



Australian Society for Veterinary Pathology
Brought to you by:
University of Melbourne
Veterinary Clinical Centre
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Publication No. PP544059/00003

EDITOR: Karl Harrigan

Number 42

September, 1995

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DEADLINE FOR NEXT VET. PATH REPORT IS NOVEMBER 30, 1995

SECRETARIAT

PO Box 114 Walkerville SA 5081

Phone: 08 3446337 (Pat Bosence) Fax: 08 3449227

ASVP EXECUTIVE 1995-1997

| | | | |
|--------------------------|-----------------|---|-------------|
| President | Ron Slocombe | University of Melbourne, School of Veterinary Science Parkville Vic 3052 | 03 3447369 |
| Secretary | Karl Harrigan | University of Melbourne, Veterinary Clinical Centre Werribee Vic 3030 | 03 97428270 |
| Treasurer | Mark Williamson | Australian Animal Health Laboratory, Ryrie Street Geelong Vic 3220 | 052 275000 |
| Committee Members | Ian Jerrett | 70 Croydon Road Surrey Hills Vic 3127 | 03 5589695 |
| | Alison Havadjia | Centaur International PO Box 1284 Bairnsdale Vic 3875 | 051 520800 |

APPOINTMENTS

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

CONVENOR - SLIDE OF THE MONTH

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

STATE REPRESENTATIVES

| | | |
|---------------------------|--|------------|
| Queensland | Bruce Hill, Rockhampton Vet Lab, QDPI Box 6014 Rockhampton MC Q 4702 | 079 360211 |
| Victoria | Malcolm Lancaster, Dept. Agric. PO Box 388, Benalla Vic 3672 | 057 611100 |
| South Australia | Ruth Reuter, VPS, PO Box 96, Plympton SA 5038 | 08 3623544 |
| New South Wales | Paul Gill, RVL Wollongbar 2480 | 066 240298 |
| Western Australia | David Forshaw, Regional Office, WA Dept Ag, Albany 6330 | |
| Northern Territory | Anton Janmaat, PO Box 79, Berrimah 0820 | 089 895511 |
| Tasmania | Barry Munday, Uni Tasmania, PO Box 1214 Launceston 7250 | 003 243812 |

1.

MINUTES

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY INC.

**ANNUAL GENERAL MEETING
VICTORIAN INSTITUTE OF ANIMAL SCIENCE, ATTWOOD**

MAY 20 1995

Meeting opened 3:25 pm.

APOLOGIES:

G Campbell, T Rothwell, P Phillips, R Webb, R Reuter, R Rahaley, K Harrigan.
Acceptance moved Rothwell, seconded Trueman

MINUTES OF 1994 AGM:

Published in Vet Path Report Number 39, July 1994 and tabled at the meeting.
Acceptance moved Gill, seconded Links

BUSINESS ARISING FROM THE 1994 MINUTES:

- Library requests for ASVP Conference Proceedings; Badham Library, University of Sydney has been included on the mailing list, requirements of other libraries not yet determined.
- NRDAP; matters covered in annual report, tabled and following in VPR.
- NATA registration; secretary has only recently contacted NATA and is awaiting a reply.
- ASVP/AVA affiliation; the motion was put to a postal vote that the ASVP pursue the affiliation with the AVA and the motion was defeated by 37 votes to 33

CORRESPONDENCE:

Routine correspondence was handled by the executive and through the VPR and tabled. Acceptance of inward correspondence moved Ross, seconded Links.
Acceptance of outward correspondence moved Trueman, seconded Samuel.

BUSINESS ARISING FROM CORRESPONDENCE:

- * ASVP/NZSVCP joint conference for 1996 in New Zealand; A facsimile transmission was received from Assoc. Prof. Alley on behalf of the New Zealand Society for Veterinary and Comparative Pathology inviting the ASVP to participate in a joint conference in Christchurch, New Zealand in June 1996 in association with the joint AVA/NZVA conference. Alastair Johnstone from Batchelar Lab reiterated the invitation and spoke briefly on the matter. There was some concern from the public sector members about getting approval to travel overseas. Moved Links, seconded Ross that **“The ASVP combine with the NZSVCP in a joint conference prior to the AVA/NZVA conference in Christchurch, New Zealand in 1996”** - motion carried.
- * VPR by Email: The possibility of sending out VPR by Email was raised by David Forshaw in correspondence. It was the general feeling of the meeting that not all members had access to a user friendly Email system and the VPR should continue publication in its current form.

2.

- * Decline in animal health services in Tasmania; Letter received from David Obendorf expressing concerns about the decline in the provision of animal health services by the Tasmanian government, particularly in the Mt. Pleasant lab. Moved Walker, seconded Ross that "**Matters raised in the letter of D. Obendorf be brought before Tasmanian Members of the ASVP and the incoming Executive act on their information to pursue the matter with the Executive Officer and Minister of the Tasmanian DPIF with the support of the Tasmanian Grazier and Farmers Association.**" motion carried. Moved Ross, seconded Giesecke that "**The executive of the ASVP bring to the attention of animal industries representatives of NAHC the current deficiencies in veterinary surveillance services in Australia,**" motion carried. Moved Glastonbury, seconded Walker that "**Incoming executive explore the possibility of developing a strategy for an animal health laboratory system in Australia and promote it to NAHC.**" motion defeated. Moved Mackie, seconded Links that "**The ASVP Executive formulate a brief (2 page max.) discussion paper altering producer organisations in Australia of deficiencies arising in Australian veterinary laboratories and promoting the role of vet pathologist in correcting these deficiencies.**" motion carried.

ANNUAL REPORTS

- Presidents Report-Tabled.
- Secretaries /VPR editors Report-Tabled.
- Treasurers Report-Tabled.

Acceptance of reports, moved Glastonbury, seconded Ross

COMMITTEE REPORTS

- Registry Committee Report was tabled. The four recommendations were accepted by the meeting. Moved Ross, seconded Seward that "**Providing the equipment is portable that the ASVP allocate up to \$2000 for purchasing a camera and transparency copier**" motion carried.
- Training Committee was tabled and Robin Giesecke spoke to the meeting. Recommendations were deferred to general business after the appointment of a coordinator.

ELECTION OF OFFICER BEARERS

The following people were nominated and elected unopposed.

President - Professor Ron Slocombe

Secretary/VPR Editor - Dr Karl Harrigan

Treasurer - Dr Mark Williamson

Committee - Alison Havadjia, Ian Jerrett

Registry Chairperson - Tony Ross

Training Coordinator - Vacant

State Representatives - Bruce Hill (QLD), Paul Gill (NSW), Malcolm Lancaster (VIC), Ruth Reuter (SA), Anton Janmaat (NT), David Foreshaw (WA), Barry Munday (TAS).

3.

GENERAL BUSINESS

- Training Committee report recommendations were adopted by the meeting. Moved Ross, seconded Links that "**The training committee members actively pursue a coordinator for the committee**" motion carried.
- Vote of thanks to Bill Hartley; A vote of thanks was moved and accepted by the meeting expressing the Society's thanks and well wishes to Bill for his contributions to the ASVP and veterinary pathology in general and his tireless efforts in the establishment and operations of both the domestic animal and comparative pathology registries.

Meeting closed 5:30pm

OUTSTANDING SUBSCRIPTIONS 1994 & 1995

ALLISON JF
FRASER G
LAMONT D
ROGERS RJ
STEWARD DJ

CONDRON R
GODWIN J
MORRISON J
SEAWRIGHT AA

CREEPER J
HUMPHREY J
REPPAS G
SMITS B

OUTSTANDING SUBSCRIPTIONS 1995 ONLY

BADCOE LM
BOULTON JC
CHOOI KF
FORSHAW D
GLAZEBROOK JS
HARPER P
HOOPER PT
JOHNSTONE AC
McCOLL K
MORTON JG
OBENDORF DL
ROBINSON WF
SEILER RJ
SLOCOMBE RF
TOWNSEND W
WATSON J

BEGG A
CHARLES JA
DUFF BC
FOSTER RA
GLEESON LJ
HARRIGON KE
HOWLETT CR
KELLY R
MELVILLE L
NORTON J
REDDACLIFF GL
SAMUEL J
SEWARD D
STRAUBE E
TRUEMAN KF
WATT DA

BELFORD C
CHICK B
FINNIE JW
GILL J
HANDLINGER JH
HASCHEK HOCK WM
JERRETT I
McCAUSLAND IP
MITCHELL PJ
NUNN MJ
RIFFKIN GG
SEARSON J
SIMS LD
TAYLOR JD
VANSELOW BA

PRESIDENT'S REPORT 1994-95

The 1995 AGM will bring to a close the current executive's term in office. In reflection, we have represented you at a time of unprecedented change in the delivery of animal health services in this country. During this time the Australian Society for Veterinary Pathology has continued to promote veterinary pathology and the skills and views of its members. Your Executive has tried to maintain a positive attitude toward change. We have been accepting of "change for the better", however, on many occasions the impetus for change has been political expediency, economic irrationalism or worse "change for change's sake" without evaluation. We cannot, as a society dedicated to the advancement of veterinary pathology, be accepting of change that erodes our values.

The National Animal Health Council (NAHC), formed by the SCARM Review (Miller Committee) has been given the task of ensuring Australia has a capable animal health service system. It is clear the future activities of the NAHC will impact on the ASVP and its members and a continuing lobby role for future Executives will exist.

The recurrent debate on affiliation with the AVA will finally be settled by the result of the recent postal vote of members. Irrespective of the outcome I would ask all members to be accepting of the result and move forward to promote the aims of the group, be it as the ASVP or as a SIG of the AVA.

I admit to a degree of relief on viewing the latest "Dangerous Goods Regulations" which now include an allocation of risk groups in its classification of infectious substances, with risk group I (low individual and community risk) not being subject to the regulations. This outcome must provide a degree of satisfaction to all the members who worked so hard on this issue and none more so than Geoff Mitchell.

The registry and training committee reports each have recommendations submitted for consideration by the members at the AGM. I encourage members to actively participate in any debate on the recommendations because they are an important platform for the future direction of our continuing education. I wish to record my sincere gratitude to the members of both committees for their unselfish dedication and commitment to the membership.

In closing, I would like to thank my fellow executive members, each State Correspondent and all the membership who have been so supportive over the last two years.

John Gibson
Honorary President

SECRETARY'S REPORT

1. SECRETARIAT ARRANGEMENTS

The Secretariat arrangements are working very well, I would like to thank Pat Bosence of the AVA office, South Australia for her help throughout the year. Members are reminded that all subscriptions and membership matters such as change of address are handled through the Secretariat.

ASVP Secretariat
PO Box 114
WALKERVILLE SA 5081

Phone: (08) 344 6337

Fax: (08) 344 9227

2. MEMBERSHIP

As of 31 March 1995, there are 160 members of the ASVP. The following are new members and are welcomed to the ASVP.

L M Genovese
R A Laing
D Middleton

R J Norman
D Williamson

Reminder notices for 1995 will be posted with the conference proceedings. Will all members please pay their annual subscriptions to the Secretariat at the above address and if mail is going astray also notify Pat of your change of address.

3. VETERINARY PATHOLOGY REPORT

Although the Vet. Path. Report has not been produced as frequently as in the past I think we have managed to keep up with the important information. I would like to thank the respective State representatives for their help over the past two years and wish the incoming Editor good luck. I would like to finally remind the members that the Vet. Path. Report is what you make of it and hopefully the success of the past will continue into the future.

J D Taylor

HONORARY SECRETARY

TREASURER'S REPORT FOR ASVP FOR 1994

This report is based on bank statements issued up to close of business on 1.5.95. The Society's ledger is available at the AGM.

Statement of Income and Expenditure of ASVP for 1994

INCOME

| | |
|---------------------|------------|
| Opening Balance | \$9480.51 |
| Petty Cash in Hand | 48.25 |
| Subscriptions | 4280.00 |
| Conference Receipts | 5440.00 |
| Interest | 314.06 |
| Miscellaneous | 111.41 |
| | \$19674.23 |

EXPENDITURE

| | |
|--------------------------------------|------------|
| Conference Costs | \$5702.45 |
| VPR/Secretariat Fees | 4193.46 |
| Government Duties, Taxes & Bank Fees | 164.65 |
| Miscellaneous | 100.80 |
| Petty Cash | 20.45 |
| | \$10181.81 |
| Petty Cash in Hand | 27.80 |
| Closing Balance | 9464.62 |
| | \$19674.23 |

Conference Participants 1994-43

Grant Campbell
Honorary Treasurer

**MODULAR TRAINING
AND CONTINUING PROFESSIONAL DEVELOPMENT
FOR VETERINARY PATHOLOGISTS IN AUSTRALIA**

REPORT TO

**ANNUAL GENERAL MEETING
AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY
19 MAY 1995**

At the 1994 AGM a series of strategies for developing modular training was accepted and \$2000 allocated to undertaking a training needs analysis. This was to provide more objective information for selecting modules, re-examining needs for an Australian (training) program, and determining possible forms of delivery of training modules. The survey was to be complementary to that undertaken with the Pathobiology Chapter of the Australian College in 1990.

The development of the survey and its subsequent analysis has been the major activity of the Training Committee in the past year.

To ensure representation and input by all those with a likely interest in an Australian program the training committee was also enlarged, as one of the strategies accepted at the AGM. The Training Committee currently consists of:

Robin Giesecke, Coordinator,
John Glastonbury (Government laboratories, Australian College of Veterinary Scientists)
Ian Links (Government laboratories)
John Mackie (formerly Government laboratories)
Rob Rahaley (private pathology services, Australian College of Veterinary Scientists)
Roger Kelly (Universities)

In the development of the survey assistance was also sought from the ASVP Executive. State representatives and the National Registry of Domestic Animal Pathology (NRDAP).

The survey involved direct mailing of 167 ASVP members on the mailing list of December 1994, including those in New Zealand, Canada, USA, Great Britain, Asia and Papua New Guinea; 45 laboratory and line managers, 22 policy makers or administrators seen to be relevant to operation of pathology services (including members of SCAHLS) and 19 managers of overseas organisations or pathology departments employing Australian members.

Considering that colleagues in New Zealand could also be interested in the concept of a modular training program aimed at Fellowship of the Australian College information on the proposed survey was sent to the executive of the New Zealand Society of Comparative and Veterinary Pathology with an invitation to be involved. This was not taken up and only ASVP members working in New Zealand, and their managers, were included in the final survey.

A full analysis of the survey results will be presented to the ASVP executive. A précis of the findings and recommendations based on them are reproduced in this report for the information of the meeting and consideration of the recommendations. The full report will be available from the Executive.

The Training Committee thanks all who gave their time to this survey.

MODULAR TRAINING AND CONTINUING PROFESSIONAL DEVELOPMENT

SURVEY OF TRAINING NEEDS OF PATHOLOGISTS AND MANAGERS 1995

SUMMARY OF FINDINGS

Eighty one (48.5%) OF 167 **pathologists** responded.

The majority of these: -

- 30 (37%) are employed in State Government laboratories
- 37 (45.6%) graduated up to 25 years ago
- 50 (61.7%) hold an Australian post graduate degree
- 29 (35.8%) hold an overseas qualification in pathology
- 27 (33.3%) hold College membership
- 7(8.6%) hold College Fellowship (71.4% of these in the Pathobiology Chapter)

Current preferred means for continuing education:

The majority, 23 (29.8%) currently give first preference to the discipline conference (i.e. ASVP meeting) for providing relevant continuing education .Only 5 (6.4%) prefer Fellowship preparation. Networking fails within the top five preferences of 51 (82.2%), library and journal use of 43 (69.3%), use of registry material of 26 (41.9%).

Incentive and assistance to undertake further study:

The greatest incentive for the majority, 39 (55.7%), to undertake further study aimed at gaining recognised specialist skills is job satisfaction, followed by recognised specialist qualification for 16 (22.8%). Time release was the assistance the majority 20 (32.5%) required.

Additional training required:

Molecular biology & histopathology are the first preference for 9 (12.6%), ultrastructure pathology and cytology each for 7(11.6%). Microbiology & immunology are included in the top five preferences. Additional training on pathology of companion animals is required by 12(21, 4%), of beef cattle, other avians (ostrich & emu) and fish each by 7 (12.5%).

Support for an Australian program:

Thirty six (44.4%) of pathologists expressed definite support for an Australian program in preference to an Australian post graduate degree or an overseas pathology qualification if it were developed within three years. Six (7.4%) gave qualified support. Thirty nine (48.1%) did not support the concept on the basis of age, other commitments, sufficient high qualifications, access to Fellowship training and unknown employer acceptance. Thirty (58.8%) were personally prepared to pay \$ 2000 toward a training program.

Resources for training:

In terms of resources which could be put into training:

Twenty six (40.6%) have recognised specialist skills in anatomical pathology, 17 (26.5%)in histopathology, 7 (10.9%) in immunology, 7(10.9%) in microbiology, 5(7.8%) in toxicology, 4 (5.1%) in molecular biology and 2 (3.1%) in clinical pathology.

Fifty (65%) have access to computer hardware capable of running multimedia programs. Forty two(53.8 %) have skills in using multiple computer programs and 24 (30.7%) have skills in using multimedia.

9.

Nineteen (283 %) of **Australian managers** responded, with collective views given by University departments and one major private pathology provider. Input was received from State and Federal Government laboratories, three state departments of Primary Industries, five Universities, one private pathology provider and the Registry of Comparative Animal Pathology.

Current preferences for ensuring continuing education of pathologists:

Discipline conferences are the first choice for 5 (27.7%) with only 4 (22.2%) including Fellowship preparation within their first five preferences. Fellowship preparation has least acceptance by government managers and most by University managers.

Qualification for specialists in veterinary pathology:

The choice for an equal number, 7 (38.8%) of managers fell on preparation for the Diploma of the American College of Veterinary Pathologists and Fellowship of the Australian College (Pathobiology Chapter). Government managers preferred the American qualification, followed by an Australian Ph.D. University and private pathology managers preferred the Fellowship of the Australian College (Pathobiology Chapter).

Disciplines in which it is seen advantageous for pathologists to have specialist knowledge and registration:

The clear first choice was anatomical pathology for 10(55.5%) managers; histopathology for 2(11.1%) and exotic disease diagnosis for 2(11.1%). For government managers the choice was anatomical pathology, for university manager's clinical pathology, for private managers, anatomical and clinical pathology.

Disciplines in which pathologists are required to have most competence:

The clear first choice was anatomical pathology and histopathology each for 8 (44.4%) managers. Microbiology, clinical biochemistry, parasitology and exotic disease diagnosis were included within the first five preferences.

Species in which further expertise needed:

Overall, more managers believed additional pathology expertise should be developed for beef cattle, then dairy cattle, fish, aquaculture species and poultry, followed by ostrich and emu, camelids, sheep, pigs and companion animals. For government managers the highest preference was for beef cattle. Universities managers did not rank their preferences.

Support for an Australian program approved as suitable preparation for Fellowship training:

There was extremely high support for such a program within three years, with 18 (94.7%) of managers supportive. Support was relevant to gaining specialist registration, training in disciplines other than anatomical pathology , being equivalent in status to the American College Diploma., providing a uniform standard of Fellowship training and not requiring staff to relocate.

Assistance for pathologists to gain additional competence:

Six (35.2%) preferred time release, 3 (7.6%) time release and financial assistance, 3 (7.6%) financial assistance. The first preference of government managers was a combination of time release and financial assistance; of university manager's time release and of private manager's financial assistance

MODULAR TRAINING AND CONTINUING PROFESSIONAL DEVELOPMENT

SURVEY CONCLUSIONS

The survey confirmed the following:

* Respondents represented a core of graduates most of whom already had high qualifications and more than 15 years post graduate experience. The majority were employed in state government laboratories. A minority had less than 15 years experience.

* Fellowship training was not the first choice for pathologists - only five giving it first preference for continuing education. However, an equal number of managers now accepted Fellowship and the Diploma of the American College as preferred qualifications for specialisation in veterinary pathology.

* There was very high support by managers for an Australian program aimed at Fellowship preparation and a lower level of support from pathologists. Only 36 pathologists expressed interest in an Australian program, 6 more dependent on the program standards and employer acceptance.

* Pathologists identified a need for further training at a species level, particularly for beef cattle, fish, aquaculture species and companion animals, and at the discipline level in molecular biology, histopathology, cytology and microbiology. Managers identified a need for further training on the same species but required pathologists to be most competent in anatomical pathology and histopathology and less in microbiology and exotic disease diagnosis.

* To undertake further study pathologists required time release and job satisfaction. The majority of managers preferred time release as assistance. A personal input of \$2000 was acceptable to the majority of pathologists.

* Resources for further training were to be found in the specialist skills held by pathologists and in material held in laboratories. Universities expressed interest in being actively involved in an Australian training program.

Both surveys have confirmed interest in an Australian training program, with Fellowship of the Australian College and specialist registration a goal. However, a need for training to cover changes in the client base of laboratories and the rise of new animal industries was identified in the later survey.

To accommodate these needs the following recommendations are made:

11.

**MODULAR TRAINING AND CONTINUING PROFESSIONAL
DEVELOPMENT**

RECOMMENDATIONS

#1. PLAN a curriculum of modules within three years to provide training in species and disciplines identified by the survey and bearing in mind the need for long term planning toward Fellowship with standards and delivery systems which will satisfy the Australian College requirements for supervised training at an approved centre.

#2. DEVELOP standards and examination procedures based on those applied by the American College and approved by the Australian College for membership and Fellowship. Consider a scheme whereby merit points are awarded for completion of modules and accumulated toward

- * (possible future need for re-registration).
- * Australian College membership,
- * Australian College fellowship.

Module scores could be based on complexity and degree of preparation required for its completion.

#3. DEVELOP a delivery system to provide flexibility, frequent interaction with supervisors and relevance to workplace requirements. Consider the role of information technology, university residencies or distance learning programs, short term postings to specialist laboratories, use of Registry resources, videoconferencing and the ASVP Conference.

#4. OBTAIN ACCEPTANCE and approval of the final proposal from:

- Major government and private pathology service providers
- ASVP members
- The Australian College of Veterinary Scientists
- The AVA Advisory Committee on Registration of Veterinary Specialists
- The Veterinary Boards Conference
- The Standing Committee on Animal Health Laboratory Standards.

#5. SEEK FUNDING from relevant industry groups, Department of Employment Education and Training etc, based on the proposed and accepted training program.

#6. CALL FOR TENDERS to provide the training program or part thereof. Consider Universities, registered specialists, the Registries.

#7. ESTABLISH a management group to coordinate module writing, trialing, review, supervision and examination processes and fees.

#8. CALL FOR ENROLMENTS in Australia and New Zealand in 1997 for the first modules to be undertaken in 1998.

Robin Giesecke
Coordinator, Training Committee

/4/95

**MODULAR TRAINING AND CONTINUING PROFESSIONAL
DEVELOPMENT**

EXPENSES

| | | | |
|---|------------------------------|--------|---------------------|
| SECRETARIAT EXPENSES: | | \$ | |
| Labour (Pat Boscence, 14 hours) | | 224.00 | |
| Copying (2,500 X 10c) | | 250.00 | |
| Labels | | 87.00 | |
| Postage (letters, drafts, questionnaires) | | 254.75 | |
| | | | <hr/> 815.75 |
| COORDINATOR'S EXPENSES: | | | |
| <u>Telephone</u> | | \$ | |
| 13/9/94 | Adelaide | 0.25 | |
| 13/9/94 | WaggaWagga | 11.60 | |
| 13/9/94 | Toowoomba | 20.31 | |
| 14/9/94 | Adelaide | 0.25 | |
| 14/9/94 | Benalla | 1.61 | |
| 14/9/94 | Benalla | 0.54 | |
| 14/9/94 | Benalla | 5.75 | |
| 16/9/94 | Brisbane | 11.46 | |
| 13/10/94 | Palmerston N (ex Wellington) | 11.00 | |
| 18/1/95 | Toowoomba | 1.08 | |
| 24/1/95 | Melbourne | 1.14 | |
| 27/2/95 | Toowoomba | 1.56 | |
| 1/95-4/95 | Adelaide (11 @ 25c) | 2.75 | |
| | | | <hr/> 69.30 |
| <u>Postage:</u> | | | |
| 11/4/94 | letter to McKenzie | 0.45 | |
| 24/4/95 | Draft to training c'tee | 4.95 | |
| | | | <hr/> 5.40 |
| <u>Copying:</u> | | | |
| 14/9/94 | 1990 survey results | 0.35 | |
| 23/4/95 | 190 @ 5c | 9.50 | |
| | | | <hr/> 9.85 |
| <u>Purchases:</u> | | | |
| 12/3/95 | 1 ream copy paper | 6.00 | |
| | | | <hr/> 6.00 |
| TOTAL EXPENDITURE | | | <hr/> 906.30 |

Coordinator's Labour approximately 106 hours

REPORT OF NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY TO ASVP AGM 1995 by Rod Reece, Registrar

MISSION STATEMENT To provide cost-effective continuing professional education and development relevant for veterinary pathologists working in the animal health industry of Australia, and to assist in maintaining and improving professional standards of veterinary pathology by providing a national registry and a quality assurance service.

HISTORY The NRDAP was established in 1990 with Bill Hartley as the first registrar (part-time). Bill retired in mid 1992 and Rod Reece was appointed (part-time) in September 1992.

FUNDING The NRDAP is funded by contributions from each of the state Departments of Agriculture or equivalents, Centaur, and several Federal bodies (Bureau of Rural Resources, AQIS, DPIE and CSIRO) coordinated through the Animal Health Committee's, Standing Committee on Animal Health Laboratory Services (SCAHLs). This provides a half-time salary, funds for travel and accommodation to run courses in each of the states on an annual basis, plus some funds for processing sections, film etc. Funds were obtained on a biannual basis in 1990-92, 1992-94, and for four years 1994-98 (to 31.01.99).

MANAGEMENT The NRDAP is managed by a committee appointed by the members of the ASVP at AGM, namely Tony Ross, Rod Reece, Keith Walker and John Glastonbury.

ACTIVITIES

CONTINUING PROFESSIONAL EDUCATION AND DEVELOPMENT

TRAINING COURSES. Tailored and structured refresher and up-date courses on veterinary pathology are delivered into each state on an annual basis. These usually last 2-4 days (but could be extended to 5) and are based on particular themes of organ or system, species and/or particular disease. In addition, small groups and individual tuition can be coordinated to coincide with these training courses. Histopathology training courses were held in Vic (attendees: Agric 3, Centaur 8, CSIRO 1, Univ 2), Qld (5), NT (3), WA (7 Agric, 4 University) and SA (4) during 1994: Tas was not in a position to have a course; NSW had two courses in 1993 (Mar & Dec) and another will be organised soon. The expertise of other providers of veterinary pathology services such as Universities have been used in some of these courses. The theme for 1994 was bone pathology and skin pathology - lectures were also given on radiology of bone tumours (Dr Becky Lonsdale, Perth), human bone disorders (Dr Prue Allan, Melb) and metabolic bone disorders (Prof Ken Jubb, Melb). Cases were also presented by the participants, and in some states a review of avian liver and kidney pathology was given.

Training courses will be offered again in 1995. The themes have not yet been finalised. Please contact Rod with requests and ideas.

SECOND OPINION SERVICE. A free second opinion service is available and has been utilised by many of the ASVP members. Replies to inquiries have been as detailed as possible, and I have endeavoured to also supply copies of relevant articles or descriptions. In addition, examples of particular diseases and/or organs were loaned from the Registry for comparative study. Several cases were received each week. **ACVSc PATHOLOGY EXAMS.** Material in the registry has been used by members preparing for specialist examinations. The transparencies, histological slides, cytological preparations and EM prints used in previous exams have been deposited in the NRDAP, with the appropriate answers. Access to these can be readily obtained.

ASVP SLIDE of the MONTH. Rod is coordinator of the ASVP Slide of the Month which is distributed to all veterinary pathology laboratories in Australia and some colleagues in other countries. This has been running reasonably well for the last year although some contributors have been unable to deliver slides during the agreed month due to other commitments: eventually cases were distributed. I have tried to standardise the format to include pathological description and morphological diagnosis.

RESOURCES

Histological sections The registry has continued to grow and now contains histological sections of cases from cattle (933), sheep (506), pigs (270), goats (153), horses (301), deer (25), dogs (487), cats (179), poultry (557) and other species (85); a total of 3496 cases as of 01.05.95. There are relatively few blocks!

Transparencies. Despite requests for clinical, gross and histological transparencies of common and unusual conditions only RVL Wollongbar, Dave Obendorf and Peter Harper have so far supplied material. Their interest and co-operation is appreciated.

FURTHER DONATIONS ARE NEEDED

Cost of copying transparencies ranges from \$1 to \$3 through normal channels but many institutes will not allow their transparencies to leave the premises. A portable transparency copier would be useful so slides could be copied at various laboratories and universities. The unit currently at EMAI produces reasonable copies but is not very robust nor readily portable. A SLR ZOOM copier to attach to a camera cost \$300 plus \$30 for fittings, but light source control can be a problem. The NRDAP does not have a camera: cost of a NIKON F-801 is \$1200 for body and \$600 for ordinary lens (tax-free).

Computer. The case records are indexed on cards and transcribed to a computer program in PARADOX. Information is retrievable by species, submitter, organ or tissue, pathology and aetiology. Refunding for the next 4 years will provide stability to enable investigation of better computing and retrieval systems.

NEW VISUAL DISPLAY TECHNOLOGIES

The 1994 ASVP AGM requested Rod to investigate and report on possibilities of some of the new visual display technologies (VET PATH REP Mar'95 p25):

LASER VIDEODISC. The NRDAP has a copy of the 6th edition, 1993 NOAH'S ARK laser videodisc from the USA (University of Georgia), which has approximately 25,000 transparencies of gross and histopathological conditions from many species around the world plus an interactive interrogation program. To utilise this we would need \$2,575 (tax-free) to purchase a Pioneer LD-V4300 player. It would be useful for teaching and training and would give access to material not readily available in Australia.

PHOTO.CD Up to 10,000 transparencies can be stored on one PHOTO.CD at one resolution (at a cost of \$1 ea). A suitable program such as Corel Draw needs to be installed on a computer with a CD reader to retrieve the images and these are of good quality. Kodak PHOTO.CD is slightly more expensive (about \$1.50 ea) but has multiple copies of each image at different resolutions so less transparencies can be stored per disc. This will only give an image, no text, so it has limited uses by itself but would be useful in conjunction with written notes. The standard programs cannot sort by text as there is no text with the figures just a sequentially numbered index. The images do not fade and are retrievable without damage. The originals can be stored safely elsewhere. Each viewer would require their own system. Hard copy text would have to be prepared and supplied separately. Separate training sets could be prepared by organ or species e.g. diseases of sheep.

INTERACTIVE CD Suitable player units should be available readily in Australia shortly - these will cost over \$1,000. A master interactive CD disc would cost \$25,000 for a 30 min program!!! This would give a series of figures, questions and answers, and other descriptive text and reviews as required. We would need to supply the transparencies, text, questions and answers, and pathways to be followed. The cost is in the programming and making the master - copies can be made for a few dollars each. Interactive CDs are currently used for Divers licence tests and some pharmaceutical company PR type displays.

FLOPPY DISC It is possible to have transparencies copied onto floppy disc (3.5in) for use with an ordinary computer at \$1 per slide (100 per HD disc). The quality of the image is reasonable but not as good as with PHOTO.CD. The package includes an indexing text with the figure so it can be retrieved and sorted by text fields - unlike the PHOTO.CD. There is a split screen of Figure with Text (sorting codes, case signalment, lesions etc) underneath. It may have some value in distribution of case material but questions

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and answers, interactive text etc would still have to be hard copy. Each user would need to purchase the program which runs it if we were to distribute courses in such a manner.

AFIP TRAINING SETS can be obtained but we would have to undertake to replace any sets which were lost or damaged. Many of the sets are valued at more than \$1-2,000 and the registry does not have funds to cover such losses. Each unit cost \$US75 to borrow within USA for handling - we would have to pay more. These sets cover a wide range of species, systems and diseases, and comprise histological slides, transparencies and notes. It is a very extensive collection. The AFIP produce a Handbook of Animal Models of Human Disease - more than 400 reviews from Am J Pathol and Comp Pathol Bull. (\$US275 + P&P for the complete set). AFIP coordinate distribution of slides contributed by various groups around the world - several Australian groups are involved but vacancies have to arise before other groups such as NRDAP could be added. They are currently using CD-ROM for their Atlases of Tumor Pathology (human).

CL DAVIS FOUNDATION FOR THE ADVANCEMENT OF VETERINARY AND COMPARATIVE PATHOLOGY. To have ready access to their material we would have to establish an Australian division of CL Davis, at an annual cost of \$US50 per person or \$250 for an institute. We would then distribute their newsletters with an Australian section. This would not replace ASVP but run parallel to it. CLD has more than 50 videos of lectures given by notable colleagues with transparencies used during the lectures dubbed into the footage as appropriate - these can be purchased (\$US55 + P&P). They have approximately 40 different study sets each comprising 100 histological sections, 100 transparencies, 1-2 illustrated lectures and self contained cross indexed catalogue arranged as an auto-tutorial. These are rotated on a monthly basis through designated study sites. We would need to pay postage as well as membership fees.

On a personal note. Rod has submitted his PhD thesis "Studies on infectious stunting syndrome of poultry" to Bristol University; "An Atlas of Avian Histopathology" by Randall CJ & Reece RL has been submitted to Mosby Books; and "Tumors of Unknown Aetiology" for Diseases of Poultry, 10th ed by Calnek et al, and "Infectious Stunting Syndrome" for Poultry Diseases, 3rd ed by Jordan & Pattison have been completed: these were done predominately out of hours. He has obtained permanent part-time work with NSW Agriculture as Veterinary Research Officer (Poultry) based at EMAI which complements the part-time NRDAP position.

Rod Reece is available most mornings

Ph 046 293333 or (direct) 046 293361; Fax 046 293400.

POSTAL ADDRESS: NRDAP EMAI PMB 8 Camden NSW 2570.

RECOMMENDATIONS OF THE MANAGEMENT COMMITTEE

1. Purchase a laserdisc player with the \$3000 allocated to Registry at the 1994 AGM, subject to successful viewing of images by the Committee.
2. Postpone obtaining and circulation of AFIP study sets until funds are available to cover losses.
3. Shelve the concept of forming an Australian Division of CLD at this stage.
4. Look at the feasibility of purchasing a camera and transparency copier to gain access to material from throughout Australia.

VICTORIA - Malcolm Lancaster

CENTAUR BAIRNSDALE

Mixed intestinal parasitism in weaner sheep - Alison Havadjia

Twelve to fifteen month old mixed crossbred weaners died from a mob of 107. They had been vaccinated, drenched (levamisole) and given a selenium injection at approximately one month of age. The deaths had occurred since that time. No scouring was noticed, but the animals appeared weak and uncoordinated prior to death.

Two animals were submitted live for autopsy. They were mildly depressed and weak, but could stand and walk when prompted. Routine ante-mortem biochemistry revealed mild to moderate azotaemia, hyperphosphataemia, hyperglobulinaemia and CPK elevations. Copper, selenium and cobalt levels were all more than adequate.

Significant gross findings were limited to a lack of body fat, pelleted faeces and mottled, enlarged kidneys in one animal.

Faecal examination revealed high strongyle egg counts (780 and 1 260 epg), high coccidial oocyst counts (300 000 and 45 000 opg) and large numbers of cryptosporidial oocysts on modified Ziehl Neelsen stained smears.

Histopathology of the intestine showed moderate villous stunting, blunting and some fusion; an increased cellularity of the lamina propria (predominantly eosinophils and lymphocytes); and scattered dilated crypts containing necrotic cells and debris. Focal areas of small and large intestine had numerous coccidial forms (predominantly macrogamonts) within enterocytes. Variable numbers of Cryptosporidia were present along the mucosal surface of the majority of the small intestinal sections examined, being particularly numerous in the ileum.

Microscopic findings in other organs were mostly mild and non specific, although there was a moderate nephrosis in the sheep with the grossly enlarged kidneys.

This case was interesting because of the variety of intestinal parasites involved and the fact that despite this, the sheep showed no diarrhoea or hypoproteinaemia, although significant intestinal pathology was present. Cryptosporidiosis appears relatively uncommon in sheep.

Nodular cutaneous xanthoma in a cat - Jacqui Tarrant

SIGNALMENT: Ten year old, castrated male Burmese cat.

CLINICAL FINDINGS: Multiple white plaques on the palpable conjunctiva. The lesions had been present in this location for approximately two years and were non-pruritic.

SAMPLES SUBMITTED: Surgical, wedge biopsies of the conjunctival lesions.

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HISTOPATHOLOGY: The epithelium is extensively eroded and covered by a layer of necrotic neutrophils with occasional intraepithelial pustules. A thick, lichenoid band of lymphoplasmacytic cells and a lesser number of neutrophils extends down in a diffuse pattern in the submucosa. The most striking features are nodular to coalescing granulomas consisting of epithelioid type macrophages and many giant cells situated superficially in the Sambucus. The nodules are focused upon adipocytes, extracellular lipid and large clefts. Both Gomori Grocott Silver stain and Ziehl Neelsen stains were negative.

MORPHOLOGIC DIAGNOSES: SUBMUCOSAL, CHRONIC, NODULAR, GRANULOMATOUS INFLAMMATION.

DIAGNOSIS: FELINE, NODULAR CUTANEOUS XANTHOMA.

COMMENTS: Considering the well demarcated, histiocytic granulomas of the lesion, the primary differential diagnosis was Feline Leprosy. A diffuse lepromatous reaction characterised by a predominance of epithelioid macrophages, a few giant cells and scattered lymphocytes and plasma cells may be encountered in this disease. A Ziehl Neelsen stain would have been expected to reveal acid fast bacilli (*Mycobacterium Lepramurium*) and was negative in this case. Nodular granulomatous inflammation in response to fungal infection was also investigated and ruled out following a negative GMS stain.

Extracellular lipid and clefts that formed the focus of the nodules were considered to be the inciting element of the reaction. Although the typical, foamy macrophages (“xanthoma” cells) were uncommon, histopathology was consistent with a diagnosis of nodular cutaneous xanthoma.

Financial constraints did not allow for investigation of a dyslipoproteinaemia. Of interest is the human condition, Xanthelasma, where lesions are restricted to the eyelids and blood cholesterol levels are ostensibly normal.

QUEENSLAND - Bruce Hill

UNIVERSITY OF QUEENSLAND, DEPARTMENT OF VETERINARY PATHOLOGY

Adenomatous hyperplasia in the placenta of a mare- Janine Barrett

A specimen from the placenta of a grey Arabian mare which had aborted 9 weeks prior to term was submitted. The attending veterinarian noted that the placenta was "very engorged" with "numerous small abscesses".

Sections showed that the white plaques seen grossly corresponded to glandular structures in the allantois supported by an abundant fibrovascular stroma. The cells of the glands were low cuboidal to columnar with vesiculated nuclei. The lumina contained small amounts of proteinaceous and cellular debris. The overlying chorion was mildly inflamed. A diagnosis of adenomatous hyperplasia was made. No significant pathogen was isolated from bacterial culture.

The aetiology of the hyperplasia is unknown as is the extent to which these adenomatous plaques contribute to abortion. Adenomatous hyperplasia of the equine placenta has most frequently been reported in association with other chronic placental lesions including placentitis and placental oedema.

References:

Hong CB, Donahue JM et al: Adenomatous hyperplasia of equine allantois epithelium. *Vet Pathol* **30**:171-175, 1993

McEntee M, Brown Y, McEntee K: Adenomatous dysplasia of the equine allantois. *Vet Pathol* **25**:387-389, 1988

VETERINARY PATHOLOGY SERVICES, BRISBANE

Ostrich fading syndrome- John Mackie

The term ostrich fading syndrome(OFS) has been coined to describe a syndrome of ill thrift and mortality in juvenile ostriches which was a major problem in several Australian states in the first few months of 1995(Anonymous 1995; Button 1995). Whether OFS is a single disease or more than one disease remains undetermined.

Sections of small intestine were reviewed from 17 ostrich chicks which died between October 1994 and March 1995. These chicks were all from one large breeding establishment which suffered a high mortality in young ostriches commencing August 1994. The peak mortality exceeded 90%. There was no contact at any time with chicks which were imported from Cocas Island Quarantine Station in January 1995.

The majority of chicks which died were approximately 4-12 weeks of age. Some suffered a variable period of anorexia, malaise and ill thrift before death and diarrhoea was noted in some chicks. Necropsy findings included intense congestion of the intestinal mucosa and the presence of excessive fluid and mucoid material in the lumen of the small intestine and caecum. Occasional chicks also had coexisting lesions such as hepatic necrosis, ulcerative lesions in the upper respiratory tract or upper gastrointestinal tract, airsacculitis or proventricular stasis.

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Microscopically, the small intestinal lamina propria of all 17 birds was congested and hypercellular with an infiltrate consisting mainly of lymphocytes, plasma cells and macrophages. Haemosiderophages were also evident in the lamina propria of 12/17 birds, predominantly in the villi. Additional changes included subepithelial proteinaceous effusion in the villi (3/17), epithelial hyperplasia (6/17), an apparent reduction in crypt density (5/17), the presence of dilated crypts containing cell debris (3/17), villous atrophy (1/17) and small foci of ulceration (3/17). Post mortem autolysis often precluded critical histologic evaluation, especially of the villi.

These histologic changes are similar to those associated with OFS by other investigators (Button 1995, Marc Kabay, personal communication) and suggest that OFS was present in Australia before 1995.

References:

Anonymous (1995) Australian Veterinary Association News, July 1995, p5
 Button, K (1995) Proceedings of the 5th Australian Ostrich Association Conference, p105

Two unusual cases of leukaemia - Geoff Mitchell

Case 1: 10 year old female Golden Retriever.

The animal was noticed “off colour” but showing minimal specific clinical signs.

| HAEMATOLOGY | | | REFERENCE |
|---------------------------|----|---------------------|-----------|
| | | | RANGE |
| | | | CANINE |
| RBCx10 ¹² /L | | 4.4 | 5.5-8.5 |
| Hb g/L | | 118 | 120-180 |
| PCV L/L | | 0.34 | 0.37-0.55 |
| MCV fl | | 76 | 60-77 |
| MCH pg/L | | 26.3 | 19-25 |
| MCHC L/L | | 345 | 300-360 |
| WCCx10 ⁹ /L | | 164.0 | 6.0-17.0 |
| %nRBC | | 0 | 0 |
| DIFFERENTIAL | % | x10 ⁹ /L | |
| BANDS | 2 | 3.3 | 0-0.2 |
| NEUTS | 32 | 52.5 | 4.0-12.0 |
| LYMPH | 55 | 90.2 | 0.9-5.0 |
| MONOS | 9 | 14.8 | 0.1-0.6 |
| EOSIN | 1 | 1.6 | 0.1-0.5 |
| BASO | 1 | 1.6 | 0-0.2 |
| OTHER | 0 | 0.0 | |
| PLASMA PROT | | 90 | 46-60 |
| g/L | | | |
| FIBRINOGEN g/L | | | 2-4 |
| RETICSx10 ⁹ /L | | 18 | <80 |
| PLATELETS | | clumped | |
| PLASMA | | | |
| 1+ | | | |
| anisocytosis | | | |

In Giemsa stained blood films, abnormal mononuclear cells showed multilobed (cerebriform) or bilobed (reniform) nuclei, multiple large nucleoli, variable pale blue cytoplasm and a variable number of small azurophilic cytoplasmic granules. Neutrophils were increased and there was a mild left shift. Toxic change and neutrophil precursors such as myelocytes or promyelocytes were absent. Initial screening tests showed that the abnormal cells were esterase and myeloperoxidase negative. In contrast, neutrophils were

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myeloperoxidase positive and monocytes were esterase positive. This result strongly suggests that the abnormal cells were lymphoid in origin. VPS has access to specific canine lymphocyte markers. The abnormal cells were positive for the following markers: CD45RA(common leucocyte antigen); CD3, TCR $\alpha\beta$, CD8 $\alpha\beta$ (T-cells) and α D (β -2 integrin, large granular lymphocytes[LGL]). These results identify the cells as CDS positive cytotoxic T-cells, subgroup large granular lymphocytes. Further investigation showed that in addition to leukaemia, the dog was exhibiting splenic enlargement due to widespread infiltration of the spleen by a similar population of large granular lymphocytes.

The large granular lymphocytosis is an infrequently recognised clinical entity affecting middle aged and older large breed dogs with a female preponderance(female:male-1.9) . Many cases have a benign clinical course with survival from up to three years without progression. Those with leukaemia and/or tissue infiltration (as in this case) often respond well to therapy with prednisone and leukeran but leukaemia and clinical signs may recur if treatment is ceased.

Initial investigation of this case by another laboratory (not VPS) resulted in a diagnosis of a severe leukamoid response, probably associated with pyometra. A laparotomy and ovariohysterectomy was performed based on this suspicion. There was neither evidence of pyometra nor any evidence of any other significant suppurative inflammation likely to lead to a leukemoid response. Neutrophils in the same smears were strongly positive for CD11 b & c (β 2 intergrins) and strongly positive for CD4. The abnormal cells were negative for CD11 b & c and CD4 thus removing any possibility that the abnormal cell population is derived from neutrophils.

Case 2: Three year old female koala

The animal was showing a generalised lymphadenopathy.

| HAEMATOLOGY | | REFERENCE RANGE | KOALA |
|------------------------|----|---------------------|-----------|
| RBCx10 ⁹ /L | | 2.0 | 2.4-4 |
| Hb g/L | | 68 | 85-134 |
| PCV L/L | | 0.20 | 0.25-0.40 |
| MCV fl | | 100 | 90-115 |
| MCH pg/L | | 34-0 | 35-45 |
| MCHC L/L | | 340 | 290-330 |
| WCCx10 ⁹ /L | | 53.7 | 4-8 |
| %nRBC | | 30 | |
| DIFFERENTIAL | % | x10 ⁹ /L | |
| BANDS | 3 | 1.6 | 0-0.1 |
| NEUTS | 10 | 5.4 | 2-7 |
| LYMPH | 3 | 1.6 | 2-7 |
| MONOS | 82 | 44.0 | 0-0.5 |
| EOSIN | 0 | 0.0 | 0-0.5 |
| BASO | 2 | 1.1 | 0-0.1 |
| OTHER | 0 | 0.0 | |

On examination of the Giemsa stained smear the abnormal cells were provisionally classified as monocytes rather than lymphocytes. Examination of myeloperoxidase and esterase stains showed that the abnormal cells were esterase positive but myeloperoxidase negative. Cells which were clearly neutrophils morphologically were myeloperoxidase positive and esterase negative. Cells which were clearly lymphocytes morphologically were negative for both esterase and myeloperoxidase. These results lead to a presumptive diagnosis of a monocytic rather than lymphocytic leukaemia. Unfortunately, specific cell surface markers for koala leucocytes are not readily available so the case was unable to be investigated in greater depth. Currently there is interest and active research on the involvement of a Retrovirus with koala leukaemia/lymphosarcoma.

WESTERN AUSTRALIA - David Forshaw

Bovine Respiratory Syncytial Virus

Several stillbirths occurred in small herd of Friesian/Murray Grey cross cattle located approximately 100 km east of Perth. At the time of investigation 5 calves were stillborn. A 6th had lived for 24-48 hours and had suckled before dying.

Necropsy of that calf revealed yellow fluid in the tarsal joints and abdomen. There was pleural and pericardial emphysema and a fibrinous clot was present in the pericardial sac. The apical, cardiac and anterior diaphragmatic lung lobes were discoloured dark pink to red. Histological examination revealed widespread interstitial pneumonia and consolidation of alveoli with proteinaceous exudate, numerous macrophages, scant neutrophils and large numbers of multinucleated syncytial cells.

No significant bacterial isolates were obtained from culture of lung tissue. Viral culture proved negative and the results of an immunofluorescent test for Bovine Respiratory Syncytial Virus on a smear of lung tissues were inconclusive. However, serum from the calf's mother tested by CFT tax respiratory Syncytial virus antibody was positive (1:80). A titre of 1:20 was found in the serum of another cow in the herd and titres of $\leq 1:10$ were found in sera from 9 other cattle.

Bovine respiratory virus infection is one of the most important causes of calf pneumonia in America and Europe. It generally occurs in weaned calves although occasional outbreaks have been recorded in sucking calves and adults. In most cases it is the bronchi and bronchioles that are affected but alveolar involvement is occasionally seen. The literature does not discuss its occurrence in neonate calves.

Traumatic Pericarditis in a Dolphin (Tursiops sp)

A stingray barb was responsible for the death of a dolphin at a popular coastal tourist resort north of Perth.

The dolphin, known locally as "Holey Fin" because of a bullet sized hole in its dorsal fin, was one of the oldest (35 years) of a group of wild dolphins to regularly visit the resort. Over the last month or so it had become listless and lost condition. During that time it was handfed but failed to recover its health and was found dead in the shallows one morning.

Necropsy revealed a fibrosed penetrating tract through the right thoracic wall and severe right sided fibrino-purulent pleuritis and pericarditis. A whitish flat object identified as the barb from a stingray was lodged in the markedly thickened pericardium with its distal end projecting into the pleural cavity. Circumstantial evidence suggests the barb broke off the tail of the ray as it struck the dolphin and lodged in the thoracic wall. Over a period of time it worked itself through the wall and penetrated the anterior lobes of the lung before penetrating the pericardial sac and re-emerging.

This is the author's second experience with marine mammals that have died from the effects of a stingray strike. In the initial case an adult male sea lion died after eating all or portion of a stingray. At necropsy there was severe focal peritonitis with gastric adhesions and extensive pleuritis, haemothorax, haemopericardium and traumatic cardiac ventriculitis. The barb of the stingray was found lodged in the right ventricle not only impairing the function of the atrio-ventricular valve but had also penetrated the base of the heart, facilitated no doubt by ventricular contraction. The stomach of this animal contained a quantity of pieces of stingray and advice by marine experts suggested that the sea lion may have been following a fishing boat that was discarding the trimmings of its catch.

Here is an excerpt from a letter sent to a government wildlife officer after an account of the dolphin case was published in a newspaper;

"I was a shark fisherman for many years and it was very common to find stingray barbs inside sharks and in their gullets when gutting. Once I was surprised to find one deeply embedded in the heart of a shark without killing it. The edge of the wound had turned white like scar tissue so I assumed it had been there for a considerable period. In many others the barbs had passed through the gullet and lodged into the flesh from the inside."

Lupin and virginiamycin associated rumenitis.

Lupins have been thought of as a very safe grain to feed to ruminants. However in recent months there have been several cases of deaths in sheep and cattle in association with the feeding of lupins. In cases where the forestomachs have been examined there has been a severe multifocal, almost full depth epithelial necrosis frequently involving the reticulum and omasum as well. In all cases investigated by the Animal Health Laboratories, the animals have been fed either 100% or lupin cereal grain ration from self feeders or troughs. It was thought the animals may have died from ammonia toxicity since unlike cereal grains, lupins store their energy as polysaccharides rather than starch. However, we have not been able to demonstrate an increase in pH and there may be other factors such as the presence of ammonium ions in the rumen liquor or the buffering action of saliva, involved.

Virginiamycin added to cereal grain rations has been thought of as an effective method of preventing lactacidosis and rumenitis in lot fed sheep and cattle. The Animal Health Laboratories has recently investigated several cases where animals have died from severe rumenitis, reticulitis and omasitis after eating cereal based concentrates to which virginiamycin has been added to the premix. The reason for this apparent failure to protect is unclear but may be related to improper mixing of the premix into the ration or the antibiotic settling out with the fines in the feeder. Histopathologically the lesions are typical of what is seen in acidosis.

There are several aspects to both types of rumenitis which should be kept in mind.

- Virginiamycin sold as Stafac 20 is not registered for use as a feed additive for sheep and cattle. The possibility that animals may die from lactacidosis/rumenitis despite its presence in the ration renders veterinarians likely to litigation if they recommend its use.
- It is well worth examining the reticulum and omasum at necropsy as a matter of routine. We have found lesions in these organs even when lesions in the rumen were absent or difficult to find.
- Submit sections of all 3 organs for histopathological examination as a matter of routine. We have found severe and extensive microscopic lesions even when there were no macroscopic changes apparent.

Cleve Main
Department of Agriculture
Animal Health Laboratories, South Perth

Copper toxicity in sheep

Copper toxicity was diagnosed as the cause of death in one of a group of stud rams kept in a shed in preparation for show displays.

Necropsy revealed dark red lungs, unclotted blood in the pericardial sac, and extensive ecchymosis involving the subcutis, epicardium and dorsal visceral pleura. The cut surface of the liver had a nutmeg appearance.

Histopathological examination revealed severe acute centrilobular hepatic necrosis with mild to moderate centrilobular haemorrhage. In many lobules the degree of necrosis approached that of massive necrosis.

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Kupffer cells were enlarged, often containing tan pigment and frequently present in small groups in centrilobular and periportal zones. Hepatocytes were enlarged and occasionally contained mitotic figures, enlarged, bizarre or multilobed nuclei. Haemoglobin was present within kidney tubules and there was mild tubular epithelial necrosis.

The ram's kidney and liver copper concentrations were 533 mg/kg and 763 mg/kg respectively. Serum GLDH activities of 7/11. Other rams in the shed ranged from 43.2 U/L-261 U/L suggesting they were suffering from sub clinical copper intoxication.

The source of copper was found to be the ration premix which was formulated commercially after analysis of grain samples. Preparation of the ration was done by the farmer. The copper concentrations of 3 undried samples of ration were estimated to be 14, 19 and 32 mg/kg. It is suspected that faulty mixing technique was responsible for the problem.

Another case of death from copper toxicity was reported (90/900) following drenching of ewes with copper sulphate to control drench resistant internal parasites. The ewes were given about 3 g of copper sulphate and held overnight in a pen containing blackberry nightshade. Deaths were noted the following day. The nightshade had been eaten. Affected sheep had diarrhoea and salivated. A haemorrhagic gastro-enteritis was seen at necropsy. Peracute necrosis of periacinar hepatocytes was evident. Kidney copper concentration was 57 mg/kg. Ingestion of blackberry nightshade may have contributed to the gross pathology seen although death due to ingestion of the plant is uncommon.

Marc Kabay
Department of Agriculture
Animal Health Laboratories, South Perth

Abscesses in feedlot cattle.

Six cattle in a feedlot near Albany had reduced weight gain and developed large bilateral swellings over the thighs and diffuse swelling of the lower hind legs within six weeks of introduction. Three died. Two were shot. One animal was killed and necropsied. Only Simmental and Simmental cross animals were affected.

When examined, it was moribund with approx 40cm diameter swellings over both quadricepses. These swellings were abscesses located underneath the tensor fascia lata and containing the necrotic remnants of the vastus group. The abscesses also dissected caudally between the semimembraneous and semitendinous muscles and contained large volumes of foul smelling brownish liquid. There was a severe necrotising cellulitis along the plantar aspect of both hind legs extending from the hock to the hoof. A focal 5mm nodule was present on a chorda tendina (sp?) of the tricuspid valve.

We cultured a heavy pure growth of *Actinomyces pyogenes* from the abscesses on both legs. A chronic active inflammatory reaction was present in the abscess walls with large colonies of gram positive coccobacilli consistent with the culture result in the exudate, but no clue to the cause was found. The nodule in on the heart valve was a subacute endocarditis containing bacterial colonies. Apparent incidental lesions were mild acute unilateral retropharyngeal lymphadenitis and mild patchy acute renal tubular necrosis.

The owner said that many animals reacted to the anthelmintic they received when introduced into the feedlot and that the abscesses were only found in animals that did not fully recover from this. He described neurological signs. Investigations by a veterinarian from the drug company failed to replicate this effect with repeat doses of the same batch of anthelmintic.

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No obvious predisposing cause could be found. Some bolts were found protruding from both sides of the race at about the same height as the lesions, and trauma is a possibility but there is no explanation why this particular batch of animals was affected when many similar cattle have gone unaffected through the feedlot before. The race was used for 5:1 vaccination and anthelmintic and vitamin B12 injection but all needles were given in the neck. Two affected and four unaffected animals were negative for mucosal disease antibody and antigen. Comments and/or accounts of similar experiences would be welcomed.

David Forshaw
Department of Agriculture
Regional Veterinary Laboratory,
Albany

Cryptosporidiosis in Guinea Pigs

Several young guinea pigs in two boxes in a conventional research colony had diarrhoea and were losing weight. A necropsy on one animal demonstrated large numbers of *Cryptosporidium* on the mucosa of the small and large intestine. In the absence of other causes of diarrhoea, the *Cryptosporidium* were considered significant in this case. There is no evidence of immunosuppression in this case.

David Pass
Animal Resource Centre
Murdoch Drive,
Murdoch, WA

NEW SOUTH WALES - Paul Gill

REGIONAL VETERINARY LABORATORY WOLLONGBAR

Progressive spinal myelinopathy of Murray Grey cattle- Paul Gill

A 2 year old Murray Grey heifer became increasingly ataxic over 4 weeks. The animal had difficulty walking and tended to walk backwards into a dog sitting position. There were no significant lesions at necropsy. Histological examination found an extensive, moderate to marked loss of myelin with ballooning of myelin sheaths in the lateral and ventral funiculi at all levels of the spinal cord.

The signalment, clinical signs and histological lesions are consistent with progressive spinal myelinopathy of Murray Grey cattle (Richards and Edwards, 1986). The disease is inherited in an autosomal recessive mode (Edwards et al, 1988). The age of onset of clinical signs varies from birth to adulthood. Cases in cattle older than 1 year are rare and have not been reported in the literature. The pathogenesis of the progressive myelin loss is unknown; a defect in central nervous myelin is postulated. It may be worthwhile examining the spinal cord in all young Murray Grey cattle which are paretic or dystatic or ataxic.

References

Richards RB & Edwards JR (1986) Vet Pathol **23** : 35
Edwards JR, Richards RB, Carrick MJ (1988) Aust Vet J **65** : 108

Toxoplasmosis in a kangaroo- John Boulton

Toxoplasmosis was fortuitously diagnosed in a young, foster reared, Eastern Grey kangaroo. For several weeks, the animal had failed to thrive but its death had been unexpected. At necropsy, an imperforate duodenal ulcer was found. There was an associated localised peritonitis. There also were bilateral cataracts which were ascribed to early hand rearing on cow's milk. No other gross lesions were detected and only the duodenum was submitted for microscopic examination. The duodenal ulcer was well circumscribed by fibrous tissue and no specific pathogen was associated with the central necrosis. However, endothelium in all layers of the intestinal wall (both at and remote from the site of the ulcer) was densely colonised by thin walled cysts of loosely packed zoites, and many individual zoites were present in the vessel lumens. Findings indicate that the illthrift was due to duodenal ulceration of unspecified cause) but death was due to acute toxoplasmosis. Bilateral cataracts possibly also contributed to the illthrift.

REGIONAL VETERINARY LABORATORY ARMIDALE

Another wireworm in ostriches - Stephen Love

Wireworm, *Libyostrongylus douglassi*, is a small round worm (4.2-5.1mm) residing in the proventriculus and gizzard of ostriches. It is the agent of verminous gastritis(vrotmaag) in young birds. A larger wireworm *L. magnus*, has been found in ostriches and rheas in the Ukraine (Huchzermeyer 1994).

L. douglassi has been found in ostriches in Australia, presumably imported with ostriches from Africa in the late 19th century. RVL Armidale possibly was the first to discover the parasite in NSW, although it is reported that the parasite was found in a collection by one L. Herbert from a bird or birds at Temora in 1911. It should be noted that wireworm larvae can be cultured from samples with zero egg counts using conventional egg counting techniques(Barton and Seward 1993, Button et al 1993).

According to Horberg et al (1995), another *Libyostrongylus* sp. has appeared on the scene, this time in ostriches in North America. Part of their paper reads as follows: "*Libyostrongylus dentatus* sp. n. is described from ostriches on farms in North Carolina and Texas. Nematodes were recovered from the posterior proventriculus and under the koilin lining of the gizzard; the parasites occurred in mixed infections with *Libyostrongylus douglassii*."

The significance of the newly reported *Libyostrongylus* sp is uncertain, but ostrich farmers and veterinarians in Australia should be aware of the presence of this species and take appropriate measures to monitor and control the parasite as outlined by Button et al (1993).

References

- Barton NJ and Seward D (1993) Aust Vet J **70**:31-32
 Button C et al (1993) Aust Vet J **70**:76
 Hoberg EP et al (1995) J Parasitol **81**:85-93
 Huchzermeyer FW (1994) Ostrich Diseases. Agricultural Research Council, Onderstepoort Veterinary Institute, Republic of South Africa

UNIVERSITY OF SYDNEY, DEPARTMENT OF VETERINARY PATHOLOGY

Zoonosis enquiry (rat) - Malcolm France

The carcass of a pet rat was submitted by a physician whose 9 year old daughter had developed a mild illness with cervical lymphadenopathy a few days prior to the rat's death. Histological examination of the brain revealed numerous gliotic granulomas consistent with protozoan encephalitis. No zoites or cysts were seen on H&E sections, however Gram staining revealed plump ovoid Gram positive bodies occurring either in large numbers within intact cells or occasionally scattered in small numbers within granulomas. Similar organisms were seen in Gram stained sections of kidney. Other organs were normal.

The findings in this rat are virtually identical to those seen with *Encephalitozoon cuniculi* infection in rabbits. This protozoan is a common pathogen of rabbits but infection in rats, mice and guinea pigs can also occur. It is not generally regarded as a zoonosis. The cause of the child's illness was not determined, although recovery was uneventful.

Enteropathy with intracellular bacteria in mice- Malcolm France

Diarrhoea, sometimes containing blood, was seen in about half a dozen mature mice in a large colony of Skh:hr/hr (hairless) mice. On gross examination there was slight reddening of the gut serosa and the caecal and colonic contents were dark and pasty. Histological examination revealed villous atrophy, crypt hyperplasia and occasional small ulcers in the ileum, and mucosal hyperplasia and focal ulceration in the caecum and colon. Careful examination of the epithelium at these sites revealed groups of small rod shaped bacteria apparently within cytoplasmic vacuoles in the enterocytes. These organisms were found to be Gram negative, weakly agrophilic and stained dark blue with Giemsa.

These findings are similar to an enteropathy associated with an atypical *E. coli* described in a severely immunodeficient strain of mice. The mice in the present case, however, are regarded as immunocompetent and usually survive well in non-SPF conditions. *Citrobacter freundii* is regarded as a cause of proliferative typhlocolitis in mice, although it colonises the surface rather than cytoplasm of the enterocytes. The cause of the present condition and the role and identity of the intracellular bacteria is unknown.

REGIONAL VETERINARY LABORATORY WAGGA WAGGA

Neuraxonal dystrophy associated with sorghum grazing - Barbara Moloney

Case 1, March 1995

History: A herd of 130 cattle produced 70 normal calves from cows in the first 3 weeks of calving, and then over the next month, 30 of the 60 first calf heifers produced affected calves. The dams were a mix of black baldy and Hereford breeds, sired by an Angus bull. They had been grazing "Jumbo" sorghum prior to the commencement of calving.

Clinical signs: Affected calves were born with a severe neurological disorder characterised by opisthotonus, slow lateral nystagmus, and clonic seizures which when aroused would progress to tonic rigidity. There was no menace reflex, so presumably these calves were blind. They were unable to stand or suckle and thus did not survive. Some calves born in the following month were arthrogryptic.

Biochemistry:

Calves; elevated aspartate aminotransferase(191 U/L), elevated bile acids (43.1 uM/L), bilirubin(43.6 uM/L), indicating some hepatocellular dysfunction.

Dams; Glutathione peroxidase levels were within normal limits in samples of both affected and non-affected dams.

Histopathology: Severe cerebral oedema and severe axonopathy with many spheroids at the level of the midbrain and caudal brain stem. Some brains showed significant demyelination of the ventral tracts of the spinal cord.

Case 2, May -June 1995

History: A mob of 75 8-month-old crossbred lambs were grazing a regrowth of "Cowpow" hybrid sorghum, which had some 2 months previously been heavily grazed by other sheep without ill effect. Three of these lambs died and 17 others were affected.

Clinical signs: Blindness, deafness, dysfunction of prehension (but continuing to eat) and limited ability to drink. Ambulatory movements were normal but there was evidence of paresis when lambs tried to rise after being forced to sit down. Bleating was more common than normal. Partial recovery occurred in 3 animals with some gaining of sight and ability to eat and drink. The other affected animals resumed normal locomotion but remained totally blind and deaf.

Biochemistry: The sorghum sample was negative for cyanide on picric acid test. Glutathione peroxidase and copper levels were normal.

Virology: There was no evidence of pestivirus or Akabane involvement.

Histopathology: Moderate multifocal axonal dystrophy with spheroids situated in the proximal segments of axons in association with nuclei of the medulla oblongata, the cerebellar roof nuclei and ventral horns of the spinal grey matter, at the thoracolumbar junction. Mild degrees of gliosis and demyelination were observed in the nuclei of the medulla and associated with the ventral horns respectively. Bradley et al (1995) J Vet Diagn Invest 7:229-236 have recently described a similar axonopathy in newborn lambs born to ewes grazing hybrid sorghum pasture, and in the young lambs grazing the sorghum. The toxin contained in the sorghum has yet to be identified. The nature and distribution of the lesions is similar to that caused by lathyrogenic compounds in Lathyrus spp plants, and it has been demonstrated that sorghum plants can produce precursors of lathyrogenic compounds in vitro.

Embolic myocarditis - Barbara Moloney

A mob of 2000 Merino lambs, 4 to 8 weeks-old, experienced an outbreak of mortality, with 10 being found dead. Gross pathology showed pus filled lesions in the heart, valvular endocarditis and severe pericarditis. Histopathologically, there was severe, acute, multifocal, embolic suppurative interstitial myocarditis and severe, subacute fibrinopurulent pericarditis. Culture of the lesions yielded *Staphylococcus aureus*. Coccoid organisms were also visible in the lesions in H&E sections. Presumably, this severe infection may have followed a navel or marking wound infection.

Plant poisoning's - Barbara Moloney

The period from November 1994 to June 1995 with the ending of a severe long dry spell has provided a wide range of disorders related to plant ingestion.

Implicated plants include;

"Dunfield" field peas with liver function tests indicating hepatopathy,
 Hairy Panic grass (*Panicum effusum*) with crystal associated cholangiopathy,
 Lesser Loosetrife (*Lythrum hyssopifolia*) causing chronic interstitial renal fibrosis,
 Redroot Amaranth or pigweed (*Amarantiius retroflexus*) associated with acute toxic renal tubular necrosis, and various plants containing pyrrolizidine alkaloids causing degenerative hepatopathy and chronic hepatogenous copper poisoning.

Nitrate/nitrite poisoning reached a peak in June with several cases associated with grazing Capeweed (*Arctotheca calendula*). Others were associated with millet (*Echinochloa* sp), sorghum and lush oats.

Lucerne redgut caused deaths on 2 farms in November and on 3 farms in June, with mortalities ranging from 1 to 4%. In some cases where necropsy was performed at the laboratory, there was evidence of mesenteric torsion with severe gaseous distension, congestion and haemorrhage of the intestine involved.

NORTHERN TERRITORY - Anton Janmaat

Mycobacterium asiaticum Infection in a Red Kangaroo - David Pritchard

We circulated Case No. 94/2343 as one of the Slide of the Month cases for May 1995. Reiterating the history, an adult female red kangaroo in captivity in a Wildlife Park developed a large suppurating mass in the pouch over a period of about one month, thought to be associated with the mammary gland. Removed surgically, it was a roughly flattened sphere approximately 10cm x 7cm, completely encapsulated, with a discharging sinus. A small piece was submitted fresh to the Laboratory in a sterile container, the remainder in formol saline. A light growth of *Staphylococcus aureus* was isolated from the pus.

Histologically, the lining skin of the pouch over the mass had eroded totally and was replaced by a thick, moderately vascular collagenous capsule with a thin layer of dried exudate over its surface. The mass itself consisted of large multilocular pyogranulomas. Numerous plasma cells with fewer lymphocytes accumulated in the connective tissue bands dividing caseo-purulent centres, there were foci of epithelioid macrophages adjacent to the fibrous bands and a large number of multinucleated giant cells. A few acid-fast bacilli were detected in association with macrophages and giant cells.

Tissue homogenate was submitted to the Australian Reference Laboratory for Bovine Tuberculosis in Perth, WA, and an isolate was eventually identified as *M. asiaticum*.

The reference Laboratory also advised as follows:

"*M. asiaticum* is a rarely isolated pathogen. It was originally isolated from normal lymph nodes in monkeys from India and was not identified as *M. asiaticum* until further studies were performed on the organisms in a laboratory in Hungary in 1970.

M. asiaticum has been isolated from cases of pulmonary mycobacteriosis in man in Australia and the USA. It has also been identified as the probable cause of joint infection in the elbow of a patient in Australia."

Psittacine Beak and Feather Disease Virus (PBFDV) infection in a Sulphur-crested Cockatoo Helen Parkes and Richard Weir

A selection of plucked feathers that were short and "clubbed" were received from a young Sulphur-crested Cockatoo with feather growth abnormalities and feather loss. Histologically there were marked changes within the feather shafts. The epithelial layers were disorganised, with loss of normal architecture, cell necrosis with nuclear fragmentation, and some cells showing margination of chromatin and prominent dark nucleoli. Abundant giant cells were present in the epithelial lining of the shaft, as well as in the pulp. Macrophages and granulocytes were also frequent and two of the feathers had large numbers of plasma cells and lymphocytes in the pulp. Intracytoplasmic structures seen with H&E in occasional epithelial cells and macrophages, stained purple/red in a Feulgen reaction, indicating the presence of DNA and suggesting viral inclusion bodies. A sample of feather debris was examined with the electron microscope, after homogenizing the feathers in PBS and adsorbing the homogenate onto a copper grid. Clusters of very small, round particles about 20nm diameter were seen, consistent with PBFDV, which has icosahedral virions of this size, containing a single-stranded, circular DNA genome.

References:

D.A. Pass and R.A. Perry (1984) The pathology of psittacine beak and feather disease Aust Vet J **61** 3 : 69-74

M.J. Studdert (1993) Circoviridae: new viruses of pigs, parrots and chickens Aust Vet J **70** 4: 121-122

A Rooster with Heart Failure - Helen Parkes

An eight month old rooster with a one month history of having "lost his crow", pale comb, progressive weakness and death, showed signs of right-sided congestive heart failure on post mortem. There was a large amount of clear, yellow abdominal fluid, an enlarged, mottled liver with fibrin tags on the surface and the right ventricle was much distended. The valves and chambers of the heart otherwise appeared grossly normal. The airsacs were thickened and opaque and there were extensive adhesions. The lungs were enlarged, with yellow borders and yellow necrotic debris on the surface.

Histologically the liver was congested with dilated sinuses and compression of hepatocytes, progressing to cell loss. The kidney also showed venous congestion. The lung showed consolidation of whole lobules with extensive cellular exudate (abundant granulocytes, foamy macrophages, lymphocytes and plasma cells) and necrosis. Two small granulomas contained foci of Gram positive cocci. A coagulase negative Staph. was isolated from the lung, but not from other tissues or the abdominal fluid.

It was not determined whether the heart failure was the primary condition and the other changes were secondary to that, or whether there was an initial infectious or inflammatory process that contributed to the heart failure.

SOUTH AUSTRALIA - Ruth Reuter

VETLAB ADELAIDE

Multiple piloleiomyomas in a cat - John Finnie

A 12 year old Russian Blue cat presented with a nodular cutaneous mass (1.75 x 1.25 x 0.75cm) on the right side of the neck. The firm lesion had apparently grown rapidly over a 2 week period and was creamish-white in colour and slightly gritty when sliced.

Microscopically the non-encapsulated tumour was composed of whorling and interlacing bundles of spindle cells adjacent to and surrounding hair follicles in the dermis. The spindle cells were identified as smooth muscle by positive immunostaining for vimentin and alpha smooth muscle actin. The muscle fascicles were either solitary or interconnected and staining with Masson's trichrome and Van Geison techniques revealed an admixture of smooth muscles and fibroblastic elements. In the centre of the majority of leiomyomas were foci of osteoid metaplasia.

In cats and dogs dermal leiomyomas and leiomyosarcomas are extremely rare, usually solitary tumours arising from the arrector pili muscle (piloleiomyomas) and less commonly, muscles of veins (angioleiomyomas). In Man solitary leiomyomas are regarded as true neoplasms, but the multiple form may represent a malformation of smooth muscle or hamartoma. Piloleiomyomas in Man are usually benign but frequently become painful and tender as a consequence of muscular contractions.

VETERINARY PATHOLOGY SERVICES ADELAIDE

Lymphosarcoma in a Border Collie - Ruth Reuter

A three year old Border Collie with a short history of retching and inappetence was presented to a local veterinary clinic in April 1993. When the oral cavity was examined, the left tonsil was found to be grossly enlarged, approximately the size of a golf ball, causing partial obstruction of the oropharynx. The right tonsil appeared normal. The owner of the dog was not willing to have a biopsy of the left tonsil taken; however, the veterinary practitioner obtained a fine needle aspirate for cytology. A diagnosis of probable lymphosarcoma was made, and the dog treated with cytarabine and prednisolone by the practitioner. The lymphadenopathy reduced very rapidly (within 48 hours).

The owner was unhappy with the diagnosis and treatment, and subsequently consulted a second veterinarian. At this consultation the dog appeared bright and alert, with no evidence of lymphadenopathy. Review of the pathology slides by a second pathologist yielded a diagnosis of reactive lymphoid hyperplasia, and treatment of the animal was discontinued. Over the next two years the dog was relatively normal apart from several episodes of pruritic dermatitis which responded to antihistamines and antibiotics. In June 1995 the skin condition became unresponsive and was treated with corticosteroids. In July the animal developed a severe upper respiratory tract condition, with subsequent generalised lymphadenopathy. Biopsies of several lymph nodes confirmed a diagnosis of lymphosarcoma.

The case illustrates some of the difficulties in interpretation of cytology of fine needle aspirates of lymph nodes, particularly from the tonsil which is continually receiving antigenic stimulation, and the value of biopsy samples. Even biopsies of this organ can be difficult to interpret.

JOBLINE

Batchelar Animal Health Laboratory, Palmerston North, New Zealand is looking for a diagnostic pathologist interested in a twelve month "sabbatical-type" appointment while one of their pathologists undertakes a year's residency overseas.

The job requires a person with good skills, residency level or MACVS standard equivalent. At Batchelor lab they would share duties with 5 other pathologists. The lab processes about 25000 cases per year, about 50/50 companion/production animals.

To quote "This is a lovely part of the country to work in. Centrally situated (an Aussie would no doubt argue that in NZ you're not actually too far from anywhere else in the country), Palmerston North is arguably NZ's main agricultural research town. We have close but informal links with the vet school at Massey which is just a hop, step and jump away up the road."

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