



VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
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DEADLINE FOR NEXT VET. PATH REPORT IS OCTOBER 31, 1993

SECRETARIAT

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President	John Gibson	Toowoomba Vet.Lab, PO Box 102, Toowoomba Q4350	076 314352
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	Geoff Mitchell	VPS, PO Box 1119, Coorparoo DC Q 4151	07 3918500
	Dick Simon	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
Coordinator	(Training Committee)	Robin Giesecke

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 VetLab, 665 Fairfield Rd, Yeerongpilly Q 4065	07 3629555
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South Australia	Ruth Renter, VPS, PO Box 96, Plympton SA 5038	08 3623544
New South Wales	Paul Gill, RVL, Wollongbar NSW 2480	066 240298
Western Australia	David Forshaw, Regional Office, WA Dept.Agric, Albany WA 6330	098 420536
Northern Territory	Anton Janmaat, PO Box 79, Berrimah NT 0828	089 895511
Tasmania	Barry Munday, Univ.Tasmania, PO Box 1214, Launceston TAS 7250	003 243232

PRESIDENT'S REPORT

The first three months of the new executive, if nothing, have been busy with several contentious issues from the AGM to deal with. Dr Tony Ross and I had brief but very positive discussions with Dr Jakob Malmo at the AVA Conference on issues raised at the AGM. The subsequent submission to the AVA and their reply is printed in this report. Dr Keith Walker and Dr John Plant have kindly accepted the task of putting together some preliminary proposals for possible re-affiliation with the AVA. An issue to be voted on at the 1994 AGM.

It is disappointing to hear of further redundancies in the Vetlab, S A. Unfortunately in these times we are all not to far removed from this reality. We have held preliminary discussions with Dr Kevin Dunn regarding an ASVP submission to the Miller committee. We are keen to target our submission at issues relevant to the committee. We would also be grateful for any comments, submissions or ideas from the membership.

Finally I would like on behalf of the membership to formally thank Dr Peter Phillips for his efforts with the Slide-of-the-Month, a job well done! Peter hands over the task to Dr Rod Reece.

John Gibson

EDITORS REPORT

My apologies for the delay with this Veterinary Pathology Report. As this is my first report I have had some teething problems and hopefully the next report will be out a little sooner after the deadline. Thanks to Gary Reddacliff for his help and advice and I hope to continue VPR's high standard.

This newsletter is the product of its member's contributions and please feel free to make any comments as to its format and content. Contributions other than just material for state reports are welcome. I was pleased to see clinical pathology case reports in the state reports and encourage members to keep good clin. path. Cases in mind.

A reminder to those with overdue membership fees appears on page 19. Please send all fees and membership applications direct to the Secretariat as this saves me the job of having to forward them on.

I look forward to your contributions.

Jim Taylor

2.

MINUTES

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY INC.

ANNUAL GENERAL MEETING
GAZEBO HOTEL, BRISBANE

15 MAY 1993

Meeting opened 4pm.

APOLOGIES: Clive Huxtable, Martin Copland, Rob Rahaley, Ruth Reuter, Chris Belford, Russell Graydon, John Finnie, Vui Ling Tham, Steve McOrist, Terry Rothwell.

Acceptance moved Ross. Seconded Gill. Carried.

MINUTES OF 1992 AGM

Published in August 1992 Edition of Vet. Path. Report and tabled.

Acceptance moved Glastonbury. Seconded Ross. Carried.

ADDITIONAL AGENDA ITEMS

- World Association of Veterinary Laboratory Diagnosticians Annual Meeting
- IATA transport regulations and implications

Both held over to General Business.

BUSINESS ARISING FROM 1992 MINUTES

- Veterinary Pathology training including Training Committee - deferred to General Business.
Moved Sims. Seconded Kelly. Carried.
- Commercialisation of membership/mailling functions - covered in Secretary's Report summarised by Reddacliff.
- Membership application of John Callahan - advised of his ineligibility but offered subscription to Vet. Path. Report. No reply to date.
- ASVPs relationship with AVA and timing of meetings - deferred to General Business.
Moved Miller. Seconded Giesecke. Carried.

3.

CORRESPONDENCE

Dealt with through VPR and Executive. No matters raised.

REPORTS

Presidents

Published in conference proceedings.

Acceptance moved Phillips. Seconded Sutton. Carried.

Treasurer's

- Published in conference proceedings.
- Membership fees for 1994 to be maintained at \$25. Moved Ross. Seconded Sims. Carried.
- R. Kelly questioned the need for an audit. L. Sims advised not required under articles of incorporation.
- Acceptance of report. Moved Ross. Seconded Trueman. Carried.

Secretary 's/VPR Editor's

- Published in conference proceedings.
- Publication of a membership list. Moved Kelly. Seconded Love. Carried.
- VPR favourable reviews from North America. Acceptance of Report moved Gill. Seconded Boulton.

NRDAP

- Published in conference proceedings.
- Thanks extended to Rod Reece and Bill Hartley for their efforts.
- Rod Reece to liaise with Wanda Haschek-Hock re: access resource material from the C L Davis Foundation for the registry. Cost of circulation in Australia met by users and Veterinary Pathology Services Pty Ltd are happy to sponsor the return cost to USA. Moved Boulton. Seconded Giesecke. Carried.
- Adoption of registry report. Moved Dowling. Seconded Reddacliff. Carried.

Slide of the month

- Peter Philips thanked membership for their contribution.
- Question of receipt of slide of the month by non-contributors raised.
- Receipt of non-contributors is not policy but will be left to the discretion of the convenor. Moved Sullivan. Seconded Taylor. Carried.
- Acceptance of report. Moved Giesecke. Seconded Sims. Carried.

4.

Training Committee

- Published in conference proceedings.
- Summary and discussion led by Robin Giesecke, issues raised included ease of production and funding of species based modules, need for continuing education and highlighting benefits to employers. Moved Glastonbury. Seconded Lee that recommendations of committee be accepted. Carried.
- Moved Robinson, Seconded Taylor that the Training Committee formulate a strategy for implementation of recommendations and possible international markets be kept in mind. Carried.
- Adoption of report. Moved Glastonbury. Seconded Seward. Carried.

ELECTION OF OFFICE BEARERS AND APPOINTMENTS

President	John Gibson		
Secretary/Editor VPR	Jim Taylor		
Treasurer	Russell Graydon		
State Correspondents	Anton Janmaat	-	NT
	Ruth Reuter	-	SA
	Deb Seward	-	VIC
	Paul Gill	-	NSW
	David Forshaw	-	WA
	Barry Munday	-	TAS
	Greg Storie	-	QLD

Registry Committee and Training Committee to continue in current structure with Tony Ross and Robin Giesecke as chairperson and coordinator respectively.

GENERAL BUSINESS

- Domestic Animal Pathology Registry Funding
 - Registry Management Committee is to apply for further funding and continue lobbying for funding at state and commonwealth levels. Moved Reddacliff. Seconded Glastonbury. Carried.
- Vet Lab South Australia - Ministers Response
 - Copies of the Ministers response to ASVPs submission on ODR were circulated. Peter Philips thanked the outgoing executive for their efforts and submission.
- Future of Government Veterinary Services
 - Discussions continued from the afternoon session.
 - New executive was given scope to co-opt help for a submission to the Miller Committee. Terry Nicholls, David Obendorf, Peter Philips, Tony Ross, Anton Janmaat and Les Sims agreed to act as state and territory contacts.

5.

- Submissions to the Miller Committee by both direct and via the AVA.
- Need to involve client groups to lobby on our behalf.
- Acknowledgment of complementary role of private labs in Animal Health Surveillance.
- Moved Robinson. Seconded Links that the Executive write to the AVA regarding representation of AVA members of the ASVP in submissions to the Miller Committee. Carried.
- Affiliation of ASVP with AVA
 - Possible affiliation of ASVP with AVA was discussed. Issues raised included type of affiliation, or example, Laboratory Diagnosticians Group and the cost to ASVP members.
 - General feeling of the meeting was ASVP conferences should retain format, timing and cost.
 - Moved Glastonbury. Seconded Robinson that the ASVP Executive drafts a position paper with regard to possible ASVP affiliations with the AVA.
 - Carried Phillips and Janmaat against.
- IATA Regulations for transport of dangerous goods and diagnostic specimens
 - Moved Ross/Cook. Seconded Kelly that Geoff Mitchell (who is on a committee to examine the matter) report to ASVP through the newsletter on new regulations for packaging of specimens. Carried.
 - Moved Cook/Boulton. Seconded Links that ASVP Executive write to Chair, Animal Health Committee and Civil Air Authority, to express concern that increasing restrictions by way of IATA Regulations (for transport of diagnostic animal specimens) severely jeopardise Animal Health Surveillance in Australia, and that AHC recommend through appropriate channels, to Department of Transport and Communications, that restrictions by weight or volume not be applied to such specimens, provided that they are packed to conform with IATA packing instructions 650. Carried.
- World Association of Veterinary Laboratory Diagnosticians
 - Les Sims to advise via VPR.
- 1994 ASVP Conference
 - Moved Taylor. Seconded Kelly that it be held in Canberra in March the weekend prior to the AVA Annual Conference.

Meeting Closed 6 pm

REPORT ON IATA REGULATIONS

Below is a synopsis of Geoff Mitchell's report to the ASVP present by Terry Nicholls as an agenda item at the SCAHLS meeting in Darwin in July.

Geoff's report is a fairly comprehensive document, some 25 pages in length available through me or Geoff to any interested parties. Members attention is drawn to the article cited from the Canadian Veterinary Journal.

The executive has written to the CAA raising issues from Geoff's report and this letter with the CAA's reply hopefully will appear in the next newsletter. Letters have also been sent to the AVA and AHC seeking their support in this matter. The letter to the AVA also seeks their support in advising its members of the requirements for packaging of diagnostic specimens, a matter which should be reiterated by Society members to their various client groups. Negotiations can be made difficult if professionals working in the animal health field are helping manufacture the bullets being fired at us. – Ed

THE IMPLICATION OF IATA TRANSPORT REGULATIONS ON ANIMAL HEALTH SERVICES IN AUSTRALIA

Background

The transport of infectious biological material by air on international flights is controlled by IATA. The Department of Transport and Communications (DTC) have recently indicated that they will be enforcing the two applicable standards, 602 and 650, in the Australian air transport industry. Because of union concerns with human diseases such as AIDS and hepatitis, it is probable that these standards will be applied to domestic road and rail transport.

Issues

- Cost increases in the carriage of veterinary diagnostic specimens for both government and private veterinary pathology laboratories will result.
- Risk assessment of veterinary diagnostic specimens indicates that most veterinary specimens are of low risk, see Special Report by Diane McKelvey, 1993, Canadian Veterinary Journal, **34**: 86-89.

Discussion of Issues

- Private veterinary laboratories depend on mailed or couriered samples for quick turn around and a cost effective service.
- Many public veterinary laboratories also provide similar services for production animals (for example, work test) and are dependent on couriers for the submission of specimens from rural practitioners to regional laboratories, and from regional to central laboratories.

7.

- Courier services in Australia are very competitive and transport costs are reasonable. The imposition of IATA container standards will increase costs in the high risk category (602) between 300 - 1200% depending on packaging requirements. The 602 requirement has a low capacity so the high volume nature of many veterinary laboratory submissions could escalate this cost even more, to a 900-3600% increase in packing/courier costs.
- A risk assessment of the majority of veterinary diagnostic specimens would place them in the low risk category (650) which, although increasing costs for road, rail and air transport, would be acceptable.
- It is important for the protection of the export and domestic trade in animals and animal products that there is no further erosion of animal disease surveillance.
 - unjustified increases in transport costs for private and public veterinary laboratory diagnostic specimens will do this, with no gain in safety to the general public and workers in the industry.
 - and a probable loss of jobs.

Recommendation

- That SCAHLS consider the current regulations, and the move to classify veterinary diagnostic specimens into the 650 category.
- That SCAHLS support immediate consultation with the DTC to clarify this issue, and to reach an acceptable resolution, as described in the Canadian Veterinary Journal **34**: 86-89.

8.

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

Incorporated in Victoria.

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Honorary President: John Gibson

Honorary Secretary: Jim Taylor

Honorary Treasurer: Russell Graydon

16 June 1993

Dr J Malmo
President
Australian Veterinary Association
134-136 Hampden Road
ARTARMON NSW 2064

DECLINE IN GOVERNMENT VETERINARY SERVICES

Dear Jakob,

As you are aware the ASVP executive represents veterinarians interested in pathology who are employed in laboratories, universities and research institutions throughout Australia. Like the AVA, our members are very concerned at the ongoing decline in government veterinary services and this issue was addressed at our recent annual conference. Brief reports on the current position in each State were presented for general discussion. The following is a precis of those reports with additional information supplied by our respective state correspondents. Copies of the entire reports are attached.

Queensland

Government Veterinary Services in Queensland have shown a significant decline in the past 5 years. Currently there are 143 staff employed in Animal Health Laboratory Services, including four regional laboratories and the Animal Research Institute, Yeerongpilly. This is 38 fewer positions than existed in January 1990. Oonoonba Veterinary Laboratory is to change from reactive diagnostic activities to structured surveillance. Should further budget cuts occur the viability of the existing diagnostic laboratory network would be in jeopardy. Animal Health field staff has also suffered severe cutbacks. Since 1989 veterinary officer numbers have fallen from 40 to 26 with 5 veterinary officer centres closing and stock inspector numbers have fallen from 141 to 96 with 18 stock offices closing. There are now less stock inspectors employed in government veterinary services than in 1971 (i.e. pre-BTEC).

Tasmania

Tasmania has 14 Agricultural districts of which 8 have district Veterinary officers with the remainder being staffed by stock inspectors. The state has one veterinary laboratory with a staff of 33. In 1990, demands for economic rationalisation resulted in fees being introduced. Enquiries, on Animal Health were referred to private veterinarians and submissions from farmers direct to laboratories were discouraged. This has resulted in an alarming decrease in production animal submissions and an increase in companion and performance animal submissions. This fact together with the loss of contact with traditional clients is eroding the fundamental role of the government veterinary service in Tasmania.

Western Australia

Failure to fill vacancies has led to the loss of 4 veterinarians in the WA government health service. Six laboratory technicians have also been lost as well as the closure of sections of the Bunbury Regional Laboratory. Services are contracting to Perth. Between 1980 and 1991 stock inspector numbers have fallen by 32%.

South Australia

In 1991 the regional veterinary laboratory at Struan was closed. In 1992 the McKinsey Organisational Development Review recommended a reduction in staff numbers at the Central Veterinary Laboratory from 54 to between 15 and 20. Companion animal work is to cease. The introduction of fee for service has contributed greatly to the serious decline in South Australia's government veterinary services. In 3 years the number of government field veterinary officers has decreased from 11 to 5 with the possibility that two more may be lost.

Northern Territory

For the last 10 to 15 years the Berrimah Laboratory has focused on the BTEC campaign and arbovirus surveillance. The present outlook for government veterinary services looks healthy, with a growing demand by field investigators for laboratory support, however, concerns exist over future funding. The Arid Zone Research Laboratory at Alice Springs has ceased operations.

Victoria

Benalla diagnostic laboratory is to cease diagnostic activity with the anticipated redundancy of 10 staff. Bairnsdale RVL has suffered a 50% reduction in staff in the last five years. Full cost recovery to be introduced by July 1993 is not considered compatible with maintaining an effective disease monitoring service.

New South Wales

The government field veterinary services of NSW are underpinned by the Rural Lands Protection Boards. A policy of non-replacement of staff vacancies exists in NSW state diagnostic laboratories. An unsuccessful attempt to close Armidale Regional Veterinary Laboratory occurred in July 1988.

I trust this information will be useful to you in your submission to the State Ministers of Agriculture etc.

ASVP/AVA REAFFILIATION

A motion was passed at the recent AGM of the ASVP directing the executive to produce a discussion paper on the reaffiliation of the ASVP and the AVA. This paper is to be presented at the next AGM after which the membership will vote on the issue. It is my understanding from our brief discussions that Dr John Plant and Dr Keith Walker will hold preliminary discussions on this matter.

AVA MEMBERS OF THE ASVP

ASVP members who are also members of the AVA expressed a desire at the AGM that they be represented by the AVA in your submissions to the various reviews being conducted into animal health services in this country. I am sure they will be heartened by your inclusion of the above information into your submission to the Ministers of Agriculture. The executive has also been directed by the membership to make a submission to the Animal Health Services Review being conducted by Dr Jim Miller.

10.

Animal Health Services in Australia are under threat as never before. Severe budget constraints, vacancy freezes, economic rationalisation, fee for service and separation packages are elements confronting our memberships in the 90's. I look forward to a cooperative association with the AVA in tackling these challenges.

Yours sincerely

John Gibson
HONONARY PRESIDENT
AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

THE AUSTRALIAN VETERINARY ASSOCIATION LTD.
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Members of AVSP.

I represent all members of the AVA in my submissions to the various reviews to which I am invited to contribute. As stated earlier, the information which you have provided has been, and will continue to be, used for the benefit of all our members.

I can assure you that I am well aware of the threats to Animal Health Services in Australia and we all need to work together to resist these threats.

Yours sincerely,

Jakob Malmo
President, Australian Veterinary Association.

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IN REPLY PLEASE QUOTE:

Dr John Gibson,
Honorary President,
Australian Society for Veterinary Pathology.
C/- Toowoomba Veterinary Laboratory
PO Box 102,
TOOWOOMBA, QLD. 4350

Dear John,

Decline in Government Veterinary Services.

Thank you for your letter of 16 June 1993 and the useful information which it contained.

I have written to all Ministers of Agriculture, CVOs, Farmer organisations, etc. pointing out the importance of maintaining adequate animal health services and asking that there be no further cut-backs, at least until the Miller Committee has presented its report and recommendations.

We have had a response from most ministers and a number of farming organisations. Your information on the situation in each state has been useful - if you receive any updated information I would appreciate receiving a copy of it.

I believe that we need to put as much pressure as we can on governments to recognise the importance of an adequate health service and to have them ensure that adequate services are maintained. We will need to use as many methods as we can to get this message across.

ASVP/AVA Reaffiliation.

I would certainly hope that this can come to pass - we are a small profession and our cause can only be helped by our presenting a united voice. We do not have the resources to allow the luxury of many divided groups.

WESTERN AUSTRALIA

Murdoch University
Clive Huxtable

FELINE DERMATOPHYTE MYCETOMA**History**

A 6 year old Persian cat (sex not given) was presented to a local veterinarian because of multiple dermal nodules over the dorsum and flanks. Similar nodules had been removed previously but had not been submitted for histopathology.

Histopathology

Histopathological examination of a nodule revealed a large focal lesion located in the dermis. Epidermis, subepidermal tissue and hair follicles were normal. The mass was composed of oval, refractile organisms of varying size. Groups of organisms were embedded in small clumps of amorphous eosinophilic material, which were surrounded by many macrophages. A mild infiltrate of neutrophils, lymphocytes and plasma cells was present at the periphery of the mass. The refractile organisms stained strongly with PAS. Hyphae were also revealed with PAS.

Diagnosis:

Feline Dermatophyte Mycetoma.

Comment:

Persian cats appear to be predisposed to this infection. The organism is thought to gain access to the dermis, from hair follicles colonised by dermatophytes following trauma. A genetic, selective immunodeficiency is postulated due to the predisposition of Persian cats to develop these lesions. The fungal species involved was not determined in the case, however, most are caused by *Microsporum canis*.

Reference: Gross, T.L. et al (1992) *Veterinary dermatopathology*. Mosby-Year Book Inc. Missouri, U.S.A.

CONGENITAL ARTERIOVENOUS ANASTOMOSES IN A DOG**History**

A 4 month old entire male Golden retriever was initially presented in 1986 with a 2 month history of a recurring fluctuant swelling at the base of the tail. The tail and perineal area were oedematous and congested and a palpable thrill was detected at the base of the tail.

When surgery was attempted a wide vascular channel, with valves was found. A catheter could be passed 20 cm into the abdomen one way, and to the tip of the tail, the other. The surgical site was closed, and as a congenital vascular anomaly was suspected, the dog was referred to Murdoch University for angiography.

Angiography revealed a large venous sinus along the ventral aspect of the tail. Arterial supply also appeared to be unusual with clusters of branching vessels situated in the most swollen area. The sinus appeared to narrow to the size of a normal vein at the base of the tail.

A congenital vascular anomaly, confined to the tail was diagnosed. The dog was also found to be a bilateral cryptorchid.

13.

Castration was performed initially and was uneventful. Amputation of the tail was performed 3 months after and was also uneventful. The day following surgery serosanguinous fluid oozed from the ventral perineum and the area became swollen. Further examination showed that some abnormal vascular tissue remained in the area. The dog was treated conservatively and improved.

The problem recurred approximately every 6 months, with oedema and swelling of the perineum lasting several days. The dog finally represented in 1993 with recurrence of the oedema, straining to defecate and passing a significant amount of fresh blood with the faeces. The dog was euthanased at the owner's request.

Post mortem

Significant abnormalities were confined to the perineum, rectum and distal colon. The perineal tissues were oedematous and contained numerous vascular channels. The dorsal and lateral walls of the distal colon and the rectal wall contained numerous dilated vascular channels. Dilated cystic structures were present on the serosal surface of the colon and the ventral surface of the bladder.

Histology

Sections of the rectal and colonic wall and perineal tissue revealed numerous dilated vascular channels lined only by endothelium. There appeared to be dilation of existing vessels rather than growth of new vessels.

Diagnosis

Congenital arteriovenous anastomoses.

Comment

These abnormalities arise because of failure of the embryonic anlage to differentiate correctly. They are more common in the extremities, but may occur anywhere. Congenital arteriovenous anastomoses are usually more extensive than predicted by angiography, therefore surgical excision is rarely curative.

References

Fairbairn, J.F. and Bernatz, P.E. (1980) Arteriovenous fistulas, in *Peripheral vascular disease*. J.L. Juergens et al ed. W.B. Saunders Company, Philadelphia, pp. 441-467.

Hosgood, G. (1989) Arteriovenous fistulas: Pathophysiology, diagnosis and treatment. *Compend. Contin. Educ. Pract. Vet.* 11 pp 625-636.

W.A. Department of Agriculture - South Perth - Ron Peet

CAMPYLOBACTER JEJUNI ABORTION IN SHEEP

Approximately 150 abortions occurred in a flock of some 1,200 ewes at Dongara (near Geraldton) in April 1993. Most aborted lambs were in last 3-4 weeks of pregnancy and ewes appeared healthy.

Original submissions to the Animal Health Laboratories were from a sacrificed ewe because no foetus was available. Submissions consisted only of a swab of purulent material from the uterus and formalin-fixed sections of her liver and kidney. These showed no significant changes, but *Campylobacter jejuni* and *Histophilus ovis* were cultured (both medium growth) from the swab.

14.

After a telephone conversation with the referring veterinarian, further samples from an aborted foetus were collected. These included fresh brain, foetal stomach contents and placenta plus a range of fixed tissues.

Campylobacter jejuni was again cultured from the placenta and foetal stomach contents. Gross and microscopic examination of the liver did not reveal the marked areas of necrosis described in the literature (Kennedy and Miller), but there were numerous focal microabscesses visible which were distinct from the haemopoietic centres.

C. jejuni is a commensal of cattle, sheep, swine, poultry, dogs, cats and many rodents. Transmission is by the oral route and abortion may occur in non-immune pregnant ewes (Kennedy and Miller).

In our experience, this abortion due to *C. jejuni* is rare in Western Australia.

Kennedy, P.C. and Miller, R.B. (1991) in "Pathology of Domestic Animals" edited by Jubb, K.V.F. Kennedy, P.C. and Palmer, N. 4 edn. Vol. 3, Academic Press, Sydney, P402.

W.A. Department of Agriculture - South Perth - John Creeper

APPARENT INTESTINAL ACCIDENT IN PIGS

In a management mix up, a 2000 head extensive piggery withheld feed for 3 days. After this time ad lib feeding was commenced with home grown grain and meatmeal from a source not used previously. The piggery started losing 10-20 pigs per day after feeding was re-established and continued to do so for 14 days, by which time a total of 200 pigs had died.

On post mortem there was sand impaction within the spiral colon, intestinal haemorrhages and bloating - described as "redgut" by the practitioner. Histologically there were severe small intestinal mucosal haemorrhages and sub mucosal congestion. Within mesenteric lymph nodes were focal areas of necrosis associated with clostridial-like organisms, indicative of a loss of gut integrity. There was no bacteriological evidence of intracellular CLO's, *Serpuina* sp or *Campylobacter* organisms.

The deaths ceased immediately following the introduction of hayrolls into the paddocks.

A diagnosis of probable intestinal obstruction was made. Several outbreaks have been previously seen where ingestion of excess sand has lead to intestinal accidents. Pathological findings have been similar to this case.

Contributor

Dr. Max Cooper, Narrogin Veterinary Hospital.

W.A. Department of Agriculture - South Perth - Marc Kabay

BONAMIOSIS IN FLAT OYSTERS (*Ostrea angasi*)

Following the first diagnosis of Bonamiosis in flat oysters from Oyster Harbour, Albany in August 1991, several follow up investigations have been conducted. Histological examination of oysters collected during February 1992 showed convincing lesions that confirmed the initial diagnosis.

Two distinct lesion types were identified. In 18 month old oysters, lesions were restricted to the gut. There was segmental ballooning degeneration and attenuation of the mucosal epithelium. Within the lesions were multiple intracellular and free organisms 2-3 um in diameter with a central dense body consistent with the morphology of *Bonamia* sp.

15.

In 4 year old predominantly female oysters, infection was systemic. There was an intense mononuclear cell infiltrate surrounding degenerating gonadal tissue. In addition, there were focal areas of necrosis. Large numbers of *Bonamia*-like organisms were present in tissue adjacent to the lesions. Lesions in the heart, as reported in New Zealand were not present.

All oysters examined showed varying degrees of mononuclear cell infiltration into the interstitial tissue. Mononuclear cells containing eosinophilic intracytoplasmic granules were common. In contrast, this cell type was absent when *Bonamia* organisms were present. This suggests that either the cell type is critical to an effective host response or that the organism causes the cell to "degranulate". However, free eosinophilic granules were not observed. Comments on the significance of this cell type would be appreciated.

VIBRIO SP SEPTICAEMIA IN SALT WATER ROCK LOBSTER (*PANULIRIS CYGNUS*)

An investigation was conducted into mortalities (up to 20%) in crayfish held in holding tanks prior to export. Affected fish were moribund with flaccid tails. In some fish, the haemolymph had coagulated around the heart.

Granulomas, varying in chronicity and containing large numbers of gram negative bacteria were present in a variety of organs including the heart, ovaries, gills, digestive gland and muscle. A pure heavy growth of an unidentified *Vibrio sp.* was consistently recovered from the haemolymph.

The syndrome appears to be stress induced. The syndrome has appeared in previous years at the end of the cray fishing season when water temperature is rising. Fish are also kept for longer periods in holding tanks and at a higher stocking density. Territorial aggression may also be an important stress factor.

W.A. Department of Agriculture - South Perth - CleveMain

OVINE CRYPTOCOCCOSIS

Cryptococcus neoformans var gattii was diagnosed by fungal culture, histopathologically and serologically in a flock of 1900 mixed age merino wethers run on a property in the extreme south west of the state.

The diagnostic lesion was found in a large mass obtained from the nostril of one of approximately 50 animals culled for so called "head cancer" this year. In previous years the annual loss from this condition has been 30-40 animals.

Lesions found in other sheep have been located in the subcutis of the forehead, around the base of the horn and the periorbital tissue. No examination has been made of the remainder of the respiratory tract or the central nervous system.

Histologically the nasal lesion is characterised by large areas of myxomatous and granulomatous tissue with numerous spicules of osseous tissue also evident. In the latter areas there were foci of severe tissue necrosis with extensive vasculitis, haemorrhage and infiltration by neutrophils, macrophages and other mononuclear cells. By contrast little inflammatory activity were seen in the myxomatous areas which contained numerous PAS positive organisms with a wide polysaccharide capsule characteristic of *Cryptococcus*.

The interest in the case is due to the occurrence of the disease in numerous sheep over succeeding years and the organism's epidemiological association with the river red gum *Eucalyptus cameldulensis* (Ellis D.H. & Pfeiffer T.J. - Lancet 1990; 336: 923-25).

16.

It has been postulated that the principle infectious propagule of this variety of the fungus is the basidiospore and that exposure to *E. cameldulensis* is required to initiate infection in man and animals. Unlike *C. neoformans* var *neoformans*, var *gattii* is said not to require impaired immunocompetance to establish infection (McAleer R. Health Department of Western Australia, pers comm).

In this case however, *E. cameldulensis* does not exist on the property where the sheep are run and in fact does not occur naturally in the south west of the state. The fungus may be associated with other flora and investigations are continuing.

CONGENITAL MUCOSAL DISEASE

Mucosal disease was diagnosed in a herd of 3000 cattle run on a coastal property in the south of the state. The problem was confined to calves born within a group of 700 1991 drop heifers. At the time of writing, 16 calves were stillborn and 6 were congenitally blind. Examination of the brain and eyes from some of these calves revealed lesions ranging from retinal hypoplasia, cerebellar hypoplasia and severe hydrancephaly. The nature of the brain lesions suggests an insult during the 90-150 days of gestation.

Serological examination has demonstrated higher MDV titres in the heifers giving birth to affected calves than those delivering clinically normal calves. MD antibody was also detected in a precolostrum serum sample taken from a blind calf and the virus has been isolated from an ill thrifty heifer running with the group.

Control measures include running 1992 maiden heifers with already infected cattle prior to mating, ensuring that bulls selected for this years heifer matings are either serologically positive for MDV or if negative, are shown by culture not to be viraemic. Use of a vaccine prepared from the MDV isolates obtained from the herd is also being considered.

CAPRINE COCCIDIOSIS INVOLVING BRUNNER'S GLANDS

Intestinal coccidiosis was diagnosed in a 3 year old feral goat held in a Geraldton feedlot. 100/200 companion goats were suffering from a green watery diarrhoea and of these, 3 had died.

Necropsy revealed evidence of "necrotic enteritis" and a heavy burden of *Haemonchus*, *Ostertagia* and *Trichostrongylus* spp.

Histopathological examination revealed marked to severe dilatation of Brunner's glands with numerous coccidial forms representing all stages of their life cycle present in the epithelial cells of many of them. The lumina of many glands contained oocysts and/or sporozoites together with low numbers of neutrophils. In other glands there was evidence of epithelial hyperplasia and hypertrophy associated with large numbers of coccidial organisms within epithelial cells. Frequently where there were large numbers of oocysts within the lumen, the structure of the gland was obscure and there was an apparent early granulomatous response. In the surrounding submucosa there was a patchy mononuclear inflammatory response, mostly macrophages and plasma cells and mostly centred around infected glands.

It is not surprising that feral goats taken from an arid environment would fall victims to parasitic diseases when placed in feedlots in a relatively moist environment. The interest in the case lies with the location of the intestinal infection.

In the writer's experience, coccidiosis involving Brunner's glands has not been previously encountered. Unfortunately no samples that would allow culture and identification of the parasites were taken from that animal. However culture attempts will be made on faecal samples from other goats in the feedlot showing evidence of diarrhoea.

W.A. Department of Agriculture – Albany - David Forshaw

MYCOBACTERIOSIS IN A PYGMY SPERM WHALE

A pygmy sperm whale beached itself near Mutton Bird Island approx 20km west of Albany. A group of people pushed the whale back out to sea. The next day, the whale re-beached close to where it originally came ashore.

The whale was euthanased by a private veterinarian. I did the PM on the beach about three hours later. The whale was a mature male about 3.5m long. There were numerous skin abrasions, most severe around the eyes. The contents of the large intestine and lower small intestine were very dark, apparently blood stained. No abnormalities were detected in the upper small intestine and stomach mucosa. In the fading light, I could see no source of the haemorrhage.

Other abnormalities included a moderate degree of pulmonary oedema and numerous parasites; a huge number of round worms in the stomach, long (to 20cm) roundworms in the skin associated with a large cystic structure and tapeworm cysts in the blubber. Specimens have been taken for identification.

Multiple granulomas up to 2cm in diameter were seen in the right submandibular node. The cut surface was pale yellow with white flecks. Histologically, there were numerous long and beaded acid fast bacilli within a necrogranuloma.

There was no microscopic lesion in the sections taken to account for the intestinal haemorrhage. No other significant lesions were seen in other organs examined including the brain, (the head was examined later at the lab for obvious reasons!) Why the whale beached is not clear.

Polymerase chain reaction test results (courtesy of Debbie Cousins of the TB reference centre in South Perth) from samples of fresh tissue suggested that the organism is **not** of the *M.tuberculosis* complex. The organism is now being cultured.

As far as I am aware, there are no recorded mycobacterial infections in cetaceans.

OUTSTANDING MEMBERSHIP FEES

Following is a list of members who as of August 1993 are not financial. Could this be remedied as soon as possible.

If your name appears on the list and you have paid your membership before August, please contact the Secretariat.

OUTSTANDING FOR 1993 - PAYMENT DUE \$25.00

ACLAND HM	ADAMS NR	BADCOE LM	BELFORD C
BOULTON JC	CAMPBELL Prof. RSF	CHOOI KF	COORDES DO
DANIELS P	ELLIS TM	FRASER G	GIESECKE PR
HOOPER PT	JACKSON ARB	JACKSON C	LADDS PW
LEHARNE L	LINKS IJ	MARSHALL DJ	McEWAN DR
McGAVIN MD	MITCHELL G	MUNTZ F	PEET R
PHILBEY AW	SAMUELS J	STAPLES P	STORIE GJ
TRUEMAN KF	VANSELOW B	WATT DA	WEBBER JJ

18.

OUTSTANDING 1992 & 1993 - PAYMENT DUE \$45.00

BEERS P	CALLAHAN JT	CROSS GM	GLEESON LJ
HINDMARSH M	HOWLETT CR	HUMPHREY J	HUXTABLE CR
JOHNSTONE A	LATTER M	LATTER M	McCOLL K
RIFFKIN GG	ROTHWELL TL	SLOCOMBE R	SMITH HV
SMITH BL	SUMMERS BA	TOWNSEND W	WICKHAM N
WILLIAMS OJ			

MAIL RETURNED: TIMMINS BJ

TAKEN OFF MAILING LIST FOR NON-PAYMENT OF 1991 ONWARDS

BARKER IK	CARRIGAN M	OLIVER RE	RAWLIN G
WILLIAMS DM			

VICTORIA - RVL BAIRNSDALE**ENTERITIS IN OSTRICHES AND EMUS – Peter Mitchell and Kit Button**

Several outbreaks of diarrhoea leading to death in ostriches have been investigated over the past few months, and we have heard of similar cases at other laboratories. Affected birds were around 3-4 weeks. There was little to see on gross post mortem - slight increases in the ratio of proventriculus contents to body weight (to about 6-8% , compared with normal values of about 5%) are common in sick ostriches. The most consistent finding (even in autolysed birds) was an increase in mononuclear cells in the lamina propria of the small and large intestine (in normal birds, few cells are present in the lamina propria). In fresh specimens, many of these cells were clearly necrotic with fragments of nuclear material, and there was some evidence of inflammatory cells and nuclear remnants in otherwise healthy epithelium. In a few cases, mild sub-epithelial vacuolation through to large spaces beneath the superficial epithelium were seen, with protein in some of the spaces. Epithelial cells covering these spaces were sometimes necrotic and sloughing. No micro-organisms were identified – virology is in progress.

An outbreak of severe haemorrhagic enteritis recently occurred in emus, with 9 deaths over a few days and another 12 affected, on a farm with about 100 birds. Affected birds were 5-6 months old. Apart from some changes due to shock, the lesions were restricted to the small intestine. The primary reaction appeared to be infiltration of the lamina propria with mononuclear cells and heterophils, many necrotic, and marked congestion. In a few places, the epithelium of the villi was necrotic, but the epithelium at the tips of most villi had sloughed, either due to autolysis or necrosis. Sub-epithelial vacuolation was occasionally present but large protein-filled spaces were not seen beneath the epithelium. However, the disease in the emus was more acute and birds died after a relatively short clinical illness. *Campylobacter jejuni* was isolated from the intestines from one of two birds - the significance of the isolate is unknown at this stage. Otherwise, no pathogens were identified. We would be interested to hear of any other similar cases.

RVL HAMILTON**CORYNEBACTERIUM RENALE - Janeen Samuel**

Blood and urine were received from a four-year-old Hereford cross cow one week post-partum. The cow showed malaise, atonic gut, grunting respiration and pale mucous membranes, and was passing red mucoid urine. The haematological picture was normal, as was serum phosphorus, but there were elevated levels of urea (102 mm/L), CK (767 U/L) and globulin (64.5 g/L). The urine contained numerous erythrocytes and yielded a pure culture of a haemolytic, gram-positive diphtheroid organism which was identified biochemically as *Corynebacterium renale*. The cow died the day after the samples were taken. This picture of severe pyelonephritis in a recently calved cow is reportedly typical for disease due to *C. renale*.

C. renale was also isolated from 2 cows which showed cystitis only; they were passing urine containing blood and pus but were not acutely ill. One of these cases occurred 4 months after the first case, on an adjoining property.

YEW POISONING - Janeen Samuel

In April, after a long period of dry weather, prunings from garden shrubs were thrown into a paddock where there were 32 Shorthorn steers in fat condition. Next morning 7 of the steers were found dead. There were no significant findings at post mortem. The clippings included large amounts of yew (*Taxus sp.*), and yew leaves were found in the abomasal contents.

As readers of Agatha Christie will know, yew contains the alkaloid taxine. This slows the heart and stops it in diastole. There are many records of deaths in cattle due to this plant, and deaths frequently occur suddenly without premonitory signs or significant lesions. Subacute poisoning may produce gastro-enteritis due to an irritant oil, but this was not observed in the survivors in this case.

Ref: Agatha Christie "A Pocketful of Rye".

SEVERE ACUTE MASTITIS - Janeen Samuel

In April and May we cultured *Pseudomonas aeruginosa* from cases of mastitis on 3 dairy farms. In all cases the cows were acutely sick and recumbent, and at least 3 of the 7 reported cases died or were euthanased. One vet reported sloughing of skin from the affected quarter and a fluorescent green diarrhoea. On two of the farms the cases all occurred in the first 2 days post partum. The case on the third farm was in a cow that had been dried off and given dry-cow therapy 5 days earlier. On one farm that had 4 cases, a new water system had been installed just before the cows were dried off; *Pseudomonas aeruginosa* was isolated from the bore supplying the system, the hot water tank and the teat washer.

These were the first cases of acute *Pseudomonas* mastitis seen at this laboratory for at least 3 years, and we have postulated that the unusually wet summer increased the chance of *Pseudomonas* being introduced into the mammary gland, from contaminated skin, at the time of administration of dry-cow therapy. We have also seen an unusual number of cases of *E. coli* mastitis: 8 cases over April, May and June. Not all of these gave adequate histories, but they included 4 cows with mastitis shortly after being dried off with dry-cow therapy, and one with mastitis one week post partum; 4 were recorded as being "very sick" or "septicaemic".

Another somewhat unusual case was a severe *Staph. aureus* mastitis in an Aberdeen Angus cow. It affected one quarter only but was accompanied by swelling of the hind legs. We rarely have submissions from mastitis in beef cows.

RVL BENDIGO**JOHNE'S DISEASE IN ALPACAS**

Johne's disease has been diagnosed in alpacas for the first time in Australia. The initial diagnosis was made in February on a yearling alpaca from a property near Bendigo. Further cases have since been diagnosed in two other young alpacas on separate properties from parent stock derived from one common property. All three were about 12 months old. In each case, the dam was imported from Chile via New Zealand.

Clinical signs were ill thrift and occasional loose faeces. The initial case had been doing poorly for several months and had failed to respond to gastro-intestinal parasite treatment. The initial case died on the property and was autopsied by the practitioner out of curiosity. The principal lesions he described were massively enlarged mesenteric lymph nodes, up to five times normal size. On cut section the nodes appeared homogeneous, were cream coloured and were of a soft consistency. Some nodes appeared to have softer centres suggesting necrosis. Prominent Peyer's patches in the small intestine were also noted. Selected specimens of lymph nodes and intestines were submitted to the laboratory. Lymphosarcoma was initially suspected of being the cause of the disease although culture of the specimens was requested for possible bacterial infection.

The second case was also submitted to this laboratory with the post mortem findings similar to the first case. One feature which was different was the firmness and degree of fibrous tissue throughout some of the mesenteric lymph nodes. The overall picture was one of massively enlarged greyish firm nodes and very obvious Peyer's patches.

Histopathology

The lymph node reaction was one of uniform replacement of lymphoid follicular structures with broad sheets of macrophages and moderate numbers of lymphocytes and Langhan's giant cells. No caseation was evident anywhere in any of the nodes or intestine. Ziehl-Neelsen stains on sections of the nodes revealed large numbers of Z-N positive organisms. Fresh tissues held by the Bacteriology lab following culture were forwarded to the Mycobacteria Reference Laboratory in WA for Polymerase Chain Reaction (PCR) enhancement which proved positive for Johne's disease within a week of submission. Mycobacterial culture confirmed the PCR result.

21.

Histopathology on the second submission was essentially the same as the initial submission with the exception of increased fibrous tissue around and throughout the mesenteric lymph nodes. Caseation was absent from all lesions.

POSSIBLE ZOLLINGER - ELLISON SYNDROME IN A CAT

A biopsy taken from an ulcerated area of the stomach of an 8 year old domestic cat was submitted for histopathology. The cat had a history of vomiting, initially of fur balls later of food. There was no history of polydipsia or polyuria and clinical examination indicated masses in the abdomen. Ulcerated areas in the stomach were found by exploratory laparotomy along with enlarged mesenteric lymph nodes. Samples of the ulcerated stomach were received for histopathology. The clinician indicated that the tissues were not immediately preserved and hence moderate autolysis had occurred.

Histopathology: The stomach mucosa which had been ulcerated was under-run by sheets of polyhedral cells with pale abundant cytoplasm and centrally placed small to medium sized dark nuclei. A fine stroma of connective tissue and blood vessels gave the tumour an endocrinal appearance. A tentative diagnosis of Gastrinoma was forwarded to the clinician.

The cat improved considerably following surgery which had removed the area of gastric ulceration. However, two months later the vomiting recommenced. Blood samples were taken from this cat and another "normal" cat and submitted for Gastrin Radio Immune Assay using a human system. (Dorevitch Pathology, Melb)

The results were:- affected cat	144 pM/L (picaMol/litre)
normal cat	<10 pM/L
normal human	<43 pM/L

These results along with the histopathological appearance typical of a Gastrinoma lead to the diagnosis of Zollinger - Ellison syndrome in the cat. To date a scan of the literature has not revealed any previously recorded cases of this syndrome in the cat.

RVL BENALLA

PROTOZOAL ENTERITIS IN AN OSTRICH - Judith S Nimmo Wilke

A young ostrich, a few months of age, had been depressed for a few days and developed a dark foul-smelling diarrhoea. It died following exploratory surgery. The intestines contained a large quantity of dark, foul-smelling fluid. Histopathology revealed very large numbers of large protozoa (40 x 60µ) with a single nucleus, a large contractile vacuole and a ciliated cytoplasmic membrane - probably *Balamidium* sp., free in the lumen and in crypts. Many crypts were dilated and there was an increase in mucus cells.

CENTRAL VETERINARY DIAGNOSTIC LABORATORY

PARASITIC UNGLUVITIS IN AN HELMETED HONEYEATER

This wild bird from the Melbourne Zoo was translocated to a new location and found dead. On post mortem worms were found embedded in the mucosa of the crop.

Histological examination revealed diffuse hepatic lipidosis. There was marked acanthosis of the crop mucosa and hyperplasia/ectasia of sub-mucosal glands. Bi-operculate eggs, 35-40µm x 50-60µm, with a 5mm diameter striated shell were present in submucosal glands. Adult worms were buried within the epithelium, surrounded by a mild heterophilic infiltrate. These worms had a chitinous body wall with coelom including a uterus and intestine. The body wall was lined with coelomyarian muscles and the uteri contained eggs.

22.

Diagnosis: Hepatic lipidosis & parasitic ungluvitis

Comments: These parasites are most consistent with capiliaria sp. The intermediate host is the earthworm.

JOBLINE

FACULTY POSITION IN VETERINARY ANATOMICAL PATHOLOGY

The Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, seeks applicants for a faculty position in veterinary anatomical pathology. This is a tenure tract position, but applicants for an immediate temporary appointment will be considered. The successful applicant must be an enthusiastic teacher, have research interests and experience in the pathogenesis of domestic animal diseases and be a competent diagnostician. Duties will include teaching at both the graduate and undergraduate level, guidance and supervision of graduate students, some diagnostic service in the context of clinical teaching, and independent research. Tenure and promotion decisions are based on performance in each of these areas. The candidate must be adaptable to the many demands on faculty in a busy academic department. Applicants must have a PhD degree or equivalent post-graduate education in research in the pathogenesis of disease and must also have had a post-graduate education in diagnostic veterinary pathology. Certification by the American College of Veterinary Pathologists or eligibility to write the ACVP board examination is desirable. For a temporary appointment, less extensive qualifications will be required. In accordance with Canadian immigration requirements, priority will be given to Canadian citizens and permanent residents. The University of Saskatchewan is committed to the principles of Employment Equity. Position #15-4-6.

Applicants should send a letter of application, **curriculum vitae** and contact information for three professional references to:

Dr Craig Riddell, Head
Department of Veterinary Pathology
Western College of Veterinary Medicine
University of Saskatchewan
Saskatoon, SK S7N 0W0
Canada

Applicants interested in an immediate temporary appointment should FAX their application to (306) 966-8747. Dr Riddell will be present at the World University Poultry Association Congress at the Sydney Hilton, Australia from August 15-19 and will be willing to meet any interested applicants at that time if convenient.

This position was circulated to all state reps for distribution in late July.

NEW SOUTH WALES - REGIONAL VETERINARY LABORATORY, WOLLONGBAR

ASVP cases - NSW

THEILERIOSIS IN AN ADULT COW - Roger Cook, Graeme Fraser, Paul Gill

Theileriosis was diagnosed as the cause of a fatal subacute haemolytic anaemia in a 6-year-old cross-bred beef cow in the tick quarantine area of northern NSW. The animal was found dead after appearing ill for 2 weeks. Jaundice, swollen kidneys and splenomegaly were noted by the practitioner at necropsy. Moderately severe renal haemosiderosis and marked splenic congestion were the main histological lesions. The cow was anaemic (PCV 0.06) and 5-10% of erythrocytes in blood smears contained pleomorphic parasites suggestive of *Theileria sp.*, mainly in ring and dot forms with the occasional pair. However, there were some organisms typical of neither *Babesia* nor *Theileria spp.* Fluorescent antibody tests on blood and brain smears performed at the Tick Fever Research Centre, Wacol, were negative for *Babesia bovis*; positive reactions with the *Babesia bigemina* conjugate were attributed to cross reactivity of the conjugate with *Theileria sp.* Reactions to serological testing (IFAT) of incontact animals were: *Theileria buffeli* (42/43) positive, *B. bigemina* (1/43) suspect *B. bovis* (1/43) suspect and (1/43 1 +), *Anaplasma* (2/43) positive. These results in conjunction with the prolonged clinical course indicated that the fatal subacute haemolytic anaemia in this cow was associated with *Theileria buffeli* infection. Fatal theileriosis in adult cows in the Northern Rivers region of NSW is very unusual. Such a low parasitaemia as in this cow is not usually associated with clinical theileriosis. Deaths in 3 mature dairy cattle in south-east Queensland were associated with 100% parasitaemia (Rogers and Callow, 1966). The atypical organisms in smears were probably *T. buffeli*, which can become bigger and more pleomorphic when proliferating rapidly in acute or subacute conditions (Callow, 1982). We thank Bert de Vos, DPI Queensland for his assistance with this investigation.

References: Rogers RJ and Callow LL (1966), Aust vet J 42: 42-46.,
Callow LL (1982) in *Animal health in Australia* Vol 5: p171

CERVICAL VERTEBRAL CANAL STENOSIS IN A GOOSE - John Boulton

Three of 60 adult Chinese geese over a 1-year period developed a neck abnormality characterised by a limp neck which curved down and backward. At necropsy the opposing right articular facets of C14/C15 were enlarged and dish-shaped with small peripheral bony nodules: the right facets of C15/C16 were similar, but less severely swollen. C15 was abnormally short and twisted to the left. At its cranial and caudal margins the intervertebral discs were lipped into the spinal canal, there being particularly severe stenosis at the C15/C16 articulation.

Local myelomalacia was found in the cervical spinal cord at the level of the articulation of the 14th and 15th cervical vertebrae. The cervical vertebral canal stenosis and local myelomalacia in this goose seems similar to that in horses and dogs with wobbler syndrome.

John's trivial pursuit question: How many cervical vertebrae does a goose normally have?

SCONE DIAGNOSTIC VETERINARY LABORATORY**DEGENERATIVE MYELOENCEPHALOPATHY IN A FOAL** - Angela Begg

A 3-month-old thoroughbred foal developed slowly progressive ataxia in all 4 legs with dysmetria of the forelegs. A cervical vertebral stenotic myelopathy was suspected. The foal was euthanased at 5 months of age by which time the ataxia had deteriorated to a 'Grade 3 wobbler level' (moderate ataxia with tendency to buckle or fall at normal gaits or when turning, backing or with neck extension). No gross pathological abnormalities were detected on post mortem examination. Histological examination of the brain and spinal cord revealed neuroaxonal dystrophy and spheroid formation due to axonal swelling in the caudal brain stem nuclei (the medial and lateral cuneate nuclei in particular), the lateral cervical nucleus in the cranial cervical spinal cord and the nucleus of the dorsal spinocerebellar tract in the thoracic spinal cord with mild Wallerian degeneration in spinal cord white matter tracts. The aetiology of this condition is unclear but Vitamin E and/or selenium deficiency, copper deficiency and exposure to pyrethrins, creosote and heavy metals have all been implicated in some cases. A familial predisposition is suspected in Morgan and standardbred horses. This disease is rare in Australia, a couple of cases having been reported in Victoria. This foal may have been exposed to creosote on recently painted stud fences. Vit E and selenium deficiencies were excluded.

VETERINARY PATHOLOGY SERVICES - SYDNEY**IATROGENIC OR ENDOGENOUS HYPERADRENOCORTICISM** - Bill Vernau

A female 6 year old Maltese presented to the veterinarian with depression, polydipsia/polyuria, anorexia and vomiting. The dog had previously been diagnosed as a 'Shaker Dog' and this was currently 'Controlled' with 5mg Prednisolone 3 x weekly.

On physical exam the dog had anterior abdominal pain, palpable hepatomegaly and severe skin disease on the ventral abdomen that looked most like calcinosis cutis. Routine blood work gave the following values:

BIOCHEMISTRY		REFERENCE
Range		
Glucose nmol/L	10.9	3.2-6.6
Urea nmol/L	17.8	2.7-6.6
Creat. nmol/L	0.11	0.06-0.18
T-Bili umol/L	11.0	0.8
ALK PHOS U/L	21970	20-70
AST U/L	240	15-70
ALT.U/L	1134	3.0-7.0
Cholest. nmol/L	10.2	3.0-7.0
Chloride nmol/L	96	105-115
Lipase U/L	2670	<500
Amylase	5102	<2500
HAEMATOLOGY		
PCV L/L	0.49	0.37-0.55
WCC x 10 ⁹ /L	35.3	6.0-17.0
	% x 10 ⁹ /L	
Bands	14 4.9	0-0.2
Neuts	74 26.1	4.0-12.0
Monos	12 4.2	0.1-0.5
Toxic neutrophils	2+	
LALP 2100		

The ALP was all LALP, suggesting steroid induction. This seemed excessive for iatrogenic hyperadrenocorticism in this case as the dose of Prednisolone was only 5mg 3 x weekly. Nevertheless this was still conceivable.

Additionally, whilst steroids can cause neutrophilia and monocytosis, they rarely cause $4.9 \times 10^9/L$ bands (left shift with toxic changes). Lipase is also inducible by steroids but rarely above 3 x the upper reference limit, in this case, the lipase was more than 5 x the upper limit. Amylase is not inducible by steroids and was 2 x increased in this case.

Therefore, the dog had pancreatitis, perhaps predisposed by the probable hyperadrenocorticism, either endogenous or iatrogenic.

The stand alone test of choice to confirm hyperadrenocorticism is the low dose dex suppression test. However, if the suspicion is high and iatrogenic hyperA needs to be differentiated from 'real' hyperA then the ACTH stimulation test is the most useful – this was therefore performed.

Baseline cortisol: 97.8nM
2hr post ACTH: 630.4nM

This result confirmed endogenous or real hyperadrenocorticism. An iatrogenic hyperA dog would show little or no stimulation. Stimulation greater than 550nM/L post ACTH confirms hyperA (although chronically sick or 'stressed' dogs will stimulate higher too).

A high dose Dex suppression (1mg/kg Dex I/V) test was performed to determine if the dog was pituitary dependent or an adrenal tumour.

Baseline cortisol: 153.9nM
8hr post desamethasone: 39.0nM

This confirms a pituitary dependent problem.

This case illustrates several useful points:

- patterns of enzyme induction, including lipase, with hyperadrenocorticism;
- how the size of the lipase increase and the left shift indicated 'other' disease, that is, pancreatitis which was very compatible with the anterior abdominal pain;
- use of the ACTH stimulation test to differentiate iatrogenic from real hyperadrenocorticism.

In this case, the distinction was important. The dog could not be taken off its steroids because severe neurologic signs recurred. Therefore, withdrawal to control the signs of hyperA and the pancreatitis was not an option. However, if the dog was a 'real' hyperadrenocorticoid dog, then it was potentially treatable. Skin histopathology confirmed florid calcinosis cutis.

LOW NA:K RATIO IN A DOG

An 8 year old spayed female German Shepherd cross was submitted with depression, anorexia, vomiting, abdominal tenderness and distension. The animal was hospitalised on i/v fluids and supportive therapy for 48 hours before blood samples were collected.

Urea mmol/L	50.1	T-Bili umol/L	47.6
Creat. mmol/L	0.59	C-Bili umol/L	29.7
Protein	68	ALP U/L	1319
Albumin g/L	29	AST U/L	370
Calcium mmol/L	2.3	CPK U/L	1011
Sodium mmol/L	137	PCV L/L	0.49
Potassium mmol/L	6.5	WBC x 10 ⁹ /L	28.7
Chloride mmol/L	96	Neuts	25.8
Bicarbonate mmol/L	12.2	Bands	0.3
Na:K ratio	21.1	Monos	1.7
		Abdominal Fluid Creatinine	1.20

INTERPRETATION

Mild neutrophilia and monocytosis. Marked azotaemia. Marked cholestasis. Hyperkalemia and hypo'saltaemia'. Rule out renal failure (USG??), Addison's, or bladder rupture. Concurrent cholangiohepatitis or tumour or peritonitis.

SUGGESTION

Reconcile inflammatory leukogram, abdominal distention and clinical chemistry. Do abdominocentesis and creatinine on fluid (1.20 mmol/L i.e. » serum (0.59)).

DIAGNOSIS

Bladder rupture with mild urine peritonitis - surgically confirmed. Likely due to trauma.

DISCUSSION

This case illustrates the need to differentiate causes of low Na⁺, high K⁺ and low Na:K ratio. Possibilities include hypoaldosteronism (Addison's), renal disease, GI disease and post renal UT rupture. (Ureter, bladder), internally leaking urachal stump may be a cause in neonates. Na:K < 25 is suggestive of Addison's (especially if the Na is low), Na:K < 22 is strongly suggestive provided other signs are compatible. However, other diseases can cause Na/K ratio less than 22. If in doubt, an ACTH stimulation test is diagnostic. Many cases will also have a low resting cortisol (< 10nmol/L).

Comparison of abdominal creatinine Vs serum creatinine is a useful test to determine presence of uroabdomen. Urea tends to equilibrate more rapidly but creatinine, a larger molecule, maintains its differential.

SOUTH AUSTRALIA - VETLAB, ADELAIDE**CAPRINE JOHNE'S DISEASE – Vui Ling Tham**

A case of caprine Johne's disease from a small property in the Adelaide Hills was reported in Veterinary Pathology Report No. 35, November/December, 1992, page 21.

In May, this year tissues from two more Anglo-Nubian does from the same property were submitted to this laboratory for examinations for Johne's disease. Both of these animals were serologically positive for Johne's disease about 6 months prior to their euthanasia.

Doe (1) had very healthy twin doe kids about one month prior to her euthanasia. She was in good body condition with no sign of scouring at the time of euthanasia. However, post-mortem examination revealed thickening of the mucosa of the terminal portion of the ileum and increased fibrous tissue on the serosal surface of this section of the ileum. The mucosa of the ileo-caecal valve was moderately thickened. No gross changes were noted in the rest of the alimentary tract or in the mesenteric lymph nodes. Histologically, aggregates of epithelioid cells containing numerous acid-fast rod-shaped bacteria were present in the mucosa, submucosa, tunica muscularis, and Peyer's patches of the jejunum, ileum, caecum and colon. Similar epithelioid cells were scattered through the cortex of the mesenteric lymph node.

Doe (2) was a companion of doe (1). She aborted and appeared depressed at the time of her euthanasia. Apart from quite marked mineralisation and necrosis of most of the mesenteric lymph nodes, there were no other gross changes on post-mortem examination. Histologically, granulomas with areas of necrosis and calcification and associated with a few acid-fast rod-shaped bacteria in epithelioid cells were noted in the mesenteric lymph nodes. A few small aggregates of epithelioid cells containing a few acid-fast rod-shaped bacteria were present in the mucosa of only the terminal portion of the ileum.

Cultures for mycobacteria from the affected intestines and mesenteric lymph nodes are in progress.

VETERINARY PATHOLOGY SERVICES (ADELAIDE)**'SUPERFICIAL NECROLYTIC DERMATITIS' IN A DOG - Ruth Reuter**

An eight-year-old neutered female *Lhasa Apso* was being minded for a month while the owner was out of town. When the dog was returned to the owner, it had ulceration of the footpads and skin-pad junction of all four feet, and reddening of the mucous membranes of the mouth and vulva. There was no apparent access to contact irritants. The veterinarian suspected an immune-mediated disorder such as pemphigus and submitted two small wedge biopsies of skin from the skin-pad junction to the laboratory for histopathology.

The sections of skin exhibited hyperkeratosis with focal ballooning degeneration, epidermal necrosis and bullous formation in the suprabasal layer. The bullae present contained erythrocytes and neutrophils. In some areas the necrosis extended into the superficial dermis. The deeper dermis was oedematous but otherwise unaffected. Although conditions such as pemphigus could not be definitely ruled out, the microscopic changes were not felt to be typical of this disorder and the possibility of an underlying systemic disease associated with a superficial necrolytic dermatitis was suggested. The veterinarian opted to try treatment with Prednisolone. Ten days later the dog began vomiting and a blood sample was submitted for biochemistry and haematology.

28.

The most significant findings were as follows:

ALK PHOS U/L	2699	(20-70)
AST	158	(15-70)
ALT	549	(15-70)
GGT	54	(0- 10)
Bile acids	58	(<20)

The isoenzyme LALP, which has been associated with influence of steroids, was 662 U/L suggesting that it was not the major type of alkaline phosphatase present. These findings indicated hepatic disease. The owners requested euthanasia, but did not wish a necropsy to be performed.

Superficial necrolytic dermatitis is an uncommon skin disease of dogs which has been associated with metabolic diseases such as diabetes and hepatic disorders. Erosions, ulcerations, crusting and exudation are seen on footpads and mucocutaneous junctions, but have also been described on the ears, elbows, hocks, ventral thorax and scrotum. The lesions tend to be bilaterally symmetrical. On clinical examination differential diagnoses would include drug eruptions, pemphigus foliaceus and zinc deficiency/generic dog food dermatosis.

* Gross TL *et al.* (1993) Superficial necrolytic dermatitis (Necrolytic migratory erythema) in dogs. *Vet Pathol* **30**: 75-81,

QUEENSLAND – VETERINARY PATHOLOGY SERVICES BRISBANE

INTESTINAL PYTHIOSIS IN A DOG

A 2 year old Kelpie was presented to a Mt Isa practice with weight loss and poor appetite. Barium contrast studies indicated delayed upper intestinal passage and stricture. On exploratory laparotomy there was uniform 1cm wide thickening of a 25cm segment of duodenum and enlargement of mesenteric lymph nodes. The affected segment was resected and submitted for histopathological examination with a tentative diagnosis of intestinal lymphosarcoma.

Histopathology revealed the thickening corresponded to a multifocal necrotising, pyogranulomatous and eosinophilic transmural enteritis centred in the muscularis. Fungal hyphae with morphology typical of *Pythium* sp were present in areas of necrosis. The overlying mucosa was atrophic and focally ulcerated. Canine gastrointestinal pythiosis may involve any part of the digestive tract, but lesions are most likely in the stomach and/or small intestine. This disease should be considered in the differential diagnosis of conditions characterised by weight loss, vomiting and/or diarrhoea, and palpable thickening of the intestinal wall. Disease with these clinical features include eosinophilic gastroenteritis, granulomatous (regional) enteritis, gastrointestinal foreign body, intussusception and intra-abdominal neoplasia (lymphosarcoma and adenocarcinoma). *Pythium* sp is the most important phycomycotic agent. This organism appears to be a primary pathogen, as infection is not associated with other debilitating disease or immune-deficiency syndrome. *Pythium* sp is an aquatic organism of tropical and subtropical areas and animals in contact with swamp water (e.g. hunting or outdoor dogs) are more frequently affected than smaller household pets.

In general, the prognostic outlook in canine gastrointestinal pythiosis is poor because most dogs are presented in an advanced stage of disease and currently used antifungal agents do not seem to be effective against *Pythium* sp in the dog. However, surgery offers a potentially successful method of therapy and this case staged a good recovery.

(Reference: Miller, R.I. (1985) Gastrointestinal phycomycosis in 63 Dogs JAVMA **186** p473-478.

MACAW WASTING DISEASE IN AUSTRALIA

A one year old green-wing macaw was autopsied after suffering persistent vomiting and chronic weight loss in quarantine. Crop stasis had been demonstrated by contrast radiological studies using barium. The main gross findings were dilation of the distal oesophagus, crop, proventriculus and gizzard with the proventriculus and gizzard containing poorly digested feed.

On histopathological examination of these organs the myenteric ganglia and nerve trunks were infiltrated by lymphocytes, plasma cells and macrophages with progression to demyelination in severely affected nerves. These changes were most prominent in the gizzard. A similar inflammatory infiltrate was variably present in the muscularis. These lesions are consistent with macaw wasting disease or neuropathic gastric dilation syndrome. This disease is well known in the US and Europe but does not appear to have been previously reported in Australia. It is most commonly seen in macaws and cockatoos, but has also been found in African greys, Amazons, canaries, eclectus parrots, and cockateils. The cause is unknown, but the nature of the lesions is suggestive of a viral aetiology. It appears to slowly spread from bird to bird but rapidly spreading local outbreaks have been reported. The incubation period may be quite prolonged and birds housed in isolated households have developed clinical signs months to years after purchase. In approximately 50% of cases (especially in macaws) central nervous system signs, consisting of ataxia, leg weakness and behavioural changes are evident. Most birds develop secondary microbial infection and these may contribute to the severity of the signs. Radiology is useful in confirming gastric dilation and contrast studies may show either retarded or shortened transit times through the proventriculus.

A strong presumptive diagnosis can be made on the basis of history, clinical signs and radiographic appearance, particularly if lead poisoning, fungal proventriculitis and foreign bodies can be ruled out. A diagnosis is confirmed by demonstrating the characteristic neural lesions in proventricular biopsies. There is no antemortem test for birds incubating this disease.

Treatment has been unsuccessful but life can be prolonged by fluid therapy, tube feeding liquid diets and eliminating secondary microbial infections. Most birds will eventually die from the neurological effects of this disease.

The clinical signs of the neuropathic gastric dilation syndrome are quite variable, encompassing depression, weight loss, regurgitation, passage of whole seeds in the droppings and nervous signs. With its identification in Australia, this condition thus becomes a significant differential diagnostic consideration in diseases of macaws, cockatoos and other susceptible species.

K. Plummer (1991) Disorders of the Digestive Tract of Cage and Aviary Birds Syd. Post-Grad Proc. pp414-415.

YEERONGPILLY VET LAB.

SALMONELLA DUBLIN DOMINATES ISOLATES FROM CATTLE

Salmonella dublin was isolated at YVL from 7 bovine faecal samples during the first quarter of 1993. Four separate herds were involved (Wacol, Dayboro, Coleyville and Mt Kilcoy). The other 2 isolates obtained from bovine faeces in this period were *S. heidelberg* and *S. tennessee*. This dominance by *S. dublin* has not been seen previously at this laboratory, although this serotype is one of the most common in cattle worldwide.

Isolates were all associated with alimentary tract disease. *S. dublin* is associated with abortions in cattle, but this manifestation of infection has not been diagnosed so far.

BLINDNESS IN GOATS – Greg Storie

Sudden onset of blindness was reported in 2 Angora goats (8 and 18 months of age) on a small holding near Beaudesert. The animals were submitted to the Yeerongpilly laboratory for necropsy. Both had dilated pupils, non-responsive to light, but no gross lesions were detected. Histological examination of optic nerves and eyes revealed marked myelin vacuolation of the optic nerves with scattered digestion chambers containing myelin macrophages, atrophy of the outer nuclear layer of the retina (often reduced to a single layer of cells compared to a normal thickness of 10-12 cells) and degeneration of the photoreceptor layer. Subsequent enquiries revealed the goats had been overdosed several weeks previously with a halogenated salicylamide anthelmintic (SEPONVER).

This case is similar to that reported by Button C *et al* (1987) - Aust vet J **64**: 226. Very similar changes are known to result from ingestion of *Stypandra imbricata* in WA and *S. glauca* in NSW.

JOHNE'S DISEASE - Greg Stone, Ross McKenzie, Laurie Dowling

Chronic diarrhoea was present in a 16 month old anglo-nubian buck in a small flock at Jimboomba. Faecal examination for worms and coccidia was negative. The animal had been imported from NSW in August 1992 and the attending veterinarian submitted a serum sample since she suspected Johne's disease. A positive titre of 1/32 was recorded to the complement fixation test. A faecal sample was immediately submitted for cultural examination and *Mycobacterium paratuberculosis* was demonstrated by DMA polymerase chain reaction on 4-month-old colonies. This test result was confirmed by the Animal Health Laboratory, Western Australia.

Subsequently in April 1993, Johne's disease has been diagnosed in an anglo-nubian goat at Eumundi after the owner suspected Johne's disease. Both flocks have been placed in quarantine and eradication measures have been instituted. These are the first diagnoses of Johne's disease in goats in Queensland.

VITAMIN A DEFICIENCY IN GRAZING CATTLE - Ross McKenzie

Vitamin A deficiency was diagnosed in a group of 1-2 year old Brahman-cross steers in a herd near Charleville in mid-February 1993. Thirty-five were affected in one paddock. Seasonal conditions were poor. Clinical signs observed were blindness, hyperlacrimation, abnormal gait, diarrhoea and episodic convulsions. Three affected animals in 1992 were treated with injections of Vitamins A, D & E and fully recovered. Serum assays for retinol (which makes up some 70-80% of serum Vitamin A) in 15 steers sampled for the current submission revealed a mean concentration of 226 ug/L (range 120-540). Normal serum retinol concentrations in cattle are > 300 ug/L. Blood glutathione peroxidase activities and serum Vitamin E concentrations were normal in the 15 steers sampled.

BRACHYGNATHIA IN GOLDEN PERCH - Peter Ketterer

High post hatching mortality and a high prevalence of brachygnathia occurred in a batch of 3-month-old golden perch. Of 20,000 fish placed, only 1,000-2,000 survived at 3 months and all survivors had a shortened upper jaw. This condition is known as 'pugheadedness' and it may be genetic in origin or caused by an environmental insult during embryonic or larval development. The latter is considered more likely since the parent fish, a single male and a single female, were not reported to be abnormal. Overcrowding and poor oxygenation due to low water flow or detrimental temperature during a critical stage of development are possible causes.

BACTERIAL INFECTION OF THE INTEGUMENT IN LARVAL MAHI MAHI- Peter Ketterer

Formalin fixed samples of larvae were submitted from 4 batches which had high mortality rates. All had microcolonies of large gram negative bacilli within the epithelial layer of the skin or in the case of the gills, beneath the epithelium of the lamellae. In order of decreasing incidence areas affected were: gills, gill cavity, oral cavity, pharynx, external integument and fins. No inflammatory reaction or ulceration was associated with the infection. The infection was considered significant because of its extensive nature. Management factors such as water quality, temperature and nutrition may have been predisposing. Fresh samples were not available to attempt isolation of the organism which may have been a *Flexibacter*/Cytophage bacterial agent.

TOOWOOMBA VET. LAB.

NERVOUS COCCIDIOSIS IN FEEDLOT CATTLE

A four thousand head feedlot at Proston lost 4 animals with nervous signs and severe bloody diarrhoea. The nervous signs consisted of apparent blindness, opisthotonus and seizures. Some 10-13% of animals were reported to have some blood in their faeces. *Eimeria zuernii* was detected in faecal samples from three of the four dead animals. Faeces from the animals looked more like a clotted blood sample with mucous and shreds of mucosa present. Brains from two of the animals had no significant abnormalities and a section of colon from one had a haemorrhagic colitis but no endogenous coccidial stages were seen because most of the epithelium had desquamated.

GENERALISED SARCOCYSTOSIS

A 6 month-old crossbred Merino lamb suddenly developed respiratory distress and colic. The lamb had a temperature of 40.8 and died within 36 hours. Histopathology revealed large numbers of sarcocysts in the skeletal muscle and a non-purulent myositis. Similar lesions were present in the myocardium. Numerous foci of gliosis were present in sections of the brain and small numbers of sarcocysts were scattered throughout. In sections of lung there was a severe non-purulent interstitial pneumonia but no sarcocysts were seen.