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VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
Queensland Department of Primary Industries
Toowoomba Veterinary Laboratory
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DEADLINE FOR NEXT VET. PATH REPORT IS SEPTEMBER 30, 1994

SECRETARIAT

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ASVP EXECUTIVE 1991-1993

President	John Gibson	Toowoomba Vet. Lab, PO Box 102 Toowoomba Q 4350	076 314352
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Treasurer	Grant Campbell	Toowoomba Vet. Lab, PO Box 102 Toowoomba Q 4350	076 314410
Committee Members			
	Ross McKenzie	Yeerongpilly Vet. Lab, 665 Fairfield Road Yeerongpilly Q 4065	07 3629432
	Geoff Mitchell	VPS, PO Box 1119, Coorparoo DC Q 4151	
	Dick Sutton	Dept. Vet. Path. University of Queensland St Lucia Q 4067	07 3772565

APPOINTMENTS

Chairperson (Registry of Domestic Animal Pathology) Tony Ross

Newsletter Editor Jim Taylor

CONVENOR - SLIDE OF THE MONTH

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

STATE REPRESENTATIVES

Queensland	Greg Storie, Yeerongpilly Vet Lab, 465 Fairfield Road, Yeerongpilly a 4065	07 3629555
Victoria	Malcolm Lancaster, Dept. Agric. PO Box 388, Benalla Vic 3672	057 622933
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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 0828	089 895511
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EDITORIAL

This edition of the Veterinary Pathology Report contains articles on a number of issues of interest to the Membership. At the 1994 AGM in Canberra a draft position paper by Keith Walker and John Plant, concerning amalgamation with the AVA, was submitted. This is found on pages 15-17. It was decided to use this draft as a starting point for general discussion by the membership, published through the Vet Path Report, and a postal vote of all members be taken just prior to the next AGM.

Members are invited to read this draft and raise any points for discussion through letters in the next VPR. Please bear in mind this draft is only a starting point and the fine details are being negotiated by representatives of both the AVA and ASVP. This is a chance for all members to contribute to the discussion. **Any submissions to me by September 30, 1994.**

Another 4 years of funding for NRDAP from the Animal Health Committee is encouraging to see in these days of decreasing resources and the efforts of all those involved are to be commended.

Deb Seward the state reporter for Victoria is taking up a position in Tasmania and Malcolm Lancaster is taking over as State Reporter. I wish Deb all the best in her new position and thank her for her contributions. I look forward to Malcolm's reports, as other members will, particularly the progress of privatisation of the Victorian Labs.

This edition also contains reminder notices for 1994 subscriptions. These remain at $25.00. If you are unsure of your membership status please contact Pat at the secretariat.

I look forward to your contributions.

Jim Taylor
Editor VPR.

OUTSTANDING FOR 1993 - PAYMENT DUE $25.00 (within Australia) - $40 (overseas)

ACLAND HM - (O/S)	BELFORD C	BOULTON JC	FRASER G
LADDS PW	LEHARNE L	LINKS IJ	MARSHALL DJ - (OS)
McEWAN DR	McGAVIN MD -($20)	MUNTZ F	
STAPLES N	VANSELOW B		

OUTSTANDING 1992 & 1993 - PAYMENT DUE $45 00

BEERS P	CALLAHAN JT	CROSS GM	HINDMARSH M
HOWLETT CR	JOHNSTONE A	LATTER M	LATTER M
RIFFK1N GG	ROTHWELL TL	SMITH BL	

These outstanding subscriptions should be included in 1994 renewal enclosed and returned ASAP to PO Box 114, Walkerville 5081. Upon receipt of outstanding amounts a copy of the 1994 Annual Proceedings will be forwarded.

MINUTES
AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY INC.

ANNUAL GENERAL MEETING
COUNTRY COMFORT MOTOR INN, CANBERRA
5 MARCH 1994

Meeting opened 3.45pm.

APOLOGIES: F Trueman, R Rogers, P Ladds, R Sutton, G Mitchell, J Mackie, R Reuter, W Robinson, T Rothwell, R Slocombe.

Acceptance moved t Ross. Seconded B Hill.

MINUTES OF 1993 AGM

Published in August 1993 Vet. Path. Report and tabled.

Acceptance moved P Gill. Seconded R McKenzie.

ADDITIONAL AGENDA ITEMS

***** Update of the Miller Review - T Nicholls.

BUSINESS ARISING FROM 1993 MINUTES

***** Future of Government Veterinary Services.

- Letter to Jacob Malmo was published in the August 93 VPR.
- Letter to SCARM Review Committee was tabled, also to be published in the VPR.

***** Affiliation of the ASVP and AVA.

- A draft discussion paper compiled by Keith Walker and John Plant was tabled at the meeting.

Moved Glastonbury. Seconded Ross.

That (1) The draft discussion be published in the next VPR.
(2) For general discussion and consideration by the membership with member's views published in the subsequent VPR.
(3) After that time a postal vote be conducted to resolve the matter.

***** Iata Regulations.

- A report to the meeting from Geoff Mitchell - Veterinary Pathology Services was tabled for publication in the next VPR.

CORRESPONDENCE

Dealt with through VPR and Executive.

- A letter from Badham Library, University of Sydney, requesting a copy of Annual Conference Proceedings was tabled.

- Moved Ross. Seconded France that ASVP Annual Conference Proceedings be sent to all Australian Vet School Libraries and the Max Henry Library after ascertaining each library's requirements.

REPORTS

Presidents

Tabled. Acceptance. Moved McKenzie. Seconded Cook.

Treasurer's

Tabled. Acceptance. Moved Ross. Seconded Campbell.

Secretary/Membership

Tabled. Acceptance. Moved Seward. Seconded Glastonbury.

VPR Editor's

Tabled. Acceptance. Moved Begg. Seconded Gill.

NRDAP

- Tabled. Rod Reece requested more tissue blocks and transparencies. Tony Ross and Terry Nicholls urged all members to lobby state CVO's for continued funding of the registry.

- Moved Boulton. Seconded Giesecke that, at the ASVP's request, the registrar liaise with CL Davis foundation through W Haschek as to the availability of material and keep members abreast of its content.

- Moved Pritchard. Seconded Miller that the ASVP executive ask the registrar to make enquiries as to the cost of insuring materials from the CL Davis foundation.

- Moved Ross. Seconded France that the executive examine the versatility and use of CD-ROM in its possible application to NRDAP and be given discretion to spend up to $3000 on its possible implementation.

Training Committee

- Report tabled and deferred to general business for further discussion.

ELECTION OF OFFICE BEARERS AND APPOINTMENTS

President	John Gibson		
Secretary/Editor VPR	Jim Taylor		
Treasurer	Grant Campbell		
State Correspondents	Greg Storie	-	QLD
	Deb Seward	-	VIC
	Ruth Reuter	-	SA
	Paul Gill	-	NSW
	David Forshaw	-	WA
	Roy Mason	-	TAS
	Anton Janmaat	-	NT
Chairperson Registry	Tony Ross	-	
Coordinator Training Committee	Robin Giesecke		

GENERAL BUSINESS

***** ASVP Training Programme

- R Giesecke highlighted major issues in her report.
  - training needs analysis
  - composition of the training committee
  - module development and use
  - application to specialist registration requirements and continuing education

- Moved Rahaley. Seconded Glastonbury that the report is adopted and the training committee is given a budget of $2000 over the next 12 months to carry out a training needs analysis and cover costs incurred by the coordinator.

***** Tendering of Government Laboratory Services

- J Gibson spoke briefly about comments reported in the "Veterinarian".

***** Dangerous Goods Regulations

- Covered in business arising from minutes and G Mitchell's report.

* NATA registration

- A letter from NATA referred to the ASVP by the AVA was tabled. The letter promoted accreditation in veterinary laboratories and gave a general outline of their accreditation process.

- Moved Rahaley. Seconded Glastonbury that the ASVP write to NATA requesting specific information of accreditation of veterinary laboratories with particular reference to the cost and personnel involved.

Meeting Closed 5.55pm.

PRESIDENT'S REPORT 1993-94

Welcome to the 1994 ASVP Scientific Conference and Annual General Meeting. Following a positive response to last years venue, your Executive was keen to locate an affordable venue with similar facilities this year. I trust the members will be pleased with the Garden City Motor Inn, Canberra. I would like to thank Terry Nichols for local support in making all the preliminary arrangements for the conference.

The theme of the conference is 'neuropathology' and we are fortunate to have Dr Rick Le Couteur and Dr Peter Harper to lead the presentations. To date we have an excellent response to the call for papers and the conference is shaping as a great opportunity to refresh our memories in this area. To ward off the effects of bureaucratic dementia.

The ASVP exists because of the dedication and commitment of its members. I would like to thank the Executive Members, Jim Taylor and Russell Graydon and Committee members Ross McKenzie, Geoff Mitchell and Dick Sutton for their support this past nine months. Unfortunately Russell Graydon has resigned his position of Treasurer to take up a consultancy in India. We will be seeking to fill this position at the AGM.

The Veterinary Pathology Report continues to provide members with a national forum for comment and scientific reporting. Jim Taylor (Editor) and the State Representatives are to be commended for their efforts. To the members who regularly contribute cases of interest, keep up the good work.

Robin Giesecke (co-coordinator) and the Training Committee, despite adversity, have continued to pursue the issue of pathology training for the ultimate benefit of members. Your efforts are much appreciated. The stringent economic conditions of employment most of us find ourselves in do little to foster training aspirations, despite the political rhetoric of governments. However, if the ASVP is to have a role in training pathologists, then the requirements for training among veterinary pathologist and their employers needs to be assessed.

The National Register of Domestic Animal Pathology and the Comparative Animal Pathology Registry continues to develop under the dedicated stewardship of Rod Reece and Bill Hartley. It is important that members support the registries at every opportunity (see VPR Nov 1993). They are a valuable resource for our continuing education.

Several issues where addressed by the executive in the past nine months, all of which will be reported on at the AGM. Issues such as the SCARM Review, IATA regulations and re-affiliation with the AVA are all issues which will impact on our future. I would like to thank those members who took the time to provide written comment on various issues. The Executive is on a fairly steep learning curve and the advice of fellow members will be eagerly sort and received.

John Gibson
Honorary President

SECRETARY'S REPORT

1. SECRETARIAT ARRANGEMENTS

The Secretariat arrangements are working very well and proving to be cost effective for the Society. I would like to think 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 17 February 1994, there are 158 members of the ASVP. The following are new members and are welcomed to the ASVP.

I G Anderson	G W Campbell
J Creeper	D Lamont
J P McGrath	J Mills
G P Reppas	J Searson

Twenty-four members have not paid their subscriptions for 1993.

HM Acland	I J Gleeson	PM Lording	GG Riffkin
P Beers	M Hindmarsh	DJ Marshall	T Rothwell
C Belford	CR Howlett	DR McKewan	BL Smith
GM Gross	AC Johnstone	F Muntz	P Staples
G Fraser	M Latter	R Peet	W Townsend
PR Giesecke	L Leharne	AW Philbey	KF Trueman

If our records do not match your own, please contact Pat at the Secretariat at the above address.

3. VETERINARY PATHOLOGY REPORT

I have kept VPR in the same format as it seems to work well. Another report is due out about now and will hopefully be in the mail shortly. Thanks to all State representatives for your efforts for contributions throughout the year and thanks to the individual contributors. Veterinary Pathology Report is what you make of it.

I look forward to your contributions over the next year and appreciate any input from members as to the reports format.

JD Taylor
HONORARY SECRETARY

TREASURER'S REPORT FOR ASVP FOR 1993

Transfer of accounts to the new Executive proceeded smoothly.

Unfortunately, due to work commitments with his new job, Russell Graydon has had to resign his position as Honorary Treasurer with the ASVP and as he has been in India since late January. I have produced the Treasurer's report on his behalf.

With help from Edla Arzey, I have finally balanced the books. A summary Statement of Income and Expenditure appears below. The Society's ledger is available at the AGM.

Statement of Income and Expenditure of ASVP for 1993

INCOME	
Opening balance	$6724.30
Subscriptions	4405.00
Publications	35.00
Conference Receipts	6968.00
	$18132.30
EXPENDITURE	
Conference costs	$6360.80
VPR/Secretariat fees	2038.29
Member awards	150.00
Bank fees and taxes	62.73
Sundries	1.72
Petty Cash	48.25
	$8651.79
CLOSING BALANCE	$9480.51
	$18132.30

Conference participants 1993-96

JD Taylor
for R Graydon
Honorary Treasurer

REPORT TO AGM ASVP March 1994

NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY

Background: The NRDAP is funded via contributions from each of the State Departments of Agriculture, CSIRO and DPIE. It is housed at the RVL, EMAI (Camden, NSW). The Registrar, Rod Reece, is employed part-time: the rest of his time is spent at the Comparative Animal Pathology Registry which is funded by and housed at Taronga Zoo.

Achievements: During 1993, histopathology workshops were held in every state. The topics presented included avian histopathology (with an emphasis on the respiratory and musculoskeletal systems), pathology of the respiratory system in general and new and emerging diseases (including pathology of native species). The feedback received from most participants was that this format was satisfactory. I made a deliberate attempt to involve private pathologists and University Departments. Their presence and/or facilities were appreciated but this policy will need to be reassessed. Slides and notes were distributed, including a re-issue of the Proceedings of the 1987 Avian Histopathology Training Course, edited by G.M.Cross, Aust. Vet. Poultry Assoc.

Histology sections: The NRDAP contains many examples of common conditions but from time to time I will request sections of good examples of pathology to expand the collection. We also need blocks because sections break, fade or the mountant deteriorates. Please also supply paperwork so that the records are complete. The main deficiency in the present system is transparencies. If you have good examples of clinical, gross, histopatho1ogy or other transparencies of any species, any condition; please send copies, or send transparencies. We can examine and copy if required, and then return the originals to you.

A second opinion service still operates, and many of the sections I receive are examined with Bill Hartley.

Remember, that these workshops count as formal continuing professional training and education/ development (CPD) programmes in pathology. As such it may be the only relevant CPD which many pathologists receive on an annual basis. It complements the Annual Meeting of ASVP and the various Saturday sessions operating in some states.

The future: The histopathology workshops for 1994 are being organised. Some of the topics suggested are comparative immunopathology (fish through to mammals), general pathology of skin and skeletal systems, other aspects of avian pathology and more on diseases of native species.

The future of the NRDAP is dependent upon continuing funding from the sponsoring state and federal bodies. We are preparing a submission to the Animal Health Committee, meeting in May 1994 to obtain funds for 1994-96. WE would appreciate your support at all levels!

Thank you.

Rod Reece,
Registrar.

MODULAR TRAINING FOR VETERINARY PATHOLOGISTS

REPORT TO

ANNUAL GENERAL MEETING
AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGISTS
5 MARCH 1994

The Training Dilemma:

The cutbacks to government laboratory services which are continuing will soon render veterinary pathologists an endangered species! John Glastonbury reports that 'of the 20 pathologists currently employed in our regional laboratories (in NSW) 15 are over the age of 40.'

There is clearly a dilemma for those remaining in, and wishing to progress professionally in the discipline.

In this climate the training committee has not been very active in the past year, but in that time several issues have been brought more sharply into focus; namely -

> That there is little point in the Society becoming involved in the Fellowship training issue unless it has objective data on the value of such training to both the pathologists themselves and to those who employ (and fund) them.
>
> That any system set up by the Society should begin on a small scale and become self supporting as soon as possible.
>
> That if there are insufficient pathologists to undertake training for Fellowship 5-6 years hence a modular system of training would not be economically viable for ASVP to develop and it may be more viable for individual pathologists to negotiate the conditions and content of their training individually with the College.
>
> If a modular system of training is not a viable one at the present time further development of the NRDAP material and training programs should be considered

DRAFT STRATEGIES FOR IMPLEMENTATION OF MODULAR TRAINING:

At the Annual General Meeting in May 1993 the committee was asked to develop strategies for implementation of its recommendation. Strategies have been developed for discussion by this meeting, as follows:

R1. Hasten slowly until the value of a Fellowship to employment is known.

* Ascertain the attitude of employers to the training of pathologists, and to the employment of pathologists with Fellowship status for 5 years and 10 years hence, through questionnaire and discussion.

* Undertake a training needs analysis of currently employed pathologists to determine number seriously interested in progression to Fellowship level in the next 5, and 10, years.

R2. Use overseas study set usage to road-test module format and delivery:

* Through the Registrar, NRDAP, and users of the Charles Davis study set material determine usage, advantages and disadvantages encountered.

* Invite users of the study sets to comment on their practicability and applicability to the Australian scene.

R3. Discuss standard to be attained and training resources available.

* Invite a representative of the University pathology departments on to the training Committee.
* Discuss with each pathology department the resources (human and material) which they would be willing to commit.
* Assist the Registrar of the NRDAP to accumulate and catalogue Registry training material.
* Evaluate the infrastructure, delivery and assessment mechanisms of distance or flexible learning systems already developed.
* Invite a representative of the Pathobiology Chapter of the College on to the training committee.

R4. Include at least one member from each state on the training committee.

The current committee has a chairman, a coordinator (non-aligned), 4 members of government laboratories and 1 member of the private pathology service. These members come from NSW, Victoria and South Australia. Given the relative isolation of some states and the value of having ownership of the process it is suggested that the committee be reconstituted to include:

a chairman or coordinator (non-aligned)
a representative of the Universities
a representative of the government laboratories
a representative of the private laboratories
the Registrar of the NRDAP
a representative of ASVP executive
a representative of the College

With State representatives or their nominee to be corresponding members where a holder of any of the above positions could not also act as a corresponding member for the State and with Phil Ladds as marketing consultant, as required. This would provide a small core committee, representation from each of the key organisations involved and state representation.

R5. Conduct a Training Needs Analysis (TNA) of employed pathologists and their employers.

Without objective data on the need for Fellowship or other training, and an appreciation of any gap between current knowledge of pathologists and the expectations of their employers' progress on development of a modular system of training is seen as an unsound course for ASVP to follow in the current climate.

* It is proposed that the TNA would be conducted through a questionnaire for all employed pathologists and their regional or funding managers. Wayne Robinson has agreed to assist in the design of the questionnaire.

R6. Support the Pathobiology Chapter of the College.

* Invite a representative of the Chapter on to the training committee to facilitate communication.

R7. Have a curriculum and module(s) in place in five years if, supported by results of a Training Needs Analysis.

Strategies:

Year 1: Conduct and analyse results of the Training Needs Analysis.

Year 2: Identify resources available for development of modules.

Year 3: Determine priority areas to be developed as modules, on basis of TNA. Draft curriculum. Apply for funding for development,

Year 4: Evaluate curriculum and framework for study and assessment with senior pathologists.

Seek accreditation for modules and training program from the College
Apply for funding to employ pathologist(s) to develop module content
Invite pathologists to develop modules
Seek applicants to commence training in the following year.

Year 5: commence training on a small scale. Review frequently.

DISCUSSION ON UNIVERSITY OF QUEENSLAND RESOURCING:

One of the recommendations of the Training committee was to discuss resources available at Universities.

During a private visit to Queensland in September last year I held informal discussions with Wayne Robinson, Roger Kelly and Dick Sutton on training issues, generally; on their availability to assist or be a representative on the training committee and the value of having bodies like the NRDAP and the College on the Committee.

Senior pathologists who might be approached in the future to develop module contents were also discussed.

MY POSITION AS COORDINATOR OF THE TRAINING COMMITTEE:

On my return from last years AGM, I was offered a TSP along with other pathologists from VETLAB, and there being no likely employment as a veterinarian, a pathologist or a training officer within the Department for at least three years I accepted the package.

This had not been contemplated when I had accepted to remain the Coordinator of the Training Committee the AGM.

While I am willing to remain as coordinator if supported by the membership, communications, development and distribution of questionnaires etc will incur greater expense than if I were employed. I have sought clarification from the executive as to reimbursement for expenses incurred on behalf of ASVP.

In conclusion 1 would like to thank John Glastonbury, John Mackie and Ian Links for their constructive contributions to the debate on development of Fellowship training.

Robin Giesecke
Co-coordinator,
Training Committee

1 March 1994

REPORT TO ASVP RE "DANGEROUS GOODS REGULATIONS AND AIR TRANSPORT"

Geoff Mitchell - Veterinary Pathology Services Pty Ltd

After an initial flurry of activity, things have quietened down considerably. Both TNT and Security Express (SE) are still accepting "Labmailers" although strictly speaking these do not yet comply with IATA Packing Instruction (PI) 650 in that they have not passed internal pressure tests. TNT insists on the use of an outer 100mm cylinder, SE does not.

The current situation is that biological products (which do not include killed vaccines!) and diagnostic specimens (which do not include formalin fixed tissue!) are **not** classified as "Dangerous Goods" by the Civil Aviation Authority (CAA), International Air Transport Authority (IATA) or International Civil Aviation Organisation (ICAO). Thus CAA has **NO** legislative power to enforce any packing requirements on the carriers and/or consignors. However, the carriers are at liberty to require compliance with IATA PI's as a condition of carriage. This is what they do at present (e.g. No foam eskies).

The future of this situation is uncertain. CAA follows the ICAO (UN) lead. There are plans for the ICAO to introduce United Nations (UN) numbers for biological products and diagnostic specimens. These will fall into four as yet unspecified WHO risk categories. The affect of these risk categories on packaging and transport is as yet unknown. In the worst case scenario, the result could be the classification of all biological products and diagnostic specimens as "Dangerous Goods" resulting in huge increases in packaging and transport costs as previously detailed. These cost increases in packaging and transport costs were underestimated in my original report. Since then I have become aware that the courier companies levy a non-reducible "Dangerous Goods Surcharge", of between $35 and $50 depending on the company, and may also change for carriage at casual or full rates not at any prearranged contract rate. This would close to double my previous estimate for cost increases and I stress would effectively prevent **ANY** transport of specimens within Australia if people stick to the regulations. My personal feeling is that much of the transport will go "underground" - obviously this is a highly undesirable situation. There are already companies in Australia soliciting Veterinary Diagnostic Specimens (e.g. Spectrum) in plastic post bags - my information from the Post Office is that this contravenes current requirements.

My recommendations are:-

1. ASVP liaises urgently with the AVA to present a united case to the CAA.

2. We present our case separately from the medical pathology interests, as this area is highly clouded by emotion re diseases such as HIV.

3. We press CAA to accept the "Canadian Model" or a similar classification based on rational risk assessment of veterinary specimens. This would result in a short list of pathogens which are recognised as "Dangerous Goods" and must be shipped as such, with the vast majority of veterinary material being classified low risk.

4. We press CAA to involve veterinary "Experts" (hopefully ASVP members) in the process of rational risk assessment. This has four parts:

    a. the risk to humans (i.e. courier staff) from accidental exposure;
    b. the risk of accidental exposure of humans;
    c. the risk to animals (see point 5. below) from accidental exposure;
    d. the risk of accidental exposure of animals once a specimen is in the courier service. (Plane crashes in the middle of sheep paddock - sheep rush up to burning wreckage and lick up vast quantities of pathogens?)

5. We convince the AVA of the difference between "Infectious" and "Contagious" (e.g. Blue Tongue virus is infectious but is not contagious as it requires a vector for transmission) and the difference between contagious to humans and contagious to animals (e.g. a culture of equine *Strep. equi* - is neither contagious nor infectious for humans).

It is vital that this process of risk assessment be implemented **BEFORE** the potential changes to the Dangerous Goods regulations. Complacency now will result in a much harder task to change the regulations once introduced.

I stress that this problem could prevent much of the disease surveillance now carried out in Australia. This surveillance is already under threat from “economic rationalization”. Further moves in this direction will increasingly involve non-government organisations in the "front-line”. Anything less than free movement of diagnostic specimens will seriously impede both government and non-government laboratory functions.

I realise that this matter has been referred to SCAHLS, however, I seriously question whether a committee which only meets annually can respond rapidly enough to this situation.

DRAFT DISCUSSION PAPER ON THE POSSIBLE AMALGAMATION OF THE ASVP WITH THE AVA

1. Advantages and Disadvantages of Amalgamation

(i) Advantages for the AVA

- an increase in AVA membership, although more than 50% of ASVP members are already members of the AVA

- it will provide SIG expertise in a broad based discipline of anatomic and clinical pathology, as distinct from the species based SIG's.

- the AVA Conference will be made more attractive with better support for papers from the expanded membership with an anatomic or clinical pathological or epidemiological approach.

- it will provide a united lobby group in areas of mutual interest e.g. diagnostic laboratory services in Australia.

(ii) Disadvantages for the AVA

- some members (less than 50%) of the ASVP are not members of the AVA and may be reluctant to join.

(iii) Advantages for the ASVP

- ASVP members who are already AVA members will be better represented and able to influence and support policy discussion through group recognition with the AVA.

- better representation outside with the agro-politicians, important in the present day with the cutback in regional diagnostic laboratories.

- the ASVP will have structured access to a wider range of views

- there is a need for AVA support if anatomical or clinical pathology are to survive, especially in the government services and at universities.

- increased membership for the SIG from the non-specialist AVA membership.

- opportunity for more members to get support to attend conferences where there is a broader range of papers available.

- opportunity for AVA Executive support in areas of concern

- income to support better speakers at conferences.

(iv) Disadvantages for the ASVP

- the cost for new ASVP members who will be required to join the AVA concurrently.

- the significantly higher cost of registration and accommodation at the AVA Annual Conference, when the ASVP meeting is held concurrently with the AVA Conference.

- problems with non-members of the AVA who wish to continue as ASVP members.

- the cost of conference registration for speakers, if this policy is continued by the AVA

Comments

1. The ASVP would not automatically qualify for Council representation, until its membership reaches 5% of the AVA membership, but has the right to exist as a specialist group. There are provisions for Council representation in special circumstances.

2. The ASVP could continue to hold a "Special Interest" Conference before the AVA Conference as is done at present, but this would not allow the SIG to obtain the full benefit of AVA membership. At present, AVA does not give support to satellite conferences of SIG's held outside the Annual Conference.

3. The ASVP would have the opportunity to market the skills and expertise of its members at a 2 or 3 day session during the AVA Annual Conference, for which it would receive some income which could be used to defray speakers costs and registration.

Recommendations

1. The ASVP should become a Special Interest Group of the AVA.

2. That for the first three years, they should be allowed Council representation. I believe that this could be allowed under Article 71, which was amended at some stage to require 5% of AVA membership before they were entitled to Council representation, unless recognised by Council as having some special skills.

3. That after three years, they would be required to meet the 5% of members to be entitled to Council representation.

4. That there be an amnesty (suggested 3 years) on the veterinarians in ASVP who are not AVA members at the present time. New ASVP members will be required to be or concurrently become AVA members.

5. When the ASVP becomes an SIG, then all Executive office bearers of the ASVP will be required to be AVA members

6. The SIG by-laws should allow for non-veterinarians to be members of the SIG, as is the case with other SIG's.

These recommendations are put forward for consideration by the ASVP and the AVA. If there is agreement in principle, then the finer details should be left to a small sub-committee from the AVA Executive and the ASVP Executive to recommend the formation of a new SIG to Policy Council.

SOUTH AUSTRALIA - Ruth Reuter

VETERINARY PATHOLOGY SERVICES (ADELAIDE)

Haemolytic Anaemia in a Poodle - Ruth Reuter

A 5 year old female poodle was presented to a local clinic with a history of sudden onset of jaundice. The dog had vomited some bile the day before, but apparently showed no other signs of illness. The temperature on presentation was 39.7°C. There was no abdominal pain identified and no known access to toxic substances.

A blood sample submitted to the laboratory gave the following results;

		Reference range
Total bilirubin (umol/L)	599.4	0-8
Conjugated bilirubin (umol/L)	323.2	0-3
Alk Phos (U/L)	266.0	20-70
AST (U/L)	118.0	15-70
CK (U/L)	927.0	70-250

All other biochemical parameters were within normal ranges.

An EDTA sample was also submitted for CBC, however, attempts to make smears or do counts were thwarted by marked spontaneous agglutination of the erythrocytes. A diagnosis of autoimmune haemolytic anemia was suggested to the veterinarian. Institution of steroid treatment gave an immediate response, and the dog is recovering well.

Spontaneous agglutination of erythrocytes in the early stages of AIHA has been associated with formation of cold agglutinins (IgM). The agglutination may be visible grossly in the syringe or on a slide.

Oestrogen Toxicity in a Cocker Spaniel - Ruth Reuter

In July 1993 blood samples were submitted from a 3 year old female Cocker Spaniel with PU/PD and incontinence. The animal had been treated every 4-5 months since 1991 with medroxyprogesterone acetate injection to control aggressive behaviour. Urinalysis showed a specific gravity of 1.0112, pH 8.5. There was moderate hyperkalemia (K 6.7 mmol/L; reference range 3.8-5.5). Total WCC was 11.4 X 10^9 with a mild left shift (band cells 0.6 x 10^9; reference range 0-0.2).

Over the next few months the incontinence continued. The urine specific gravity varied from 1.005 to 1.027, the pH was relatively stable at 6.5. On one occasion culture of the urine resulted in a pure growth of haemolytic *E. coli* (10^4- $l0^5$/ml). A T4 level was 37.1 nmol/L (reference range 12-50) and serum cortisol was 44.5 nmol/1 (reference <200). The cholesterol was moderately elevated (12.4 mmol/1; reference range 3-7).

In November of 1993 the dog was represented to the clinic with an ocular and nasal discharge and respiratory difficulty. Xray of the thorax showed mild thickening of the peribronchial areas. *Staphylococcus intermedius* was isolated from a nasal swab and *Pseudomonas* from an oral swab. A CBC at this time gave the following results;

		Reference range
RBC X 10^{12}	4.4	5.5-8.5
Hbg/L	103	120-180
PCVL/L	0.30	.37-.55
WCC X 10^{9}	6.1	6.0-17.0
% nRBC	0	0
Relics X 10^{9}	44	<80

Other parameters were unremarkable. The findings suggested the possibility of bone marrow suppression. A call to the clinician elicited the fact that the dog was receiving stilboestrol every third day for incontinence with little effect. The drug was immediately discontinued and antibiotic treatment instituted. The incontinence subsequently disappeared and the dog has made a good recovery.

Oestrogen-induced bone marrow toxicity has been described in dogs in association with treatment of perianal gland adenomas, prostatic hyperplasia, urinary incontinence, pseudopregnancy, anestrus and mismating. The changes seen include thrombocytopenia, leukopenia and anemia and can depend on the time of sampling, the dose of the compound and the sensitivity of the particular animal.

VETLAB (ADELAIDE)

Two Haemangioendotheliomas in a Bovine Brain - John Finnie

An unusual vascular neoplasm in the brain of a calf that resembled epithelioid and spindle cell variants of haemangioendothelioma in man was recently diagnosed. This tumour has apparently not previously been reported in domestic animals.

A 5-month-old Angus Hereford cross steer had been circulating to the left for about a month. The head was always inclined 45 degrees to the left with ptosis and dorsal deviation of the left eye. When no improvement was detected the animal was killed. At necropsy, 2 firm discrete masses, 1.5 cm in diameter, were situated 1cm apart in the deep white matter of the left cerebral hemisphere.

Microscopically, the lesions were composed of small proliferating blood vessels lined by plump endothelial cells and resembled vascular channels in early angiogenesis. The lining cells were epithelioid and were quite uniform in appearance. The plump endothelial cells were sometimes grouped in small solid nests or short cords, and a few lumina appeared as slit-like spaces. Erythrocytes were plentiful in patent neoformed vascular lumina and were also observed in a few very primitive lumina. Evidence of the formation of a vascular lumen was commonly seen in single endothelial cells, and these miniature intracytoplasmic lumina were represented by cytoplasmic vacuoles. The distinct vessels and nests of epithelioid cells merged with intervening spindle cells, which formed fascicles without obvious lumina. The endothelial origin was confirmed by immunohistochemical studies, particularly by the demonstration of the endothelium-specific marker, Factor VIII antigen in the cytoplasm of most cells.

This tumour was designated a haemangioendothelioma because of its close resemblance to this type of vascular tumour in man, the latter being uncommon, of borderline low-grade malignancy, and histologically intermediate in appearance between haemangioma and angiosarcoma. Primary haemangiomas in the CNS of domestic animals are rare, and most cerebral vascular masses are either metastatic angiosarcomas or hamartomas. Since no vascular tumour was found in other sites in this calf at necropsy, it seems likely that the lesion arose in the brain.

Enzinger FM and Weiss SW (1988). Soft Tissue Tumours, 2nd Edit. CV Mosby Company, St Louis, pp 83-101, 533-44.

NEW SOUTH WALES - Paul Gill

Anaemia and Septicaemia Associated with Tick Infestation in a Juvenile Alpaca - GL Reddacliff

A 2 week old alpaca cria was "suddenly found dead", with no reported signs of illness.

Reported Gross Necropsy Findings: More than 50 ticks (subsequently identified as the common bush tick, *Haemaphysalis longicornis*) in various stages of engorgement were present on the axilla, ventral abdomen, neck and flanks. There were extensive subcutaneous haemorrhages in the axilla and ventral abdomen, and smaller doughnut-shaped haemorrhages on the neck and flank associated with the ticks. Internally the carcass was pale, as were the liver and kidney. The heart was also pale, with small haemorrhages on the endocardium. Superficial lymph nodes were haemorrhagic. Small amounts of clear straw-coloured fluid were present in the abdominal and pleural cavities. Yellow-creamy fluid with some gas bubbles was noted in the proximal half of the small intestine. No other lesions noted. Anaemia and enterotoxaemia were considered as possible causes of death by the submitting practitioner.

Histopathology: Liver had mild diffuse sinusoidal leukocytosis, and mild scattered individual cell necrosis. There multiple tiny foci of agonal subendocardial haemorrhage on the heart. There was fibrinoid degeneration in the periarteriolar sheaths of the spleen, which was otherwise normal, with abundant germinal centres. Peripheral lymph nodes were severely congested, with diffuse, mild individual cell necrosis and scattered foci of fibrinous exudate. There was also multifocal granulocyte infiltration and moderate extra medullary haematopoiesis. Lungs had a moderate acute interstitial pneumonia, with fibrinous alveolar exudate, intra-alveolar inflammatory cells, including prominent macrophages, and diffusely increased cellularity of alveolar walls. There were no significant lesions in the gut, kidney or brain. Skin had multifocal to extensive acute haemorrhage, and an acute vasculitis in the deep dermis and hypodermis, with generally increased perivascular cellularity.

Bacteriology: Smears from various levels in the small intestine failed to reveal any increase in gram positive rods, and the counter-immuno-electrophoresis test for epsilon toxin was negative. A pure growth of *Escherichia coli* was obtained from the intestinal content, liver and heart.

Comment: The pathology and bacteriology suggested an enterohepatic septicaemia in an anaemic animal. Was the extramedullary haematopoiesis a result of blood loss anaemia from the ticks, or was the anaemic appearance of the carcass simply due to extensive agonal subcutaneous haemorrhage? Presumably the tick worry predisposed this animal to septicaemia. There was no history to suggest failure to obtain sufficient colostrum. Bush ticks have been very prevalent around the Sydney region over this past summer. Anecdotal reports of mortalities in neonates of many species due to heavy tick infestations are frequent, but the pathogenesis of such mortalities is not defined.

REGIONAL VETERINARY LABORATORY, WOLLONGBAR.

Crescentric Glomerulonephrosis in Pigs - Paul Gill

Kidneys were submitted from two 20 week old pigs which had died unexpectedly in two separate piggeries. Both the kidneys were enlarged (160mm x 70mm, 180g), firm and mottled by multiple, pale, pinpoint foci over the surface and through the cortex. Histologically there are diffuse glomerular changes including segmental fibrin deposition, capsular fibrosis, mesangial fibrosis and sclerosis, septic thrombosis and haemorrhage, segmental hypertrophy and proliferation of the capsular epithelium, fibrinous exudation into Bowman's space and synechiosis. Tubular lesions include dilation, formation of protein castes and precipitation of urate calculi. The morphological diagnosis is glomerulonephritis, proliferative, chronic, marked. The lesions resemble those of postinfectious crescentric glomerulonephritis in humans (pen. comm. Dr David Papadimos).

As *Streptococcus suis* infections had been diagnosed in other pigs from both piggeries, it is postulated that these kidney lesions may be the result of earlier *Strep, suis* infections. These lesions are unusual in our experience and I would appreciate contact from other veterinary pathologists who have seen similar lesions.

Multiple Eye Defects in a Calf - Roger Cook

Eyes were submitted from a congenitally blind crossbred calf. The left eye was enlarged (bupthalmus) with a prominent cornea (keratoconus). The lens was thin (2mm) and white in both eyes. Histologically goniodysgenesis and anterior and posterior cataract are evident in the left eye. We were unable to determine whether the defects were the result of a genetic abnormality or due to an in utero insult e.g. pestivirus.

Cryptosporidiosis in Calves - Stephen Love and Carol Quinn

There was a marked increase in the number of diagnoses of Cryptosporidium associated calf scours at RVL Armidale last year, particularly in late spring. In all, we had 24 accessions in which this organism was identified in faeces submitted from scouring calves. Rather than representing an epidemic of cryptosporidiosis, this probably is largely due to an increased interest in this pathogen by field veterinarians, particularly Rural Lands Protection Board District Veterinarians. This in turn, is doubtless related to the Beldico/Elt-Vet Tetra Kit test, which for a number of months has been offered by our serology lab, headed by Carol Quinn.

The Eli-Vet Tetra Kit combines four tests (ELISAs): Eli-Vet Rotavirus test, Coronavirus test, K99 test and the Eli-Vet Cryptosporidium test. Depending on the reference of "gold standards" employed, the manufacturer claims the following sensitivities/specificities: rotavirus - 93-97% sensitivity/94-100% specificity; coronavirus - 90%/88%; K99 - 42%/87%; and cryptosporidium - 94%/97% regarding the evaluation of the K99 component, the manufacturers state that the agglutination test, used as the reference method in this case, is probably inadequate because it is unable to distinguish between K99 and ATT25 attachment factors. Notwithstanding this, the Cryptosporidium test looks good on paper. In a limited number of comparisons, we also have found that it correlates well with other tests (e.g. FAT and modified Ziehl-Neelsen) for this organism.

It appears that calf scours, particularly in calves older than one week, are too often contributed to *E coli* in this part of the world, particularly by farmers. Perhaps opportunists such as *cryptosporidia* as well as other pathogens and risk factor, have been underrated. REGIONAL VETERINARY LABORATORY, ARMIDALE

Aspergillosis in Ostriches - Stephen Love.

RVL Armidale is collaborating with the Department of Animal Science, University of New England (Dr Harry Gill, Senior Research Fellow) in a study of various aspects of aspergillosis in ostriches. The project is supported by the Rural Industries Research and Development Corporation and the Australian Ostrich Association.

Aspergillosis is considered to be an important disease of ostriches but basic epidemiological data (prevalence, possible risk factors etc) is lacking. Additionally, the disease is notoriously difficult to accurately diagnose, at least in live birds. We aim to collect and analyse data on such problems, with a view to providing information to veterinarians and the industry.

We seek the assistance of veterinary laboratories in Australia with the following:

* If possible, could you send us copies of all lab reports relating to ostriches, including those in which aspergillosis is diagnosed or suspected, from (and including) 1990 and also for the coming year (1994)? We would need copies of all ostrich case reports, not just those relating to aspergillosis, so that the "backdrop" or context of the problem under study can be defined. Information relating to individual owners would of course be kept confidential.

* In addition to the above, we also request that you send us Aspergillus isolates from all ostrich cases in 1994 in which this organism is involved. To assist your laboratory in this matter, we can arrange to pay for the transport costs of these isolates.

Please send copies of reports, and isolates, to:

Stephen Love
Regional Veterinary Laboratory
NSW Agriculture
Trevenna Road (Private Mail Bag), University of New England, ARMIDALE 2351.

We hope that you can assist us in this way. Please contact us indicating whether you are willing and able to cooperate, and if so, the name of a suitable contact person in your organisation.

For further information or comments, please contact Stephen Love (RVL Armidale, phone 067 701800, fax 067 701830} or Harry Gill (UNE, phone 067 732227, fax 067 733275). We look forward to hearing from you.

DIAGNOSTIC VETERINARY LABORATORY, SCONE

Atypical Interstitial Pneumonia in Foals - Angela Begg

Three of 300 foals on a thoroughbred stud died over a 2-week period in January with a rapidly developing respiratory disease. Affected foals were 8-10 weeks of age and were all in different pasture improved paddocks in groups of 10 to 20 mares and foals. Supplementary feeding with grain and lucerne hay was common to all groups. All foals had received Equimec anthelmintic paste in December but no other treatment. All foals were found in terminal respiratory distress with only one foal being treated for 12 hours before it died. Gross and histological changes were similar in all foals. A generalised pneumonia was present and was characterised grossly by severely distended firm plum-coloured finely mottled lungs which did not collapse. A small amount of excess serosanguinous pleural fluid was present. Two of the foals had a small single localised area of chronic lung abscessation due to *R. equi.* Extensive bacterial culture of other parts of the lungs and cell culture for equine herpes virus were negative. Histologically, severe generalised subacute atypical interstitial pneumonia was present and was characterised by acute alveolar epithelial necrosis with extensive hyaline membrane formation and areas of intense proliferation of Type II alveolar epithelial cells with early alveolar epithelialisation. Some epithelial necrosis of terminal bronchioles with exudation was present but larger airways were not involved. Dense perivascular and interstitial lymphocytic/plasmacytic infiltrates, interstitial and pleural oedema, and variable vascular engorgement with small alveolar haemorrhages were other features. Neutrophilic infiltrates and fibroplasia were not dominant features. Other significant changes included splenic lymphoid hyperplasia and peracute hepatic periacinar degeneration/necrosis (terminal change?) with variable sinusoidal and periportal lymphoid/neutrophilic infiltrates. The generalised lung changes were not typical of the normal pathological processes associated with *R. equi* infection, but were more suggestive of toxic lung pathology or possibly a hypersensitivity reaction to *R. equi* antigens. As yet no toxin has been identified. Paraquat toxicosis has been excluded.

Any observations on similar disease syndromes in foals or suggestions regarding possible aetiologies would be appreciated.

REGIONAL VETERINARY LABORATORY, ORANGE

Feather Mites in Ostriches - Michael Hindmarsh

Feather mites, *Strithiopherolichus bicaudatas*, have been identified in the feathers of ostriches in Victoria and on the north coast of NSW.

Affected feathers have a marked reduction in the length of barb; mites can be found in the quill tunnel. Line drawings of the mite can be obtained from Michael Hindmarsh at RVL Orange.

Multifocal Symmetrical Encephalopathy in a Simmental Calf

A 3-month-old Simmental bull calf was presented for post mortem examination after pyrexia, paresis and one month of recumbency in a sling. Necropsy revealed severe necrosis in the inner abductor muscles of the hind legs and some pale foci on the midbrain.

Histopathology revealed bilateral areas of malacia in the caudate nucleus and the putamen and a normal spinal cord. These lesions are consistent with the description of multifocal symmetrical encephalopathy by Peter Harper, *el al*, Veterinary Record (1989) **124**: 122-123.

This case and all previous cases are related to the bull "Extra". A repeat mating occurred 2 months ago of the same bull and cow that produced this calf. The next progeny will be closely observed for this disease.

Regional Veterinary Laboratory, Wagga Wagga - John Glastonbury

Cattle

Haemoglobinuria was investigated in a local dairy herd. One recent case had a temperature of 40.0°C and pale but jaundiced mucous membranes. Haematology found:

RBC	1.59 x	10^{12}/L
HB	48	g/L
HT	0.138	L/L
MCV	87	fL
MCH	30.2	pg
MCHC	348	g/L

Smears revealed anisocytosis, poikilocytosis and many microcytes. Levels of serum phosphorus, calcium, magnesium and copper were normal while titres of <30 were obtained in microscopic agglutination tests for *Leptospira pomona* and *hardjo*.

Following a week's illness, the cow died. Severe haemoglobinuric nephrosis, severe hepatic bile stasis, moderate acute periacinar hepatic necrosis and mild degrees of hepatic as well as renal haemosiderosis were found histologically.

The aetiology finally came to light when it was revealed that the pasture which the herd was grazing contained a luxuriant growth of wild radish, *Raphanus raphanistrum* L. Haemoglobinuria and intravascular haemolysis, presumably of the Heinz body type, have been associated with the ingestion of this plant by livestock in Western Australia and Queensland.

Septicaemic salmonellosis due to **Salmonella Dublin** was responsible for the sudden death of 10 2-year-old Murray Grey steers on feed.

Heavy growths of the organism were recovered from the liver, spleen, lung and bronchial lymph node of 1 case.

Histological examination revealed severe acute multifocal necrotising splenitis, moderate acute multifocal necrotising hepatitis, mild hepatic bile stasis and evidence of disseminated intravascular coagulopathy.

Late-term abortion in 4 of 30 Angus heifers was attributed to infection with **Listeria monocytogenes**. The one aborted foetus, examined in detail, underwent rapid autolysis and excessive volumes of blood stained peritoneal and pericardial fluids and severe diffuse pulmonary congestion with the terminal airways containing yellow/green suppurative casts were the principal post mortem findings.

Heavy growths of *L. monocytogenes* were obtained from the foetal stomach, liver, spleen, lung and pleural fluid. Histological findings included moderate acute suppurative foetal bronchopneumonia and mild acute multifocal necrotising hepatitis.

Sheep

Crystal-associated cholangiohepatopathy and photosensitisation caused a fear of bluetongue and severely interrupted our Christmas Eve beers. Twelve of 500 sheep introduced to southern New South Wales from Coonabarabran 3 weeks previously developed pyrexia, facial swelling and congestion of coronary bands.

During necropsy affected animals had severe photosensitisation of the hairless areas of the body, generalised icterus and swollen friable livers.

Acicular clefts in bile ducts, bile ductules and hepatocytes as well as swollen perisinusoidal cells were found histologically. The history indicated that dikkor and geeldikkop were possible but access to *Panicum* spp was greater than to *Tribulus terrestris*.

A Series of Dogs with Primary Lung Carcinoma - Malcolm France

In the 3 months from December 1993 to February 1994, primary lung carcinoma has been confirmed at necropsy in 5 dogs seen at the University of Sydney Veterinary Clinic.

The dogs ranged in age from 8 to 13 years, 3 were Labradors, 1 a Labrador cross and 1 a cattle dog-bull terrier cross; all came from the Sydney metropolitan area. All dogs had a history of cough with a generally poor response to antibiotics over a period ranging from 2 weeks to 6 months. A range of radiographic changes was seen. Cytological preparations made from transtracheal aspirates and/or fine needle aspirates of lung masses led to an antemortem diagnosis of ‘carcinoma’ in 3 dogs, ‘neoplasia’ in 1 dog, while only inflammatory cells were seen in the other dog.

Grossly, neoplastic invasion of the lung was extensive in all cases, although patterns ranged from multinodular to diffuse, mainly single lobe involvement. Involvement was greater in the right lobes in 4 cases and in the left lobes in 1 case. A striking feature in 4 dogs was a strong propensity to invade lymphatic vessels, particularly around the airways and larger blood vessels. This produced marked mural thickening of these structures; in one case, chains of small nodules were visible on the pleural surface. Surprisingly, lymph node enlargement was only modest.

As with many types of neoplasia, there is no universally accepted system for classifying primary lung carcinoma. The term ‘bronchogenic carcinoma’ is widely used for human lung cancer, although this does not usually refer to a particular histological type, but rather to the common site of origin of several types of lung cancer all of which arise in the major bronchi. Generally speaking, classifications usually include 3 differentiated types (squamous, glandular or cuboidal/columnar cells) and 2 anaplastic types (large cell and

small cell); of course, combinations of these can be found within a given neoplasm. Of the 5 cases in this series, 2 were cuboidal/columnar (usually termed bronchiolo-alveolar carcinoma), 1 was mixed (adenosquamous carcinoma) and 1 was undifferentiated (anaplastic carcinoma, large cell type). Extrathoracic metastasis was seen only in the anaplastic carcinoma (brain and muscle), and it was this neoplasm which lacked the obvious lymphatic vessel invasion.

Primary lung carcinoma is rare in animals; however, at this stage there is nothing to suggest that this run of cases is the result of anything other than chance.

WESTERN AUSTRALIA - David Forshaw

Murdoch University Veterinary School

Polioencephalomalacia in an Alpaca

An 18 month old castrated male alpaca suddenly displayed ataxia, left hind limb lameness and hunched back. It was treated with steroids and antibiotics and removed from the mob of males. Twelve hours later it exhibited lateral recumbency and opisthotonus. It was very distressed and had a temperature of 39.6C. It was treated with fluids and pethidine and then anaesthetised to radiograph the spine. Recovery appeared normal but 5-10 minutes later it exhibited tetanic spasms and apnoea and then died.

Lesions were confined to the brain. There were haemorrhages in the grey matter of the lateral and dorsal gyri from the forebrain to the occipital lobes and in the oculomotor, lateral geniculate and vestibular nuclei. Histologically there was necrosis, oedema and haemorrhage, and marked activation of cerebral vessels in affected areas.

The haemorrhage was marked in this case but it is not known whether this was potentiated by the treatment and manipulations (e.g. anaesthesia) that the animal was subjected to. Although haemorrhage is not generally prominent in ruminants with PEM it does occur in amprolium induced PEM in lambs and is a common feature of thiamine deficiency in carnivores.

Oesophageal Lesions in Ostrich

Four eight month old ostrich developed swellings in the lower neck 4 weeks prior to examination. The swellings were firm, not painful and were moveable with the oesophagus. Endoscopy revealed pale, rough, swollen areas 5x3 centimetres in the oesophagus. At surgery these areas were shelled out leaving an ulcerated base covered by granulation tissue. Histologically the shelled out material was composed of necrotic exudate in which fungal hyphae and numerous bacterial colonies were present. Trichomonads were not demonstrated on wet preparations or in section.

It is not known whether the fungal infections are primary or secondary to trauma. Trauma to the oesophageal mucosa is a distinct possibility as the birds are free-ranging on pasture that contains a lot of Dryandra, a plant with very prickly leaves.

Has anybody got anything to offer on this case?

Acute Myopathy in a Rex Rabbit - (C.R. Huxtable)

A five months old male Rex rabbit was first presented with sudden onset of clinical weakness, from which it recovered in a few days with a multi-vitamin and mineral supplement. Serum creatine kinase activity was markedly elevated initially and fell to normal over 5 days. Serum potassium was low (4mM) and remained low in spite of clinical recovery.

One month later, there was again sudden onset of profound flaccid paresis, with serum CK elevated to 5x normal. Euthanasia and immediate necropsy were performed. Grossly there was no significant abnormality. Histologically, there was acute segmental myonecrosis in samples taken from pectoral and pelvic girdle groups. Liver selenium and Vitamin Å levels were within "the normal range for sheep".

Anecdotal evidence indicates that this problem is fairly common, and it seems likely to have a nutritional/metabolic basis. Can anyone offer information or comments please?

Leukoencephalopathy in a Mixed Breed Dog

A 7 year old, speyed border collie-cross bitch was presented to Murdoch University Veterinary Hospital with a 5 month history of behavioural and neurological disorder. The dog sometimes failed to recognise the owner, and had become aggressive towards other dogs and children. The owner also reported that the dog occasionally circled to the right, misjudged distances and lost its balance.

Complete clinical, ophthalmological and neurological examinations failed to reveal any abnormalities. A behavioural abnormality was therefore postulated.

The dog was euthanased at the owner's request and a necropsy performed. The dog was found to be in good body condition. Significant lesions were confined to the brain, where there were 1mm diameter cavitations within the white matter tracts of the frontal lobe on the right side.

Histological examination of the brain revealed marked symmetrical loss of myelin and axons from the cortical white matter tracts. Lesions were most severe in the frontal lobes. Lost axons were replaced by glial scar tissue and there was prominent gemistocytic astrocytosis.

A diagnosis of severe sclerosing leukoencephalopathy was made, but the aetiology of the lesion was not determined.

W.A. Department of Agriculture
Albany

Eosinophilic Rumenitis in a Cow - David Forshaw

Severe acute necrotizing rumenitis and omasitis was the diagnosis in a single Simmental cow which died suddenly while grazing a dry kikuyu paddock. The lesions consisted of widespread subcorneal infiltrates of polymorph dominant exudate, the majority of which were eosinophils. Numerous protozoa were present in the lumen. The cow was negative for mucosal disease antibody by gel diffusion and negative for antigen by capture ELISA. The rumen pH was not measured.

The cause of these lesions is not clear. The eosinophilic nature of the exudate is puzzling. The sub corneal nature of the lesions is consistent with the descriptions of kikuyu poisoning but as there is a single cow affected and the feed is anything but lush I think this is unlikely. Any suggestions?

VICTORIA - Deb Seward

AAHL

A Pulmonary Adenoma in an Australian Sheep - Peter Hooper

Pulmonary adenomatosis or jaagsiekte is an infectious (retroviral) neoplastic disease of sheep exotic to Australia. There was therefore considerable concern when a single lesion consistent with the morphological diagnosis of pulmonary adenoma was observed in a sheep in a quarantine station holding sheep derived from imported embryos.

The lesion was on and immediately under the pleural surface of the lateral aspect of the left cranial lobe of the lungs. It was round, well circumscribed, about 1.2 cm in diameter, grey/brown and slightly granular in appearance. There were no other visible lesions in the remainder of the lungs. Histologically, the single lesion was distinctly an adenoma. Its epithelial cells were cuboidal to low columnar, and were mostly smaller and more cuboidal than the bronchiolar cells which had been surrounded by the tumour. It was not encapsulated, rather the neoplastic tissue was spreading into the surrounding lung tissue along the alveoli and up to and against the pleura. In some areas the "acini" were almost "empty" where they were lined by smaller, more basophilic cells, while elsewhere there were masses of foamy macrophages filling the acinar spaces which were lined by taller more vesicular cells.

The sheep, an 8 year old Suffolk-Merino cross ewe, was not of imported stock but rather kept as a combined teaser and sentinel animal. No similar lesions were found in any of the stock bred from imported embryos, or in any other sentinel stock. If the lesion was caused by the retrovirus of pulmonary adenomatosis, the lesion should have arisen by spread from an animal with well developed disease. All sheep were kept in close surveillance including post mortem and histological examinations of the lungs to exclude maedi-visna and pulmonary adenomatosis, and no such potentially infectious animal was seen.

In the absence of a reasonable source of retroviral infection, the cause of this single lesion was more likely to be spontaneous rather than retroviral. Few animals have been reported with probable spontaneous pulmonary adenomas, presumably because the retroviral disease is ubiquitous in all continents other than Australasia and most diagnoses would therefore assume the widespread retroviral aetiology.

HAMILTON VETERINARY LABORATORY

Actinomyces Pyogenes Septicaemia - Janeen Samuel

A non-pregnant Friesian heifer was seen to die suddenly. The owners reported that immediately before death it had convulsions, laboured breathing and an audible heart beat. The veterinary practitioner saw it half an hour after death and noted that there was blood in the anterior chambers of both eyes; when he opened the carcass he found it very cyanosed. He did not do a full post mortem for fear of anthrax but sent samples, including the head, to this laboratory.

Smears for anthrax were negative. All tissues received appeared congested, particularly the brain and meninges. There were ecchymoses on the epicardium and the surface of the kidney and pale spots within the kidney parenchyma. Actinomyces pyogenes was isolated from the anterior chamber fluid, brain, kidney, spleen and lung. Growth was heaviest from kidney and brain. Coliforms were also grown in low numbers from all these tissues except the eye fluid.

Histological examination showed numerous micro-abscesses throughout the brain and the renal cortex. These consisted of necrotic inflammatory cells with colonies of bacteria at their core. They were very discrete, with almost no transitional zone between abscess and normal tissue. There were also some colonies of bacteria in the cerebral cortex with no associated cellular reaction. The brain showed generalised vascular congestion with some vessels showing cuffing by leucocytes. In the kidney there was some interstitial nephritis at the cortico-medullary junction. The heart had scattered small foci of myonecrosis with mononuclear infiltrates, but no bacteria were present. In the eye, the ciliary body was very congested, and there was bleeding into the anterior and posterior chambers, but no evidence of inflammation or of bacteria.

This was an unusual presentation of *A. pyogenes* infection. We postulated that there may have been a primary abscess, for example in the lungs, which had ruptured into the blood-stream and thus seeded large numbers of bacteria into the organs. Alternatively, there could have been an *A. pyogenes* lesion on a heart valve, as the heart was not fully examined.

BENALLA VETERINARY LABORATORY

Necrotizing Enterocolitis in Young Ostriches - John Mackie & Judith Wilkie

A local breeding establishment experienced an outbreak of diarrhoea and deaths in young ostriches, 2 weeks to 2 months of age. The morbidity over a 2-month period was 70% and the case fatality rate was 100%. On post-mortem there was some pasting of the vent and large intestinal contents were pasty to fluid. There were flecks of fibrin on the large intestinal mucosa of one bird. The proventriculus contained a large amount of grass.

Histologically there was a segmental necrotizing enterocolitis with crypt necrosis and secondary superficial bacterial overgrowth. The most severe lesions were in the large intestine. Bacterial culture of small intestinal and large intestinal contents yielded profuse mixed growths of predominantly E.coli. Enrichment cultures for Salmonella and Yersinia were negative. Electron microscopy and virus isolation are pending. There is one recent report of coronaviral enteritis in an ostrich chick in the USA (Frank RK & Carpenter JW *Journal of Zoo & Wildlife Medicine* 23:103 (1992). We would be interested in hearing whether anyone has seen similar lesions in young ostriches and whether any aetiological agents have been incriminated.

Uveitis and Sequelae in Foals - John Mackie

On a thoroughbred stud, 6/30 foals aged 1-3 months developed severe anterior uveitis with hypopyon. Clinical cases occurred over a 4 week period. The uveitis resolved in all foals after several days to 3 weeks of systemic treatment with NSAID. Three of the treated foals developed chronic gastrointestinal ulceration, primarily of the large intestine, and subcutaneous swellings over the fetlock and hock regions. Joints were not obviously affected. Histologically in the region of the subcutaneous swelling there was a necro-suppurative cellulitis associated with a necrotizing vasculitis in the subcutis and dermis. No bacteria were isolated from fluid aspirated from the areas of cellulitis and special stains of fixed tissue were negative for bacteria and fungi. Apart from an EHV titre of 3 (SN test) in one foal, serology for EHV, EVA, EIA, Leptospira pomona, Leptospira bratislava and Chlamydia psittaci was negative. Fluorescent antibody testing for the deposition of immunoglobulin or complement in the vascular lesion is pending. One hypothesis is that a primary, clinically silent, streptococcal bacteraemia led to the uveitis which was followed by an immune-mediated cutaneous vasculitis against streptococcal antigens. One difficulty with this hypothesis is the simultaneous occurrence of an immune-mediated vasculitis in more than one animal. Any comments would be appreciated.

Intracellular Infection in an Emu - Malcolm Lancaster

A 3-4 year old female emu was very depressed one day and dead the next, despite oxytetracycline treatment. On necropsy the bird had large fat depots, a large caseous cast in the gall bladder, little intestinal content and faecal staining around the vent.

Histologically, many cells were distended by intracytoplasmic aggregations of faintly basophilic circular to oval structures (approx.1 micrometre across). These organisms were particularly common in the lungs but were present in most of the organs examined. The identity of individual parasitised cells was not clear in many instances but some were definitely endothelial cells. This fact, together with the general location of the other affected cells, suggested that most of the parasitised cells were endothelial cells. Reaction was very limited except in the brain where there was multifocal gliosis apparently centred on collapsed parasitised capillaries. A few non-endothelial parasitised cells (microglial cells?) were also evident in the brain.

The organisms resemble those seen in Muscovy ducks in NE Victoria (and elsewhere). While ducks did not feature in the history, the farmer did report that several galahs had been found dead on the farm around the time of the emu's demise.

BENDIGO VETERINARY LABORATORY

Hepatic Failure Due to Lesser Loosestrife Poisoning in Sheep - Rod Badman

The sudden onset of deaths in a mob of 630 mixed aged ewes on a property in north central Victoria prompted the owner to submit several animals for post mortem. The sheep had been grazing canola (rapeseed) stubble for approximately 2 weeks prior to the first deaths. Because of this, plant poisoning was not immediately suspected. Severe acute hepatic necrosis indicated by an accentuated lobular pattern, moderate jaundice, excess straw coloured fluid in body cavities and marked pulmonary oedema were the major autopsy findings. Severe periacinar necrosis, in many lobules total necrosis, and early bile ductular proliferation were the outstanding histological changes.

Blue-green algal poisoning was considered a possible cause due to favourable weather conditions for algal growth preceding the deaths and because the severity of liver necrosis could have obscured the periportal component of necrosis. Water samples from the source paddock proved negative for toxic algae but close examination of the paddock indicated that only two types of plants were present; dry canola stubble which has little nutritional value and an erect green plant about 30cms high which was positively identified as Lesser Loosestrife.

Lesser Loosestrife is a widely distributed weed in the grain growing areas of Victoria, and is common in stubbles when seasonal conditions of good spring and early summer rain occur. It is considered to have very low palatability and is generally not eaten by sheep. In the circumstances on this property, the lack of good alternative feed was thought to be the reason for the sheep eating sufficient Loosestrife to cause toxicity.

BAIRNSDALE VETERINARY LABORATORY

Gastro-intestinal Problems in Emus - Peter Mitchell, Kit Button, Nick Barton

We have found little published information on the diseases of emus, so we offer the following observations as a contribution towards our understanding of these birds.

1. Diet and the digestive tract

Emus are described as "omnivorous, taking sparsely distributed foods of high nutritional quality, principally seeds, fruits, insects and growing tips of plants but fermentation of small quantities of fibre is important for energy requirements" (Handbook of Australian, New Zealand and Antarctic Birds, Vol. 1, 1992). The digestive tract of emus reflects their natural diet. The following table documents the body weight (kg) and weight (g) of contents of the proventriculus (PV) and gizzard (G) and weight of stones (ST) in gizzard of emus examined at this laboratory:

Case	BW	PV	G	ST	Comments
1	26	783	0	0	Grass, banksias & Cyprus cones in PV. Intussusception
2	18	0	+	6	Large nuts in G. Haemorrhagic enteritis - see VPR 37:20 - note we have since isolated *C. jejuni* in 5/8 emus without enteritis.
3	18	scant	+	+	Matt f grass, wire stick and pebbles, perforation of G by wire.
4	14	0	+	+	Plastic in G
5		0	153	0	Grass, wheat and chestnuts in G.
6	21	1630 in both			76 Possible slight impaction.
7	16	0	350	3	Grass cord
8	12	0	460	278	Grass and stones in G.
9	13	0	273	120	Grass cord.
10	18	+	+	3	Grass cord
11	7	0	140	0	Grass cord and piece of glass in G.
12	6	0	299	53	Grass cord

The proventriculus does not contain any gravel (cf. ostriches), and was often empty in the birds we examined, suggesting that food spends little time in this chamber. The muscular wall of the gizzard is relatively thinner than in ostriches. Stones may be present in the gizzard, but were not consistently present, and only three had amounts of stone comparable with ostriches (around 1% of body weight). The relative lengths of the intestines are 90% small intestine, 3% caeca (paired narrow tubes) and 7% colon (straight tube about 30 cm long in adult birds) - this compares with 36%, 7% and 57%, respectively, for ostriches. Passage of digesta through the intestines is only 4 to 5 h (cf. 36 h for ostriches). These differences suggest that emus rely on the easily digested component of their diet for most of their nutrient requirements, and are not as well equipped as ostriches for handling a fibrous diet.

2. "Grass cords"

We have seen 5 cases characterised by the formation of mats of fibrous grass in the gizzard extending as a cord into the small intestine:

7: Cord extended and tapered 15cm into the small intestine. Probably subclinical - contributed to poor condition.

9: Cord extended 10cm into intestine. Also subclinical - contributed (with worms) to poor condition.

10: Cord was 85 cm long, protruded into the proventriculus and extended 70 cm in the intestines, to within 30cm of the colon. Koilin layer in the gizzard was macerated, mixed with mucus. The small intestine was concertinaed by the cord, with a 10cm intussusception. Intestines also had many worms and *Salmonella* group B.

11: Cord extended 62 cm past the gizzard with 18cm hanging from the cloacal. Both ends were expanded - at the gizzard and at the cloaca - and the entire intestinal tract was concertinaed to a length of 44 cm with one major intussusception. The koilin layer near the intestinal opening was disrupted and peeling. Intestines also had *Salmonella* group B. Owner reported losing other birds with material hanging from the cloaca.

12: Same property as 11. Gizzard contained mass of mainly loose fibrous vegetable material, with a more matted cord extending through the duodenal opening and down the intestine. The first part in the intestine was narrow (1.0-1.5cm diameter) and tightly twisted. The distal part was looser, forming a large wide mass up to 5cm diameter. The anterior 72cm of intestine was concertinaed into 18cm between the mass in the duodenum and the distal mass of fibre in the intestine. Parts of the intestinal wall were necrotic.

The grass cords probably formed when large amounts of fibrous plant material (individual fibres to 20 cm) matted in the gizzard - the movement of the gizzard and intestines probably encouraged the continuation of the process. No other materials (such as wire) were found in the cords. Two clinical cases also had *Salmonella* and worms. Significant amounts of stone were present in the gizzard of only two birds. The disease is recognised in zoos, and is prevented by cutting all grass in enclosures.

3. Intestinal parasitism

Series of post mortem examinations from two properties revealed large numbers of worms. Based on the names of specimens in Australian museums, these were assumed to be *Dromaeostrongylus bicuspis* and *Trichostrongylus tenuis* (nematodes), *Raillietina australis* (a cestode) and Philopfuhalmus sp. (a conical-shaped trematode). Worm counts and faecal egg counts were:

Case No.		4	5	6	7	8	9	10
D. *bicuspis*	adult	2120	0	0	400	500	1500	2000
	larvae	50	0	0	700	1000	300	2600
T. *tenuis*	adult	550	0	0	0	700	800	800
	immature	0	0	0	200	300	100	0
R. *australis*		many	some	0	0	0	0	0
Philophthalmus sp.	mature	860	0	50	10	110	0	0
	immature	0	0	0	0	90	0	0
FEC (strongyle epg)		3720	0	0		30	360	0

Four others (2, 3, 11 and 12) had no eggs in faeces. Two (4 and 8) had foci of macrophages and giant cells in the liver, suggesting migrating parasites.

We do not know what constitutes a lethal worm burden in emus. All were in poor condition. 4, 5, 8 and 9 died with no other pathology, 6 had possible impacted stomachs and intestinal torsion, 7 had subclinical grass cord, 10 had *Salmonella* group B and clinical grass cord. It seems likely that these worm burdens at least contributed to the poor condition of the birds. Emus are selective feeders on widely distributed food items. Forcing animals to feed on the ground and reducing the home range/increasing population density would encourage the development of heavy worm burdens.

5. Comments

All birds were in poor condition. Many were muddy and some had broken body feathers and bare areas. Many of the diseases listed above were possibly subclinical, causing illthrift - death may have followed cold wet weather, or may have been the inevitable outcome of a variety of diseases and poor nutrition.

Emus are described as omnivorous. Given the simplicity of the digestive tract and some of the digestive tract problems that we encounter, we need to look harder at the diet of captive emus and question their management as grazing animals.

***E. coli* Septicaemia and Adenovirus Infection in Emus - Peter Mitchell**

Three 2-3 week old emu chicks died over three days - 2 were submitted for autopsy. Both chicks had very wet lungs, excess fluid in the pericardium, and yellow to khaki coloured livers. Intestines appeared normal in A; chick B had traces of blood in the lower small intestine and the large intestine appeared thickened.

On histology, livers showed severe periacinar necrosis with a few mixed inflammatory cells in necrotic areas and infiltration of portal triads with mixed inflammatory cells. Many hepatocyte nuclei in B were enlarged and contained dark purple inclusions. Both lungs had areas of infiltration of alveoli and alveolar septa with heterophils and necrotic cells. Kidneys had several foci of inflammation and necrosis, with marked glomerular thrombosis in B. Intestinal mucosa of both birds contained foci of inflammatory cells and necrosis in the lamina propria.

E. coli was isolated from a range of tissues from both birds. However, the liver necrosis appeared too severe and extensive to be caused just by the septicaemia - the inclusions suggested a disease similar to inclusion body hepatitis (thanks to Rod Reece at the Pathology Registry in Sydney for comments). Adenovirus particles were identified in the liver by Alex Hyett at AAHL.

Hepatopathy in Koalas - Peter Mitchell

Between September 1993 and January 1994, 21 koalas from Raymond Island were submitted for post mortem examination. Another 13 koalas were found dead but were too autolysed for examination. Three of 17 koalas with radio collars were among those found dead.

Liver disease was identified in 17 of the koalas. Pathology was characterised by pooling of blood, some necrosis and increased mononuclear cells in periacinar areas, swollen Kupffer cells, vacuolation of hepatocytes in some cases, karyomegaly, slight bile duct proliferation and infiltration of portal areas with small mononuclear cells. Some livers showed more severe but localised coagulative necrosis of entire lobules. Fibrosis was present around hepatic veins and scattered through the parenchyma, but this also occurs in most koala livers. The pathology suggested toxic liver damage. The liver damage was not as severe as in many toxic hepatopathies in domestic animals, but koalas may be more susceptible to liver damage because they live on a narrow energy balance and consume a high load of toxins that are normally detoxified in the liver.

This was a significant disease outbreak in a population of about 450 koalas. A review of past cases showed that a similar hepatopathy occurred in previous years, at about the same time of the year. The only consistent epidemiological observation is the occurrence of the disease in late spring/early summer, which coincides with the growing time for the eucalypts on the island. It is difficult to conceive that koalas which are so well adapted to eucalypt leaves could be poisoned by those leaves. I am seeking a keen toxicologist to investigate the problem.

***Haemaphysalis longicornis* (Bush Tick) in Gippsland - Nick Barton, Mike Terry**

H. longicornis the "bush tick" or "New Zealand cattle tick" has been present in Gippsland at least since 1976, and probably much longer. During most of this period, except for sporadic instances on occasional properties, numbers have remained low. This contrasts with the situation in the central and northern coastal regions of New South Wales, where it is recognised as a serious pest of livestock. It is a three host tick which can affect cattle, horses, dogs, sheep and humans.

1990/91 saw the first of a succession of wet summers in East Gippsland; this created suitable conditions for the survival of adults, eggs and larvae. By late spring of 1992 there were complaints of high numbers of nymphal ticks on a small number of properties: 12 months later the tick was present, often in high numbers, on many properties throughout Gippsland. Mechanism of spread is uncertain: transfer on infested livestock is probable, but it is stated to occur on wild birds in New Zealand. Foxes are also probable hosts.

Registration of some chemicals effective against this parasite has recently been extended to include Victoria, but a return to dry summers is needed to reduce this tick to its former curiosity status.

Goldfish Ulcer Disease

Goldfish ulcer disease is a septicaemic infection caused by various non-pigment-producing strains of the gram negative bacterium *Aeromonas salmonicida*. The disease arrived in Australia with imported goldfish about fifteen years ago and presents initially as multiple, small, skin erosions with erythematous borders on the sides of the fish. The lesions coalesce and progress to form deep ulcers. The disease is often fatal. Severely affected fish may also exhibit protrusion of the eyes, oedema of visceral organs and peritoneal effusion.

A similar disease, furunculosis, is seen in salmonids including trout and salmon. Strains of *A. salmonicida*, which produce brown pigment on bacteriological media, can be recovered from these lesions.

Laboratory transmission studies have demonstrated that *A. salmonicida* recovered from goldfish lesions can damage trout and salmon; however, there are no documented cases of this occurring in the field.

The potential for interstate movement of fish to spread Goldfish Ulcer Disease and the possibility of occurrence of the disease on salmonid farms is of concern. Control methods have been requested by the Animal Health Committee. There is at present a total ban on importation of gold fish to Tasmania.

This case study, submitted by Grant Rawlin, illustrates an attempt to control goldfish ulcer disease on a Victorian property.

VICTORIAN INSTITUTE OF ANIMAL SCIENCE

Goldfish Ulcer Disease Control in Victoria - Grant Rawlin

Background

In 1991, the Sub-Committee on Fish Health expressed concern at the presence of Goldfish Ulcer Disease in a major Victorian goldfish farm. Since that time a disease control scheme has been implemented involving chemotherapeutics, environmental decontamination and quarantine procedures. The disease control program appears to have been successful as of Summer 1993/94 and further monitoring is continuing.

Design

Year 1: A section of the property was separated by fencing and by water source. The three dams involved were emptied over summer, one was bulldozed to remove 10cm of soil and 2 were rotary hoed twice over the summer period. In autumn the dams were repopulated with susceptible goldfish from Coffs Harbour. The populations of goldfish in these dams were sampled the next summer to assess presence of goldfish ulcer disease.

All breeder and grower fish in the general population of fish (approximately 2.5 million fish) were fed Flumaquinalone 10 kg/tonne twice.

Year 2: No ulcer disease was seen in the general farm population summer 1993/94. Several fish were found to have lesions among the Coffs Harbour sentinel fish in one dam. This dam never dried out fully the previous summer and not all the bottom was able to be rotary hoed.

Results

No infected fish have been seen in the general farm population in treated fish. Fish from treated parent stock have been placed in dams previously occupied by infected then treated stock and no disease was seen over the summer period in these untreated young stock.

Infected fish were only seen in the quarantine area in the dam which did not completely dry out.

Discussion

Strong evidence has indicated that the organism can be removed from the environment by desiccation. There is also evidence that clinical disease is treatable by the use of Flumaquinalone. There is preliminary evidence that the organism can be eradicated from latent carriers by the use of Flumaquin.

Further Work

Latency reactivation work is continuing with the assistance of AAHL. The Fish Farm is taking part in the Victorian fish health accreditation scheme as per the recommendations of the Goldfish Ulcer Disease working party of the Sub-committee of Fish Health.

Victorian Fish Health Accreditation Scheme

In accordance with the design of guidelines of the Goldfish Ulcer disease Working party, an accreditation scheme began in October 1993. 5 visits over each year with 3 visits over Spring and Summer are planned and are continuing. Visits to fish farms are made by Fish Health staff of the Department of Conservation and Natural Resources and the Department of Agriculture. Inspections are made of the facilities, a disease history taken and as large a sample of stock as possible is observed. Managers are bound to report unusual deaths and ulcerative lesions.

At present 20 fin fish farms subscribe to this service, including salmonid, native fish and aquarium fish producers. Reception of the new scheme has been enthusiastic from all farmers involved. Supply of fish to DCNR now requires compliance to the scheme.

AHL RUAKURA

BLAD in New Zealand - Bronwyn Smits

Bovine leukocyte adhesion deficiency or BLAD is an inherited defect of neutrophil function described in humans, Red Setters and Holstein-Friesians. It is an autosomal recessive trait, which we have diagnosed three times at this Laboratory and had two of these cases confirmed by a commercial company in Utah, U.S.A. I believe the Lynfield Animal Health laboratory in Auckland has also had a suspected case, based on histology and haematology (Pers. Comm. Derek Belton, LAHL).

The following is a brief case summary of one of these cases, with the presentation being fairly typical.

A young calf (2-3 weeks) was depressed, anorexic and scouring. Leukocyte counts were markedly elevated (170×10^9/L) due to neutrophilia (144×10^9/L) and left shift (bands 10×10^9/L). Fibrinogen and monocyte counts were also elevated. Necropsy showed multifocal ulceration of the alimentary tract, especially the mucosa overlying gut-associated lymphoid tissue. Samples were negative for Salmonella and Bovine Virus Diarrhoea Virus. Grossly, BVD-MD was a major consideration.

Histologically the ulcerated areas consisted of necrotic material and numerous bacteria, with a base of granulation tissue amongst remnants of lymphoid tissue. A characteristic feature was lack of inflammation at the ulcerated sites; yet blood vessels of all layers of the gut were packed with neutrophils. Similarly, vessels in the lung were filled with neutrophils, giving a cellular appearance to the septa.

Blood samples were tested for the defective gene, which was suspected to be in N.Z. as semen of carrier bulls from U.S.A. had been used here. The genetic defect results in reduced or absent surface expression of integrins adhesion molecules on leukocytes, interfering with their ability to adhere and migrate from vessels. As a result, affected animals have extremely sparse leukocyte infiltration at extravascular sites of bacterial infection and suffer from life-threatening bacterial infections.

(Acknowledgement: Rob Fairley and Roger Ellison)

References

Ellison, RS, Fairley, RA and Burton, LJ (1993) Abstracts of the 22nd Conference of the New Zealand Society for Veterinary and Comparative Pathology. NZ Vet Journal 41(1):45.

NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY

FUTURE. The future of the NRDAP has been assured by the Animal Health Committee agreeing to fund the Registry for the next four years. In the current financial climate, that was a major achievement and the NRDAP Committee thanked all our supporters for their lobbying etc.

BLOCKS as well as HE stained slides are still required of common and unusual conditions in domestic animals.

TRANSPARENCIES of clinical and gross pathology would be appreciated! This is YOUR collection so YOU need to contribute to make it work. The International Veterinary Pathology Slide Bank has a motto 'Just take one more for the Registry'. It is far cheaper than duplicating and the quality is much better. DO IT FOR US!!!

COURSES in the next few months, pathology training courses will be held in each state. We will continue with avian pathology and the pathology of native species: review and show and tell sessions will be focused on bone pathology and skin pathology. If you have further suggestions please contact me.

COMPARATIVE ANIMAL PATHOLOGY REGISTRY

FUTURE. The veterinary services at Taronga Zoo are being restructured and that includes a review of the function, staffing and funding of the Registry.

NAME. The CAPR was given this name before I arrived; it is probably not the most appropriate name. Your suggestions and comments would be welcome. Attention is drawn to the following: it is housed at and funded by Taronga Zoo; more than 70% of the material relates to native species; it does not include domestic animals (and therefore, is not truly comparative); it was established by Bill Hartley; the material at the CAPR is indexed and catalogued, but is it a registry, that is, 'a central agency for the collection of pathological material and related data in a specified field of pathology, so organised that the data can be properly processed and made available for study' [Blood and Studdert (1988) Bailliere's Comprehensive Veterinary Dictionary].

PLEASE send further cases of common and unusual cases of pathology in native and exotic animals. NOTE that blocks and transparencies would be most welcome.

***** THESE ARE YOUR RESOURCES - USE THEM *****

AUSTRALIAN ANIMAL PATHOLOGY COLLECTIONS

As I was very largely involved in the initiation and implementation of both national collections and in view of the brief reference to my involvement in the November 1993 ASVP, it might be of interest to some members for me to summarise the pertinent facts relative to this involvement.

Regarding the Taronga Pathology Collection (inappropriately now named CAPR - but does not contain any goats): This after 8 years consists of a unique and fairly comprehensive collection of materials relating to the diseases - and includes normals - of mainly native terrestrial and aquatic fauna of the South West Pacific. It is used by undergraduates and postgraduates wishing to expand their knowledge and in lectures and training courses. It also receives many cases in a consultant capacity. So far 20 papers have emanated from materials contained therein. Some ASVP members continue to generously support it with case materials. I have bullied and badgered others to obtain current and archival materials with variable success. In the 7 years I was building this collection up, I examined about 75,000 sections and from these selected, reported, coded and filed on card and computer some 5,455 cases. In the last 15 months, since Rod Reece's arrival, 753 additional cases have been entered, of which I have contributed about 40%. (I was asked to stay on at Taronga as a consultant and to help Rod one day a week for 6 months. As at mid January 1994 I am still employed.)

Regarding the ASVP associated domestic animal pathology collection (DAPC): I initially made available to it, on loan, a mass of relevant materials which I had collected nationally and internationally over nearly 40 years. Many ASVP members kindly helped to extend this collection during its formative stages. In the 3 years I was involved with its creation I examined very many cases and from these 2,815 was selected, coded and filed. This material augmented with my transparencies, was used in many inter and intra-state training courses. I gave many second opinions and sometimes disagreed with a submitter's diagnosis. In the last 15 months 200 cases (mostly chooks) have been added to it. Quality being more important than quantity. About 50% of the histologic sections in this collection came from me and are housed at EMAI. I have a personal collection of around ten thousand colour transparencies covering many aspects of the pathology of domestic animals and this includes exotic diseases and poisonous plants. This is not housed at EMAI.

Any materials in my collection are available on request to approved pathologists or institutions for training for courses, lectures, etc.:- for as long as the DAPC continues to be fully financed, that I have priority of use of any materials contained therein and that I am still employed as a consultant at Taronga.

It has long been my hope that my personal collection of pathologic materials of domestic animals, which is quite specialised in some areas, should one day, in whole or part; find a permanent home in one or other of the Australian Veterinary schools. This is now under investigation.

Yes, I am still very much involved in investigative histopathology in the fields in which I have had considerable experience both on the national and international scene. I have a microscope at home and will be pleased at any time to look at tissue sections, to give a second opinion or share ignorance. This will make me a very proud old pathologist.

ADDENDUM

Mention was made in the same Veterinary Pathology Report that for reasons given, it was regrettable that native fauna material was being examined by medical pathologists. I am in general agreement with this. However, some readers may be interested to know that at the present (we are hoping for a change) almost no native fauna or zoological park case materials are made available from private veterinary pathologists to the Taronga Pathology Collection. Whereas, all pathologic materials from one large fauna park, which are processed and reported - at no cost - by medical pathologists, because they are interested in comparative pathology and have other associations with that particular park - are made available to me in a consultant capacity and selected sections are entered into the Taronga Pathology Collection.

The state of excellence and continued viability of the Taronga Pathology Collection depends not only on the experience and dedication of its staff but increasingly now on the interest and co-operation of private veterinary pathologists in making suitable materials available to it on a regular basis.

Bill Hartley, 42 Valetta Street, MOSS VALE 2577.

FURTHER FUNDING FOR REGISTRY

Current funding for the Registry finished at the end of August 1994. The Management Committee of the National Registry of Domestic Animal Pathology submitted a proposal for further funding of the Registry to the April 1994 meeting of Animal Health Committee. The Committee consists of the Chief Veterinary Officers of the Commonwealth, State and Territories plus CSIRO.

ASVP members around Australia lobbied their CVO's to support the Registry. Last month the effort was rewarded and we were delighted to learn that AHC had agreed to a further 4 years funding for the Registry.

The budget is "lean" and similar to the current period of funding. It includes a half-time salary for the Registrar, travel and accommodation to each state end territory in each year and small amounts for photography and histological processing.

NSW Agriculture has agreed to continue to house the Registry at EMAI and provide it with the usual secretarial end support services.

Concurrently the Zoological Society of NSW is considering continued funding of the Comparative Animal Pathology Registry, which is the registry for zoo and wildlife species.

ASVP is pleased that AHC has continued to recognise and support this important national initiative in domestic animal pathology.

Tony Ross, Chairperson, Management Committee, July 1994.

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

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Honorary Treasurer: Russell Graydon

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23 February 1993

Chairman
SCARM Working Group on Animal Health Services
C/- GPO Box 46
BRISBANE Q 4001

Dear Chairman,

AUSTRALIA'S LONG TERM ANIMAL HEALTH REQUIREMENTS

I write to you on behalf of the members of the Australian Society for Veterinary Pathology. Our members share a common interest in veterinary pathology and are drawn from both the public and private sector. They are employed in diagnostic laboratories, universities and research institutions throughout Australia and overseas. Our members are the providers of animal health services and are therefore interested stakeholders in the outcome of the SCARM review.

There are a number of issues which fall within the terms of reference of the review which concern our members. We offer the following comments for your consideration from a position of experience through our direct involvement in animal health services both within Australia and overseas.

Current situation:

Government animal health services in Australia are in a state of serious decline. Policies of economic rationalisation, non-replacement of staff, retrenchment and severe budget constraints have reduced staffing and resources in many animal health laboratories to critical levels. The continued depletion of resources can only compromise the ability of governments to monitor and report on the health status of Australian livestock.

Queensland now has fewer animal health staff than before the Brucellosis and Tuberculosis Eradication campaign which began in 1971. Other states have suffered even greater staff reductions, ranging from 19% to greater than 50% in some cases. This skeletonising of regional laboratories has created severe deficiencies in their ability to handle major endemic/exotic disease outbreaks. It has been argued that any deficiencies can be met by temporary secondment of staff from interstate and/or overseas, such as occurred in the recent Avian Influenza outbreaks in Victoria. However, this is clear evidence to our trading partners, the Office International des Epizootics (OIE) and the rest of the world that Australia's animal health services are bordering on being inadequate.

Australia has long supported the tenet that for agriculture-based third world economies to develop, an efficient animal health service is essential. It is ironic that Australia is providing financial and logistical support to develop animal health services in other countries, while Australian governments, state and federal, are complicit in dismantling the nation's regional laboratory network.

Over the past decade it has become obvious to our society that we are an ageing profession, largely due to the policy of non-replacement of staff by governments. Diagnostic laboratories traditionally have been the initial training ground for a majority of our members. If the present recruitment restrictions continue, Australia's animal health laboratory system will face a critical shortage of professional staff. Animal health laboratories in Australia are not fully recognised or utilised as the important training resource they are. In many other countries diagnostic laboratories are integrated closely with teaching institutions, often being physically housed within such institutions. The ASVP believes that animal health laboratories in Australia could be more effectively used as a training resource.

How should animal health services be provided?

We believe that any national animal health service must be based on a viable network of fully staffed and adequately resourced diagnostic laboratories, both public and private. The services provided by diagnostic laboratories underpin the field operations of any national animal health service. Government regional laboratories are involved in endemic disease monitoring (active and passive); surveillance for exotic disease; detection of new and emerging disease or shifts in endemic disease expression; support for disease control programs; health certification of live animal exports; support for veterinary public health programs and maintenance of professional standards and expertise. Our members in private veterinary laboratories also provide many of these functions, especially where companion and performance animals are involved. However, many of the public benefit functions of government laboratories are not cost-effective for private laboratories.

One failing of the present animal health system is the lack of a formal national reporting requirement and for this reason we fully support the concept of a National Animal Health Information System (NAHIS). The Animal Health Committee (AHC) Task Force Report on NAHIS has recognised the important role of regional laboratories in disease surveillance by recommending that NAHIS be based primarily on data from state/territory animal health field and laboratory services.

Elsewhere in the world our trading partners have diagnostic and research laboratory networks that use sophisticated laboratory methods and risk analysis techniques to monitor not only their own animal health status, but also imported Australian products. It is against these standards that our animal health services will be judged and it is against these standards we must compete. Our trading partners use both active and passive disease surveillance mechanisms to produce quantitative and qualitative assessments of their animal health status. In Australia, active surveillance is seen as an alternative rather than an adjunct to the passive surveillance role of diagnostic laboratories. It is unfortunate in Australia that many state governments view active disease surveillance programs as budget control measures rather than as quantitative disease monitoring systems. While active and passive disease surveillance programs have strengths and weaknesses, together they form a complementary system which when used to its full potential provides an effective and efficient animal health system.

To **not** provide a viable animal health system in Australia risks our trading partners invoking OIE requirements, for a national disease reporting capability, as a non-tariff trade barrier to our livestock industry exports. The inability of countries to substantiate disease freedom statements was a matter for heated debate between American and Mexican delegates at the 1st International Conference on Porcine Reproductive and Respiratory Disease, Minnesota. Diseases, residues and food quality restrictions (the most recent example being *E.coli O157:H7* contamination) will increasingly be the trade barriers of the future.

How should the service be funded?

It is the view of the ASVP that the beneficiaries of animal health services should fund the service in proportion to the benefit received. We submit that the major beneficiaries of the animal health laboratory system in this country are the state and federal economies. Laboratory functions such as endemic and exotic disease surveillance, support of veterinary public health programs, endemic disease control, exotic disease preparedness and maintenance of market access all produce benefits that accrue to the nation. We therefore believe that a community responsibility exists for governments, both state and federal, to accord animal health services the appropriate priority and funding they deserve in an agriculture-based economy such as ours. Increasingly it appears that governments are prepared to abrogate their community responsibilities in favour of short-term fiscal gains. While the Australian economy continues to be reliant on export revenue generated by our primary industries it is indefensible to fail to meet such responsibilities.

The most serious threat to the animal health services in this country is posed by the theories of economic rationalists. Born out of financial mismanagement, their theories dictate that governments are not responsible for community needs and that the "user pay" philosophy should apply irrespective of the outcome. Many of our members in South Australia, Victoria and Tasmania know from bitter experience the consequences of the blind adherence to the "user pay" philosophy. Attempts at full-cost recovery by government laboratories inevitably lead to a shift in emphasis away from commercial livestock toward companion and performance animals. This is the province of our private sector members who are better resourced to service this area. When the induced shift away from their core business occurs in government laboratories it provides justification for the continued down-sizing of the service. What is not appreciated by the powers-that-be is the degree of loss of essential endemic disease and animal health surveillance information.

We recognise that industry also benefits from animal health service activities and where substantial private benefit is gained then clearly a case can be sustained for industry bodies and/or individuals funding these activities. Already significant revenue is collected from industry for disease accreditation schemes, show/sale/movement testing, health monitoring and export testing. It is a fact, that many individuals are prepared to accept the cost of a service where there is a direct private benefit to the individual; however, it is rare that individuals are prepared to accept the cost of services linked only to public benefit. This is rightly the obligation of governments.

Australia enjoys a privileged animal health status being free of many of the major disease scourges present in other countries. It would be a tragedy for all Australians if the first cases of an exotic disease are missed because the cost to an individual outweighed the perceived benefit to the community.

No doubt many of our comments have been expressed by others, but as a society whose members have a vested interest in the future of animal health services we are keen to contribute. I am happy to expand further on any of the points mentioned in this submission.

Yours sincerely,

John Gibson
PRESIDENT
AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

CENTAUR INTERNATIONAL PTY LTD

Centaur International Pty Ltd is a newly established independent company specialising in providing laboratory based diagnostic services to the livestock industries, the agricultural community, private veterinary practices and government agencies.

Centaur has recently taken over the management and operation of the four veterinary diagnostic laboratory facilities previously operated by the Department of Agriculture. The facilities are located in regional Victoria in the rural centres of Hamilton, Bendigo, Benalla and Bairnsdale.

Centaur's aim is to manage the laboratories as a single entity, to encourage the development of scientific excellence at a regional level and to expand and improve the services delivered to include contract research and development, and environmental assessment and management. Diagnosis veterinary pathology will, however, remain the core function of the laboratories.

There are currently 60 specialist and technical staff employed by the organisation and Centaur aims to expand this to a level of approximately 200 by 1998.

Applications are invited for the following positions:

Laboratory Director, Bendigo

We are seeking an experienced laboratory veterinarian with:

- a high Level of motivation and positive client interaction skills
- an ability to recognise opportunities for expansion
- the ability to co-ordinate and motivate a team of experienced veterinarians and scientists

Specialisation and broad experience in veterinary pathology and clinical pathology are essential. Post graduate qualifications are highly desirable.

Veterinary Pathologists

Centaur International is seeking highly motivated veterinarians to join a diagnostic team servicing the needs of both the agricultural industries and companion animals throughout Victoria. Specialised clinical pathology and/or pathology experience is desirable; however, positions and training also exist for recent graduates with an interest in these areas.

Veterinary Parasitologist

A position exists at the Hamilton Laboratory for a veterinarian with experience and/or post graduate raining in veterinary parasitology. Knowledge of the sheep industries and farm management would be an advantage.

Attractive remuneration packages will be offered commensurate with qualifications and experience. Interstate and overseas applicants are encouraged to apply for any of the above positions.

Employment with Centaur offers successful candidates an unparalleled and exciting opportunity to be involved in the start-up of a new company.

For applications or further information please contact:

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