks to Dr Reece for his contribution as Registrar and assistance to members was carried by acclamation.

(c) **"Slide of the Month" Program - Convenor's report**

1. The ASVP Slide of the Month has continued to operate through what appears to have been a difficult year for many of our colleagues and their places of employment.

2. During 1996, several contributors who agreed to supply cases for distribution had to withdraw, others circulated the material later than anticipated.

3. The cases by month are:

    - Dec 95: Vet Lab SA, 3057/95 kingfisher and 6897/95 dunnart with mycobacteriosis; and 10766/9f4 cat with piloleiomyoma.

- Jan 96: VPS Brisbane, B119156F and/or B306222B dogs with helicobacter gastritis; B533656 cockatiel and enteric cochlosomiasis.
- Feb 96: Vet Path Massey Univ NZ, 24166-94 stitchbird with systemic coccidiosis, 21860-94 Galloway with epidermolysis bullosa.
- Mar 96: Trop Vet Sci Townsville, 95-235-2 crocodile with avitaminosis A, 96-5 Bedlington terrier with chronic cuprosis.
- Apr 96: No slides.
- May 96: ARI Yeerongpilly, 96-125583 perch with red spot, 95-139320 Murray Grey congenital haemolytic anaemia.
- Jun 96: No slides.
- Jul 96: No slides.
- Aug 96: Vet Path Murdoch Univ WA, 96-1454 butterfly fish with mycobacteria, 96-1347 budgerigar with oesophageal trichomoniasis.
- Sep 96: No slides.
- Oct 96: No slides.
- Nov 96: No slides.
- Dec 96: South Perth Lab WA, P96-2067 bull herpes encephalitis, P96-106 koi carp with gonadal tumour.

Rod Reece
Coordinator ASVP SOTM

The incoming Registrar of the National Registry of Domestic Animal Pathology will take over as Convenor of the "Slide of the Month" program in future. Dr Reece volunteered to act as caretaker of the program until the new Registrar is appointed. His offer was gratefully accepted.

It was noted that similar programs in other countries had collapsed. We should make every effort to preserve our own program.

(d) **Veterinary Pathology Report - Editor's Report**

I had hoped to achieve a more expeditious and timely publication of the Vet Path Report in 1997. Unfortunately, publication every 3-4 months as intended has not been achieved.

Again, the Society's State Representatives are to be thanked and congratulated for their efforts in obtaining the information that makes up most of the Report. Members are reminded that there can be no Report if they fail to submit information for publication in it.

I have had some difficulty with material for publication submitted via facsimile. Often they have been of very poor quality, sometimes largely illegible, sometimes incomplete. The best transmissions still require retyping prior to publication. In future, it would be useful for the material to be submitted in letter format or on disk and in the style of the VPR presentations. I'm sure this will help to make the task of the future VPR editor(s) much easier.

Karl Harrigan
VPR EDITOR

Acceptance of reports moved C. Main, seconded J. Taylor. Carried, none opposed.

9. Office Bearers and Committee Appointments

The following people had been nominated and were elected unopposed:

- President: Dr Clive Huxtable
- Secretary: Dr Cleve Main
- Treasurer: Dr Jeremy Allen
- Committee Members: Dr B Richards, no other nominations. The incoming Executive may co-opt appropriate candidates if necessary.
- Chairperson, National Registry of Domestic Animal Pathology: Dr Tony Ross
- "Slide of the Month" Convenor: Dr Judith Wilkie
- State Representatives:

Queensland:	Dr Bruce Hill
New South Wales:	Dr Paul Gill
Victoria:	Dr Malcolm Lancaster
Tasmania:	Dr Roy Mason
South Australia:	Dr Ruth Reuter
Western Australia:	Dr David Forshaw
Northern Territory:	Dr Anton Janmaat

10. General Business

(a) Diagnostic criteria for Ovine Johne's Disease

Several members expressed concern over the current guidelines for the histopathological diagnosis/exclusion of ovine Johne's disease.

It was pointed out that diagnostic protocols/guidelines were under review and members should address their concerns to SCAHLS Chairman, Dr Keith Murray.

Consensus was that the incoming Executive should review members' concerns and draw them to the attention of the appropriate authorities. When finalised, the criteria for histopathological diagnosis might be published in the Veterinary Pathology Report.

(b) Preservation of histopathology slides, paraffin blocks, reports and other reference material previously/currently located at recently closed veterinary diagnostic laboratories.

It was noted that, following the closure of several laboratories and reassignment of the buildings to other purposes, there is a high probability of this material being lost/discarded.

It was reported that in NSW, significant material from the Wagga and Armidale laboratories was being retained by the Department of EMAI.

The situation in Victoria was less clear as a result of the control of the laboratories having initially passed into private hands which has now lapsed. Some material from the Benalla Laboratory had been retained at Attwood.

Consensus was that the incoming Executive should send letters to the appropriate authorities expressing concern and seeking assurances that this valuable material will be retained.

(c) Letter published in the Canadian Veterinary Journal expressing concern at the reduction in government support of animal disease surveillance in Canada.

Copies of this letter were circulated at the meeting for the information of members. Consensus was that efforts should be made to publish a copy of the letter in the Veterinary Pathology Report.

(d) "Path Mail"

Members' attention was drawn to this excellent innovation developed by Dr B Richards. "Path Mail" provides a mechanism for rapidly alerting diagnosticians to issues in veterinary pathology, for asking questions and obtaining responses expeditiously.

(e) Availability of ASVP membership list to outside bodies.

The Executive had received several requests from certain organisations for provision of a list of members' names and addresses. These had been declined on the basis of confidentiality.

Following discussion consensus was that the ASVP membership list should remain confidential and not made available to outside organisations. The ASVP Secretariat may disseminate information to the entire membership on behalf of outside bodies providing adequate recompense was provided to cover the costs involved.

(f) ASVP involvement in providing/administering quality assurance programs for veterinary histopathology.

The National Registry of Domestic Animal Pathology probably offers the best mechanism for implementation of these QA programs. Future ASVP involvement is likely to be via the Registry.

(g) Possible ASVP Web Site.

The possible/probable advantages of the ASVP developing/maintaining a web site were discussed.

It was suggested that the incoming Executive examine the possibility of doing so.

(h) ASVP policy on members failing to renew subscriptions.

Consensus was that members who fail to renew their ASVP subscription should be allowed 1 year's grace. The ASVP Secretariat will provide subscription notices (current and arrears) to the member at the address recorded on the membership list. Subsequent failure to pay subscriptions outstanding for more than 1 year should result in removal of the offender's name from the membership list.

(i) Next Meeting.

It was agreed that the 1998 Annual General Meeting and Conference will be held in Sydney in association with the 1998 AVA Conference, probably 15/16 May.

There being no other business, the meeting was closed at 5:55pm. In closing, the President, on behalf of both the Executive and membership, thanked the local Brisbane committee for organisation of an excellent conference.

INFORMATION SHEET # 24 - LICENSING & ASSIGNING RIGHTS

This information sheet is mainly for owners of copyright who intend to grant copyright rights to a person or organisation. It will also be of interest to people who are acquiring rights from a copyright owner.

We give a brief introduction to the ways in which copyright rights can be granted, and matters to be considered when granting rights. In most cases, the granting of rights will form part of a contract, and we note some general points about contracts.

This information sheet is only an introductory guide to the sorts of issues you need to consider when granting or acquiring copyright rights. In most cases, you should get legal advice before finalising any agreement or transaction relating to copyright.

In some cases, the Copyright Council's staff lawyers can give free legal advice about copyright law. A staff lawyer gives advice by telephone from Monday to Thursday, 9am to midday, and 2pm to 5pm (Sydney time). You may also post or fax a written enquiry (please include your phone number). If you are seeking advice about a contract you have been offered, we generally need to see the contract before giving any advice.

Ways of dealing with copyright rights

Copyright is not one "right", but rather a "bundle" of rights. For all material protected by copyright, the copyright owner has the exclusive right to reproduce or make copies. Depending on the type of material, the copyright owner usually has other rights as well - for example, to perform in public and broadcast.

Copyright rights may be assigned (which means someone else becomes the owner) or **licensed**. When being assigned or licensed, copyright may be divided in any number of ways, including by territory, time and type of use.

Assignment

To be effective, an assignment must be in writing and signed by or on behalf of the owner of copyright. For example, it is common for film production companies to obtain an assignment of copyright in a screenplay from the screen writer. Thus, the film production company becomes the owner of copyright in the screenplay.

Licence

For the purpose of copyright, you grant an "exclusive licence" if the licence is in writing and signed by you, and the licensee is the only person entitled to use the work in the way covered by the licence. For example, it is common in book publishing agreements for a writer to grant the publisher an exclusive licence to print and publish the writer's novel. The writer is not entitled to license another publisher to publish the same novel.

An exclusive licensee has similar rights to the owner of copyright, and may take legal action for infringement by third parties. In the example above, if the copyright in the novel is infringed, both the publisher and the writer may institute legal proceedings.

A licence may also be non-exclusive. If you grant a non-exclusive licence to do something with your work, you may continue to use your work in that way and to grant non-exclusive licences to others to use your work in that way. For example, if you granted a non-exclusive licence to a publisher to reproduce your illustration in a book, you may also grant other publishers non-exclusive licences to reproduce your illustration in their books and you may reproduce the illustration yourself.

Some points about contracts

Generally, a contract need not be in writing to be legally binding. However, contracts which assign copyright or grant an exclusive licence must be in writing and signed by the copyright owner to effectively grant rights. It is also advisable that agreements dealing with non-exclusive uses of copyright material are recorded in writing; the potential for dispute about what was agreed will be minimised, and there is evidence of what was agreed.

In order for a contract to be binding, the following four elements must be present:

- An offer. For example, a publisher offers an artist $200 for a licence to include the artist's work in a book.

- An unconditional acceptance of the offer. If the artist asks for a free copy of the book in addition to the fee, this is regarded as a counter offer and the contract is not made until this or some later offer is accepted.

- Some "valuable consideration" or benefit must pass between the parties. In this case, the publisher receives the benefit of using the work in the book and the artists receives a payment for this use. However, the consideration does not need to be financial. Receiving a free copy of the book could be sufficient.

- An intention to be legally bound.

Points to note

- Once a contract is final, neither party can vary it without the consent of the other.
- If the contract is in writing, there is a presumption that the written contract contains all the terms. It is difficult to argue later that orally agreed conditions form part of the agreement between the parties.
- A contract is only binding on the parties who have made the agreement. Obligation cannot usually be imposed on people who are not party to the contract.
- Only individuals, incorporated bodies (companies and associations) and partnerships can enter legally binding agreements.
- You should always obtain legal advice before finalising any contract or signing any document. It is usually difficult to escape the obligations of a contract once it has been signed.
- **Stamp duty:** All states and territories have stamp duty legislation. Duty is imposed either at an ad valorem rate (according to the value of the property) or a fixed nominal rate. Where an agreement is executed which relates to copyright, stamp duty may be payable on that document. Generally, only nominal duty would be payable. If a document is liable to be stamped, it cannot be relied on in court proceedings until it has been properly stamped. Enquiries in relation to stamp duty may be made with the relevant state or territory revenue office.

Granting copyright rights matters to consider

The following matters should generally be covered in a contract in which copyright rights are granted. We refer persons granting the rights as the "grantor", and to the person acquiring rights as the "grantee".

Parties: The grantor and the grantee must be clearly identified. If either of the parties is trading as a business, you may need to include the name or names of the people or company behind the business - for example:

This contract is between John Moretti of 10 Seaside Drive, Flower Bay, NSW 2222 and Maria Brown and Gillian Singer, trading as Great Aspirations, of 6/45 Riverview Terrace, downtown, VIC 3333.

If either of the parties is incorporated, you may need to include the Australian Company Number (CAN) or Australian Registered Body Number (ARBN).

Describe material in which rights are being granted: The material for which rights are being granted must be clearly described or identified. In some cases, you may attach a copy of a work to the agreement.

Copyright owner: The agreement should state the name of the owner of copyright in the material. This will usually be the grantor. In some cases, however, the grantor may be granting rights in another capacity - for example, as the copyright owner's agent, or as an exclusive licensee.

Rights granted: The agreement must set out clearly what rights the grantor is granting to the grantee. In some cases, it may be a good idea to confirm what rights (if any) are retained by the grantor.

Duration: The agreement should state how long the rights are granted for.

Territory: The agreement should state where the rights may be exercised - for example, Australia and New Zealand. Generally, you should grant rights only for those territories in which the grantee has the experience and expertise to exploit the work to your benefit.

Payment: There are different ways of being paid for copyright, including by an up-front fee, or by a percentage of income from sales of the work (royalties). In some cases, there are recommended rates of industry standards - for example, in relation to book publishing and music publishing.

The agreement must set out how payments are calculated and when they are to be made.

Obligation to publish and market: If you are to be paid a percentage of the income from sales of your work, then there should be a clear obligation in the agreement for the grantee to publish and market your work within an agreed time frame, and to continue to do so.

Accounting and inspection of accounts; If you are to receive a percentage of income received by the grantee (for example, a percentage of income from book sales), then the grantee should be required to give you information on a regular basis (for example, every three months) about how much income it has received, and how much is due to you. The agreement should also require the grantee to allow you to inspect its relevant accounts on request.

Attribution and copyright notice: In most cases, the agreement should require the grantee to ensure that your name appears with reproductions of your work. If you remain the owner of copyright, the agreement should require the grantee to ensure that the copyright notice with your name appears with reproductions of your work. The copyright notice is the symbol © with the name of the copyright owner and the year. It is not necessary for protection in Australia, but operates as a notice that the work is protected by copyright.

Alterations: In most cases, the agreement should require the grantee to get your permission before making any changes to the work.

Warranty: Many agreements include a warranty from the grantor that the work is original and does not infringe anyone else's copyright.

Assigning rights: The agreement should prohibit the grantee from assigning or licensing the rights granted in the agreement to someone else without your consent.

In some cases, it may be appropriate for the grantee to be entitled to authorise certain uses of your work by others (sometimes referred to as sub-licensing). For example, book publishers are commonly entitled under publishing agreements to authorise reproduction of extracts of a work in works published by other publishers.

Termination: Where an agreement allows ongoing use of a work, it must deal with the circumstances in which you may stop further use of the work.

If you are being paid royalties, the agreement should entitle you to terminate the agreement and the entitlement to continue using the work, if the grantee stops publishing or marketing the work. In agreements which have this sort of provision, the grantor must usually give the grantee notice of intention to terminate.

The agreement may need to state what the grantee may do with undistributed articles made under the contract (for example, books or T-shirts which have been made but not sold). Where the agreement allows the grantee to grant rights to others (sub-license), the agreement may need to state what effect termination of the agreement has on these sub-licences.

If the grantee is a company, it is a good idea for the agreement to provide that the agreement and any rights granted under it will automatically terminate if the company goes into liquidation.

Arbitration: You may want to include a clause which provides for arbitration in the vent of a dispute.

Sign and date of contract: The agreement should be dated, and signed by all parties.

Further information about copyright

The Copyright Council publishes a series of pamphlets and information sheets, such as : *An Introduction to Copyright in Australia and Writers and Copyright*. The Council also publishes a series of more detailed publications called *Bulletins*, such as: *Music & Copyright* and *Artists & Copyright*. These cost $20 each.

For further information about our publications, please ask us to send you our publication list. Please contact us if you would like further information about the Copyright Council or its services.

Photocopying this information sheet

P#24 August 1996

The Australian Copyright Council has been assisted by the Commonwealth Government through the Australia Council, its arts funding and advisory body. The Copyright Council also gratefully acknowledges the support of the Australian Film Commission.

Dental Abnormalities in a Steer with Chronic Mucosal Disease

Anita Gordon, Yeerongpilly Veterinary Laboratory (QDPI)

A stunted two year old Murray Grey steer was presented for necropsy with a history of intermittent scouring and loss of condition over the past two to three months. The herd had been recently drenched with Ivomec and Nilzan. The very observant stock inspector involved with this case pointed out that the steer's first permanent incisors had not yet erupted.

At necropsy, there was pitting of the crowns of the deciduous incisors and irregular wear of the occlusal surfaces. The cheek teeth were unevenly worn with deeply interlocking upper and lower arcades. The presence of buccal, glossal and oesophageal ulcers, together with a scurfy dermatitis of the lower limbs, suggested chronic mucosal disease. A significant burden of *Haemonchus placei* was also present. Histology of skin and upper alimentary lesions revealed necrosis of individual or small groups of epithelial cells. The diagnosis of persistent bovine pestivirus infection was confirmed with antigen capture ELISA.

Bovine pestivirus infection is described as a cause of enamel hypoplasia by Barker et al (1993): look under Odontodystrophies in J, K and P.

Lymphosarcoma in a Young Fruit Bat

Anita Gordon and Hume Field, Yeerongpilly Veterinary Laboratory (QDPI)

A captive-bred six month old male grey-headed flying fox (*Pteropus poliocephalus*) was part of an experimental colony being serologically and virologically monitored for bat paramyxovirus (BPV) infection. (BPV, recently isolated from several species of fruit bats in Queensland, appears to be identical to equine morbillivirus). The bat was found dead without any apparent premonitory signs. At necropsy, there was moderate abdominal haemorrhage which emanated from a 3cm diameter multilobulated tumour mass, which was displacing the left kidney, ureter and adrenal gland ventrally. On cut surface, the tumour was pale and soft with grossly visible areas of haemorrhage and necrosis. Some lymph nodes (mediastinal, gastric) were enlarged and haemorrhagic, whereas others (mesenteric) were grossly normal. Both kidneys were pale and the left kidney was slightly hydronephrotic.

Sections of tumour revealed a lymphoblastic lymphosarcoma characterised by large sheets of large round cells with large hyperchromatic or vesicular nuclei, often with a single, very large nucleolus. There were high rates of apoptosis and mitosis together with confluent areas of tumour necrosis. Tumour cells were present in the subcapsular, interstitial and hilar parenchyma of the kidneys and there was widespread necrosis of distal tubular and collecting duct epithelium (presumably as a result of ischaemia).

The animal appeared to have been leukaemic as tumour cells were present within blood vessels in a number of tissues (lung, myocardium, liver). The spleen appeared to be uninvolved. Tumour cells effaced lymph nodes architecture of the gastric and mediastinal nodes and occupied the subcapsular sinus in a mesenteric node.

This bat had previously been found positive for antibodies to equine morbillivirus (as had its dam) and was negative for Australian bat lyssavirus by fluorescent antibody test. At this stage, there is no intention to search for retrovirus.

Reference: Halpin, K., Young, P. & Field, H (1996) Identification of likely natural hosts for equine morbillivirus. Communicable Diseases Intelligence 20,476.

Hypocalcaemia in a Dog

Peter Lording, Central Veterinary Diagnostic Laboratory, Mount Waverley

A nine year old spayed female German Shepherd was presented to the referring veterinarian having been off colour for one week. On the day of presentation, the dog had diarrhoea, continual tetanic-like seizures and collapse. Temperature, pulse and respiration were normal. The seizures were not activated by stimulation. The owners did renovate an old house approximately 18 months previously and the differential diagnoses considered were electrolyte disturbance secondary to diarrhoea, hypocalcaemia, hypoglycaemia and lead poisoning.

Blood samples were collected and submitted for a total body function profile and the following results were obtained:

HAEMATOLOGY	**Patient Results**	**Normal Values**	**BIOCHEMISTRY**	**Patient Results**	**Normal Values**
RBC x 10^{12}/L	7.36	5.50-8.50	BiliT. Umol/L	1	2-15
Hb g/L	149	120-180	Protein g/L	69	54-78
PCV L/L	0.49	0.37-0.55	Albumin g/L	31	24.38
MCV fl	66	60-77	Globulins g.L	38	28-42
MCH pg	20	19-24	Sodium mmol/L	147	138-156
MCHC g/L	304	300-360	Potassium mmol/L	4.7	3.8-5.8
			Chloride mmol/L	110	100-115
WBC x 10^{9} / L	9.30	6.00-1700	Bicarbonate mmol/L	21.4	18.-24
Seg. Neutrophils	6.98	3.00-11.50	Urea mmol/L	5.5	3.6-8.9
Lymphocytes	1.39	1.00-4.80	Creatinine mmol/L	0.09	0.06-0.15
Monocytes	0.93	0.15-1.35	Calcium mmol/L	1.02	2.10-2.80
Platelets x 1010^{9} / L	172	200-500	Phosphorous mmol/L	2.32	0.87-2.10
RBC morphology	Normal	-	ALT IU/L	42	5-80

HAEMATOLOGY	Patient Results	Normal Values	BIOCHEMISTRY	Patient Results	Normal Values
Basophilic stippling was not detected			AST IU/L	46	10-80
WBC morphology	Normal	-	ALP IU/L	13	10-120
Platelets	Adequate	-	CK IU/L	280	50-400
			LDH IU/L	51	50-400
			Glucose mmol/L	5.2	3.3-6.7
			Cholesterol mmol/L	3.08	3.9-7.8

DISCUSSION:

The significant findings were decreased serum calcium and increased serum phosphorus. The calcium was repeated on the sample initially submitted and the result was 1.10 mmol/L. The clinical history, physical examination findings and laboratory data were consistent with hypoparathyroidism.

A frozen serum was submitted for a parathyroid hormone assay and the result was <1ng/L (normal reference range for dogs 10-90ng/L). The dog was supplemented with calcium and vitamin D and 14 days after the initial samples were collected, calcium was 2.05 mmol/L and phosphorous 2.26 mmol/L. At this stage, the dog was clinically normal.

Further results 33 days after initial sample collected were calcium 2.27 mmol/L and phosphorous 2.10 mmol/L. The owners reported that the dog had not been so well for a long time!

Hypoparathyroidism is an absolute or relative deficiency of parathyroid hormone secretion resulting in hypocalcaemia. The prevalence of hypoparathyroidism in dogs is not known but the disease is uncommon and is generally due to idiopathic immune-mediated parathyroiditis. The disease is seen in dogs of most ages (mean age 6 years, range 6 weeks to 12 years) and breeds commonly affected include toy poodles, miniature schnauzers, German shepherds, Labrador retrievers and Scottish terriers. The disease occurs primarily in female dogs. In cats, spontaneous disease is also rare but it is not uncommon in cats after thyroidectomy.

Clinical signs are caused by low levels of ionised calcium which affects various body systems. Neuromuscular signs include seizures, tetany, ataxia and weakness caused by increased neuromuscular activity as a result of diminished neuronal membrane stability. Cardiovascular changes are bradycardia, and on ECG, prolongation of the ST and Q-T segments. Gastrointestinal signs include anorexia and vomiting and other possible changes are polyuria and polydipsia of unknown pathogenesis, panting, face rubbing and posterior lenticular cataracts. Up to 20% of hypoparathyroid animals may be normal on physical examination.

Haematology, biochemistry and urinalysis are generally normal except for hypocalcaemia (<1.60 mmol/L) and normal or mild to moderate hyperphosphataemia. The value of the laboratory tests is confirming hypocalcaemia but also ruling out other differential diagnoses. Serum albumin must be carefully noted because hypoalbuminaemia is the most common cause of hypocalcaemia. If the albumin is low, it is necessary to correct the serum calcium by the following formula:

Corrected calcium = calcium (mg/dl) - albumin (g/dl) + 3.5

The only other disease process that produces hypocalcaemia and concurrent hyperphosphataemia is renal failure which is easily distinguished from hypoparathyroidism by the present of azotaemia.

Other differentials for seizure disorders are syncope, hepatoencephalopathy, hypoglycaemia, epilepsy, neurotoxicities, neoplasia and inflammatory diseases of the CNS. Disorders that may cause weakness include congenital cardiac anomalies, arrhythmias, heart failure, pericardial effusion, hypoadrenocorticism, hypoglycaemia, anaemia, hypokalaemia (especially cats), hypothyroidism, myasthenia gravis, polymyositis, spinal cord disease, tick paralysis, botulism, organophosphate toxicity and lead poisoning.

Serum parathyroid hormone is undetectable or in very low concentration in dogs with hypoparathyroidism. Patients with other processes causing hypocalcaemia, e.g renal failure, have normal to high concentrations of parathyroid hormone.

Circulating serum calcium is 50% protein bound, 40% ionized and 10% complexed with other substances. Only the ionized fraction is available to tissues and changes in this fraction are responsible for clinical problems (hypercalcaemia and hypocalcaemia). Measurement of ionized calcium is difficult for routine performance and biochemistry results record only total serum calcium values. The pathogenesis of hypocalcaemia is varied, there are numerous diseases associated with low calcium levels and a wide variety of clinical signs is seen.

Low calcium results should be rechecked to rule out laboratory error. Serum calcium levels can be significantly increased by lipaemia but levels may be decreased by alkalosis. The main rule outs for hypocalcaemia include primary hypoparathyroidism, eclampsia, renal failure, ethylene glycol toxicity, acute pancreatitis, intestinal malabsorption and nutritional secondary hyperparathyroidism.

Dogs with hypocalcaemia should be hospitalised for medical management of the disorder until clinical signs of hypocalcaemia are controlled and serum calcium concentration is at least 1.75 mmol/L. Emergency treatment is best done with 10% solution of calcium gluconate given slowly to effect over a 10 minute period (5-15mg/kg or 0.5-1.5ml/kg). Heart rate should be monitored, and ideally ECG monitored as well, with administration of calcium gluconate stopped if bradycardia or shortening of the Q-T interval occurs. Short-term treatment after the correction of tetany can be done with 10% solution of calcium gluconate, either by:

a) constant rate intravenous infusion of 60-90mg/kg/day added to IV fluids, or

b) subcutaneous administration 3-4 times daily of the dose needed to initially control tetany after dilution of the calcium gluconate in an equal volume of saline.

Long term treatment involves indefinite administration of vitamin D and calcium. The dose of vitamin D should be varied on the basis of serum calcium concentrations and shorter acting preparations are preferred to enable faster correction of hypercalcaemia if over dosage occurs. Maximum oral doses of calcium and reduced amounts of vitamin D is preferable although calcium preparations given orally can cause gastrointestinal disturbances.

There are numerous potential hazards with injectable calcium, such as incompatibility with tetracyclines, cephalosporins, methylprednisolone, sodium succinate, dobutamine, metoclopramide and amphotericin B. Intravenous calcium may cause arrhythmias in dogs on digitalis and may antagonise the effects of calcium channel blocking agents. Thiazide diuretics, given in conjunction with large doses of calcium, may result in hypercalcaemia.

Serum calcium concentrations should be monitored monthly for the first six months, and then every 2-4 months after stabilisation. The aim of treatment is to maintain serum calcium between 2.00-2.50 mmol/L. Ongoing complications include persistent hypocalcaemia or overcorrection causing hypercalcaemia which can lead to nephrocalcinosis and renal failure. The prognosis for long term survival is excellent when owner compliance is good and serum calcium is closely monitored. Adjustments in the doses of oral calcium and vitamin D can be expected, especially during the initial 2-6 months of treatment.

Encephalitozoonosis in a pet rabbit

Kathleen Johnston, Central Veterinary Diagnostic Laboratory, Mount Waverley

A 6 week old Dwarf female rabbit was presented dead to a clinician. Death had been sudden and the rabbit had been vaccinated for calicivirus 5 days prior to death. Similarly, earlier in the year, a 2 year old rabbit from the same household had been vaccinated for calicivirus and had died 9 days later.

The entire rabbit was presented to CVDL for autopsy. The rabbit was in good condition with food in the stomach and intestines, further indicating acute death. There were no gross pathological changes. No pathogens were isolated from lungs, spleen and liver. The ELISA test on fresh liver for calicivirus antigen was negative.

Histological lesions occurred in the kidneys, striated muscle, lungs and brain. There was a non-suppurative interstitial nephritis with multifocal acute to subacute tubular epithelial necrosis. Within both necrotic and intact tubular epithelium were trophozoites 1μm long or pseudocysts 8-12μm in diameter. There was multifocal striated muscle necrosis with heterophilic inflammation and focally intense heterophilic interstitial pneumonia. Within the brain were multifocal zones of necrosis and astrocytosis.

The diagnosis was encephalitozoonosis. *Encephalitozoon cuniculi*, a microsporidium, is an obligate intracellular, protozoan parasite. Rabbits are the principal hosts though other mammals can be infected. Transmission is urine-oral in a rabbit colony, exchange being common between a doe and her young. Severity of infection depends on host resistance and infective dose.

Following ingestion, the spores pass via infected mononuclear cells into the systemic circulation thus to the organs of high blood flow such as lung, liver and kidney. Most infections are chronic and sub-clinical. Progressive weight loss or neurological signs may be observed. Encephalitozoon has a tropism for the central nervous system and kidney although lungs, heart and liver may be affected.

Diagnosis is made by the identification of the lesions and the protozoal organisms in tissue sections. They can be differentiated from *Toxoplasma spp*. by being gram positive and staining with carbol fuchsin. Serological tests include modified India ink immunoreaction test, indirect immunofluorescence microscopy and a dot ELISA test.

Although infection is often latent, *E. cuniculi* is a significant pathogen in rabbits due to its ability to cause decreased weight gains in young rabbits, to lower resistance to illness and deaths due to other diseases and its ability to affect the immune response. Except in severely immunosuppressed humans, there is no convincing evidence of zoonotic potential.

References:

Percy, D.H., Barthold, S.W. Pathology of laboratory rodents and rabbits. 1st Ed. 1993. Iowa State University Press: 210-214.

Walden, N.B. Rabbits: A compendium 1990. University of Sydney Post Graduate Foundation: 40-41.

Harkness, J.E., Wagner, J.E. The biology and medicine of rabbits and rodents. 3rd Ed. 1989. Lea & Febiger: 132-134.

Intestinal Cryptococcosis

Judith Wilkie, Victorian Veterinary Pathology Services

A 2 year old pug from the western district of Victoria presented with vomiting of one week's duration which was worsening despite treatment. Haematology and serum biochemistry were unremarkable. Exploratory laparotomy revealed an intussusception around a mass in the small bowel.

Histopathological examination identified the mass as a mucoid granuloma with the typical "soap bubble" appearance and numerous budding yeasts with thick capsules characteristic of cryptococcosis. The lesion extended from the mucosa, which was replaced focally by inflammatory and granulation tissue, through the full extent of the intestinal wall.

STATE REPORTS SOUTH AUSTRALIA - RUTH REUTER

Veterinary Diagnostic Laboratory Services in South Australia - Ruth Reuter

There has been much concern expressed by a large number of people over the decline of funding and uncertainty of the future of diagnostic laboratory services in several of the States in the past months. South Australia has been no exception, with VETLAB the subject of numerous reviews and discussions over the last few years.

In February of this year, the South Australian government decided to outsource the management of its laboratory services and called for tenders for this purpose. The VPS tender was successful and a contract to take over management of VETLAB for 5 years was signed on 15 May. The contract takes effect from 1 July 1997.

Under the terms of the contract, VPS will relocate to the VETLAB site at Glenside, SA. We will be sharing the facilities there with several other groups from Primary Industries South Australia. The two organisations will be run jointly, but virtually separated according to the case material. VPS will handle companion and performance animals, while VETLAB will handle commercial production animals, birds and fish.

We are obviously very excited by this event and the challenge it presents for our operation in South Australia. We are strongly committed to providing all clients of both organisations with a high level of service. All VPS pathologists have extensive backgrounds in production animal pathology and we are recruiting additional people for the operation (see advertisement in this issue of Vet Path Report). We will also have the back up support of our members in Brisbane and Sydney.

VPS Adelaide/VETLAB address is:
33 Flemington Street, Glenside, SA 5065 or PO Box 445, Glenside, SA, 5065.

Putative Feline Cutaneous Entomophthoromycosis

John Finnie, VETLAB

A 3-year-old male, Persian cat from Broken Hill presented with several indurated, 1-1.5cm diameter, cutaneous masses on the back. These deep (0.5cm) subcutaneous lesions had draining sinuses and were situated on the shoulder, mid-dorsum and near the base of the tail.

Microscopically, there are numerous, generally well circumscribed, granulomas in the dermis extending down into the subcutis and sometimes to the epidermis with focal ulceration. The granulomas were composed of epithelioid macrophages and multinucleated giant cells and were usually surrounded by a rim of neutrophils. In the necrotic centre of the granulomas were numerous septate, and sometimes branching, fungal hyphae which often appeared as oval, largely non-staining spaces (5-20μ in diameter) with a well defined wall when cut in transverse section. These hyphae, which were much better visualised in PAS and GMS stained sections, were surrounded by a large eosinophilic cuff (Splendore-Hoeppli phenomenon) that was PAS-positive. Rarely, the fungi had an internal structure of numerous basophilic granules.

A diagnosis based on fungal culture was not possible in this case as all skin lesions were totally excised and submitted formalin-fixed. However, the fungus was assigned on distinctive morphological criteria to the order Entomophthorales of the Zygomycetes. There are two diseases associated with species of the order Entomophthorales, basidiobolomycosis caused by *Basidiobolus haptosporus (ranarum)* and conidiobolomycosis due to *Conidiobolus coronata* infection. In the veterinary literature, entomophthoromycosis was often grouped with mucormycosis and pythiosis under the terms “phycomycosis” but this taxonomic classification is no longer tenable.

B. haptosporus and *C. coronata* have a very similar morphological appearance. Both have septate, branching hyphae with a prominent perihyphal sleeve of eosinophilic Splendore-Hoeppli material, the latter probably an antigen-antibody immune complex precipitate. Since the subcutaneous lesions of *B. haptosporus* are most commonly found on the trunk and head (where contact with contaminated soil is facilitated), the skin lesions on the back of this cat were probably caused by this fungal species. *C. coronata*, by contrast, is a rhinoentomophthoromycosis that occurs exclusively in the nasal region.

Multicentric Lymphosarcoma in an Alpaca

R Mason, Tasmanian Animal & Water Quality Diagnostic Service Laboratory, Launceston

A two year old castrated alpaca was found in sternal recumbency at 9:30am. The animal at this time had a heart rate of 120/minute, a respiration rate of 90/min and rectal temperature of 42°C. It appeared uncomfortable when standing and refused food and water. The owner noticed small urticarial-like patches around the head and over most of the body plus a small amount of bleeding from the nose.

The animal received intense treatment throughout the day. It was given combinations of various antibiotics, electrolyte fluids, vitamins, corticosteroids and adrenaline. The alpaca died at around 9:30pm the same day and it was submitted for autopsy the following day.

There was subcutaneous oedema, especially of the lower limbs, plus some haemorrhage. The pericardial sac contained excess fluid, the lungs were oedematous and there was froth in the airways. The spleen was enlarged, soft and pulpy. There were multiple pale, rather diffuse, foci throughout the myocardium which were most apparent in the subepicardial myocardium of the right ventricle. All lymph nodes were enlarged and soft with blotches of reddening. There were haemorrhages beneath both the pleural and peritoneal lining and in fat depots.

Although the overall appearance presented as a possible severe acute infection, no bacteria were detected in stained smears or cultures of blood or tissues.

The histopathological diagnosis was multicentric lymphosarcoma. In some sections, which included moderate sized blood vessels, clusters of enlarged mononuclear leucocytes were present suggestive of a leukaemic state. The myocardial lesions seen grossly were due to neoplastic infiltration by malignant lymphoid cells. Unfortunately, no blood was collected from the live animal for a haematological examination.

Shifting lameness, dyspnoea, fever and recurrent infections can be clinical features associated with leukaemias - systemic lymphosarcomas. Death may be a consequence of infection, haemorrhage - anaemia, organ dysfunction secondary to neoplastic cell infiltration and proliferation, etc.

The fever in this animal may have been due to neoplasia or infection for although no bacteria were detected, they may have been overwhelmed by the intense treatment received.

A multicentric lymphosarcoma has been described in a llama from the USA. ME Fowler, D Gillespie and J Harkema (1985) JAVMA Vol 187, N. 11 p.1245.

STATE REPORTS NEW SOUTH WALES - PAUL GILL

NEWS:

Prof. Malcolm Nairn is currently interim Vice Chancellor of the University of New England. Malcolm was formerly Professor of Clinical Pathology, then Deputy Vice Chancellor at Murdoch University, WA.

Acid run-off linked to Botulism in Water Birds

John Boulton, Regional Veterinary Laboratory, Wollongbar

Botulism has been incriminated in the deaths of about 20 pelicans in the Richmond River over the past 2 years. Gulls and terns have also been affected. The affected birds become listless and recumbent on land or water, develop green-brown diarrhoea and die after several days despite supportive therapy.

An adult male pelican brought to RVL Wollongbar had muscular weakness and spinal and anal flexes which were intact but slow. At necropsy it had hydropericardium (10ml serous fluid) but no other significant gross or microscopic lesions. Its gastrointestinal tract contained various Helminth parasites (not considered significant) and no recent ingesta, indicating inability to feed and resulting in the bile-stained faeces.

Mouse inoculation tests for *Cl. Botulinum* type C toxin were positive. Mice inoculated with serum or pericardial fluid, with and without types A and D antitoxins, developed “wasp waists” and died. Mice inoculated with fluid and type C antitoxin remained healthy. ELISA tests for type C and D toxins were negative. *Cl. Botulinum* was not recovered from the bird’s intestinal tract.

The birds’ death typically follow periods of heavy rain, when acid water runs into the river from acid sulphate soils drained for agriculture. This acid run-off kills many fish which are scavenged by birds of the affected species. The lethal toxin is probably elaborated when *Cl. Botulinum* multiplies in the decomposing fish.

Avian Hepatic Vasculopathy

George Reppas, Regional Veterinary Laboratory, EMAI

As part of Marek’s vaccine trial, a six week old SPF cockerel that had been injected with vaccine diluent was necropsied. Macroscopic findings included enlargement and multifocal petechial haemorrhages of the thymus. Microscopically, the thymic parenchyma was reactive and congested. Other significant findings included a moderately severe multifocal necrotising vasculitis within the liver. Further probing of the history revealed that this SPF bird upon completion of the vaccination trial, was placed in the same room as a guinea pig colony several days prior to necropsy. It was hypothesised that the guinea pig colony may have provided an infectious antigenic stimulus for this SPF bird.

Bovine Malignant Catarrh

George Reppas, Regional Veterinary Laboratory, EMAI

Case 1:

A 12 month old heifer from Albury was presented with a 2 week history of weight loss without diarrhoea, nasal discharges, erythemia and sloughing of the oral mucosa and muzzle, marked pyrexia, severe coronitis and hoof separation. A moderate neutropaenia and marked hyperfibrinogenaemia supported diagnosis of a severe inflammatory process. No further macroscopic lesions were detected at necropsy. Microscopic findings included severe subacute renal arteritis at the corticomedullary junction and severe subacute ulcerative coronitis.

Case 2:

A 6 month old Brahman x Shorthorn heifer from Menangle that was destroyed due to ill-thrift. Grossly the carcass was in poor condition. Bilateral corneal oedema was present and had been noticed about 2 months earlier. The intimal surface of the aorta from the semilunar valves to about the level of the adrenal glands was raised and roughened. Microscopically there was a widespread, severe vasculitis/perivasculitis (including the cornea) which often included fibrinoid necrosis of the vessel walls. The sections of aorta displayed mineralisation and fragmentation of the muscularis (possibly an unusual manifestation of the widespread vasculitis).

The interesting feature about both these cases was their protracted clinical history.

Bovine Respiratory Syncytial Virus Pneumonia

George Reppas, Regional Veterinary Laboratory, EMAI

Pneumonia in a 7 day old Limousin calf was thought to be due to BRSV infection. Suggestive microscopic findings were an acute segmental necrotising bronchiolitis along with the presence of numerous alveolar syncytial cells. Three of the 5 herd mates yielded positive reactions in the BRSV ELISA.

In another case, calves developed coughing about 2 weeks after birth and died despite antimicrobial therapy. Microscopic findings included a severe necrotising bacterial (*Actinomyces pyogenes* isolated) bronchopneumonia and associated generalised congestion and alveolar oedema. However, less affected parenchymal areas displayed prominent alveolar syncytial giant cell formation which suggested some involvement of BRSV.

Two dyspnoeic 8 week old Angus x Murray Grey calves from another property, that died with treatment, were necropsied. Pertinent macroscopic findings included diffusely congested and oedematous lungs with froth filled airways and scattered fibrin tags over the pulmonary pleurae. Both calves were well conditioned. Bacteriological examination failed to isolate any organisms. Microscopic findings included a severe subacute interstitial pneumonia with fibrin exudation into

the alveoli and proliferation of alveolar epithelial cells with a tendency to form large alveolar syncytial giant cells suggesting BRSV infection.

Avian Myeloid Neoplasia

George Reppas, Regional Veterinary Laboratory, EMAI

A 22 week old broiler breeder cockerel was necropsied because of a soft tissue mass adherent to the breast bone. Microscopic examination of the tissue mass failed to identify the type of tissue affected as normal architecture was replaced by a population of fairly uniform round cells and a few fibrous septae. The round cells had abundant granular and refractive eosinophilic cytoplasm and displayed variable nuclear morphology (polylobed to large vesiculated round nuclei). A high mitotic index was evident. Myelomas frequently arise in rich haemopoietic tissue and breast bone is one such area in the bird.

Porcine Foetal Pneumonitis and Abortion

Tony Ross, Regional Veterinary Laboratory, EMAI

Early and late abortions and stillbirths were suddenly observed in 10 litters in a 400 sow large white piggery on the northern tablelands of NSW. Eleven foetuses from approximately 60 days to age to full term were examined together with sera from 4 sows. Serological examinations were negative for *Leptospira Pomona* and *L. tarassovi*. Seven foetuses of several sizes showed acute interstitial or bronchopneumonia. One foetus had a mild multifocal epicarditis and the one placenta examined showed a subacute diffuse placentitis.

Parvovirus antigen and antibody examinations were negative in 8 sets of fluids and tissues and EMC virus isolation was negative in all 11 hearts examined.

There was clear evidence of an infectious foetopathy of unknown origin. The foetal lung lesion has been seen with porcine respiratory and reproductive syndrome. A wide range of foetal tissues and fluids plus acute and convalescent sow sera were referred to the Australian Animal Health Laboratory, Geelong for PRRS examination. No antibodies to PRRS were found in sow sera and foetal tissue pools were used for virus isolation of PRRS, Aujeszky's disease and classical swine fever in a range of cell culture lines. No cytopathic effects were seen after three passes and no immunostaining was seen for PRRS virus. It is possible that the foetal pathology could have been caused by congenital toxoplasmosis, chlamydiosis or a non-cross reactive strain of leptospirosis. Suggestions are most welcome.

Zinc and Caprine Dermatopathies

Tony Ross, Regional Veterinary Laboratory, EMAI

An investigation into generalised hyperkeratosis of the skin and abnormal hoof growth in British Alpine dairy goats on the central coast of NSW lead to examination for a number of parasitological, nutritional and environmental factors.

Zinc deficiency was considered a possibility as it had been associated with alopecia and hyperkeratosis in Angora goats in South Australia. Ten sera were referred to Vetlab, South Australia for zinc assay. The results were 16.6 $\pm$ 4.3 μmol Zn/1 which was interpreted as normal. However, interpretation of these results proved to be somewhat difficult due to the following factors:

1. Zinc levels in blood are very labile and simple estimations of it alone are likely to be misleading (Radostits et al Veterinary Medicine 1994 p.1405).

2. Zinc levels in blood can fall precipitously at parturition and with hyperthermia (as above).

3. Zinc levels fall to about 50% of normal after 1 week on a highly zinc deficient diet (as above).

4. Significant zinc contamination has occurred from vaccutainer bungs of all types except specific trace element vaccutainers - blue tops (Vetlab SA).

5. Published data on serum and plasma zinc levels in goats is relatively scarce. Therefore, some useful information from the literature appears below together with a conversion from SI to non-SI units.

a) Goats / alopecia / hyperkeratosis South Australia
(Reuter et al 1987, AVJ 64:351)

Unaffected goats	>21.4
Affected goats	7.35 - 11.64 μmol/l
Partially affected goats	10.11 - 14.10 μmol/l

b) Seasonal dermatosis Florida dairy goats
(McDowell et al 1991, Small Ruminant Research 5:327)

Affected goats	x = 8.27 μmol/l
Unaffected goats	x = 12.71 μmol/l

c) Normal range for British Ruminants
(Mills et al, Br J Nut 1969 21:751)

Normal	12.25 - 18.38 μmol/l

d) <u>Sheep</u>

(Underwood, The Mineral Nutrition of Livestock, 2nd edition 1981)

Normal range	> 12 μmol/l
Deficient range	≤ 6 μmol/l

Note: $\frac{\mu mol/l\ Zn/1}{1000} \times 65.3 = ppm = mg\ Zn/kg$

In summary, as a guide it would appear that zinc deficiency in goats could be suspected when serum or plasma levels fall below 10 - 12 μmol/l.

STATE REPORTS WESTERN AUSTRALIA - DAVID FORSHAW

Facial Eczema in an Alpaca

J. Creeper, Animal Health Laboratories, South Perth

Facial Eczema was diagnosed in an Alpaca on a property on the south coast of W.A. The Alpaca was one of twenty that had been grazing pasture that carried *Pithomyces chartarum* loads of 100,000 spores per gram (70,000 is considered a toxic limit). The particular animal showed mild jaundice, inappetence and in the terminal stages, lateral recumbency. Plasma GGT, bilirubin and AST were markedly elevated in the Alpaca that died and were elevated in over half of the remainder of the group, some of which were showing signs of photosensitisation.

WANTED TO BORROW

A copy of Veterinary Dermatopathology by Thelma Gross et al (currently out of print). Anyone with a spare copy or one gathering dust on the shelf, please contact:

Alison Havadjia, Phone 044 220 281 (h), 044 215 370 (w)

STATE REPORTS NORTHERN TERRITORY - ANTON JANMAAT

An Evaluation of Johne's Disease ELISA Testing in Northern Australian Cattle

Anton Janmaat, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

The final report of the MRC project "An evaluation of Johne's disease ELISA testing in Northern Australian cattle" was completed in March 1997. The NT part of the project involved the ELISA testing of 508 mature beef cattle from various properties in the NT and the Kimberley and processing of tissues for bacteriological culture and histopathological examination of 13 positive and seven high negative reactors. In Queensland there were eight positive and eight High negative reactors in 541 cattle. None of the reactors showed evidence of Johne's disease, suggesting they were all "false" reactors and the specificity of the ELISA becomes 98.0 [(1048-21)/1048]. This specificity is acceptable for export testing but it does not make this ELISA an ideal test to show disease freedom.

TB or Not TB

Anton Janmaat, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

In 1996 there were 65 granulomatous lesions submitted through the National Granuloma Submission Program from the four domestic and export NT abattoirs. Five of these submissions showed lesions consistent with tuberculosis - four from buffalo and one from cattle. The buffalo came from a known infected herd; the cattle lesion was designated "indeterminate" after histopathological examination and grew *Mycobacterium bovis* on culture. With the end of the Wet, the 1997 slaughter season has been going for a few weeks and unfortunately another case of tuberculosis in a NGSP submission has been diagnosed already. The animal involved is a four to five years old steer from an Alice Springs property. There has not been a confirmed case of TB in the Alice Springs region since 1987.

Tuberculosis was a differential diagnosis of lung lesions in a feral pig. Histological examination showed a pyogranulomatous inflammatory process associated with Gram-ve bacteria. These were shown to be *Pasteurella multocida* on culture. Tuberculosis was also a differential diagnosis in a dog with a large solid abdominal mass, about 8 cm in diameter, attached to loops of intestine and involving the mesenteric lymph nodes. The mass turned out to be a fungal granuloma and *Pythium insidiosum* was isolated from the lesion. This fungus was also isolated from another dog's tail which had suddenly gone necrotic.

Abscesses in the spleens from three pigs yielded *Burkholderia pseudomallei* on culture. One pig also showed bronchial lymph node involvement and another had abscessation of liver and portal lymph nodes.

Specimens from two deer from the same property showed tuberculosis-like lesions, i.e tubercular in shape with central caseation and mineralisation and surrounded by macrophages (and giant cells in one lesion), plasma cells, lymphocytes and fibrous tissue. *M. Fortuitum* was isolated from the mandibular, retropharyngeal and prescapular lymph nodes in one animal. *B. pseudomallei* was isolated from the splenic lesion, i.e the one with giant cells, in the second animal. Unfortunately the on-farm infrastructure precluded testing the remainder of the animals for antibodies to these two bacteria. Other agents which cause tuberculosis-like lesions in lymph nodes of deer are described in the November 1996 issue of the Veterinarian, pp 10-12.

We can't leave the crocs out of this and mycobacteriosis was diagnosed histopathologically in a group of farmed yearling saltwater crocodiles which had shown increased mortalities in March this year. The early lesion consists of 30-40 macrophages surrounded by a thin layer of lymphocytes, plasma cells and the occasional granulocyte; the whole lesion is about 1/8 of a 10x40 field. There may or may not be central necrosis. Larger lesions consist of groups of the above, about ¼ of a 10x10 field. Acid-fast bacteria are usually plentiful. We received three crocodiles and lesions were found in the lung, kidney and surrounding the small retained yolk sac of one, and in the lung and liver of another animal. The yolk sac of the first animal also contained numerous acid-fast bacteria among its bacterial flora. The lesions were only detected grossly in the second animal as "mm sized microabscesses" in the lung and "white spots" in the liver, but mycobacteriosis was not thought of as a differential diagnosis and tissues were submitted for routine culture only. Nothing grew.

UNITED KINGDOM LUCY GENOVESE

Introduction

Lucy Genovese, Abbey Veterinary Services, Newton Abbot, Devon

Last year I wrote to Karl Harrigan regarding the possibility of contributing case reports to the Veterinary Pathology Report. He indicated that this was OK and suggested that we should also include a little bit about ourselves.

Abbey Veterinary Services was established in 1984 and is now one of the largest specialised, purely diagnostic veterinary histopathology laboratories in Europe. We now receive in excess of 30,000 submissions each year. The majority of our submissions are from companion animals with a smaller number from farm and laboratory animals; we also provide pathology services to the major zoological collections in the UK. We now also provide services to many parts of the world, especially Europe, but also the Middle East and Hong Kong. Abbey Veterinary Services was established by Trevor Whitbread, BSc., BVSc., MRCVS., who qualified from the University of Liverpool in 1978. He lectured in pathology at the University of Liverpool and then joined a large general veterinary diagnostic laboratory before establishing his own laboratory in 1984. There are two other full time pathologists. Judith Hargreaves MVB, MRCVS, MRCPath., graduated from Dublin in 1983 and myself, a 1990 Murdoch graduate. Dr Vanda Lucke BVSC., PhD, FRCVS., FRCPath., who was Head of the Pathology Department at Bristol University comes to us two days a week.

We are now looking for another diagnostic pathologist. See Jobline, this issue.

Calcinosis Cutis in a Cat

Lucy Genovese, Abbey Veterinary Services, Newton Abbot, Devon

Cutaneous mineralisation is a rare condition in the cat with only five cases reported in the literature. All of these cases describe changes similar to that seen with calcinosis circumscripta. Three of the cases were solitary lesions which either resolved spontaneously or for which surgical excision was curative. These may have been areas of dystrophic calcification as in one of the cases reported there was a history of an injection given in the site. The two remaining cases were associated with chronic renal disease with one case of pedal calcinosis circumscripta and one with a solitary lesion beneath the tongue.

Recently a 6 year old neutered female domestic shorthaired cat was presented with acute vomiting and multiple firm plaques within the skin of the abdomen. Radiography revealed a thickened stomach wall, decreased mineralisation of the skull and a generalised decrease in mineralisation o the skeleton. Serum biochemistry revealed azotemia, hyperphosphatemia, hyper-cholesterolaemia and hypo-albuminemia.

There was also mild increase in serum ALP and ALT.

Skin biopsies showed multifocal mineralisation of dermal connective tissue with foci of granular basophilic material within the superficial dermis. Collagen fibres at the periphery of these foci showed basophilia gradually blended with the more eosinophilic appearance of the normal adjacent collagen. A small number of lymphocytes, macrophages and very occasional eosinophils surrounded these foci. At the time the biopsies were taken, no pedal lesions were observed.

On the basis of the biochemistry, histology and radiographic changes a diagnosis of renal failure with possible secondary hyperparathyroidism was made. Treatment was conservative but the cat continued to deteriorate and the owner elected euthanasia.

At post mortem, the cat was in good body condition. There were multiple firm plaques within the skin of the trunk, face and upper limbs but the lower limbs and digits appeared to be spared. Both parathyroids were enlarged, both kidneys appeared slightly small and possibly slightly paler than normal. There was softening of the bones of the skull and the distal portion of the mandible could be bent (“rubber jaw”).

Histologically sections from both kidneys showed changes consistent with moderate to marked chronic interstitial nephritis. Sections from the parathyroid glands showed hyperplasia of the chief cells. Further sections of skin showed changes similar to that seen in the original skin biopsies. Sections taken from the digits showed changes more consistent with calcinosis circumscripta with large, focal areas of mineralisation surrounded by fibrosis and moderate numbers of inflammatory cells with macrophages dominating, accompanied by lymphocytes.

In reviewing our database over the last three years, we have identified another 5 cases of cutaneous mineralisation in cats. Two were focal lesions of calcinosis, one in the skin of the thigh, the second in the dorsal neck. Both animals had received injections in this site. Excision of the lesions was curative. The remaining three cases were consistent with pedal calcinosis cutis. All three cats were diagnosed with chronic renal failure and were euthanased shortly afterwards. Reviewing the literature and reviewing the cases submitted to us, calcinosis circumscripta appears to be the more common presentation of cutaneous mineralisation in the cat. Focal lesions appear to have quite a good prognosis. Pedal calcinosis circumscripta in the small number of cases that we have seen is associated with chronic renal disease and cats actually entering renal failure. The actual survival time following a diagnosis of pedal mineralisation similar to calcinosis cutis appears to be quite short. Widespread cutaneous mineralisation similar to calcinosis cutis appears to be quite short. Widespread cutaneous mineralisation similar to calcinosis cutis appears to be extremely rare in the cat and I have not been able to find a reference to this in the literature. Our case was associated with metastatic mineralisation due to chronic renal failure and renal secondary hyperparathyroidism.

EDITOR'S NOTE

The following two previously published reports have been reprinted in this copy of the Veterinary Pathology Report as requested/directed in the Minutes of the ASVP Annual General Meeting 3 May 1997.

1. "Dr Obendorf issues resolved" - reprinted from "Prime News", Staff Newsletter of the Department of Primary Industry and Fisheries, Tasmania, June 1997. The article is reprinted with permission provided by both Dr Obendorf and the Minister for Primary Industry and Fisheries, Tasmania.

2. "Crisis in our midst" - reprinted from the Canadian Veterinary Journal, volume 38, February 1997. The editor gratefully acknowledges permission to reprint the letter provided by both the authors and the editor of the Canadian Veterinary Journal.

Dr Obendorf issues resolved

After protracted disputation between the Department and Dr David Obendorf, all issues have now been resolved.

In 1992 and 1993 Dr Obendorf, a veterinary pathologist with 15 years service in the Department, expressed his professional concerns about departmental policy regarding animal health. In October 1996 the Department ceased providing routine diagnostic services for companion and performance animals at the Mt Pleasant Laboratory. Dr Obendorf has consistently maintained that the provision of these services is not the fundamental role of an agricultural veterinary laboratory, and that the Department should focus its resources on the provision of quality animal health services to support Tasmania's commercial livestock industries.

During the period of dispute, Dr Obendorf experienced deep distress and anxiety as a result of what he perceived to be personal attacks on him. As a result Dr Obendorf commenced litigation against the Department alleging defamation and unlawful discrimination. In May 1995 fifty DPIF employees signed a letter to the Secretary expressing their concern at the perceived disciplinary action taken against Dr Obendorf and the effect this action was having on him, the Department and the community standing of the DPIF.

Dr Obendorf appealed successfully to the Commissioner for Review on the basis that he had been treated in an unfair and inequitable manner by his employer, in that the Department allowed the publication of statements about him without his knowledge of consent. The Department, as a result of that finding, has now apologised to Dr Obendorf for its action.

Dr Obendorf lodged a complaint with the Human Rights and Equal Opportunity Commission alleging discrimination in employment. Through a process of reconciliation the matters at issue were resolved. The Department has also agreed to arrange some instruction on the operation of the *Disability Discrimination Act* for its staff and to develop guidelines concerning its obligation

thereunder. The current administration of the Department deeply regrets and deplores any personal imputation which might have been made about Dr Obendorf.

With the completion of negotiations Dr Obendorf has decided to leave the Department and in doing so he wishes to express his heartfelt appreciation for the support of his friends, work colleagues, veterinarians, farmers and members of the general public. The Secretary of the Department, Mr Evans wishes Dr Obendorf well in the future. The Department reaffirms its opinion that Dr Obendorf's competency and professionalism are beyond question and believes that this will stand David in good stead in his future career endeavours.

COMMENTARY

Crisis in our midst

John Coates, Barbara Horney, Maria Spinato

At the recent annual meeting of the Canadian Association of Veterinary Pathologists in Charlottetown, a motion was passed unanimously by our members declaring their concern and dismay regarding the level of governmental, public, and veterinary support for veterinary diagnostic laboratories in various areas of Canada.

Increasingly, our members perceive reduced government support for a variety of activities within the veterinary field, whether it be the maintenance of veterinary diagnostic laboratories at the provincial level, continued financial support for veterinary colleges, maintenance of federal programs to monitor and eradicate reportable diseases, or the monitoring of national meat inspection programs.

While privatization or abandonment of some activities presently performed by public veterinary laboratories may be considered by some individuals to be possible or even desirable, there is a significant hazard that, by doing this, the diagnostic capability of this country will deteriorate into a disjointed network under the control of a patchwork amalgam of public sector and various private groups. It is difficult to perceive how this new structure will achieve the desirable goal of a rapid, accurate, and coordinated assessment of animal disease in domestic livestock and wildlife.

A nationwide system of provincially and federally funded veterinary laboratories has been an integral pat of our ability to offer diagnostic assistance to veterinary practitioners and livestock producers. In the past, services have included daily diagnosis of animal disease in a wide variety of domestic and wildlife species, monitoring for potential entry of exotic disease into Canada, control of zoonotic disease within the country, and assurance of a healthy food supply for both national consumption and export. These are all aspects of our profession that have received too little attention both nationally and within our own profession. Many of these areas of research and diagnosis are in sectors that touch directly upon the public health aspects of veterinary science. Obviously, our purpose must be to assume a more proactive role in presenting the diverse contributions of the veterinary professions and its various disciplines, including pathology, to the rest of society and the decision-makers of Canada. Without concerted action now, we believe that the long term future of veterinary medicine is at risk through reduced public support, as are the prospects of those who work within its various disciplines.

Reduction or permissive atrophy of our diagnostic system can only lead to eventual disarray and dysfunction of Canada's agricultural well-being, with potentially disastrous economic consequences. Is it merely coincidence that the outbreak of bovine spongiform encephalopathy (BSE) in Britain followed a period of downsizing in the laboratory system and relaxing of regulatory standards? In their zeal to attain fiscal responsibility, we fear that both provincial and federal government ministries may cut programs that currently protect this country from the devastating effects of diseases that can close export markets. In today's global economy, trading

partners react quickly to perceived threats to their livestock industry or human health, as evidenced so clearly by the European response to BSE. If we no longer recognize the existence of a disease because of a successful vaccination or monitoring program, do we assume that the disease has been eradicated and that the program can now be cancelled? Perhaps we have done our job too well! So well that the consequences of the absence of these activities may not be fully comprehended by governments or the public. Canada's livestock industry has achieved a high level of health through great struggle and careful planning over the years. We cannot afford to relax our efforts now.

Unfortunately, some veterinary clinicians are unwittingly contributing to the deterioration of laboratory services. Because veterinarians are performing more diagnostic tests in-clinic, procedures that generate significant income are no longer being conducted in laboratories. Perhaps our colleagues do not realize that revenues generated by these tests are used to subsidize or offset the costs of more expensive procedures, such as microbiology and large animal necropsies. Governments take this economic bottom line into consideration when deciding whether or not to continue with diagnostic services. If this trend continues, there will undoubtedly be an increase in the price of these remaining costly tests to practitioners. Increased fees inevitably result in reduced submission of samples, with deleterious effects on passive surveillance for disease. Diagnostic laboratories offer scientific expertise, technical support, and quality control to veterinarians and producers. Let us not dilute the quality of specialization now available in the veterinary profession.

A society that has little appreciation of the immense contributions of veterinary medicine, including the discipline of pathology, to the nation's welfare may not consider our efforts to be worthy of public financial support. Members of the CAVP-ACPV hope that this letter has outlined some of the problems that diagnosticians are currently facing. It is difficult to respond to accusations by critics that such concerns are merely symptoms of self-interest and job preservation; however, events of the past few years have convinced us that we are approaching a time of crisis in the diagnostic sector. If our laboratory system is dismantled or fragmented, it is unlikely that it will be replaced by a better structure.

As veterinary professionals, we need to work together to preserve the quality of diagnostic services that support clinical medicine, public health, and the livestock industry in Canada. Members of the CAVP-ACPV look to the CVMA executive and all veterinarians to present our concerns at the various industry and regulatory meetings in which they participate. We need to begin this process now. Certainly, if we do not speak out, no one else will do it for us.

Executive Committee, Canadian Association of Veterinary Pathologists, 4840 Wascana Parkway, Regina, Saskatchewan S45 OB1.

Com Vet J Volume 38, February 1997.

JOBLINE

WANTED!!!
VETERINARY PATHOLOGIST

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

The present team includes three specialist veterinary pathologists, all with North American training and 15 technicians. The laboratory currently receives 30,000 cases annually and supplies a full range of diagnostic services to small and large animal veterinarians and two zoological gardens.

Essential to the position is a veterinary degree suitable for NZ registration and post graduate training and expertise in anatomical pathology with at least Board eligibility. Preference will be given to those applicants certified with the American College of Veterinary Pathologists or those planning on certifying this September. The position involves substantial client interaction, so excellent communication skills are essential.

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

Salary is commensurate with qualifications and experience, starting at NZ $64,000.

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

The Laboratory Manager
Alpha Scientific Ltd
141 Ellis Street
Hamilton, New Zealand
Fax 64-7-8462346 Ph 64-7-8462266
Email ghoggard@alpha-scientific.co.nz

DIAGNOSTIC HISTOPATHOLOGIST REQUIRED

We are now looking for another Diagnostic Pathologist to join us here at Abbey Veterinary Services. Experience is essential. We are a friendly and flexible working environment. Research interests and CPD are encouraged. We are situated in a lovely part of the country, close to the sea and also under the shadow of Dartmoor. For further details contact Trevor Whitbread - phone number 01626 53598, fax number 01626 335135.

An outline of Abbey Veterinary Services is contained in the report from United Kingdom by Genovese.

Abbey Veterinary Services
10 Oak Place
Newton Abbot
Devon, TQ12 2HW
United Kingdom

VETERINARY PATHOLOGIST

Qualifications: A degree in Veterinary Science registrable in South Australia.

Experience and competence in diagnostic histopathology (both companion and farm animals) and clinical pathology. Good communication skills and a focus on service delivery to VPS clients.

Salary: From $45,000 pa according to experience.

Duties: The appointee will work under the direction of the Chief Pathologist and participate in the routine roster for diagnostic necropsy, histopathology and clinical pathology. This roster will be shared with two Specialist Anatomical Pathologists and a Clinical Pathologist providing an excellent learning environment. Opportunity and encouragement to prepare for ACVSc Fellowship examination will be given.

Applications and requests for further information relating to either position should be directed to:

Dr Robert Rahaley
Chief Pathologist
Veterinary Pathology Services
PO Box 445
Glenside SA 5056

Phone: 08 8372 3700
Fax: 08 8372 3777
Email: rahaley@vetpath.com.au

VETERINARY ANATOMIC OR CLINICAL PATHOLOGIST

A full time position exists at our Sydney laboratory for an experienced pathologist able to support our surgical pathology case load and to assist with routine interpretation of haematological and biochemical data.

VPS is a dynamic company committed to providing the highest possible level of pathology support to veterinarians in private practice, industry, university and other areas. VPS is Australia's largest private veterinary pathology practice employing 10 Pathologists or Clinical

Pathologists and currently serving over 1200 veterinary practices in all states and in some overseas countries.

We are looking for a person who:

- shares our commitment to excellence
- can interact positively with our clients
- is prepared to work hard for suitable reward, and
- can continue the expansion of our Sydney based services.

Post graduate qualifications suitable for registration as a veterinary specialist are preferred. Ideally the appointee will have skills in both anatomic and clinical pathology, but VPS is able to provide some training in clinical pathology to a motivated surgical pathologist.

The level of appointment (Associate or Specialist) and remuneration will be commensurate with qualifications and experience. The salary is negotiable from $60,000 (Senior Resident) plus a 5% site allowance of working in Sydney. Other benefits of the position include: superannuation, 4 weeks annual leave, one week conference leave pa and sick leave.

Further information is available from:

Dr Jim Sutherland
Veterinary Pathology Services
PO Box 62
North Ryde NSW 2113

Phone: (02) 9805 0944
(02) 9805 0629
