AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

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VETERINARY PATHOLOGY REPORT
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EDITOR: Cleve Main

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ASVP EXECUTIVE 1997-98

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APPOINTMENTS

Chairman - Registry of Domestic Animal Pathology	Tony Ross
Newsletter Editor	Cleve Main
Working Party on Proficiency testing	Barry Richards (convenor)
	John Mackie
	Roger Kelly
	Judith Nimmo-Wilkie

CONVENOR - SLIDE OF MONTH

Rod Reece	EMAI, Private Mail Bag 8 CAMDEN NSW 2570	02 4640 6333

President's Report

The AGM in Melbourne in May was a great success. It was my first conference for a number of years and I thoroughly enjoyed the science and the ability to renew old friendships and make some new ones. On behalf of the members I wish to thank Ron Slocombe and Karl Harrigan for organising the conference and putting together a stimulating programme. I would also like to thank the sponsors whose contributions go a long way to making the conference a financial success. The sponsors this year were VPS Brisbane, CVDL Melbourne, Victorian Veterinary Pathology Services and AAHL(CSIRO).

There are a few items from the AGM that need highlighting. Firstly, the resignation of Clive Huxtable from the Presidency. Clive has moved to Cornell University and was unable to carry on as President. This is most unfortunate for us as he has always been a very active member of the ASVP and he was an active President. I was very sorry to see him go after 20 years of collaboration although I must admit that after the fourth farewell I was ready to push him on to the plane. We all wish you and Jill great times Clive and hope you are back with us in a few years. Clive is now our official North American correspondent. Let's hope he doesn't end up like Alistaire Cook who now seems to be sending his Letters from America from the grave.

The second issue that I want to mention is the formation of a Working Group on Professional Standards. The group consists of Roger Kelly, John Mackie, Judith Nimmo-Wilkie and is convened by Barry Richards. The terms of reference are to expand and develop the preliminary business plan entitled Animal Pathology Standards in Australia. It is the plan of this group to have their report ready for decisions to be made at next years AGM which will be held on Saturday and Sunday 24th and 25th June 2000 in the Esplanade Hotel in Fremantle.

Yes, next years AGM is in Perth and for all those who will groan and say "that's a long way to go" it's no further than we have to go each year. Start planning to be here and start organising something to present because it's going to be a terrific conference.

That's all I have to say at the moment. It's good to be more actively involved with the ASVP again having spent most of my time with lab animal people over the last 11 years, but they are a great bunch too.

Regards

Dave Pass
Honorary President 1999/2000

EDITORIAL

Well, this is the first issue of the Veterinary Pathology Report for 99/2000. The 1999 Annual General Meeting is receding into the distance. We have a new President, David Pass, and we have said farewell to Past President Clive Huxtable who has now become our North American correspondent.

In this issue are the Minutes of 1999 AGM and all associated reports. Also included are some comments from our Treasurer regarding payment of membership dues. Note that overseas members now have the opportunity to pay by credit card. Jeremy has also included a financial statement which shows that the Annual Conference made a slight profit. Well done Ron Slocombe, Karl Harrigan and all those who helped make it the success it was. Thank you also to the conference sponsors whose support was so vital.

As you will see from the minutes, your Executive has been given some work to do. The question of medical laboratories carrying out veterinary testing was debated. A working group has been formed to look into development of continuing education with a long term objective of maintaining proficiency standards. The Society has become an Associate member of FASTS and we will use the next year or so to assess the benefits. We have been asked to ensure that the histopathology collection at Hamilton is retained for archival purposes.

The future of the Slide of the Month Service provided by the Registry was also discussed. Advice has been given to the Executive that many people and Institutions who regularly receive the slides do not actually contribute cases and some Australian laboratories are reluctant to contribute because of the growing costs in terms of preparing the slides and posting them out.

There are probably many of you that do not need to receive a personal set each month because the laboratory that you work in already receives a set. A large proportion of the sets prepared each month go to overseas recipients and while their interest is appreciated, we must find ways to recoup some of the expenses in providing that service. Many of the overseas recipients are in fact, not members of the Society.

As an initial step to try and rationalise the service, we are asking each person or Institution whether they wish to continue to receive the Slide of the Month. If you wish to continue receiving a set, please consider contributing to the service by offering to provide a case (mammalian and avian/piscine) when asked to do so. All overseas recipients who are not ASVP members will be contacted and asked the same question. The matter must be resolved and I am asking you to indicate on the form supplied with this issue of the Report whether you wish to continue to receive the SOM, and whether you are willing to contribute a case once every year or so.

Cleve Main
Honorary Secretary

Subject: FASTS Release

"SCIENCE MEETS PARLIAMENT" DAY

Australian scientists will descend in numbers on Parliament House in Canberra later this year, but there won't be a white lab coat in sight.

The Federation of Australian Scientific and Technological Societies (FASTS) has set Wednesday November 24 as the day when 150 scientists and technologists will meet their local Members and Senators at Parliament House.

FASTS' President, Professor Peter Cullen said scientists have one clear message to deliver to Parliamentarians: funding for research and development is an investment in Australia's future.

"Science, technology and innovation should be at the centre of the Australian economy," he said, "Research isn't a cost, it's an investment.

"Studies in the USA show how strongly industry depends on public science in developing the next generation of products and processes. Every year in the USA, 180,000 jobs are created as technology is transferred from the laboratory to industry.

"The journal Nature reported a recent economic analysis which suggests that half the fastest growing US companies are ‘knowledge companies’, selling the knowledge and skills of their employees rather than manufacturing products or providing services.

"Australia's future in a tough world depends on science and technology."

He said politicians needed the best possible advice on science and research, because so few of them had scientific backgrounds.

Our politicians are former lawyers, economists, teachers, farmers or trade unionists-hardly a scientist among them," he said. "On November 24 we’ll be knocking on their doors, with solid examples of how scientific research has benefited the nation."

Professor Cullen said Parliament House would be a labcoat-free zone on November 24.

"Labcoats and words like ‘boffin’ exist largely in the mind of Hollywood. Scientists and technologists aren’t remote academic Figures - they are real people, with real solutions to the problems that confront Australia today," he said.

The "Science meets Parliament" Day is based on the Congressional Visits Day in the US. Last year, 200 scientists and engineers from across America made more than 215 visits to House and Senate offices to underscore the long-term importance of S&T to the nation.

The American scientists from research and educational institutions were joined by others from the world of business.

Professor Peter Cullen said that scientists wishing to participate should contact their Society.

He has written to the Speaker and the President of the Senate seeking their advice on how to make the day a success. Plans included a reception for all Members and Senators and the launch of FASTS’ new Policy Document.

Professor Cullen said that he was inviting other bodies with an interest in boosting the presence of scientific research in the Australian economy and environment to join FASTS on the day.

Comments from the Treasurer

I would like to thank all members who responded so well to the accounts for subscriptions sent out in April/May this year. A list of those who still haven't paid their 1999/2000 subscriptions is presented below.

For the interest of members I also include an Income/Expenditure statement for the annual conference. Ron Slocombe and Karl Harrigan need to be congratulated for bringing in an excellent conference on budget.

We have had enquiries in the past from overseas members about the development of an easier means of paying their subscriptions; I have arranged that overseas members can now pay their subscriptions by credit card, using the payment voucher presented below. Please note that this is only for overseas members and the vouchers must be sent to me.

Financial Statement for the Annual Conference 1999

Income		
Registrations	$3290	
Sponsorship	$1700	
		$4990
Expenditure		
Hire of venue	$1160	
Catering	$2680	
Honorariums and expenses for invited speakers	$1043.25	
		$4883.25
Surplus		**$106.75**

(Printing of the Proceedings was a Society cost because all members get a copy. The printing cost $606.56.)

Members Who Have Not Paid Their 1999/2000 Subscriptions
(as at 13 September 1999)

Alley, MR
Badcoe, LM
Bailey, G
Beers, P
Boulton, JC
Car, BD
Carlisle Melissa
Carrigan, M
Charles, JA
Chick, B
Donnelly, TM
Dowling, LA
Drew, C
Duff, BC
Forshaw, D
France, MP
Fraser, G
Friend, SS
Genovese, LM
Gibson, JA
Gleeson, LJ
Graydon, R
Hamid, H
Harrigan, KE
Havadjia, A
Hooper, PT
Howell, JMC
Jardine, J
Jerrett, I
Johnston, KM
Johnstone, AC
Kabay, MJ
Ketterer, PJ
Laing, RA
Lamont, D
Lee, JM
Lilly, E
Lording PM
Mackie, J
Marshall, Jeff
McGavin, MD
McNicholl, KA
Miller, RI
Mitchell, G
Muntz, F
Norman, RJ
Nunn, MJ
Obendorf, DL
Pass, DA
Pierce, RJ
Richards, RB
Robinson, W
Rose, KA
Rozmanec, M
Seward, D
Sims, LD
Smith, HV
Straube, E
Sullivan, N
Taylor, DP
Taylor, JD
Thomas, J
Townsend, W
Trueman, KF
Watson, J
Watt, Da
Webber, JJ
Whittington, R
Williams, OJ
Woodgate, RG

Note: This list only refers to fees for 1999/2000

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY
ANNUAL GENERAL MEETING - MAY 1999

PRESIDENT'S REPORT

As we gather for another AGM, like me you are probably saying, "where did that year go to"? Go it certainly did, and it is time now to look back and see what has or has not been achieved by the Executive on behalf of the Society. In general it has been a year of debate and planning but of frustratingly little concrete progress.

First an expression of thanks - to the members of the executive for their effort and commitment, to those society members who have fed in comments and ideas over the past year, and to Ron Slocombe and Karl Harrigan for organising the conference.

Thanks too to Phil Ladds for delivering the Continuing Education "road show" and his report to the Society on the issues of CE and QA based on a questionnaire and discussion with our members. In recommendation 5.5 of that report, a consensus view is documented of the need to link proficiency testing with continuing education and professional development. A discussion paper on principles for proficiency testing was included in the Veterinary Pathology Report number 51 last December, and I trust members have given it their attention and thought.

I hope we can resolve several important issues at this meeting. In my opinion the priority order for resolution of these is first to decide on the substance and format of a QA/CE/PT/PD programme (see Ladds report recommendation 5.2), and to then move to consider how it should be funded and who might deliver it. Another pressing issue is to establish minimal qualifications required for recognition as a "veterinary pathologist", and at a higher level, as a veterinary pathologist qualified to sign-off on final reports. Members of the Society have expressed a range of views. In this context the Ladds report makes the point (item 3.4) that "by 2001 NATA will require a signatory in anatomical pathology to be a registered Specialist Veterinary Pathologist, or be an MACVSc, or have a higher degree in a relevant discipline".

If this meeting can produce a resolution of these issues it will have achieved much.

Last year I expressed the view that it was important for us to have a clear definition of ourselves, and I believe this now exists within the substance of our submission to the AAHC national review of laboratory services. This submission was also included in the Veterinary Pathology Report 51 and I commend it to the attention of members. The point is made that while anatomic pathology is at the core of our interest, the Society emphasises the essential links with allied disciplines. This leads me to own up to some of the objectives for the last year which were not achieved, namely the production of a booklet or pamphlet about the Society similar to the one produced by the American Association of Veterinary Laboratory Diagnosticians, and a web home page. It is hoped that both of these will eventuate in the coming year, and will utilise the material referred to above.

At last year's AGM, the "APSA" initiative received some pointed criticism, but was accepted in principle along with a number of provisos. The Executive was hopeful that AAHC would quickly act on the recommendations of its national review group, but regrettably this has not happened and the process seems to be stalled. At this point there is no clear indication of when a decision might be made.

The executive was in contact with the ACVSc regarding its requirements for candidates undertaking Fellowship training. Our viewpoint is that the current requirements are inhibitory to many of our members and unrealistic in the Australian context. This issue remains unresolved and the Executive seeks the opinion and ideas of members for future action.

During the year the Executive established contact with FASTS (Federation of Australian Scientific and Technological Societies) and as a result a representative of that organisation will speak at our conference. Members should then have a better idea of what FASTS can offer the ASVP.

The executive also pursued the question of affiliation with the C.L. Davis Foundation. I was in touch with Prof. Don McGavin who has long been acting as a link for us in the US. After consideration of available information the executive decided that affiliation was not a priority for us at this stage, and will not be sought.

On a final personal note, it is probably known to most members that I am departing these shores for a time and will not be able to complete the final year of my term as President This I regret, but I will certainly keep an active interest in the Society while overseas and will be more than willing to further its interest in whatever way I can.

Clive Huxtable
Honorary President

SECRETARY'S REPORT

Committee met six times during the year, dealing with a wide range of topics including membership of the C.L. Davis Foundation, FASTS, APSA and quality assurance. Other matters included the ACVS fellowship training program, the continuing role of the Slide of the Month series and the role of medical pathology laboratories in carrying out veterinary work. Some of these topics are listed for discussion under general business.

Several matters arising from the Sydney AGM have been dealt with by Committee. Matters regarding APSA and QA have been already been discussed in the President's report to the meeting. As agreed at the Sydney AGM, Committee looked into affiliation with FASTS (Federation of Australian Science & Technology Societies). The presence of FASTS representative Prof. Snow Barlow at this conference is a manifestation of our continuing dialogue with the Federation.

The membership list has increased slightly to 157 members, but that includes people who are not financial and may have their names removed in the very near future. The Brisbane AGM determined that people who were not financial for 2 consecutive years should have their membership terminated. It is pleasing to see that members moving overseas or returning home to other countries are electing to remain with the Society. Unfortunately there are still many of us that have not paid their 1998/99 subscriptions and this problem needs to be resolved. I cannot believe it results from deliberate attempts to avoid paying. More likely it is a consequence of not receiving a formal invoice towards the end of the financial year. This was largely due to the previous perilous state of our membership database, a problem which has now been fixed. Nevertheless we have several names on our list with no address. Some of these are dormant and will be removed, but there are a few people who paid their subs last year but whom we do not have a mailing address for. These people cannot be receiving their ASVP Report and it does not seem to worry them. Remember, if you change your address, please let me know.

Most of you will be aware of the problems that occurred with the South Australian Secretariat last year. These arose from the absence of a Manager and a consequential lack of direction and guidance for the then office secretary. All of this resulted in failure to maintain an up to date list of members, failure to issue invoices and failure to bank many of the subscription cheques that were received. The appointment of a new manager and office secretary together with personal representations on the Society's behalf by Martin Copland and Rob Rahaley saw a huge improvement in performance. The state of our records at the Secretariat has improved to an extent that their records and those of the Executive are almost reconciled. Many thanks go to their new staff member Chandra Atkinson who has worked hard to restore them to a credible state.

Cleve Main
Honorary Secretary

LEWIS & WALKER
CHARTERED ACCOUNTANTS

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AUDIT REPORT

We have audited the attached statement of Income and Expenditure for the financial year ended 30 June 1998 for the Australian Society for Veterinary Pathology.

The scope of our audit has included verification of actual cash receipts and expenditure only together with verification of the bank account balance as at 30 June 1998. We have not examined any other assets or liabilities which may be in existence or any figures or balances of previous years.

In our opinion the cash book of the Society has been regularly kept during the year and the attached Statement of Income and Expenditure has been properly drawn up to reflect the cash position of the Society as at 30 June 1998.

LEWIS & WALKER
Chartered Accountants

DAVID GEORGE KERR
(Director)

Dated: 30/4/99

Australian Society for Veterinary Pathology

Annual General Meeting May 1999

TREASURER'S REPORT

Financial Members

As at 1 April 1999 the Society had 106 financial members.

Income and Expenditure

Two sets of Income and Expenditure Statements are presented. The first is the Audited Statement for the 1997/98 financial year (the Auditors Report is attached), and the second for the current financial year to 1 April 1999. The full audited Statement for 1998/99 will be presented at the AGM in 2000.

Statement of Income and Expenditure of ASVP for 1997/98

Opening Balance			**$6,307.07**
INCOME			
Subscriptions		$3,705.00	
Conference (1997) Registration and Income		$3,710.00	
Advertising in VPR		$150.00	
Interest	$32.63		
		$7,597.63	$13,904.70
EXPENDITURE			
Veterinary Pathology Report		$1,403.09	
Secretarial Services		$570.00	
Conference (1997) Proceedings (printing)		$382.42	
Conference (1998) Deposits and Mail out		$604.60	
President's Attendance at the 1998 AGM		$591.40	
Representation at the Veterinary Pathology QA Workshop		$1,414.20	
Ministry of Fair Trading (Incorporation requirements)		$64.00	
Bank Fees and Charges		$218.46	
		$5,248.20	
Closing Balance			**$8,656.50**

Notes: 1. Most expenditure for the 1997 Conference fell in 1996/97, whereas all income fell in 1997/98.

2. Some expenditure for the 1998 Conference fell in 1997/98, while other expenses and the income fell in 1998/99.

Statement of Income and Expenditure of ASVP from 1 July 1998 to 1 April 1999

Opening Balance			**$8,656.50**
INCOME			
Subscriptions		$1,380.00	
Conference (1998) Deposit Repaid		$500.00	
Conference (1998) Surplus		$680.39	
WA Pathology Seminar Surplus		$60.00	
Interest	$10.31		
		$2,630.70	$11,287.20
EXPENDITURE			
Veterinary Pathology Report		$792.50	
Secretarial Services		$225.94	
Conference (1998) Proceedings (printing)		$521.00	
Ministry of Fair Trading (Incorporation requirements)		$32.00	
Bank Fees and Charges		$171.19	
		$1,742.63	
Closing Balance			**$9,544.57**

Notes: 1. Subscriptions are down because not all members have paid for 1998/99, and the invoices for 1999/00 subscriptions were sent out in April.

2. The Secretary has implemented cost saving changes in the preparation of the Veterinary Pathology Report, and the cost of only two are included in the period reported.

3. Expenditure for the 1999 Conference has fallen outside the period reported.

4. The Bank has refused our application to have Bank Fees waived for this account. Consideration may be given to moving the account to a bank or building society that will not charge us fees.

5. During the year the Secretariat had problems with staff changes and the ASVP records they had became quite disorganised. It took several months to remedy the situation and during this period no fees were charged. The first account for services in 1998/99 was received in April and does not appear in the report above.

Jeremy Allen
Honorary Treasurer

BULLETIN EDITOR'S REPORT

Three editions of the Report were issued during the year. All submissions have come via e-mail in Word format and as far as I am concerned, this method really is the "way to go". It simplifies compiling and formatting considerably. The only glitches that have occurred have been due to human error at this end and I apologise to members in New South Wales and Victoria whose contributions were not included on two occasions. From my point of view these were serious errors as I have more than once berated the membership for not contributing to the Report.

Thirteen members now receive their Reports by e-mail and as to date there have been no adverse comments. The first attempt revealed several unforeseen problems. Despite repeated attempts "failed transmission" messages kept appearing on my computer screen. This problem appears to have been resolved by deleting graphics in the form of Society and State logos from the Report before it is sent. Any form of graphics adds considerable size to the file. I would appreciate feedback from those members regarding electronic Reports. Positive or negative, I want to know.

Some members are still not receiving their reports. In several cases this has been due to the poor state of our membership mailing list. This problem has almost been resolved, but there are a few members whose mailing address is not listed on our books. If any of you in the audience is one of these or you know of some member who is not receiving their Report, please notify me.

Finally I must thank all of you who at some time during the year contributed to the Report. Without your efforts there would be no Report. Special thanks must go the State representatives and to the West Australian representative David Forshaw for his efforts in getting the material to me. I must also acknowledge the efforts of Susan Edmonston-Fearn for her work in compiling and formatting the Reports.

Cleve Main
Honorary Editor 5 May 1999

REGISTRY REPORT

Your National Registry Management Committee delivered continuing education courses for 1997/98 by contracting Dr Philip Ladds to present them in each state and territory. The major theme was the male and female reproductive tract of domestic animals and the minor theme was the emerging Menangle Virus disease in pigs.

The 1998/99 courses are being delivered in April - June 1999 by Dr Philip Ladds. He has contracted to provide a course on the lymphoreticular system of domestic animals. The minor theme was Avian Influenza and Newcastle Disease. The Chief Veterinary Officer of NSW Dick Jane agreed to pay for a training set of slides for Avian Influenza and Viral NDV for each state and territory. Funds were provided from the cost sharing agreement for NDV eradication.

Suggestions for course themes in 1999/2000 are welcome (Ph: 02 4640 6312).

The position of part-time Registrar has not been advertised on the advice of the executive and the committee. It was hoped that by late 1998 or early 1999 the Australian Animal Health Council would have formally recognised the need for continuing education in veterinary pathology. The Society would then put a joint bid to both AAHL and Veterinary Committee for further funding and support. It is hoped this opportunity will arise during 1999. In the meantime there are adequate funds on hand to contract an eminent pathologist and pay for travel to each state in 1999/2000.

The committee thanks Dr Rod Reece for continuing (in the absence of a Registrar) to co-ordinate the slide of the month service and answer enquiries concerning the Registry. The Registry collection is available for study for those who wish to visit it at EMAI.

A. D. Ross
Chairperson

Australian Society for Veterinary Pathology
Minutes of Annual General Meeting
Held at the University of Melbourne
Swanson Street Melbourne 4.50pm 15/5/99

ATTENDANCE - 26 members

APOLOGIES - Phil Ladds, Jenny Charles, J. Jardine, Terry Nicholls, Terry Rothwell, Peter Hooper, John Glastonbury, Steve Hum, Dick Sutton, Wayne Robinson, Don Seaman, Jeff Marshall, John Jordan, Roy Mason, Rob Rahaley, Leslie Reddacliff, Rod Reece, Mary Barton.

MINUTES OF 1998 AGM - Moved R Miller/ J Finnie - Accepted.

BUSINESS ARISING -

Histopathology Collection, Hamilton - Janeen Samual voiced her concerns over the fate of the histopathology collection (blocks & slides) remaining at Hamilton. A discussion on the value of the collection followed. Tony Ross advocated that the collection be reviewed and that only the valuable slides and blocks be retained. It was suggested that Bill Hartley might carry out the review. J. Samual emphasised that there was nobody left in Victoria who was able to carry out the review. Furthermore, permission from the CVO Victoria would be required for somebody to access the collection.

ACTION - ASVP Executive to follow up.

ASVP Training Committee - Clive Huxtable advocated that training courses such as the one presented by Phil Ladds on the reproductive system could be put onto a CD.

ACTION - Executive to discuss and inform the membership.

Involvement of allied disciplines and their relationship to the ASVP - Some of the membership voiced concerns that the proposed Australian Pathology Standards Association (APSA) will carry out functions which should be done by the ASVP. The President informed the meeting that APSA will be an arm of the Society, not a separate and competing organisation.

PRESIDENTS REPORT - In his report, the President acknowledged Phil Ladds report on quality assurance (QA) and continuing education (CE). The Society's priority is to define the substance and format of QA, CE, professional development; its funding and delivery. In NATA accredited laboratories, signatories to pathology reports need to have a higher qualification. After touching on other aspects of his written report, the President informed the meeting he was not able to offer himself for re-election as he was about to leave Australia to take up a post in the USA.

Moved T. Ross/J Mackie that the report be accepted and that with his agreement, Clive becomes a North American correspondent for the Society - Accepted.

SECRETARY'S REPORT - Referring to his written report, the secretary outlined some of the problems related to the Secretariat based in the AVA SA office. He reported that things had improved with the appointment of an office manager and a new secretarial officer. He also alluded to the fact that many of those listed as ASVP members were not financial.

Moved P Phillips/R Kelly - Accepted.

TREASURER'S REPORT - In talking to his report, the Treasurer outlined the sound financial position of the Society, and supported the Secretary's comments regarding unfinancial members. He also attributed some of the Society's position to the fact that the costs of producing ASVP Report had been kept to a minimum. He had no success in his attempts to convince banks that the Society should be exempt from bank fees. A general discussion on bank fees and Government charges followed and it was pointed out that the Registry account is free of Government charges. The meeting suggested that the Treasurer move the account to a Building Society or something similar.

Moved K Harrigan/ J Mackie - Accepted.

EDITORS REPORT. The editor referred to minor problems which occurred during the year and apologised for the fact that on 2 occasions, articles submitted for publication had not been printed. As instructed at 1998 AGM, members now have the opportunity of receiving the Report by email. Thirteen members now receive their report by this method.

Moved K Harrigan/T Ross -Accepted.

SLIDE OF THE MONTH AND REGISTRY MANAGEMENT REPORT

Speaking to his report, the Chairman informed the meeting that the Management Committee had contracted Dr. Phillip Ladds to deliver a series of CE courses on the male and female reproductive systems in 1997/98 and the lymphoreticular system in 1998/99. A set of training slides on AI and NDV would be provided to each State and paid for out of the cost sharing agreement for NDV eradication. The Committee thanked Dr. Rod Reece for his continuing work on the Slide of the Month Series.

Moved P Phillips/ R Kelly - Accepted.

CORRESPONDENCE

Inwards —
Geoff Mitchell - Role of Medical Pathology laboratories. *
John Mackie - Proficiency testing. *
Malcolm France (email) - Acknowledgement of pathologists in publications. *
Roger Kelly - Brisbane as venue for ASVP AGM 2004. *

* Listed as items to be discussed under general business.

Veterinary testing carried out by Medical Laboratories.

The secretary1 explained that the item was put on the agenda following receipt of a letter of concern from VPS Laboratories in Brisbane. Karl Harrigan told the meeting that during his time as secretary, he had written to the Queensland Veterinary Surgeons Board about the problem. Clive Huxtable mentioned that some veterinarians are reporting that Medical Laboratories will no longer carry out tests for them and that is causing some problems. Tony Ross commented that laboratories should cross refer. Jeremy Allen told the meeting that AHL Western Australia submitted material to laboratories for testing and AHL in turn, often do tests for medical labs. No problems were experienced because neither group attempted to make a diagnosis based on their results, they merely reported them. A general discussion took place and it was generally agreed that as long as the medical laboratory made no attempt at diagnosis, no breach of the Vet Surgeons Act would occur.

ACTION - Members to write a letter to offending laboratories if they so wish, pointing out that when they provide a diagnosis they are in breach of the Veterinary surgeons Act.

Proficiency Testing

Inclusion of this item followed a receipt of a letter from John Mackie from the Brisbane VPS Laboratories. Speaking to his letter Dr. Mackie pointed out that a fish pathologist may not be good at examining sections of dog skin. Proficiency testing should concentrate on appropriate training and qualification and good quality continuing education. The ASVP should support and provide good quality CE. In conclusion he agreed that the steps outlined in the draft document were generally OK.

Clive Huxtable pointed out that proficiency testing was not going to be in the form of a High Bar examination. The Society needed to agree to a model and test it. There would be no penalty for failure. Corrective procedures are likely to be in the form of continuing education.

Barry Richards added that the Government was no longer in the game of training pathologists. As things stood the quality and service provided in support of the livestock industry is likely to suffer. North America went down the "qualifications" track but Australia doesn't have the capacity to train to this level. The number of people going into pathology is decreasing alarmingly.

Judith Wilkie offered the opinion that Proficiency Testing should be part of Continuing Education and that a set of training slides would be of value - even across areas of interest. The set could be linked to "stated learning objectives".

Barry Richards added that there was documentary evidence that standards are falling and that a small working group should be formed now. The impression that standards were falling was supported by Tony Ross and Janeen Samuals especially in the area of gross pathology.

Both Roger Kelly and Ron Slocombe agreed that proficiency testing was a minefield and advocated that CE was the "way to go".

Barry Richards gave an update on the slow progress of the Society's submission to AAHC. Ron Slocombe added that AAHC by its failure to support the conference had shown it had little interest in the future of veterinary pathology.

Clive Huxtable added that we should retain the concept of proficiency testing, but keep it in the background. He supported the idea of a working group.

The following members were proposed as members of the working group - Barry Richards (convenor)

John Mackie
Roger Kelly
Judith Nimmo-Wilkie

The group will decide its own terms of reference.

It was suggested that in broad terms the Society establish this working group to look into development of continuing education with long term objective of maintaining proficiency standards.

The members nominated were duly elected.

Slide of the Month - The Secretary outlined the problem experienced by the coordinator. Some laboratories although willing to receive the SOM are reluctant to provide a set of slides for distribution amongst the membership because of the expense in terms of labour, postage and preparation.

A general discussion followed in which it was suggested that the list be "culled". Tony Ross suggested a 2 tier system where those institutions that chose not to contribute would pay to receive the slides and those which did contribute did not.

J Wilkie commented that that the slides are far too cheap (part of membership) and suggested there should be a financial contribution from each laboratory.

ACTION Rod Reece to provide a "State of the Nation" report for the next issue of the ASVP Report.

ACVSc Training Program.

Clive Huxtable and Barry Richards advised the meeting of the problems related to pathologists wishing to sit for Fellowship examinations. J Mackie asked what the Veterinary Surgeons Board true requirements were. The guidelines were formed in the 1980's .

Note: No resolution made.

FASTS

The meeting discussed the address by FASTS representative Professor Snow Barlow. The suggestion by Tony Ross that the Society take up Associate Membership for 2 years was accepted

Moved D Pass/' B Richards - Carried.

Sponsorship

Ron Slocombe raised the matter of sponsorship and pointed out how valuable the generous assistance of VPS Brisbane, AAHL Geelong, Victorian Veterinary Pathology Services and the Central Diagnostic Laboratory Melbourne had been to conference.

President Clive Huxtable expressed the Society's gratitude and the meeting responded accordingly.

NOTE: Agenda items relating to correspondence received from Malcolm France and Roger Kelly was withdrawn because of time limits.

ELECTION OF OFFICE BEARERS

President - David Pass being the only nominee was duly elected.

All other positions were duly re-elected

Venue of Next Meeting.

It is has been customary for the AGM to be held in the same city as that of the AVA conference. This year was an exception because the previous AGM had decided that insufficient members would go to Hobart to ensure a good conference. Tony Ross suggested that for the same reasons, the year 2000 conference should not be in Perth. This view was supported by several members, but a straw poll conducted indicated that sufficient members would indeed travel to Perth.

The meeting concluded at 7.00 pm.

Victoria - Malcolm Lancaster

Encephalomalacia in chickens - Malcolm Lancaster VIAS Attwood

Several groups of week-old chickens showing neurological signs have been investigated over the last year, from different properties. The common histological finding is the presence of large areas of malacia in the brain stem and sometimes in the cerebrum and optic lobes, often with a symmetrical distribution. Hyaline thrombi are present in capillaries in affected areas. This condition has been attributed to *Enterococcus* infection by European workers, but the disease has not been reproduced with bacteria of this genus. Enterococci have been isolated from cases at VIAS. The condition needs to be differentiated from Vitamin E deficiency, which characteristically affects the cerebellum in birds older than 10 days.

Chronic Active Hepatitis and Copper Storage in Blue Heeler Dog - Judith Nimmo Wilkie, Victorian Veterinary Pathology Services.

An eight-year-old speyed female blue heeler presented with a presumptive diagnosis of hepatocutaneous syndrome and elevated liver enzymes for the past two years. At the time of presentation the levels were ALP 787 IU/L (23-212), ALT 958 IU/L (10-100).

A liver biopsy revealed a chronic active hepatitis with lymphocytes accompanied by neutrophils in the portal areas and infiltrating the parenchyma in places in association with a piecemeal necrosis. There were also random fatty nodules some surrounded by lymphocytes and neutrophils as well as random clusters of neutrophils. Foamy pigment-bearing macrophages surrounded by mixed inflammatory cells were present in some portal areas. Hepatocytes contained refractile greenish brown granules. The granules were positive for copper with rhodanine stains. The granules were negative for bile, lipofuscin and haemosiderin with special stains. Portal macrophages and Kupffer cells were strongly positive for haemosiderin however. Liver copper was 7mmol/Kg wet weight (normal range 0.08 -2.09).

The dog was placed on a low copper diet, zinc acetate and trientin (a copper chelator). After three months of treatment liver enzymes were significantly lower (ALP 264 IUI/L and ALT 264 IU/L).

It is not known whether this is a primary copper storage disease (which has not been described in this breed) or whether this is secondary to the liver damage. Copper levels are often elevated in Dobermans with chronic active hepatitis and elevated levels may precede onset. The role of copper in the pathogenesis of liver disease in the Doberman is unclear. It has been implicated in that breed as apparently familial and associated with accumulation of large concentrations of hepatic copper in that breed and also in West Highland White Terriers (as well as in the classic Bedlington Terrier disease where it is well-documented). Other breeds in which copper toxicosis has been suspected are Labradors, Cocker Spaniels, German Shepherds, Schnauzers, Kerry Blue Terriers, Pekinese, Keeshonds and crossbreeds.

Thornburg, L.P. et al.: High liver copper levels in two doberman pinschers with subacute hepatitis. JAAHA 20:1003, 1984
Thornburg. L.P et al.: The diagnosis and treatment of copper toxicosis in dogs. Canine Practice 11:36,1984
Thornburg L.P et al.: Copper toxicosis in dogs. Part 3: Diagnosis and treatment. Canine Pract 13 :10,1986.

Mycosis Fungoides in a Tasmanian Devil - Judith S. Nimmo Wilkie, Victorian Veterinary Pathology Services,

A two-year-old female Tasmanian devil from a private zoo was found dead after a short illness involving bloody diarrhoea. It also had a skin rash. Post-mortem findings included mottled lungs, bloody intestinal content and patchy cutaneous erythema. Fixed specimens of lung, skin, spleen and small intestines were submitted for examination. The skin showed a diffuse lymphocytic infiltration extending from the middle to the superficial dermis and extensively infiltrating the epidermis where the lymphocytes were remaining

in small clusters in the basal and inner layers. Hair follicle epithelium was also affected. The lymphocytes were of medium size and many had folded or convoluted nuclei. The spleen showed extra-medullary haematopoiesis. No significant lesions were seen in the intestine. The lung was oedematous. Tasmanian devils in captivity are prone to neoplastic disease. Lymphosarcoma has been described in this species in Australia but not the cutaneous form. Cutaneous lymphoma has been reported in a Tasmanian devil in the USA but was not characterised as to type.

Griner, L.A. (1979) Neoplasms in Tasmanian Devils. J National Cancer Institute

South Australia - Ruth Reuter

Isospora Infection in a Puppy - Julia Lucas, Veterinary Pathology Services, Adelaide

An eight week old Labrador puppy presented with haemorrhagic diarrhoea and vomiting. The pup collapsed and died 24 hours later. This was one of a group of puppies from a puppy farm and purchased by a pet shop. Several puppies developed diarrhoea and two developed haemorrhagic diarrhoea. Other puppies in the group were less severely affected and this was the only puppy to die. A necropsy was performed by the referring clinician. Post mortem findings included multiple intussusceptions of the distal small intestine that had not been palpated ante mortem.

Histological examination of three sections of the small intestine identified severe suppurative and eosinophilic enteritis associated with large numbers of coccidia in the intestinal mucosa. The coccidial organisms were located within the enterocytes and lamina propria. At the base of the crypts the enterocytes had an increased mitotic rate and appeared hyperplastic. In the lamina propria there were moderate to large numbers of eosinophils, neutrophils, lymphocytes and plasma cells. A faecal float confirmed that the coccidia seen in sections were morphologically consistent with *Isospora canis.*

In addition the tips of the intestinal villi were necrotic and had large numbers of fine filamentous bacteria colonising the surface. The bacteria extended deeply into the crypts. This was interpreted as a secondary bacterial overgrowth.

Coccidian parasites of the dog include *Isospora* and *Hammondia*. Differentiation may be based on oocyst size, *Isospora canis* - 34-40 µm, *Isospora ohioensis* - 20-27 µm and *Hammondia heydorni* - 11-13 µm[1]. Other coccidian parasites include *Toxoplasma, Cryptosporidium* and *Sarcocystis* .

Isospora infects the distal third of the small intestine, and may also extend into the large intestine[2]. Epithelial cells are destroyed by the schizogenous and gametogenous phases[1]. Infection by *Isospora spp.* is infrequently associated with clinical disease, however, in young puppies in unhygienic surrounding there is potential for ingestion of large numbers of organisms[1]. In this case infection may result in severe haemorrhagic diarrhoea[1]. Confirmation of *Isospora* as the aetiological agent requires demonstration of large numbers of organisms in the intestinal mucosa[1].

The large numbers of coccidia in the sections examined indicates these were an important aetiological agent in this animal. The coccidia would have accounted for the severe, bloody diarrhoea observed grossly and would have been the underlying cause of the intussusception that accounted for the death of this puppy.

It is unclear if the bacteria in this section are secondary to the coccidia. However, the large numbers of bacteria suggested that they were significant, and would have contributed to the clinical signs in this animal.

1. Dunsmore JD, 1990, *Clinical Parasitology of Dogs*, University of Sydney Post Graduate Foundation, Sydney.
2. Levine ND, 1985, *Veterinary Protozoology*, Iowa State University Press, Ames.

Western Australia - David Forshaw

Congenital leucodystrophy in calves linked to dams grazing Tagasaste

(Chamaecytisus palmensis) - Anima! Health Laboratory - Agriculture Western Australia.

In a previous issue (No. 51 Dec. 1998), a case of suspect Oligodendroglial dystrophy in two calves was reported.

"The first of these calves displayed hypermetric placement of the forelimbs and severe hind limb ataxia. The second calf, an Angus/Hereford cross could stand quite easily, but displayed coarse trembling of its hind limbs and mild to moderate hind limb ataxia when forced to walk. Fear, blink, and withdrawal reflexes were present in both animals. In the brains of those calves there were moderate to severe white matter lesions in the internal capsule, mid brain, medulla and cerebellar white matter, severity increasing towards the mid and hind brain. Lesions were also present in the optic tract and chiasma but not the optic nerve or retina. Severe lesions were present in all white matter tracts for the entire length of the spinal cord, but not in the cauda equina or peripheral nerves.

In H & E stained sections, the lesions appeared as vacuoles and faintly eosinophilic granular plaques approximately 40-50 u in diameter. In PAS stained sections they were slightly pink and did not stain at all in LFB sections, in some places the plaques contained peripheral nuclei resembling those of oligodendroglia. The presence of segments of normal axons within the plaques was demonstrated by use of LFB-Holmes silver stain. Apart from the presence of an occasional macrophage within empty axonal tubules, there was no inflammatory reaction. Glial cell reaction was negligible apart from the presence of what appeared to be isolated dense shrunken nuclei of microglia in the vicinity of the injured axon."

There were remarkable similarities between the histopathological lesions seen in these calves to those described in progressive ataxia of Charolais cattle (1).

Since then, similar cases have been reported on several farms in the west midlands area of the State. Clinical signs vary in severity. At their most extreme expression, calves are not able to stand and exhibit coarse muscular tremors, especially in the hindquarters. The limbs of these calves scramble wildly if attempts are made to assist them to their feet. The milder manifestation of the disease is expressed by tremors of the musculature of the hind quarters which become exaggerated when the calf is stimulated. There is no breed or sex disposition.

All affected calves have histopathological lesions consistent with those previously described in the white matter of the internal capsule, cerebellar white matter and spinal cord. Ultrastructurally the plaques consist of intramyelinic expansions containing numerous vesicular membranous profiles and myelin bodies dispersed throughout a granular matrix possibly containing remnants of microtubules. The remnant myelin sheaths surrounding these foci are usually very thin, containing a few lamellae only. Surrounding the plaques, numerous glial cells and their processes are similarly affected with numerous vesicular profiles. Oligodendrocytes are affected and possibly also astrocytes. Some smaller diameter myelinated fibres have periaxonal vesiculation that appears to derive from the inner lamella of myelin sheaths. In the brain of some calves there are focal plaques of disorganised and tangled myelin.

The cause of the problem is not known, but may be associated with a toxin contained in tagasaste (*Chamaecytisus palmensis*) the plant the dams were grazing during pregnancy.

Tagasaste also known as tree lucerne is a leguminous fodder shrub planted to provide feed for stock, mainly in the West Midlands and Greenough areas in WA. Its main advantage is its ability to grow in deep sandy coastal soils and when correctly fertilised has increased stocking rates from less than one dry sheep equivalent per hectare to up to six and enabled producers to greatly expand their beef cattle enterprises. The nutritional benefits of the plant are well known but the apparent toxic effects are less well understood and may be precipitated by other factors.

A stagger syndrome in cattle grazing tagasaste is well recognised in Western Australia and cases have been investigated by AHL. No gross or microscopic lesions in affected cattle have been found and clinical chemistry is unremarkable. The cause is unknown and cattle usually recover if left alone (even if they continue to graze tagasaste).

1. Progressive ataxia of Charolais cattle associated with disordered myelin. WF Blakemore and AC Palmer (1974) Acta Neuropathol 29:127-139.

Chinkerinchee Poisoning - Animal Health Laboratory - Agriculture Western Australia.

Although Chinkerinchee (Star of Bethlehem, *Ornithogalum thyrsoides)* poisoning is a well recognised disease in South Africa, it was not until May of this year that, the first Australian case was diagnosed at Tambellup. Chinkerinchee is a bulbous perennial herb with broad fleshy strap like leaves and attractive star shaped flowers which have escaped from garden cultivation into local farmland.

Within 4 days of introduction to the paddock containing the plant several hoggets died and another 2 exhibited hind limb incoordination. About a third of the flock developed a putrid diarrhoea. Microscopic examination of tissues from affected sheep revealed multifocal cardiomyopathy and non-specific changes in the small intestine. These changes are consistent with cardiac glycoside toxicity, the suspected toxic principle in Chinkerinchee.

The owner of the sheep had experienced problems with the plant in previous years, but never after the beginning of July. This suggests that the plant may only be toxic for a short period following the break of the season.

Lesser Loosestrife (Lythrum hyssopifolia) Toxicity in Sheep - Animal Health Laboratory - Agriculture Western Australia.

10/300 and 20/390 weaner lambs died on 2 farms in the Tenterden and Tambellup areas where both flocks had been grazing predominantly canola stubble during late December.

Results of histopathological examination of tissues from some of the first lambs dead established that they suffered severe and acute liver necrosis with concurrent severe necrosis of the kidney tubules. Lambs continued to die or became sick even though they had been removed from the stubble. A surprising laboratory finding was that although these lambs later died and had severe destruction of the kidney tubules, there was no evidence of liver damage.

This finding suggests that the lambs died from ingestion of a plant toxin which at certain concentrations induced the severe and often fatal liver lesions. If the lamb survived that insult or only consumed sub lethal amounts of the toxin, it was converted by the liver into another form which was toxic to the kidney. This toxin continued to exert an effect even though the lambs had been removed to another food source.

While initial investigations centred on the possibility of algal toxicity, molybdenum or phosphorus poisoning or even that the canola stubble may have been toxic, examination of the paddocks of both farms revealed the presence of the plant commonly known as Lesser Loosestrife. This plant had established itself in moister areas of the canola paddocks and at the time that the sheep had been introduced was still actively growing, providing a green alternative to the dry and possibly less palatable canola stubble. The plant is

known to be toxic and similar liver and kidney lesions have been described in sheep in New South Wales in 1991.

Probable Salt poisoning/Water deprivation in 6 week old pigs - Mandy O'Hara[1], Dave Forshaw[1,2] and Jan Thomas[1]
1 Division of Veterinary and Biomedical Science Murdoch University
2 Animal Health Laboratory Agriculture WA

Two pens of 10, 6 week old weaner pigs were used for blood sample collection. Forty eight hours after blood collection 5 pigs in one pen developed nervous signs and 1 pig died. None of the 10 pigs in the adjacent pen developed nervous signs. Postmortem examination of the dead pig demonstrated acute gastric venous infarction, multiple acute renal infarction, generalised pulmonary congestion and oedema and multifocal chronic hepatitis. The brain appeared swollen and congested. Bacteriology was unrewarding.

Significant histological changes were restricted to the brain, which demonstrated a moderate, predominantly cerebral, eosinophilic encephalitis and mild to moderate diffuse eosinophilic leptomeningitis. There was also laminar cortical oedema with vascular reactivity. A preliminary diagnosis of water deprivation/salt poisoning was made. Examination of the water drippers revealed corroded and blocked drippers in the pen containing the pigs that demonstrated neurological signs. The drippers in the pen containing clinically normal pigs were free flowing.

Despite supportive medical care of the affected pigs, clinical deterioration resulted in the euthanasia of 7 out of the 10 pigs in the affected pen. Examination of 2 of the 7 pigs prior to euthanasia (2 and 3 weeks after the first onset of neurological signs respectively) identified an absence of both menace and pupillary light responses in both pigs. Gross pathology was similar in both pigs and was restricted to the brain. There was severe deep laminar cerebrocortical necrosis and cavitation, which was most severe in the frontal lobes. Histological examination confirmed the gross findings and demonstrated a marked gitter cell infiltrate and gliosis associated with the cavitation. Unlike the acute lesion there was no eosinophil infiltrate and there was marked neovascularisation and mineralisation of vessel walls within the parenchyma and meninges adjacent to the area of cavitation. Wallerian degeneration was also observed within the cerebral white matter and there was multifocal gliosis within the basal nuclei.

Lesions were progressive and of greater severity in the pig euthanased 3 weeks after the initial onset of neurological signs. In this pig there was also bilaterally symmetrical focal cavitation within the ventral midbrain, with multifocal giant cell formation observed in one area of the cerebral poliomalacia and vascular mineralisation.

Both laminar cerebrocortical cavitation and perivascular infiltrates of eosinophils have been described in association with salt poisoning (either in combination or independently), but the development of marked vascular mineralisation and giant cell formation have not. Although it is possible there was more than one disease condition in these pigs, the presence of neurological signs only in those pigs demonstrated to have an obstruction to the water supply, (and in otherwise identical management conditions to a pen of clinically normal pigs) is highly suggestive of water deprivation/salt toxicity.

Northern Territory - Anton Janmaat

Mortalities in juvenile barramundi: coccidiosis/ichthyobodiasis/nephrocalcinosis - John Humphrey, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

Healthy, hatchery reared juvenile barramundi *Lates calcarifer*, stocked into earthen rearing ponds underwent a mass mortality resulting in total loss of stock over a period of approximately 7 days. A massive superficial infection of the intestinal epithelium by a coccidian consistent with *Epieimeria* sp was present in all fish examined. A range of merogonous and gametogenous stages were present on the epithelial surfaces and large numbers of unsporulated oocysts were evident in the lumen. The lamina propria showed a mild to moderate inflammatory response and mild oedema. The epithelium underlying the parasites showed attenuation. In addition, fish had mild to moderate infections of the integument and gills with the pathogenic external protozoan *Ichthyobodo (Costia)* sp. All fish examined showed mild to severe degeneration and mineralisation of renal tubular tissues. Examination of hatchery stocks of the same cohort showed no parasitism or nephrocalcinosis. The cause of the nephrocalcinosis was not determined.

Epizootic ulcerative syndrome (ulcerative mycosis or red spot) in wild fish - John Humphrey, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

Numerous estuarine and marine fish species were reported with ulcerative lesions typical of epizootic ulcerative syndrome. The syndrome was confirmed in a mullet from Ludmilla Creek, Darwin. Histopathological examination showed the typical invasive fungal hyphae of *Aphanomyces invaderans* with severe granulomatous dermatitis, myositis and myonecrosis. It is remarkable that widespread ulcerative mycosis was reported in Queensland fish at approximately the same time. Bacterial culture of the lesions resulted in the isolation of mixed bacteria including *Vibrio* species.

Myxoboliasis in eel-tailed catfish - John Humphrey, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

A single eel-tailed catfish captured in a local stream was submitted for examination with a history of numerous fish of the same species developing lumps under the skin followed by ulceration. Other in-contact species were reported to be unaffected. The submitted fish showed generalised severe ulcerative lesions on the integument with underlying intra-muscular haemorrhages. Histopathological examination showed a severe dermatitis and myositis associated with large numbers of the protozoan *Myxobolus* sp.

Queensland - Jim Taylor

"Broken Back" syndrome in Barramundi - Greg Storie, QDPI Yeerongpilly

Several barramundi farms in Queensland employing re-circulation systems have experienced intermittent losses due to this condition. Onset is acute with affected fish swimming erratically, often on their sides or upside down, at various depths in the tank. Death follows in 3-4 days. Usually, the only significant gross abnormalities detected at necropsy are 1 or 2 small foci of haemorrhage overlying the ventral aspect of the vertebral column. Deeper dissection reveals underlying vertebral fractures, Histological examination reveals the bone to be osteoporotic compared with control fish of the same age. It is postulated that there is a dietry-related weakness in the bones of these fish and that spontaneous fractures are occurring when the fish are handled (e.g. at grading), frightened (causing acute extreme muscle contraction) or due to increased activity at the time of feeding. Affected farms were found to have purchased bulk feed which was stored for long periods under unsuitable conditions. Analysis of feed revealed very low levels of vits.A and E. Inclusion of a vitamin/mineral premix and supplementary vit.C reduced the problem by 30-50% but did not completely eliminate it.

Neurological complications following rapid intravenous correction of hyponatraemia in a dog - Richard Ploeg, University of Queensland

A three year old male Tenterfield terrier was presented to the referring veterinarian following a two week history of weakness, lethargy and intermittent vomiting. An initial biochemical profile revealed hyponatraemia, hypochloraemia and prerenal azotaemia. A tentative diagnosis of hypoadrenocorticism was made and subsequently confirmed by an ACTH stimulation test. Intravenous fluid therapy with 0.9% NaCl was commenced at presentation at a rate of twice maintenance requirements and glucocorticoid/ mineralocorticoid therapy was initiated following the confirmation of hypoadrenocorticism. Approximately twenty four hours after presentation the serum sodium had gone from 117mmol/L to 134mmol/L and intravenous fluids were discontinued soon after. For the next 24 hours the dog appeared normal and was offered food with a small amount of sodium chloride "sprinkled on top" and was given access to water. The dog subsequently began convulsing and was killed.

Significant findings at post mortem were cerebellar coning, bilaterally small adrenal glands (5mm x 3mm). ulceration and oedema of the fundic gastric mucosa. Histologically primary/idiopathic hypoadrenocorticism was confirmed and brain sections revealed variable acute oedema most severe in the cerebellar cortex.

The history and pathological findings are consistent with cerebral oedema resulting from the rapid correction of hypovolaemia in the face of hyponatraemia and compounding acute water intoxication.

Myelinolytic lesions are worth looking for in cases such as this where there has been a rapid shift in effective osmolality with the subsequent onset of progressive neurological signs. Myelinolysis has been reported in dogs following the correction of hyponatraemia at a rate >15mmol/L/day and is a condition analogous to central pontine myelinolysis in humans (1). In dogs the lesions are found within the central thalamic nuclei and rostral commisures and consist of a loss of myelin in association with the degeneration of glia but with the preservation of neurones and axons (1).

These lesions take several days to develop and given that the rate of correction of the deficit in the outlined case was 17mmol/L, it is possible that true myelinolysis may have developed if the animal had lived for longer. This case illustrates that lesions of myelinolysis may be preceded by the development of acute cerebral oedema following the correction of hyponatraemia.

(1) O'Brien, D.P, Kroll, R.A., Johnson, G.C., Covert, S.J. and Nelson, M.J. Myelinolysis after correction of hyponatraemia in two dogs. J. Vet lnt Med. 1994, **8**(1), pp 40-48.

Eperythrozoonosis in lambs - Jim Taylor, QDPI Toowoomba Veterinary Laboratory.

About two hundred 10-month-old lambs in a mob of 1300 were showing signs of ill thrift a month after purchase and introduction to a new lucerne pasture. The lambs had been drenched prior to introduction.

A typical live animal was submitted for necropsy. Clinical findings included a moderate regenerative anaemia with numerous *Eperythrozoon ovis* in the smear, (PCV 18%, Hb 5.4 g/dl) and moderate haemoglobinuria. No oocysts or helminth eggs were detected in faeces.

At necropsy the lamb had a moderate serous peritoneal and pericardial effusion and mild submandibular oedema. The spleen appeared congested, although the animal was euthanased with barbiturates and the urinary bladder contents were haemoglobinuric. The only histological lesion of note was mild haemosiderosis of the renal tubular epithelium.

While illthrift and anaemia are typical signs of Eperythrozoonosis in lambs, haemoglobinuria is not considered typical.

Cysticercosis (Beef Measles} in feed lot cattle - Jim Taylor, QDPI Toowoomba Veterinary Laboratory

A mob of about 2000, two to three year old steers were purchased for finishing and slaughter.

Shortly after the start of processing a number of cattle were found to have mineralized lesions up to 5mm diameter in the heart and skeletal muscles. Most lesions are mineralised eosinophilic granulomas; some contain remnants of degenerating cysticercus in the form of calcareous corpuscles. In a few, viable *Cysticercus bovis* have been found.

Inspectors and processors estimate 50% of the mob slaughtered to date have lesions. The cattle originated from a property that irrigated pasture with sewage effluent.

Sporadic Bovine Encephalomyelitis in weaner heifers - Jim Taylor, QDP1 Toowoomba Veterinary Laboratory.

Twenty seven 8 month old Santa Gertrudis cattle were weaned into a set of yards and fed for four days with native pasture hay and molasses. The steers were sold and the remaining 13 heifers were turned out onto a small Mitchell grass paddock. About 6 days later, 6 of the heifers were noted lame with swelling of the coronary bands or fetlocks and upon examination were found to be febrile. One heifer progressed to neurological signs and was euthanased and a postmortem performed.

Grossly a mild fibrinous pleural effusion and flocculent material in the fetlock joints were described. The most significant histological lesions were present in the brain. There was a diffuse meningeal and perivascular mononuclear infiltrate with multifocal necrotising and thrombosing vasculitis, particularly in small vessels, with malacia, gliosis and polymorph infiltration of the associated neuropil. Cultures for *Haemophilus spp*. and *Listeria spp*. were negative and no bacteria were evident in Gram stained sections. An enzyme immunoassay (EIA) for Chlamydia, using a commercial kit was positive on brain tissue and pleural exudate.

New South Wales - Paul Gill

Abortion in sheep - John Glastonbury, Regional Veterinary Laboratory Camden, Elizabeth Macarthur Agricultural Institute

A commercial experimental farm in western Sydney runs 260 Merino ewes to breed nematode free sheep. The flock was mated to commence lambing on 7 June 1999. From 25 May until 26 June 1999, there were 23 late term abortions or parturient deaths. The flock grazes small paddocks and hand feeding was started on 25 May 1999.

During the period of the outbreak, 11 foetuses, and fixed tissues from a lamb which died at 2 weeks of age, were submitted to the laboratory. The foetuses weighed between 1.4 to 3.0kg. Eight of the foetuses had not breathed or had only partially aerated lungs, one had breathed and walked but not fed, one was mummified and the remaining foetus had not walked and was eviscerated. Excessive volumes of straw coloured fluid were noted in the serous body cavities of three foetuses and one had excessive volumes of blood stained serous fluid and subcutaneous haemorrhages. Foetal membranes were received from three foetuses, and in each instance, they appeared grossly normal.

Apart from varying degrees of visceral congestion and non-aeration of the lungs, significant histological findings were mostly restricted to the brains. Six of the foetuses had small numbers of focal lesions, consisting of a central zone of necrosis surrounded by reactive glial cells. These lesions were most common in the brain stem, seven of nine lesions, and occurred in both grey and white matter. The remaining two were located in the cerebral cortex and the corpus callosum. Small foci of acute leucoencephalomalacia were observed in the other three brains - deep cerebral white matter, corpus callosum and the thalamus. The only noteworthy finding in the three foetal membranes was the detection of isolated cysts with a morphology consistent with *Toxoplasma gondii* in one. Only liver and lung were submitted from the post parturient death and moderate chronic interstitial pneumonia was found in the latter.

Ancillary laboratory testing gave conflicting results. Serous fluids from seven of the foetuses were subjected to the latex agglutination test for *T. gondii*; six were negative and one yielded a reaction of 1:16. The pestivirus antigen capture ELISA (PACE) was performed on spleens from seven foetuses, with only one yielding a positive result. No antibodies to *Leptospira hardjo* and *pomona* were detected in serous fluids from three foetuses and the pestiviral gel diffusion precipitin test (GDPT) was negative when performed on fluids from two foetuses, including the one with the positive PACE. Routine bacteriological culturing of tissues from each foetus yielded negative results.

Because of the positive PACE, the virologists solicited blood samples from the ewes and cattle being pastured in close proximity to the sheep, 36 and 25 samples respectively. Three of the sheep samples yielded titres of 2 in the pestivirus GDPT and all other samples were negative. Two of the GDPT positive animals yielded titres of 128 and 512, respectively, in the pestivirus virus neutralisation test (VNT); the remaining 34 samples tested were negative. Heparin blood samples were collected from five apparently normal lambs and none yielded pestivirus following culture. Twelve of the sheep samples were selected at random for the latex agglutination test for toxoplasmosis; the results are presented in the following table.

Table: Latex agglutination test for toxoplasmosis

Titre	Number of samples
1:4	1
1:64	5
1:256	5
1:1024	1

In my opinion these abortions were the result of *T. gondii* infection and it was not until the virologists came up with the positive PACE that I commenced to have some doubts. Nevertheless, this exercise does demonstrate how difficult it can be to absolutely confirm the diagnosis.

Points in favour of the diagnosis of toxoplasmosis are the epidemiology of the outbreak, the fact that toxoplasmosis was "confirmed" as the cause of ovine abortions in another flock on the farm in 1996 and the histological findings in the brains of aborted lambs. Around 95% of lambs aborted due to toxoplasmosis have either focal granulomatous encephalitis or focal leucoencephalomalacia; I consider the former to be almost pathognomonic for the disease, whereas the latter can also be seen in pestiviral infection. Multifocal necrotising placentitis is supposed to occur in about 50% of toxoplasmal abortions. Only three of 11 foetal membranes were examined from these cases so it is probably within statistical bounds that the change was not found. In my experience, the latex test performed on foetal fluids gives inconsistent results. As the latex test detects both IgM and IgG, the maternal serological results are of doubtful significance, especially in view of the previous history of the disease on the farm. However, the relatively high titres are consistent with toxoplasmosis and mitigate against the alternative possibility of *Neospora caninum* infection, which we are yet to diagnose in New South Wales sheep. The interstitial pneumonia found in the lamb that died post-natally could well be a legacy of toxoplasmosis.

The pathogenesis of the lesions in the central nervous system is interesting. Focal leucoencephalomalacia in the periventricular white matter is generally thought to be a consequence of hypoxia. In cases with severe placentitis, this is easily explainable. However, the placenta was examined in one of the present cases with leucoencephalomalacia and was found to be normal; another possibility would be that it results from direct damage induced by the protozoan. No protozoa and/or cysts could be detected in haematoxylin and eosin stained sections of the present material and unstained sections have been forwarded to Mt Pleasant for immunoperoxidase staining. The granulomas could be the reparative stages of the encephalomalacia or represent an inflammatory response to the presence of the protozoa. The fact that they occurred in both the white and grey matter would tend to eliminate the former.

Despite the initial excitement, evidence in favour of a major role for pestivirus was not great. The virus was not recovered from the blood of five lambs, only three of 36 ewes had serological evidence of previous exposure and hairy birth coats were not readily apparent.

The way forward in the confirmation of Toxoplasma induced abortion would appear to rest with molecular biological techniques. PCR technology would be of great value, and hopefully, the immunohistochemistry supports the above theory.

Mycotic pneumonia in mice with defective phagocytes - Malcolm France, University of Sydney

Illthrift was noted in a few animals from a colony of mice deficient in an enzyme important in the respiratory burst exhibited by neutrophils and macrophages during phagocytosis of micro-organisms. On gross examination, there were numerous pulmonary abscesses from which the fungus *Paecilomyces variotii* was cultured. In addition to abscesses containing fungi, histological examination revealed the presence of numerous eosinophilic crystals accompanied by macrophages and giant cells in adjacent parenchyma.

The genetic defect in these mice is homologous to the most common form of an inherited phagocyte disorder of children known as chronic granulomatous disease. Affected children suffer from a range of opportunistic infections; these are mostly staphs, *E. coli* and *Aspergillus* sp although there is one recorded case of a child infected with *P. variotii*. While the source of infection in this mouse colony was not determined, *P. variotii* is a common environmental fungus and it is thought that it might have entered the facility on non-autoclaved feed pellets or sunflower seeds fed to the mice as a treat. The eosinophilic crystals are representative of a sporadic disease peculiar to mice known as 'acidophil macrophage pneumonia'. The condition is associated with some mutant genotypes and probably added to these animals' predisposition to pulmonary disease.

Hearts, Flowers and Enterotoxaemia - Paul Gill, Regional Veterinary Laboratory, Wollongbar

Clostridium perfringens epsilon toxin is rarely detected in the intestinal contents of adult cattle in our experience. We have recently had 2 cases where epsilon toxin was detected in the intestinal contents of cattle which had died due to *Bryophyllum sp* poisoning. Multifocal myocardial necrosis was confirmed in one case, the other involved many animals and was clinically and epidemiologically consistent with *Bryophyllum sp* poisoning. In neither case was brain submitted for histological examination. I would be interested in hearing from anyone else who has made similar observations.

"A Victim of General Paresis"

The following stanzas were submitted as part of an assignment by Andrew Lynch, a 3rd year veterinary student at the University of Sydney. The question concerned specimen collection at necropsy of an animal suffering from generalised weakness. The poem is reproduced with the author's permission.

Victim of General Paresis.
I'll try not to make this a thesis,
But so non-specific a cause,
That I'd dare not to pause
To do everything short of centesis.

Of course every post mortem I start,
I fix lung, liver and heart;
A small chunk of spleen
And some kidney I glean,
And a few bits of gut might be smart.

If solving the mystery looked tough,
And these samples proved not enough,
I'd bleed a large vein.
And take out the brain.
Not worrying 'bout being too rough'.

Microbiology would do no harm
If nothing had yet raised alarm;
So each tissue I've stated,
I'd freeze just abated,
And send to the culturing farm.

Not much carcass is left on the tray;
Yet that was quicker than the average spay!
We've much evidence to sift.
To resolve the ill-thrift -
Then we'll lap up the owner's pay."

Tasmania - Roy Mason

Neospora caninum indirect fluorescent antibody testing - Pat Statham and Roy Mason

We have been successful in recently obtaining a serum sample from a dog with active myositic and neurological neospora infection. Using the IFAT for neospora we obtained a reciprocal titre of > 3200. A literature report detailed IFAT titres of 800 or higher in dogs 37 days after experimental infection.

We now offer to other laboratories both bovine and canine neospora IFAT serological testing.

Note that our experience has shown that in neospora abortion in cattle aborting cows may not maintain a significant titre for long periods post abortion. Therefore we do not recommend the test to retrospectively diagnose neospora abortion, rather the test should be used on cows at the time of or soon after abortion.

Reference: McAllister MM *et al*: Dogs are definitive hosts of Neospora caninum (1998): **International Journal of Pathology**, vol 28 pp1473-1478.

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