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VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
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Veterinary Clinical Centre
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ASVP EXECUTIVE 1995-1997

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Treasurer	Mark Williamson	Australian Animal Health Laboratory Ryrie Street GEELONG VIC 3220	03 5227 5000

Committee Members

Ian Jerrett	Gippsland Veterinary Pathology Service PO Box 1464 BAIRNSDALE VIC 3875	03 5150 3407
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APPOINTMENTS

Chairperson (Registry of Domestic Animal Pathology)	Tony Ross
Newsletter Editor	Karl Harrigan
Coordinator (Training Committee)	Judith Wilkie

CONVENOR - SLIDE OF THE MONTH

Rod Reece	National Registry of Domestic Animal Pathology, EMAI, Private Mail Bag 8 CAMDEN NSW 2570	046 29 3327

STATE REPRESENTATIVES

Queensland	Bruce Hill, Rockhampton Vet Lab,QDPI Box 6014 ROCKHAMPTON QLD 4702	079 36 0211
Victoria	Malcolm Lancaster, VIAS 475 Mickleham Road ATTWOOD VIC 3049	03 921 74200
South Australia	Ruth Reuter, VPS PO Box 96, PLYMPTON 96	088 362 3544
New South Wales	Paul Gill RVL WOLLONGBAR NSW 2480	066 26 1261
Western Australia	David Forshaw, Regional Office, WA Dept Ag ALBANY WA 6330	09 842 0500
Northern Territory	Anton Janmaat PO Box 990 DARWIN NT 0801	088 999 2240
Tasmania	Roy Mason, TAWQDS PO Box 46, KINGS MEADOWS TAS 7249	03 6336 5216

Notes from the President

There are several new matters that arose as a consequence of the last AGM, and these are documented in the minutes published below. "The Executive is interested in member's opinions on these matters, and if you are unable to come to the meeting in Brisbane, then please contact the members of the Executive directly. The meeting in Brisbane holds promise as an exciting and topical one, with the first day dedicated to both the theoretical and practical aspects of emerging diagnostic technologies.

The meeting also marks the completion of the term for the current Executive and officers of the ASVP. In due course, the Executive will be contacting all the Officers and State Representatives to ascertain their willingness to continue in these roles. In addition, I call for nominations from the membership for replacement of the Executive (President, Secretary, Treasurer and Committee Members) and also for nominations for all of the other positions associated with the ASVP.

On behalf of the Executive and the local organising committee, we hope to see you in Brisbane in May.

Ron Slocombe
President, ASVP

ASVP Secretariat

The Secretariat in Adelaide has been ably conducted by Mrs. Pat Bosence for many years. Pat retired last year and the position was taken over by Ms Julietta Cerin. Ms Cerin resigned recently and the Secretariat is now being provided by Ms Amanda White.

MINUTES
AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY INC.
ANNUAL GENERAL MEETING

UNIVERSITY OF MELBOURNE, VETERINARY CLINICAL CENTRE
PRINCES HIGHWAY, WERRIBEE
26 NOVEMBER 1996

1. The meeting opened at 8:00pm, 22 members in attendance.

2. Apologies:

F Doughty, I Jerrett, M Barton, R Campbell, D Obendorf, R Miller, G Mitchell, J Mackie, M Nowak, R Kelly, R Sutton, P Lording, D Forshaw, J Rothwell, J Glastonbury, K Walker, A Ross, R Whittington, W Hartley, S Hum, P Gill, P Hooper

3. Minutes of 1995 AGM:

Published in Veterinary Pathology Report Number 42, September 1995 and circulated at the meeting. Acceptance moved J Seaman, seconded B Hill.
Carried, none opposed.

4. Business arising from the Minutes:

(a) Motion by Drs Ross/Giesecke that "Matters raised in the letter of D Obendorf be brought before Tasmanian members of the ASVP and the incoming Executive act on their information to pursue the matter with the Executive Officer and Minister of Tasmanian DPIF with the support of the Tasmanian Graziers' and Farmers' Association". Discussion deferred to Item 6.

(b) Motion by Drs Glastonbury/Walker that "The Executive of the ASVP bring to the attention of animal industries representatives of NAHC the current deficiencies in veterinary surveillance services in Australia". Discussion deferred to Item 6.

(c) Motion by Drs Mackie/Links that "The ASVP Executive formulate a brief (two page maximum) discussion paper alerting producer organisations in Australia of deficiencies arising in Australian veterinary laboratories and promoting the role of veterinary pathologists in correcting these deficiencies". Executive yet to complete this undertaking.

5. Correspondence:

Routine correspondence was handled by the Executive. Some appropriate and more significant items had been published in the Veterinary Pathology Report and/or tabled at the meeting.

Some of the more notable items brought to the attention of members were:

(a) ASVP submission to the Standing Committee on State Development, New South Wales, regarding the proposed closure of NSW regional veterinary laboratories. Copy of report received 4 September 1996.

(b) Letters to the Tasmanian Department of Primary Industries and Fisheries in reference to Item 4(a) above. Reply received 12 July 1996.

(c) Letters to the Australian Animal Health Council etc in reference to Item 4(b) above. Reply received 24 June 1996.

(d) Letters to five Tasmanian members of ASVP in reference to Item 4(a) above. One only reply, 24 June 1996.

(e) Letters from Dr D Obendorf. Letter dated 12 November 1996 tabled and circulated at meeting in accordance with Dr Obendorf's request.

(f) Letters from National Association of Testing Authorities, Australia (NATA), published in the Veterinary Pathology Report Number 44, September 1996 and circulated at the meeting.

(g) Letter from Quality Assurance Services (QAS) 24 November 1996 circulated at the meeting.

Acceptance of inward correspondence moved W Robinson, seconded P Phillips.
Carried, none opposed.

Acceptance of outward correspondence moved W Robinson, seconded R Reuter.
Carried, none opposed.

6. Business arising from Correspondence:

(a) ASVP submission to the NSW Standing Committee on State Development. The meeting noted the submission and the subsequent appearance of Professor Slocombe and other members before the Committee.

It also noted that despite the positive nature of the Committee's report both of the laboratories have closed. Nonetheless the NSW laboratories appreciated the ASVP contribution to the inquiry.

(b) In reference to the various correspondences listed under Items 5(b), 5(d) and 5(e) above. Professor Slocombe reported having two separate conversations with Dr Obendorf in Christchurch, June 1996, and thought that he had discussed the matters thoroughly. A number of members suggested that it was probably unlikely that the ASVP would have any further impact on the situation in Tasmania especially considering the outcomes for government veterinary laboratory services in other states.

It was generally felt that the ASVP had done the best that it could have been expected to do in the circumstances and that it would not be beneficial to continue to pursue the issues with the Tasmanian ministry.

Moved W Robinson, seconded S Friend that "This meeting supports the action taken by the ASVP Executive in relation to the matters raised by Dr D Obendorf in reference to the DPIF Tasmania". Carried, none opposed.

It was resolved that the Executive should write to Dr Obendorf indicating the extent of this discussion particularly that following the tabling of his letter of 12 November 1996 and of the responses to the ASVP's letters to the Minister for the DPIF, Tasmania and Tasmanian members of the ASVP together with copies of appropriate non-confidential correspondence.

(c) In reference to correspondence with the Australian Animal Health Council listed under Item 5(c) above it was noted that the document drawing the attention of animal industry members of the Australian Animal Health Council to the current deficiencies in veterinary surveillance services in Australia had not yet been finalised. Some of the members present volunteered to provide contributions to assist the expeditious completion of the document.

(d) In reference to the correspondence from NATA and QAS listed under Items 5(f) and 5(g) above it was noted that both organisations requested ASVP endorsement of their laboratory proficiency testing programs; one requested exclusive endorsement and there appeared to be other organisations being set up to provide similar quality assurance programs.

The advantages and disadvantages of these proposals were discussed at length and while the meeting agreed that the ASVP should support in general the implementation of QA programs, it should not provide official endorsement of any particular organisation(s). It was generally felt that the participation of certain ASVP members in the development of some of these programs did not obligate the ASVP to provide any official endorsement.

Moved B Richards, seconded J Wilkie that "The ASVP supports the concept of the introduction of quality assurance programs to Australian veterinary laboratories but declines to provide endorsement to any particular organisation".
Carried, none opposed.

7. Annual Reports:

Reports for the year to June 1996 from the President, Secretary/VPR Editor, Treasurer and Training Committee Co-ordinator had been published in the Veterinary Pathology Report Number 44, September 1996 and copies were circulated at the meeting.

Moved W Robinson, seconded B Richards that the reports be accepted.
Carried, none opposed.

8. Committee Reports:

(a) Training Committee Report.
The meeting noted the work already done by the Training Committee and in particular by Dr Robin Giesecke.

Moved P Phillips, seconded S Friend that "The ASVP acknowledges the considerable contribution made to the Society by Dr Giesecke through her work with the Training Committee. It also wishes to express sincere sympathy on the recent death of her husband".
Carried unanimously.

Discussion ensued in support of continuing the work of the Training Committee and in the expeditious development of training modules as outlined in the Training Committee Report. Adoption of some programs being currently used/developed by Veterinary Pathology, University of Queensland was thought to be a mechanism whereby at least some training modules might be made available in the near future. In the first instance production of the module(s) could be funded by Veterinary Pathology, University of Queensland. The problem of ownership of the intellectual property rights pertaining to the module(s) was considered; some members indicated the desirability of the property rights residing with the ASVP. The development of at least one training module might serve as a pilot for the entire program.

Moved W Robinson, seconded R Reuter that "The Training Committee expedite the development of at least one training module".
Carried, none opposed.

Consensus was that ownership of the intellectual property rights to such modules would be explored by the Training Committee in association with the ASVP Executive. It was agreed that this rider would qualify the motion proposed by Robinson/Reuter.

(b) National Registry of Domestic Animal Pathology, Registrar's Report. The report has been published in the Veterinary Pathology Report Number 44, September 1996 and copies were circulated at the meeting.

Moved R Reuter, seconded A Janmaat that "The Registrar's report be accepted".
Carried, none opposed.

The Registrar reported receipt of $2,000.00 and that a camera and transparency copying equipment were being purchased.

9. Office Bearers and Committee Appointments.

Moved P Phillips, seconded R Reece that "The following 1995/96 appointments continue for 1996/97".

President	R Slocombe
Secretary / Editor Veterinary Pathology Report	K Harrigan
Treasurer	M Williamson
Committee	A Havadjia
	I Jerrett
Registry Chairperson	A Ross
"Slide-of-the-Month" Convenor	R Reece

And "J Wilkie be appointed as Training Committee Co-ordinator".
Carried, none opposed.

State Representatives for 1996/97 are:

Queensland	B Hill
New South Wales	P Gill
Victoria	M Lancaster
South Australia	R Reuter
Northern Territory	A Janmaat
Western Australia	D Forshaw

It was pointed out that Dr B Munday will not be able to continue as the representative for Tasmania. Dr Munday was thanked for his contribution as State Representative.

Moved W Robinson, seconded J Gibson that "Dr R Mason be appointed as State Representative for Tasmania in 1996/97".
Carried, none opposed.

10. General Business:

(a) ASVP Secretariat.
The Secretariat in Adelaide has been ably conducted by Mrs. Pat Bosence for many years. Pat retired from the position recently whereupon the ASVP Executive sent her a bouquet of flowers and a note of thanks for her contributions to the running and well-being of the Society.

Moved K Harrigan, seconded P Phillips that "The ASVP congratulates and thanks Mrs. Pat Bosence for her contributions to the Society and wishes her well in her retirement".
Carried unanimously.

(b) "Slide-of-the-Month" program.
Laboratory changes and staffing difficulties have resulted in some irregularities in this program over recent months. Some of the smaller laboratories may have difficulty preparing and mailing a large number of slides. It was suggested that at least two laboratories should be scheduled for supplying cases each month. If one laboratory was unable to provide cases the second one could do so. The program convenor will arrange this. The Registry may also be able to assist with mailing slides if an individual laboratory is unable to cope. Moreover the larger institution laboratories should be able to assist with the preparation and/or mailing of slides.

Consensus was that the "Slide-of-the-Month" program was a valuable asset and every effort should be made to preserve it.

(c) New Members:
Drs R Woodgate, J Wilkie, P Holz and F Uzal who have become members of the ASVP since the last Annual General Meeting were officially welcomed by the President.

11. Any Other Business:

Affiliation of the ASVP with the Australian Veterinary Association was raised.

Moved R Slocombe, seconded J Wilkie that "Consideration of ASVP/AVA affiliation be made an Agenda item for the next Annual General Meeting".
Carried, none opposed.

Moved W Robinson, seconded S Friend that "The ASVP Executive correspond with the Australian Veterinary Association regarding special interest group affiliation with the AVA especially in relation to ASVP veterinary members who are not also members of the AVA".
Carried, none opposed.

Consensus was that the ASVP Executive should prepare a position paper on ASVP/AVA affiliation for the next Annual General Meeting which would be sent out with the Agenda for that meeting.

12. Next Meeting:

It was agreed that the 1997 Annual General Meeting will be held in Brisbane in association with the 1997 AVA conference. The meeting will be held on the Saturday and Sunday immediately preceding the AVA conference with the first day devoted to one or more special themes, the second day available for free communications.

Topics suggested for inclusion as special themes were veterinary clinical pathology and cytology, immunocytochemistry, PCR technology, new/emerging infections such as equine morbillivirus, lyssavirus and bovine spongiform encephalopathy.

Dr B Hill as Queensland State Representative will arrange a local organising committee which will negotiate a draft program and suitable venue for the meeting.

There being no other business, the meeting was closed at 10:15pm

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY INC.

ANNUAL GENERAL MEETING 1997

1. The ASVP Annual General Meeting 1997 will be held in Brisbane on Saturday and Sunday 3rd and 4th May. This is the weekend immediately preceding the Australian Veterinary Association conference in Brisbane.

2. The venue for the meeting is the Bellevue Hotel, 103 George Street, Brisbane, phone (07) 3221 6044, facsimile (07) 3221 7474, toll free 1800 065 064. Registration fee for the meeting will be $35 per day. Please make cheques payable to ASVP and forward with registration to the Honorary Secretary as soon as possible.

3. The theme of the Saturday program will be "Diagnostic Techniques problems and solutions". Professor Robinson and the local organising committee have assembled a range of speakers to provide an overview of diagnostic techniques varying from routine to the esoteric. It will include updates on the use of PCR, immunohistochemistry, toxicology and immunology to assist diagnosis.

4. The Sunday program will be a potpourri of short research and case report presentations as in previous years.

5. Members (and visitors) intending to present reports in the Sunday program should submit the title of their presentation to the Honorary Secretary as soon as possible. An abstract of the presentation will be required before 17th April 1997 for publication in the conference proceedings.

6. A conference dinner has been organised for the Saturday evening at Weekes Restaurant, Mercure Hotel, 85 North Quay, Brisbane (approximately five minutes from the conference venue).

7. Accommodation at the Bellevue Hotel is available at $112 (single and shared). This is the discounted "corporate rate" available to ASVP members. There is limited car parking available at the Bellevue Hotel but the Festival Car Park is around the corner in Charlotte Street, approximate cost per day is $8 during the week and $5 on weekends.

8. The 1996/97 Annual General Meeting of the ASVP will be held in conjunction with the conference.

South Australia - Ruth Reuter

An Unusual Reproductive Problem in the Guinea Pig - Ruth Reuter, VPS, Adelaide

Most people who have kept guinea pigs (usually at the urging of a smaller member of the family!) would feel that the major reproductive problem of the guinea pig is reproductive excess rather than reproductive failure. Females can reach puberty at five to six weeks of age, although the average is around 70 days. The oestrus cycle is 15-17 days, and with a gestation period of approximately 68 days and litter size of up to six piglets, the potential for a population explosion is high.

Apart from provision of dietary Vitamin C and general good management and hygiene, there are few problems which affect the breeding guinea pig under normal circumstances. Sporadic bacterial infections are relatively uncommon and viral agents not ordinarily of significance.

A two year old female guinea pig was euthanased soon after giving birth to one dead piglet. The submitting veterinarian had found four more piglets in the uterus, two normal and two grossly abnormal. This was representative of a problem of abortions, stillbirths and abnormalities being encountered by a group of show guinea pig breeders in the region. The two abnormal piglets were submitted to the laboratory for examination.

On gross examination one of the foetuses was partially resorbed, the other appeared oedematous. They were fully haired with development of incisors consistent with term foetuses. The larger piglet exhibited a 1.0cm long defect in the skin over the back. The dorsal vertebral arches were missing, exposing the underlying spinal cord which was small and flattened. The brain was protruding through the cranium as a fluid-containing sac with a small amount of soft tissue. There was marked mineralisation of the placental cotyledons. A diagnosis of multiple foetal anomalies, including spina bifida, exencephaly, myelomalacia and anasarca, was made.

Although sporadic malformations occur, the involvement of several breeders and numerous litters suggested this was not the case. Exposure of pregnant guinea pigs to a variety of chemicals and drugs, particularly between days 12-16 of gestation, can produce abortions, resorptions and foetal malformations. Placental mineralisation occurs sporadically in a range of species with no obvious ill effect to the fetus. However, in the extent observed here, the level of oxygenation would be compromised.

Examination of the ration being fed to the animals revealed that several of the owners were also horse enthusiasts. In order to keep their guinea pigs in the best show condition, they were feeding guinea pig pellets supplemented with fresh vegetables, oat chaff and several commercially available equine supplements. These preparations contained high levels of Vitamin A and Vitamin D as well as other trace minerals and protein.

Vitamin D has been associated with excessive mineralisation of tissue, such as was seen in the placenta. The level of Vitamin D required by the guinea pig is very low, approximately 1.6 lU/kg of feed, which is well under the level of 2,000 IU/kg present in one of the supplements.

Vitamin A in levels ranging from 20,000 to 50,000 IU has been associated with abortion and foetal malformations of the type seen here. One of the supplements being fed contained 20,000 IU Vitamin A per kilo. In addition the guinea pigs were receiving complete guinea pig pellets, carrots, silverbeet, tomato, celery, lettuce, cabbage, brussel sprout leaves and beetroot.

Moral of the story - Over nutrition can cause as many problems in the animal world as it does in Western society!!!

Oral Eosinophilic Granuloma in the Dog - Ruth Reuter, VPS, Adelaide

Eosinophilic granuloma is a relatively common condition in the skin or oral cavity of cats. Lesions can be seen on the thighs, forelimbs, neck, ear and thorax. The lower lip, chin and footpads can also be affected. The cause of the condition is not known. However, hypersensitivity and immune mediated disorders have been suggested to predispose to this reaction. A differential diagnosis can include insect-bite hypersensitivity and food allergy.

In the past two months, VPS has seen several cases in dogs which closely resemble the feline condition. The most recent of these was a seven year old Maltese terrier with a circular, firm mass on the lip. The animal had been treated with antibiotics with no observable effect. On biopsy there was an extensive cellular infiltration of the dermis with eosinophils predominating, accompanied by macrophages and some giant cells. Foci of collagen degeneration were present. Specific aetiologic agents such as foreign bodies were not found in the sections.

Oral eosinophilic granuloma has been described in the dog, however the condition is relatively rare. A familial tendency has been documented in the Siberian husky, and sporadic cases have been reported in other breeds. Most of these cases have involved plaque-like lesions or ulcers, particularly on the tongue or soft palate. In the cases seen at VPS, the spectrum of lesions has included the tongue and palate, as well as the lip and buccal mucosa. None of these animals have been huskies.

A syndrome of ulcerative eosinophilic stomatitis affecting the palate in Cavalier-King-Charles spaniels has been described in North America. Steroid treatment had a beneficial effect in two cases. A third resolved spontaneously.

Reference:

Joffe, D. J. and Allen, A. L. (1995), J. Amer. An. Hosp. Assoc. **31**:34-37.

Death of a Night Heron - Ruth Reuter, VPS, Adelaide

On September 19, 1996 an adult male night (rufous) heron was submitted to VPS Adelaide for necropsy. The bird was in full plumage and had been found dead in a park enclosure in Adelaide that morning. The bird was in good body condition. On external examination irregular patches of yellow curled feathers were seen over the sternum and in small numbers on the back. There was haemorrhage in the left periocular area.

When the skin was peeled back, extensive oedema with branching haemorrhages were obvious in the subcutaneous area particularly on the right side, associated with the right jugular vein, and extending through the right pectoral muscles. The left lung was severely congested with blood dots throughout the airways. The liver was pale and friable with parenchymal haemorrhages. There was a mid shaft fracture of the left femur. The stomach was distended with portions of undigested shellfish. J

The pathologic changes were typical of those seen in cases of electrocution, where birds or animals have contacted power lines or been struck by lightning. The path of the electrical discharge would have been from the right sternal area diagonally through the muscles, and left lung. The femoral fracture would have been associated with extreme contraction of surrounding muscles.

The night heron lives near swamps, ornamental ponds or rivers. It is nocturnal, feeding at night and roosting in trees close to water by day. In the Adelaide area they have been observed roosting and nesting on powerlines. In the week before the bird was found dead there had been severe electrical storms in this area. The pathologic changes suggest that this bird had been feeding shortly before it was struck by lightning during the storm or blown into a powerline, resulting in electrocution.

Feline Heartworm - Ruth Reuter, VPS, Adelaide

An eight and a half year old domestic male cat was recently presented to VPS for necropsy. He had shown no illness until the previous evening, when he vomited after eating. The next morning he was found dead. The cat was in good body condition with large deposits of fat in the storage depots. The heart was grossly normal, however, there were two adult heartworms present, one in the anterior vena cava and the other in the pulmonary artery. The lungs were rubbery with excess fluid in the trachea and bronchial lumens. The airways were thick-walled and prominent on cross-section.

Antemortem diagnosis of feline heartworm disease is difficult due to the lack of a reliable clinical test. Clinical signs are non-specific, with vomiting, gagging, lethargy and inappetence occasionally seen. Sudden death is common. Typical symptoms of respiratory distress, coughing and cardiac symptoms seen in the dog are not commonly seen in the cat.

Clinical pathology may include mild anaemia, occasional hyperglobulinaemia or eosinophilia, and rarely basophilia. Usually less than ten adult heartworms are present and microfilariae are reportedly seen in less than 1% of cases. Antigen-detecting tests used in the dog often fail to detect cats carrying only one or two worms.

References:

Atkins, C. E. *et al*. (1996), Guidelines for the Diagnosis, Treatment and Prevention of Heartworm (Dirofilaria immitis) Infection in Cats, Am. Heartworm Soc Bull. **23**:1-7.
Holmes, R. A. (1994), Feline Heartworm Disease: Diagnostic and Treatment Methods. Vet. Tech. Cont. Ed. Article #1, **15**:213-219.

A New Animal Model of Traumatic Head Injury - John Finnie, Vetlab

The Adelaide Head Injury Study Group, to which Vetlab has contributed, has recently developed a new animal model of traumatic closed head injury. Impaction of the temporal region of an unconstrained sheep head with the heavy mushroom-head of a humane stunner produced sudden, rotational acceleration, which is considered to be responsible for diffuse axonal injury (DAI) and subdural haematoma (the commonest causes of death and irreversible brain damage in human head injury).

This new head impact model produced axonal and vascular damage resembling, in many respects, that found in man after a traumatic, blunt, non-missile impact to the head. Temporal lobe contusions were found beneath the impact site and some sheep also had contralateral contusions. Subarachnoid haemorrhage was regularly present with widespread parenchymal haemorrhage. Amyloid precursor protein (APP) immunostaining revealed early (one hour post-impact) and widely distributed axonal spheroids, similar to DAI in man. APP is normally produced in neurons, is conveyed by fast axoplasmic transport and functions are believed to include cell adhesion, growth and response to injury. Cytoskeletal disruption of the axon results in accumulation of APP and positive immunostaining when it reaches detectable levels. APP is regarded as the most sensitive marker of axonal damage and does not stain normal axons.

With respect to experimental neurotrauma studies, the relatively large brain of sheep (approximately 120g) is important as shearing forces and inertial loading are related to brain mass and animals with small brains can tolerate much greater rotational and acceleration/deceleration forces than subhuman primates and man. There is also a slender margin between a strong blow to the head and fatal injury in animals with small brains. Furthermore, sheep have a gyrencephalic brain (resembling more the human brain) with a much more complex pattern of gyri and sulci than the almost lissencephalic appearance of rodent brains.

Reference:

Lewis, S. B., Finnie, J. W., Blumbergs, P. C., Scott, G., Manavis, J,, Brown, C., Reilly, P. L., Jones, N. R. and McLean, A. J. (1996). A head impact model of early axonal injury in the sheep. J Neurotrauma 13:505-514.

Victoria - Malcolm Lancaster

Lyssavirus and morbillivirus infections in Victorian bats - John Humphrey, Malcolm Lancaster, V1AS Attwood

Thirteen grey headed flying foxes (*Pteropus poliocephalus*) and five microbats have been examined at VIAS Attwood over the last two months. Several of the bats were from frozen museum collections, collected as far back as 1992. Electrocution on high voltage power lines was the commonest cause of death.

Perivascular cuffing was seen in the brain of two of the flying foxes, with neutrophils, macrophages and lymphoid cells present. One of these bats, collected in December of 1996, had small Negri bodies in a few neurons of the cerebrum. Lyssaviral antigen was detected by immunohistochemistry and lyssavirus was cultured from thawed frozen brain. The second bat, one of four found dead under a particular eucalypt tree over a three week period in 1995, had a few hyperchromatic neurons with a pyknotic nucleus. No Negri bodies were observed, and immunohistochemistry was negative for lyssaviral antigen.

One captive flying fox was submitted after morbillivirus antibodies were detected serologically. No lesions were seen grossly or histologically.

Sialodacyroadenitis virus and cilia-associated respiratory (CAR) bacillus infections in rats - Malcolm Lancaster, VIAS Attwood

Two populations of rats with a common source had a history of chronic respiratory disease. Severe bronchitis was observed grossly and histologically. A mycoplasma (assumed to be Mycoplasma pulmonis) was cultured from the rats of one colony.

Warthin-Starry silver staining showed numerous organisms morphologically consistent with CAR bacillus in the cilia of the bronchial epithelium of rats from both colonies. Necrosis and attenuation of acinar cells in the lacrimal and salivary glands in rats from one colony was characteristic of sialodacryoadenitis virus (corona virus) infection.

Serological investigation revealed titres to *Mycoplasma pulmonis*, CAR bacillus and coronavirus. All three agents are respiratory pathogens, and mixed infections are likely to produce a more severe clinical syndrome.

Inclusion-body hepatitis in young emus - John Humphrey, VIAS Attwood

Ten out of 100 four month old emus died suddenly. Two birds were necropsied and a swollen speckled liver was seen in each. Liver, kidney and heart were submitted for histopathological examination. Many individual hepatocytes were rounded up and necrotic. Other hepatocytes were swollen and vacuolated, and some of these hepatocytes contained an intranuclear basophilic inclusion, occupying a large proportion of the nuclear volume.

Liver tissue will be examined electron microscopically in an attempt to confirm a viral aetiology for this syndrome.

Yersiniosis in Chinook (Pacific) Salmon *Oncorhynchus tshawytscha* -John Humphrey, VIAS Attwood

High mortalities were present in a population of fingerling Chinook salmon reared at a freshwater fish hatchery in October 1996. Clinically, fish showed lethargy, lack of response to external stimuli and death. Occasional fish had intra-ocular haemorrhage.

Histopathological examination showed large numbers of bacilliform bacteria in the interstitium of the kidney, in the myocardium, in vascular spaces of the liver and occasionally in the vascular spaces of the secondary gill lamellae. Degenerative changes and the occasional necrotic cell were present in the renal haematopoietic tissue. Moderate hypertrophy of the secondary gill lamellar epithelium was present with separation of the epithelium from the underlying vascular sinus.

The Gram-negative organism *Yersinia ruckeri* was isolated from visceral organs of the fish in pure culture. This organism is a well described cause of septicaemic disease of salmonid fish frequently referred to elsewhere as "enteric redmouth disease". Acute infections are characterised by a fulminant bacteraemia and death in the absence of significant histopathological changes.

Gossypol toxicity in calves - Judith Nimmo Wilkie, Peter Phillips, Victorian Veterinary Pathology Services

Two outbreaks of gossypol poisoning in calves were diagnosed in calves in October and November of 1996. One was in the north-east of Victoria and the other in Gippsland. In both instances calves were dying suddenly without premonitory clinical signs. Post mortem findings were minimal although some animals had slightly swollen livers, ascites and pulmonary congestion and oedema.

Histologically the principal lesion was a severe, acute, periacinar hepatic necrosis with replacement haemorrhage. Pulmonary oedema was also a common finding with secondary bacterial pneumonia in some cases. Heart lesions were not seen.

In the Gippsland outbreak, deaths did not commence until about four days after the gossypol feeding had ceased, and continued for several weeks thereafter. Peracute deaths continued for two weeks after feeding of gossypol had ceased but by this time a few animals were showing less acute signs, namely depression and dyspnoea.

Gossypol is believed to be a primary cardiotoxin, but the evidence in these cases was of a direct hepatotoxic effect, with cardiac and related pulmonary lesions occurring later in the course of the disease if the animals survived long enough. These findings are consistent with those in pre-ruminant calves in New South Wales reported by Zelski *et al*.

Reference:

Zelski, R. Z., Rothwell, J. T., Moore, R. E. and Kennedy, D. J. Gossypol toxicity in pre-ruminant calves. AVJ **72**:394-398, 1995.

New South Wales - Paul Gill

"Vale", RVL's Wagga and Aimidale

Haemolytic Anaemia of Murray Grey Calves - Paul Gill, Regional Veterinary Laboratory, Wollongbar

A two month old female Murray Grey calf was thin, jaundiced and anaemic (PCV 0.09 L/L). Examination of blood films revealed marked anisocytosis, 2% reticulocytes and 4% Heinz bodies.

Necropsy findings included a globose, flaccid heart, dark greenish-brown renal cortices, dark yellow lungs, and a dark brown liver. The urine contained bilirubin and blood. Serum bilirubin concentrations were elevated (T. bilirubin 186 µmol/L) as were serum liver enzyme activities (GGT 153 U/L, ALT 517 U/L). Histological findings included a densely cellular bone marrow with many metarubricytes, diffuse periacinar fatty change with hepatocyte loss, with canalicular cholestasis in mid-zonal and periportal areas and bilaterally symmetrical myelinic vacuolation of the white matter of the substantia nigra. These findings are similar to those reported for congenital haemolytic anaemia and jaundice of Murray Grey calves, believed to be due to an inherited glucose-6-phosphate dehydrogenase deficiency.

Reference:
Nicholls, T. J., Pritchard, D. H., Jerrett, I. V. and McKee, J. J. (1992). Aust. Vet. J., **69**:39-40.

University of Sydney - Malcolm France

A review of neurological conditions seen in laboratory rats over the last three years found that half (six out of twelve) involved suppurative meningitis. Five out of these six animals also had suppurative otitis media. Although culture was not attempted, bacteria were visible in the meningeal exudate on H and E sections in three cases and B and B staining showed that these were Gram negative rods. No organisms were seen in the other three cases.

The disease seen in these rats does not appear to be contagious since each case occurred as an isolated incident in separate animal facilities. The association with otitis media, however, appears to be important. We routinely examine decalcified sections of skull in rodent necropsies because of the high prevalence of otitis media in respiratory disease. The above cases demonstrate that this practice can also be of value with investigating animals presenting with signs referable to other body systems.

Elizabeth Macarthur Institute - John Glastonbury

Ovine Lymphosarcoma

A five year old Merino ram was found weak in a paddock on a farm using a "holistic grazing strategy". Grossly the mediastinal lymph nodes were severely enlarged, and microscopically, the parenchyma of the node was obliterated by sheets of well differentiated, small lymphocytes. Other histopathological findings of interest were moderate, subacute, generalised, periacincar hepatic necrosis and mild, acute, multifocal, suppurative, embolic nephritis and pneumonia. The lymphoid neoplasm may have caused a degree of immunological compromise.

Ovine white liver disease

This vitamin B12/cobalt responsive condition was proposed as a possible cause of weakness, diarrhoea, illthrift and occasional deaths in Merino hoggets at Cooma. Suggestive histopathological observations in the liver were moderate fatty change, generalised biliary hyperplasia, mild individual hepatocyte necrosis and accumulation of ceroid-like pigment in Kupffer cells. Mild status spongiosis was noted in the crus cerebri.

Uterine adenocarcinoma

It was interesting to receive an extensive range of specimens from a field case of this condition as opposed to the regulation piece of lung expected under the Granuloma Submission Program. A five year old, illthrifty Hereford cow, which had been coughing for one month was destroyed.

The body and one horn of the uterus were thickened and fibrotic. The ovary on the opposite side was considerably enlarged and multiple tumours were evident in the mesentery, liver and lungs. One kidney contained multiple infarcts.

A section of the uterine horn showed a scirrhous adenocarcinoma arising in the endometrium, leading to segmental erosion and tracking through the myometrium to cause full thickness obliteration, segmentally. The tumours detected grossly were found to be metastases with similar histological features.

Colonic atresia

A male Friesian calf was observed to be born and suck normally, despite having very mild brachygnathia inferior. Over the succeeding three days, the calf became increasingly depressed and was recumbent prior to euthanasia.

Post mortem examination found that the descending colon came to a "blind ending" approximately 30cm proximal to the anus. The abomasum and the entire intestinal tract were distended with gas, becoming more severely so in the descending colon. Microscopic observations were attenuation of the large intestinal tunica mucosa and moderate, acute, erosive, necrotic ileitis.

Erysipelas in Emus

Twenty of 270 12 month old emus died after being depressed and pyrexic for 24 hours. Prominent septic emboli were evident in glomerular capillaries and to a lesser extent in hepatic sinusoids. Profuse growths of *Erysipelothrix rhusiopathiae* were obtained from the liver and kidneys of two birds.

Additional histological observations of interest in the portal triads of one bird were septic emboli in the vasa vasorum of portal vessels, segmental non-suppurative vasculitis and obliterative vasculitis. These vascular changes have been described as one of the manifestations of chronic erysipelas. If this is the case, this bird must have become re-infected to succumb from acute septicaemia.

Yersiniosis in Water Buffalo

A five month old water buffalo, being maintained in a zoo on the south coast developed anorexia and diarrhoea. Microscopic examination of faeces detected neither nematode eggs nor coccidia.

Severe, acute, segmental, erosive, necrotic enteritis, associated with microabscesses containing prominent bacterial colonies in the lamina propria adjacent to the necks of crypts of Lieberkuhn, was found in the jejunum and ileum. A heavy growth of *Yersinia pseudotuberculosis* was obtained from faeces, the only material submitted for microbiology.

Update on Taronga Zoo Pathology Collection - Bill Hartley

This collection has been in existence for just over 11 years and Bill Hartley has been directly or peripherally involved with its development from its inception, until he was terminated at the end of 1995.

As at November 1996 this pathology collection contains over 7,700 cases with histologic sections, of which about 75% are derived from the terrestrial and aquatic fauna of Australia and New Zealand. The collection includes pathologic materials from 850 macropods, 120 monotremes, 365 koalas and wombats, 485 possums and gliders, 335 dasyurids, 140 bandicoots and bilbies, 95 bats, 280 snakes, 200 lizards, 140 marine mammals and 2,780 birds. Case information is still contained in a simple card filing system and is readily retrievable.

In spite of staffing peculiarities and financial stringencies placed on the unit, over the last several years the collection has managed to exist and expand. It now probably contains pathologic materials from most of the disease entities of captive and free ranging native fauna species occurring in Australasia. Very many new diseases have been identified, and some of these have been reported in journals. Materials contained in the collection are available on loan for lecturing purposes, etc., and a consultancy service still exists.

Due to the failure to fill the position of veterinary pathologist in 1996 and the departure of Paul Canfield overseas, Bill Hartley offered to come out of retirement in September 1996 and rejoin Taronga Zoo part-time with the intention of trying to ensure the survival of the Pathology Collection. The position of full-time veterinary pathologist is to be re-advertised in about December 1996.

Tasmania - Roy Mason

Enteric Microsporidian-like Infection in an Ostrich Chick - Roy Mason, Animal & Plant Health Assessment Branch

Two young live ostrich chicks were received as part of an investigation into chick losses occurring at about 14 days of age. Chick weights on receipt were 604 and 662 grams respectively. This report relates to the heavier chick.

A small residual yolk sac of about 3cm diameter remained. The liver was fatty, the gizzard and proventriculus contained a mix of plant and proprietary feed material. The small intestine was empty and the large intestine contained a watery to mucoid fluid, plus at the distal and small, pale and soft faecal pellets.

The lamina propria of the small intestine was moderately infiltrated by lymphohistiocytic cells. The villous enterocytes contained small apical intracytoplasmic vacuoles, especially in enterocytes at the villous tips. The vacuoles contained oval to roundish bodies. Similar epithelial vacuolation with similar internal bodies were present in areas of colonic epithelium. In addition there was focal early colonic epithelial erosion associated with a fibrinous and inflammatory cell effusion plus a mix of bacterial forms present in the effusing material.

The round to oval intracellular bodies possessed a strongly PAS positive staining spot consistent with a polar cap. The distribution and cytological features suggest the bodies are microsporidian parasites.

Microsporidia are common parasites of fish and insects. They have been described in humans causing ocular and enteric disease in immuno-compromised patients. They have also been described in lovebirds (*Agapornis*).

Cell destruction is believed to occur when the parasitophorous vacuoles discharge their contents. Consequently enterocytic destruction in the ostrich chick could lead to integumentary breaks, seen focally in the colon, with resultant secondary infections and extensions to multifocal enteric necrosis. Multifocal enteric necrosis, especially affecting villous tips, has been seen previously here in ostrich chicks but no cause could be defined. It is tempting to speculate that microspordian infection, or equivalent, may be an initiating pathogen.

Because the bird was received alive tissues were removed and formalin fixed soon after euthanasia. Under normal circumstances birds arrive dead, often 24 hours or more after death. Enterocyte sloughing and other autolytic changes could make ready recognition of enteric microsporidian infection difficult or impossible under these conditions.

References:

Cali, A. General Microspordian Features and Recent Findings on AIDS Isolates (1991). J. Protozool, **38**:625.

Lorn, J. and Dykova, I, (1992). Developments in Aquaculture and Fisheries. Vol 26, Protozoan Parasites of Fishes. Elsevier Amsterdam P125.

Norton, J. H. and Prior, H. C. Microsporidiosis in a peach-faced lovebird (*Agapornis roseicollis*) (1994). Aust. Vet. J. 71:23.

Randall, C. J., Lees, S., Higgins, R. J. and Harcourt-Brown, N. H. Microsporidian Infection in Lovebirds (*Agapornis* spp.) (1986). Avian Pathology, 15:223.

Cervical Vertebral Gout with Associated Compression of the Spinal Cord in Ostriches - Roy Mason, Animal & Plant Health Assessment Branch

Three cases have been referred to our laboratory. All have been in adult (two, three and eight year old) female ostriches.

Consistent presenting signs were an unsteady gait and progressively worsening ataxia. Other signs included "kinks" in the neck (two), a treading action when standing (one) and a swaying motion of the head and neck (one).

As the presenting signs worsened the birds lost more condition.

Misadventure was reported for two birds, both becoming tangled in fences. In one case it was the opinion of the owner that it was this incident that precipitated the problem.

All autopsies were undertaken in the field by veterinary practitioners. No mention was made of there being any lesion or tissue change other than superficial cutaneous abrasions and knobbly swellings associated with the cervical vertebrae.

The swellings occurred at the intervertebral joints and were most prominent at the C2/C3, C3/C4 and C4/C5 joints. There were milder swellings associated with some other cervical intervertebral joints.

The content of the swellings, which were bounded by relatively minor encapsulation, was a bacteriologically sterile pultaceous white material which extended between the joint surfaces and protruded into the vertebral canal as discrete bulges producing localised spinal cord compression. The material was chemically analysed as uric acid.

This problem does not appear to have been reported previously in ostriches.

Articular gout can occur in association with visceral gout or independently.

Visceral gout occurs as a result of impaired renal function or urinary tract obstruction. In only one of the three cases examined was kidney tissue also submitted. This bird had been clinically affected for several months but there was no histological evidence of renal disease. None of the practitioner reports accompanying the submitted material reported on the presence of visceral gout, obvious renal disease or articular lesions at any other location. It therefore appears these three cases are cases of primary articular gout. Primary articular gout has been induced by high protein diets in turkeys. In addition, there is also evidence of articular gout having a heritable cause.

The cause of these cases of cervical gout is unknown. However, given that the ostrich industry has been based on a fairly small genetic base, with breeding flocks in the formative years consisting of single pairs of birds, then where expansion through local acquisition of birds occurs, birds are likely to be closely bred and the occurrence of heritable based disorders cannot be dismissed. In addition the ideal protein requirements for ostrich may not be met by diets based on many of the raw ingredients used for poultry.

Flounder Diseases - Barry Munday, Department of Aquaculture, University of Tasmania

1. Herpes virus dermatitis detected in adult greenback flounder which had been severely stressed during translocation.

Lesions varied in extent and were most common on the fins which appeared opaque and showed moderate erosion between the rays. On the dorsal surface some fish had pale plaques up to 15mm in diameter. Enlarged bizarre cells were present at the eroded edge of the lesions and samples sent by Mt Pleasant Labs, to AAHL were found by transmission EM to contain herpes virus particles.

A few fish died, but most recovered and it is presumed that this is an endemic virus which becomes activated under stressful conditions.

2. "Egg peritonitis" has been commonly found in broodfish which have not been stripped when "ripe". The peritonitis is characterised by adhesions between the retained egg mass and the colonic wall. Some of these fish become debilitated and susceptible to opportunistic parasites and bacteria.

Fish Pathology Registry

This facility was suggested for funding during the second triennium of the Aquaculture CRC. The application was not successful.

Ulcerative Mycosis of Platypus - Barry Munday, Department of Aquaculture, University of Tasmania

In a recent case the only lesion found was a nodule (approximately 2mm diameter) on the web of a forefoot. There was no evidence of ulceration. This observation, together with the regular occurrence of fungal pneumonia in ulcerated animals, has led to an alternative pathogenetic mechanism being hypothesised. The suggestion is that platypus inhale spores of *Mucor amphibiorum* which establish (at least temporarily) in the lungs of platypus and eventually enter the blood stream and spread to various parts of the body, but only proliferate where temperatures are <34°C.

Nutritional Muscular Dystrophy in Calves - D Seward

Low Selenium and Vitamin E levels were found in a three month old Jersey calf from Western Creek in Central Northern Tasmania.

		Normal values	
Glutathione peroxidase activity		>5U/gHb	>30U/gHb
Serum Vitamin E	0.55mg/L	>2.0mg/L	

Of a group of 35 calves, four died and two others went down with clinical signs of muscle weakness over a two week period in late December. A post mortem examination performed by the practitioner on one calf revealed bilaterally symmetrical pale patches/streaks in the skeletal muscles of the axilla and inner thighs. The urine had dark brownish discolouration. Coagulative necrosis of muscle fibres and renal changes indicative of myoglobin nephrosis were evident on histological examination. The myocardium was not available for examination. Clinical, gross and histological findings were consistent with nutritional muscular dystrophy. Despite low levels of Selenium in the Western Creek area, white muscle disease in calves is rarely diagnosed. There was no history of recent superphosphate or sulphur application to the pastures. (Calcium sulphate in superphosphate is known to reduce Selenium uptake). Lush pastures high in unsaturated fatty acids (e.g. linoleic acid) following the unseasonable wet weather during late November/early December may have increased the demand for Vitamin E and precipitated the outbreak.

Queensland - Bruce Hill

Suspect *Leucocytozoon* sp. infection in an Emu - Giant Campbell, Toowoomba Veterinary Laboratory (QDPI)

A six week old emu was presented in extremis with a history of scouring for four or five days. At necropsy, fibrin plaques were present in the left and right posterior airsacs and lungs were pale and fluid filled. The spleen was much enlarged and mottled. There was bilateral enlargement of the kidneys which were covered with mucofibrinous material and distended with urates. Peritonitis, with greenish fluid and fibrin plaques and strands in the abdominal cavity, was noted. The liver was swollen, with a greenish mottled appearance and the presence of occasional pale foci (1 to 3mm diameter) on the surface. There was hyperaemia/haemorrhage of the wall of one caecum which contained a caseous cast.

Erysipelas was considered a very strong possibility, however 24 hours after necropsy there was no evidence of *Erysipelothrix* sp on blood plates and histological examination had revealed a severe protozoan infection. Severe degenerative changes were noted in the liver, spleen and kidney where round basophilic bodies about ten microns in diameter were present in blood vessels in large numbers. These organisms were also in large numbers in the lung with minimal change in the tissue. The morphology of these organisms was suggestive of *Leucocytozoon* sp. though they were not positively identified. No other cases were detected in the flock.

Mycoplasmal polyarthritis in persistently MD infected calves - Grant Campbell, Toowoomba Veterinary Laboratory (QDPI)

A two month old Hereford calf was presented with a history of scouring and fairly sudden development of severe polyarthritis. Of the 50 calves in the group, nine had recently died as a result of severe scouring and at least three had developed acute polyarthritis.

Necropsy revealed swelling of all major long bones arthroses and opening a number of these showed the presence of fibrin plaques and floccules and brown discoloured synovial fluid. The small intestine showed distension with frothy yellow contents for about 50% of its length. Histological examination of synovial membranes and associated tissues showed a severe mixed cellular inflammatory response. Lymphocytes predominated in the synovia and parasynovial tissue while polymorphs were more evident in some deeper tissues. Aerobic culture of joints grew no organisms, however a roup seven Mycoplasma was grown from two sites. Further investigation revealed that a number of calves within the group were persistently infected with pestivirus on an antigen capture ELISA and it is surmised that pestivirus was playing a part in the apparent outbreak of mycoplasmal polyarthritis.

***Ichthyophthirius multifiliis* in Silver Perch - John Gibson, Toowoomba Veterinary Laboratory (QDPDI)**

Ichthyophthirius multifiliis was diagnosed as the cause of a sudden fish kill in one tank of a commercial farm. The farm breeds and raises Silver Perch (*Bidyanus bidyanus)* for live export trade to Japan. Some 80,000 fish are present on the farm and the kill involved 123, 18 month old fish in one tank. Two recently dead perch were submitted for necropsy. Both fish had hyperaemia of the skin which was noticeable on the tail and fins. The skin was covered with thick mucus that contained small white "salt-like" granules. The gill filaments were pale, thicker than normal and with similar white granules on the surface. Wet preparations of the gills and skin revealed massive numbers of a large ciliate protozoan consistent with *Ichthyophthirius multifiliis*. Lesser numbers of a much smaller protozoan consistent with *Trichodina* sp. were also present. Histologically the gills had severe epithelial hyperplasia with clubbing of many lamellae. There were no lesions in other organs.

Suspect Lymphosarcoma in a Horse - Grant Campbell, Toowoomba Veterinary Laboratory (QDPDI)

Numerous irregular swellings, 1 to 5cm in diameter were reported beneath the skin of a 16 year old mare. The greatest concentration of lesions was on the ventral torso. These lesions had been first noted three months previously and had enlarged slowly. A biopsy was submitted and on histological examination was seen to consist of sheets of cells having small to medium sized, fairly regular, dark staining nuclei and moderate amounts of cytoplasm. Mitotic figures were fairly common. This histological picture was suggestive of lymphoma/lymphosarcoma. Such tumours are either rare or rarely reported in the horse and the submitting vet was contacted for information on clinical progression. Though the horse was maintaining good health the lesions were increasing in size and number.

Enzootic Ataxia in Lambs - John Gibson, Toowoomba Veterinary Laboratory (QDPI)

Enzootic ataxia was diagnosed in a mob of 420 Dorset cross lambs. Ten lambs were affected with marked ataxia in the first weeks of life. One lamb submitted for necropsy was alert, had severe forelimb and hind limb ataxia but with a strong righting reflex. It was only just able to stand with a peculiar crouching "sway back" stance. Histologically there was mild to severe Wallerian degeneration of white matter tracts in the dorsal aspect of the lateral funiculus and ventral funiculis adjacent to the central fissure. The lesion was most severe in the cervical and lumbar enlargements. There were scattered necrosis of Purkinje neurones and white matter tracts in the cerebellar peduncles were also affected. Serum copper levels in affected lambs ranged from 63 to 328µg/L and the liver copper of the necropsied lamb was 5ppm.

Lyssavirus Meningoencephalitis in a Microchiropteran bat - Jim Taylor, Toowoomba Veterinary Laboratory (QDPI)

A member of the public contacted QDPI to report a flying fox on the ground and seemingly unable to fly. On arrival at the scene, the bat was not a flying fox (sub-order Megachiropteran) but a small insectivorous bat (sub-order Microchiropteran), later identified as a Yellow Bellied Sheath Tail (*Saccolaimus flaviventris*). During transportation to the lab the bat aborted a full term foetus. At necropsy apart from mild oedema of the pelvic fascia, there were no significant abnormalities. Histologically the brain had a very mild meningoencephalitis with occasional mild , mononuclear perivascular cuffs in the meninges and neuropil and very mild scattered foci of gliosis with an occasional polymorph.

Brain impression smears were positive to Lyssavirus using a fluorescent antibody test performed at AAHL.