VETERINARY PATHOLOGY REPORT

3 Baron-Hay Court, South Perth, Western Australia 6151
Postal Address: Locked Bag 4, Bentley Delivery Service WA 6983
Publication No. PP544059/0003

EDITOR: Cleve Main

Number 51 **December 1998**

PAGE **CONTENTS**

SECRETARIAT

PO Box 114 Walkerville SA 5081
Phone: 08 8344 6337 (Chandra Aitken) Fax: 08 8344 9227

ASVP EXECUTIVE 1997-98

President:	Clive Huxtable	School of Veterinary Studies Murdoch University MURDOCH WA 6130	08 9360 2297
Secretary:	Cleve Main	Animal Health Laboratories 3 Baron Hay Court SOUTH PERTH WA 6151	08 9368 3351
Treasurer:	Jeremy Allen	Animal Health Laboratories 3 Baron Hay Court SOUTH PERTH WA 6151	08 9368 3466
Committee Members			
	Barry Richards	Animal Health Laboratories 3 Baron Hay Court SOUTH PERTH WA 6151	08 9368 3424
	John Jardine	Vetpath Laboratory Services 9 Lyall Street REDCLIFFE WA 6104	08 9277 9847

APPOINTMENTS

Chairman – Registry of Domestic Animal Pathology	Tony Ross
Newsletter Editor	Cleve Main
Coordinator – Training Committee	Judith Wilkie

CONVENTOR – SLIDE OF MONTH

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

President's Report

The major event for the Executive since the AGM has been interaction with the National Review of Laboratory services launched by the AAHC. No doubt many of you will have also interacted with it in some way. Barry Richards is of course one of the three members of the Review team, and I represented the Society when the team visited Murdoch University during the Western Australian leg of its national tour. In addition the society has made a written submission to the Review, which was initially drafted by me and refined by the Executive. This submission is included in this issue of the ASVP Report for the information of members. The impact of recommendations made when the Review submits its report towards the end of this year will be of great significance for the Society. We should be hopeful that there will be some positive outcomes for all of us. In the meantime I would appreciate receiving information and critical comment from you. Several members have already obliged in this way, so join them and feed us your opinions and knowledge.

Another task we are getting started on is the venue and program for the 1999 meeting. The plan is to schedule it for Melbourne on the weekend before the AVA meets in Hobart. This year's meeting set a high standard all round and gives the next organizing committee something to aim for. One thing I would like to see – as many presentations as possible by the young members, particularly those in training.

That's it for now – best wishes to you all

A/Prof. Clive R. Huxtable
PRESIDENT

Editorial

Well it's that time of year again and in the hope that this issue of the report gets to you somewhere near Christmas, I wish you all the best for Christmas and the New Year.

Most of you will be aware that we have had some problems with the Secretariat in South Australia. In short, membership and postal address lists were not up to date. Cheques were not being banked on time and communications between the Executive and the Secretariat were becoming strained.

I am pleased to report that things have improved. New staff have been employed and they are making every effort to sort out the "mess". It will not happen overnight, so we must give them time to do the job. Having said that, if you have a problem such as your subs not being acknowledged or if you know of somebody not receiving their mail or ASVP Report, let me know.

While on the subject of the ASVP Report, it disappoints me that quite often contributions have been difficult to get. Here we have a situation where Lucy Genovese sends in contributions from the UK, yet some of our more populous States find it difficult to do so. We are, after all, only looking for short articles and case reports. We are not looking for AVJ standards. Surely it's not all that difficult, especially if it is planned a month or so ahead. If you have an interesting case, let's hear about it. If you have an opinion on something, especially if it concerns veterinary pathology, drop me a line and I'll print it.

Are you receiving your "Slide of the Month"? If you're not, let me or Rod Reece know and we'll fix the problem. Do you still require your SOM? Do you, for example, work in an institution which receives its own set of slides? Have you ever thought about contributing to the SOM series? Most of you would have had cases which would be of interest to us all. Give it some thought.

In conclusion, I thought I'd let you know that the next ASVP conference will be in Melbourne immediately preceding the AVA conference in Tasmania. Ron Slocombe has agreed to head the organizing committee and we look forward to an interesting session. Don't forget that conference time coincides with renewal of subscriptions. You will see elsewhere in this issue that Treasurer Jeremy Allen has provided a list of current members. If you have not paid your subs, you will receive a reminder and if you are more than one year in arrears, you will receive a request to pay your back dues. We are a small organisation and we cannot afford to carry unfinancial members.

Don't forget, it was at the Brisbane AGM, a vote to remove the names of people who had not paid their subs for two or more years was carried.

Cleve Main
(HON SECRETARY/EDITOR)

Review of Australian Veterinary Laboratories

The review of Australian animal health laboratories, commissioned by the Australian Animal Health Council (AAHC), has been completed and a final report was presented on 30th November 1998. The major recommendations of significance to the ASVP relate to the suggested formation of a national laboratory program under the auspices of AAHC. It is recommended that the program have a full-time Manager/Coordinator and a management group with representation from State and Commonwealth governments, CSIRO, private laboratories and the livestock industry. The program would be involved in coordinating a number of laboratory activities of national importance including standard diagnostic techniques, methods enhancement, quality assurance for specific tests, skills maintenance, training, research, exotic disease preparedness, rapid response capability and national reference laboratories

Of most interest to our Society is the recommendation that **"all laboratories should collaborate in identifying long term training needs for laboratory specialists as soon possible. The aggregated training requirements derived from this exercise should be taken to potential training providers so that the long-term training needs of the network can be put to tender".** This was in recognition of the impending shortage of formally trained veterinary pathologists in Australia and the need for continuing education for existing veterinary pathologists. The ASVP will have a role to play in provision of services to meet these needs.

These recommendations were put to a workshop of AAHC members and key stakeholders in October 1998 and subsequent feedback indicated there was broad general agreement. The AAHC Executive will now consider formal adoption of the recommendations and begin the process of implementation. We can only hope that the progress is quick and decisive. The ASVP will tender for the provision of specialist training in veterinary pathology along the lines previously reported in this newsletter. The ASVP Executive will seek the advice and opinions of members during the tender process.

For members' information, the AAHC Board now has a new look with several changes taking place at the AGM in October. The new Chairman of the Board is Dr Raoul Nieper, formerly Director General of QDPI, who replaced Dr Alan Donald. New veterinarians on the Board include Dr Lex Carroll of Queensland and Dr Ian McCausland of NSW. Other Board members are Mrs Keryl Enright (a primary producer from WA), Mr Michael Nicholls (a Wool Council of Australia member from NSW), Mr Harvey Parker (with producer and Executive involvement in the horse industry, from Victoria) and Mr Tim Roseby (Department of Agriculture, Forestry and Fisheries, Canberra).

Barry Richards (December 1998)

DISCUSSION PAPER

Guidelines for the Implementation of Proficiency Testing in Anatomic Pathology in Australia

Background

Members will be aware that Dr Phil Ladds undertook a contract with the society during 1998 to deliver a continuing education package in all states. The contract also required that while performing this mission he should canvass and report on the opinions of Society members on broad questions of the state of veterinary pathology in Australia and quality assurance processes that might be implemented. A major component of such processes wou7ld be a system of professional proficiency testing (PT) in anatomic pathology.

Dr Ladds duly submitted his report to the Society in October 1998, and it revealed that while there was widespread support in principle for a proficiency testing mechanism, there was, not unexpectedly, a divergent range of opinion on its nature and application.

In the view of the Executive this is an important issue which needs to be resolved as soon a possible. The position of the ASVP should be that in the immediate future, proficiency and qualifications must be recognised and appropriately rewarded by the consumers of the expertise. There must be a positive move to stop selling pathology training and expertise short by accepting low "minimal levels" of qualifications and proficiency. A credible PT process should demonstrate that any sub-standard performance will be exposed. A complementary professional development/CE program should maintain proficiency at demonstrably high levels.

Accordingly, on the basis of the Ladds report, a set of principles for
proficiency testing has been drafted by the Executive and is detailed below for the information of members.

This will be an agenda item at the AGM and it is the hope of the Executive that these principles, or an amended version thereof, will be officially adopted by the society at that time.

DRAFT PRINCIPLES FOR PROFICIENCY TESTING (PT) IN ANATOMIC PATHOLOGY

* A basic PT "module" should contain material which falls into one of three categories. (Material should not be confined to histopathology – some evaluation of gross lesions should be included).

  The three categories would be:

  1. The Examples or "atypical" lesions of common Australasian conditions.
  2. Classic examples of conditions which might be rare/exotic, but which are of potential relevance to Australia and New Zealand.
  3. Cases which are good fundamental exercises in interpretation of basic processes.

* In such a basic module the focus would be on "economic livestock" (broadly defined). However, categories 2 and 3 would allow for the use of any species. A basic module could also be supplemented by some additional cases of species-specific material which could be elective.

  In addition the PT program would be closely linked to the CE-focused slide-of-the-month program Which covers a diversity of species.

* A basic PT module should contain **at least** 10 cases – 6 of category 1, and 2 each of categories 2 and 3 or similar proportions if the total was more than 10 cases.

* Cases for each module could be reviewed by a panel of 3 or 4 experienced consultant pathologists – not all need necessarily be located in Australia or New Zealand. This would set the general context for the desired responses which would be evaluated as:

  1) Preferred diagnosis
  2) Acceptable diagnosis
  3) Unacceptable diagnosis.

Some attention might also be paid to morphologic versus aetiologic diagnosis, and plausible differential diagnoses.

* "Unsatisfactory" performance might be >20% of diagnoses in the "unacceptable" category.

* The testing process would be "brokered" by the successful tenderer to the AAHC for the provision of a national quality assurance program for veterinary diagnostic services (see accompanying report in this issue).

* The test should be directed to laboratories/institutions whose senior people would then arrange for their staff to take it, and for the return of the completed tests. Information on individual performance would have to be restricted to a range of persons to be decided.

* The PT exercise could initially be voluntary, but would eventually become part of mandatory QA processes.

The comments of members on the above are sought and will be welcomed by the Executive prior to the AGM.

A/Prof. Clive R. Huxtable
PRESIDENT

NEW SOUTH WALES – Paul Gill

NSW Histopathology Group Meetings for 1999

This group has met three times a year for several years now. Meetings consist of informal case presentations and anyone with an interest in histopathology is encouraged to attend. The meetings for 1999 are as follows:

Saturday 13 February 24,	-	EMAI, Menangle
Saturday 14 August	-	EMAI, Menangle
Saturday 13 November	-	Vet School, Sydney Campus

For further details, please contact Malcolm France (Phone: 02 9351 2023, or email: m.france@vetp.usyd.edu.au).

Cardiomyopathy in gene knockout mice – Malcolm France, University of Sydney

A colony of RAG-1 knockout mice has suffered ongoing losses among its adults. Affected mice are depressed and occasionally may exhibit dyspnoea. At necropsy the heart is enlarged (sometimes marked so) and dark red/purple. Histologically there is patchy necrosis and diffuse fibrosis of the myocardium and large endocardial thromboses are present in the ventricle or atrium of some animals. Results of microbiology examinations have been negative.

The RAG-1 knockout mouse is a severely immunodeficient genotype created through genetic engineering and is one of the more widely used varieties of gene knockout mouse. This condition is therefore of some concern although anecdotal evidence suggests that the problem is not a direct consequence of the mutant gene but rather an effect of the genetic background.

An unusual Encephalomalacia in an 18 month old steer – Paul Gill, Regional Veterinary Laboratory, Wollongbar

An 18 month old, depastured, Hereford steer died after appearing blind for about a week. Coronal sections of fixed brain revealed bilaterally symmetrical, soft, dark areas of about 3mm in diameter in the pyramidal lobes, dorsolateral aspect of the anterior medulla, ventral pons and ventrolateral medulla (near the facial nerve nucleus). Histologically, these foci were focally extensive areas of malacia, ringed by gemistocytes. Vessels within the affected area are congested, and there is occasional, moderate perivascular haemorrhage.

Bilaterally symmetrical foci of malacia have been described in the internal capsule and adjacent basal nuclei, thalamus, midbrain and cerebellar peduncles of young calves. This steer is 18 months old, and the bilaterally symmetrical foci are distributed in the medulla/pons. Your ideas and comments are sought (email: paul.gill@agric.nsw.gov.au).

NORTHERN TERRITORY – Anton Janmaat

Mycoplasma infection in pigs – Helen Parkes, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

Until recently, there has only been one commercial piggery in the Top End, and for many years it has been run virtually as a closed operation. A second piggery has recently commenced operations, and as well as breeding, it imports weaner and grower pigs from southern Australia for finishing up here. The original piggery also recently bought some young boars from South Australia. In August/September 1998, the new piggery reported problems with pigs coughing, and pleuritis was reported at slaughter. At about the same time, the original piggery started seeing disease, and a few deaths in sows.

In October, when BVL became involved, the original piggery was losing more than 40 pigs per week (average pigs born per week is about 150). Grower pigs were dying acutely, with few clinical signs and were presented for post-mortem examination in good nutritional condition. Sucking weaner pigs showed a more prolonged course of disease, and were thin and dehydrated at post-mortem. All pigs examined showed lung lesions. Lungs were moist and oozed excess fluid and had varying degrees of dark pink consolidation of the ventral area of the cranial lung lobes. Fibrinous pleuritis, pericarditis and peritonitis were variable findings but were particularly prominent in the weaner pigs. Meningitis was noted in some grower pigs.

Histological examination of the lungs consistently showed bronchopneumonia with a lobular distribution. Bronchioles contained abundant mucopurulent exudates. Peribronchiolar inflammation was prominent in some animals, and was mainly mononuclear, particularly lymphocytes and plasma cells. Alveolar septa were thickened, with increased mononuclear cells, and alveoli contained fluid and sometimes abundant alveolar macrophages. In other animals there was purulent alveolar exudate. Fibrinous pericarditis was particularly severe in the weaner pigs. The brains of two grower pigs were examined and both showed meningitis with a thick layer of mixed inflammatory cells and congestion of the meninges. One of these also showed mild to moderate perivascular cuffing with mononuclear cells in the medulla, midbrain and cerebellum.

Bacterial culture grew *Strep.suis* type 2 from most of the lungs examined, as well as from samples of pericardial fluid and brain swabs. Although *Actinobacillus pleuropneumoniae* was looked for it was not isolated. Neither were we able to isolate a mycoplasma. However, stored serum taken in late 1997 and early 1998 from sows on the original piggery was available. Twenty of these stored samples plus 25 samples taken since the disease outbreak (from pigs at slaughter) were sent to RVL Menangle for *Mycoplasma hyopneumoniae* ELISA. All 20 samples taken before the outbreak were negative for *M. hyopneumoniae* antibodies but of the 25 samples taken since the outbreak 18 were considered positive, 3 were inconclusive and 4 were negative.

Our interpretation of this information is that, due probably to isolation and luck, the original piggery was free of *Mycoplasma hyopneumoniae* at least until early this year. Following importation of pigs into a new piggery in the Top End and a few boars into the original piggery, *M. hyopneumoniae* has been introduced. In association with *Strep.suis* type 2, this has caused acute disease in grower pigs, and more chronic disease in sows and weaner pigs.

This was an unfortunate way to discover we had one of the few *M. hyopneumoniae*-free piggeries in Australia.

QUEENSLAND – Jim Taylor

Suspect Nodavirus infection in Barramundi fingerlings – Wendy Townsend QDPI, Yeerongpilly Veterinary Laboratory

A fish farmer noticed that some recently purchased Barramundi (*Lates Calcarifer*) fingerlings were showing odd swimming behaviour, including floating upside down. Swim bladder problems were suspected and a sample of the affected fish sent to the laboratory. Histological examination of the fingerlings (estimated to be about 28 days old) failed to show any swim bladder abnormalities, however, two of the eight fish showed vacuolation of the brain and/or retina, a finding consistent with nodavirus infection.

Nodavirus infection, or viral nervous necrosis, is a disease of larval and juvenile Barramundi. The disease produces vacuolation and cell necrosis in the CNS and retina. Affected fish exhibit a range of neurological signs including darting and corkscrew swimming. Such signs are often accompanied by anorexia and pale colouration.

The most characteristic histological lesion seen is vacuolation in the grey matter of the brain, which appears to be intracytoplasmic. Other lesions include pyknosis, shrinkage and basophilia of affected cells, focal pyknosis and karyorrhexis of neural cells, granularity of neurophil, accumulation of eosinophilic material in macrophages and blood vessel walls and the presence of mononuclear cell infiltrates. Basophilic, intracytoplasmic inclusion bodies have also been reported. Vacuolation of the retina tends to be most prominent in the bipolar and ganglionic layers, although small vacuoles can be found in the rod and cone layer (reviewed by Munday and Nakai 1997).

Mundal BL and Nakai T (1997). Special topic review Nodaviruses as pathogens in larval and juvenile marine finfish. *World Journal of Microbiology and Biotechnology* **13**, 375-381.

"Scrub ataxia" in a Braham calf – Wendy Townsend, QDPI, Yeerongpilly Veterinary Laboratory

A local farmer contacted the laboratory following problems with his Brahman calves. Three of 45 animals had been affected to date, one of which had died. All animals were under 6-7 months of age and still suckling. Clinical signs included dribbling and staggering. As the condition progressed, the animals became increasingly recumbent, eventually refusing to rise. Dermatitis was reported to be present around the face of affected animals. One calf was submitted for post-mortem examination. Gross findings were unremarkable apart from localised dermatitis around the face, unilateral corneal ulceration and abrasions on the fetlock and carpal joints.

On histological examination Wallerian degeneration was evident in the cervical, thoracic and lumbar spinal cord, with the most severe changes occurring in the thoracic segment. No particular pattern could be associated with the lesions. The liver showed some disruption of the normal architecture with the normal cord-like arrangement of hepatocytes being difficult to discern in areas. Some hepatocytes showed marked swelling and their nuclei appeared pale, vesicular and larger than normal, although the latter change was not severe enough to suggest megalocytosis. Cytoplasmic vacuolation and occasional degenerate/apoptotic cells were also noted. Sections of hyperkeratotic skin contained dermatophilus. Clinical pathology revealed liver and muscle damage. Serum copper was normal.

A definitive diagnosis was not obtained in this case. The possibility of *Macrozamia* or *Xanthorrhea* toxicity was ruled out. The clinical signs, history and spinal lesions were suggestive of a condition known colloquially as "scrub ataxia". This is seen in suckling calves, usually at the time when feed is scarce. The most consistent lesion in the affected animals is Wallerian degeneration in the spinal cord. Toxic plants have been implicated, but no aetiology has yet been proven.

We would welcome any comments from others who have seen similar cases.

Riboflavin deficiency in broiler chicks – Greg Storie, QDPI, Yeerongpilly Veterinary Laboratory

A group of 1440, 13-day-old meat chickens exhibited poor growth rates and 220 developed difficulty walking. Affected birds appeared bright but sat with legs sprawled, often with one leg pointing forwards and the other backwards. There was inward curling of the toes. Sciatic nerves were swollen. Histological examination revealed a severe bilateral sciatic nerve neuropathy characterized by myelin degeneration with accompanying axonal swelling and fragmentation. Schwann cell hypertrophy and hyperplasia was marked. There were scattered foci of pericapillary lymphocyte infiltration. The lesions are characteristic of riboflavin deficiency. An error in premix formulation was suspected.

Pigeon Herpesvirus infection – Greg Storie, QDPI, Yerrongpilly Veterinary Laboratory

Forty squab died and 40 were ill in a group of 160 young birds n a pigeon rearing establishment near Biloela in central Queensland. Three hundred breeding adults were unaffected. The owner reported that sick squab developed respiratory signs and brown pus in the mouth prior to death. A moderately autolysed carcass was presented to the laboratory. Necropsy revealed an adherent yellow caseous exudates on the surface of the pharynx and upper oesophagus. Histologically there was a severe subacute diffuse necrotizing pharyngitis and oesophagitis. A diphtheritic plaque of necrotic mucosa and inflammatory cells contained numerous bacterial colonies. Many of the remaining epithelial cells contained eosinophilic intranuclear inclusions pathognomonic for pigeon herpesvirus.

SOUTH AUSTRALIA – Ruth Reuter

Myxobacterial infection in young snapper – Ruth Reuter, VPS/VETLAB, Adelaide

Snapper are a popular table fish in Australia. Currently there is interest in aquaculture of this species in South Australia and several farms have been established for this purpose. Recently one of these farms developed a problem with fingerlings showing loss of appetite, erratic swimming, lethargy and reddening around the mouth and nose. A second younger batch appeared unaffected. The fish were held in a series of separate tanks. Suspicion fell on the feed which was an old batch and had not been stored in ideal conditions. However, both batches of fish were receiving the same feed.

Samples of live and dead fish from both batches were submitted for examination. The live affected fish were gasping, the opercula were open and scales were discoloured and darkened. The live unaffected fish were active and appeared grossly normal. Histological examination of tissues revealed large masses of slender bacterial rods matted on the surface of the skin around the mouth and extending into the oral cavity. The underlying skin and muscle were degenerating. The bacteria resembled Myxobacteria, the cause of "Cold Water Disease" or "Columnaris Disease".

Myxobacteria are commonly found in soil and water, and can be primary or secondary pathogen in both freshwater and marine fishes. At present there is still some discussion over nomenclature, but *Flexibacter* and *Cytophaga* are most frequently used to name the organisms found. Infections of this type are usually associated with stress factors such as transport and handling, overcrowding, excessive exposure to intense UV light or sub-optimal water temperatures. Treatment with oxytetracycline has been reported to be beneficial. Increasing the water temperature has also been recommended in some reports.

Pilchard mortality in South Australia – Ruth Reuter, VPS/VETLAB Adelaide

The recent mortality of pilchards, particularly in South Australian waters, made headlines across the country and stirred an intense media and public interest here. Some of this is "dying down" now that the major event seems to have passed. However, we are still getting a trickle of animals in for post-mortem as a result of the publicity. The range so far includes leatherjackets, pufferfish, seahorses, seadragons, jellyfish, penguins, eels, cuttlefish and anchovies. It has certainly challenged my knowledge of anatomy and histology of these species! A great learning experience!

TASMANIA – Roy Mason

Diagnosis of Rabbit Haemorrhagic Disease: Rabbit Calicivirus Infection from badly predated carcases – Pat Statham, Roy Mason and Deborah Seward

Following the unplanned introduction of RHD into Tasmania there was a need to examine dead rabbit carcasses for evidence of RCV infection to define location and spread of infection. Whole carcasses provide the organ(s) of choice (liver, spleen, Lung) for viral antigen detection using the capture ELISA method. However, where a carcass has been predated to the extent that internal viscera has gone then the carcass appears to be useless for detection of infection.

We have tested a number of rabbits for calicivirus using both liver and bone marrow and have obtained a good correlation between the results. Generally though bone marrow gives a lower optical density reading in positive rabbits (but still well above the positive/negative cut-off) than the respective liver with the capture ELISA test. Negative samples have always given an optical density that is unequivocally negative. We also looked at skeletal muscle extracts from known positive carcasses but on all occasions they gave negative e results.

We believe that bone marrow can be a useful tissue to test for the presence of RHD/RCV infection when no other su8itable tissue is available.

For testing we use about 1 gram of bone marrow recovered from a long bone such as the femur. Marrow is removed by breaking the bone with bone forceps and scraping out the marrow with a wooden applicator stick. The marrow is then tested using the same procedure as for liver.

WESTERN AUSTRALIA – David Forshaw

Suspect Oligodendroglial Dystrophy in Aberdeen Angus Calves – Cleve Main – Animal Health Laboratories, South Perth

A nervous disorder in neonate Aberdeen Angus calves is under investigation. Initial reports described calves which were unable to stand, suffering muscle twitching and convulsions. These calves invariably died within 4-5 days of birth. Brain and visceral samples were submitted from one of these calves and two other calves were presented alive for examination and necropsy. The first of these calves displayed hypermetric placement of the forelimbs and severe hind limb ataxia. The second calf, an Angus/Hereford cross could stand quite easily, but displayed coarse trembling of its hind limbs and mid to moderate hind limb ataxia when forced to walk. Fear, blink and withdrawal reflexes were present in both calves. The cross-bred calf had a strong suckling reflex and had been bottle fed since birth.

Similar histopathological changes were present in all calves, varying only in severity.

In the brain there were moderate to severe white matter lesions in the internal capsule, mid brain, medulla and cerebellar white matter, severity increasing towards the mid and hind brain. Lesions were also present in the optic tract and chiasma but not the optic nerve or retina. Severe lesions were present in all white matter tracts for the entire length of the spinal cord, but not in the cauda equina or peripheral nerves.

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

The lesions are similar in many respects to those described in progressive ataxia of Charolais cattle. In that disease as with this one, the pathogenesis appears to be a dystrophic one affecting oligodendroglia. In progressive ataxia, clinical signs are evident at 8-24 months of age and progress over a period of 1-2 years, whereas in these calves the condition is present at birth. The mildest clinical signs and histopathological lesions were seen in the cross-bred calf and although it is tempting to speculate that the disease may be a genetic one, clearly it could not be an autosomal recessive condition.

More on fungal infections in frogs – Chytridiomycosis – Cleve Main – Animal Health Laboratories, South Perth

In a previous edition, mortalities in frogs from the Perth Zoo from overwhelming infections by the fungus *Mucor amphibiorum* were described. As part of a follow-up investigation, tissues from another tree frog (*Litoria adelaidensis*) were examined histopathologically. The only lesion found was confined to folds of skin associated with the proximal hind limbs. These were confined to folds of skin associated with the proximal hind limbs. These were characterized by marked sub-acute to chronic dermatitis with hyperkeratosis, acanthosis and the presence of numerous small organisms in the epidermal layer. They varied in diameter (20-30µ) and were enveloped by a clear capsule. The number of bodies within a capsule varied from a single large nucleus-like body to aggregates of smaller dense bodies.

The organisms are consistent with the Chytridriomycete fungus described by *Berger* et al. 1988. In their excellent paper, the authors describe lesions in over 100 frogs from various parts of Australia and from the rainforests of Central America. Members of the phylum Chytridiomycota are ubiquitous heterotrophic fungi found primarily in soil and water and usually have a saprophytic role. Some genera are obligate parasites of fungi and algae and plants. The chytrid reported in their paper is the first member of the Phylum Chytridiomycote to be recognised as a parasite of the phylum vertebra.

The extent of the disease in Western Australian amphibians is unclear. Only one other case has been previously reported. A cooperative investigation with the Western Australian Museum has so far failed to locate any other frogs suffering from the disease.

DEPARTMENT OF PRIMARY INDUSTRIES

MANAGER, YEERONGPILLY VETERINARY LABORATORY

The position of Manager, Yeerongpilly Veterinary Laboratory is being re-advertised. The position is within the Animal Health & Welfare Division of the Department's Animal & Plant Health Service.

Centre: **Animal Research Institute, Yeerongpilly, Brisbane.**

Salary: **$56,194-$60,253 p.a.**

Key duties:

Manage the Yeerongpilly Veterinary Laboratory which provides laboratory support to active and passive disease surveillance programs and to exotic and emergency disease resolution. Involvement in development and maintenance of skills and in achieving laboratory accreditation of the Department's statewide laboratory network is required.

Skills/Abilities:

A registerable degree in veterinary science preferably with postgraduate qualifications in pathology is required.

For further information including a Position Description please contact Russell Rogers by phone on (07) 3239 3835; by fax on (07) 3239 3558; or by e-mail at rogersr@dpi.qld.gov.au

CURRENT FINANCIAL MEMBERS

As at 9 December 1998

Allen	JG	Ladds	PW	Haschek-Hock	VM	Sutherland	RJ
Arzey	KE	Love	SC	Havadjia	A	Taylor	JD
Bailey	G	Mackie	J	Hemsley	S	Tham	VL
Baxendell	S	Main	Cleve	Hill	BD	Townsend	W
Beers	P	Marshall	Jeff	Holz	P	Uzal	F
Begg	A	McOrist	S	Hum	S	Vanselow	BA
Boulton	JC	Miller	RI	Huxtable	CR	Watson	J
Campbell	Prof R	Mills	J	Janmaat	A	Whiteley	P
Campbell	G	Mitchell	G	Jardine	J	Whittington	R
Canfield	PJ	Moloney	B	Jerrett	I	Williams	OJ
Carlisle	M	Munday	BL	Jones	B	Woodgate	RG
Carrigan	M	Norton	J	Kelly	R		
Charles	JA	Obendorf	DL				
Chick	B	O'Hara	AJ				
Clark	P	Parsons	J				
Coleman	G	Pass	DA				
Connolly	JH	Philbey	AW				
Cook	R	Phillips	PH				
Copland	MD	Pierce	RJ				
Creeper	J	Rahaley	RS				
Donnelly	TM	Reddacliff	LA				
Doughty	FR	Reppas	GP				
Drew	C	Reuter	RE				
Ellis	TM	Richards	RB				
Finnie	JW	Riffkin	GG				
Forshaw	D	Robinson	W				
France	MP	Rose	KA				
Fraser	G	Ross	AD				
Friend	SS	Rothwell	J				
Genovese	LM	Rozmanec	M				
Gibson	JA	Scott	PC				
Gill	PA	Seaman	J				
Glastonbury	JR	Seward	D				
Gogolewski	RP	Slocombe	J				
Gordon	AN	Slocombe	RF				
Hampson	DJ	Storie	JF				
Harper	P	Straube	E				
Harrigan	KE	Sullivan	N				
Hartley Summers		BA					

SUBMISSION TO AAHC GROUP MADE ON BEHALF OF THE AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

Introduction

The Australian Society for Veterinary Pathology (ASVP) sees itself as having an important contribution to make to any debate on, or investigation of the Nation's animal disease diagnostic resources and their state of preparedness or otherwise. The Society thus welcomes the opportunity to make a submission to the present review instigated by the AAHC, believing itself able to assist the review in an objective and informed manner. As will be apparent in what follows, the Society perceives this review to be timely. It views with increasing concern the diversity of effects wrought by the rapid pace of political, economic and technological change over recent years, and their implications for supplying and maintaining a necessary body of expertise in animal disease diagnosis and monitoring. The aim of the Society is to be part of a process of constructive and soundly reasoned adaptation to new challenges, and it offers its collaboration and co-operation to this end.

Background

The Society

The ASVP is the sole national professional body representing practitioners of veterinary pathology and closely related diagnostic specialties. Its current financial membership stands at 150 but it should be noted that not all the members of the Society are necessarily working full-time. The members of the Society come from the State diagnostic laboratory systems, the five Australian university veterinary institutions and various private sector diagnostic laboratories. It is an incorporated Society administered by an honorary Executive which is appointed usually for a three-year term. It serves its membership through regular newsletters, an annual scientific meeting, a monthly diagnostic case circulation and an email list. In addition it sponsors a national registry of domestic animal pathology and annual continuing education events across the country.

Although the focus of the Society is upon veterinary anatomic pathology (defined below), eligibility for membership is open to anyone who has an interest in "veterinary pathology" in its broadest context. This includes such specialists as clinical pathologists, microbiologists, parasitologists, immunologists, toxicologists and so on. Many of the current members represent these disciplines.

The disciplines

Modern veterinary anatomic pathology is that field of diagnosis founded upon the interpretation of animal disease in the context of gross, microscopic and ultrastructural morphology. As such it is one of the direct descendants of the discipline of pathology created in the 19th century by Rudolph Virchow. The founding principles enunciated by Virchow continue to be enhanced by the grafting-on of new knowledge and technologies and the base must include some consideration of immunology and the new fields of molecular and cell biology and molecular genetics.

The discipline with which anatomic pathology has perhaps the most extensive interface is clinical pathology, the emphasis of which is on the laboratory manifestations of disease. Obviously in techniques such as cytology and histopathology there is an overlapping boundary between the two.

The disciplines which focus on the cause of disease, such as bacteriology and virology are clearly intimately linked to anatomic pathology, and anatomic pathologists need a sound general familiarity with them.

The important thing is for these various specialities to recognise their relationships and interact accordingly. The ASVP represents a vehicle for such interaction.

The basis of specialization in anatomic pathology

It might be asked "what is the manifestation of real expertise in an anatomic pathologist?" The answer could be "not only broad capability in the diagnosis of classic diseases but also the experience and ability to define processes, recognise and ignore the insignificant, interpret the atypical and unusual, and follow up with the appropriate action."

As pointed out above, this area of expertise overlaps with several allied areas. If one were asked to identify the core expertise which sets anatomic pathology apart and for which the specialist would be sought out for consultation by someone from an allied speciality, one would probably nominate skills in the analysis of tissue and cell architecture in disease.

To become a credible specialist in veterinary anatomic pathology, an individual must first gain a registerable degree in veterinary science/medicine. This must be followed by a prescribed period of intensive training of several years duration. During this training the individual must acquire a deep understanding of basic disease mechanisms, and the ability to recognise and interpret them. This in turn necessitates an in-depth knowledge of the gross, microscopic and sub-cellular anatomy of the cells, tissues and organs of the common domestic mammalian and avian species, and a wide knowledge of their major specific diseases and the agents which cause them.

As an adjunct to their training, anatomic pathologists must also retain a general familiarity with the clinical and laboratory manifestations of disease.

This is no mean task, and with the increasing exploitation of, and interest in wildlife and exotic species there is parallel pressure for sub-specialisation into areas such as avian, aquatic, zoo and wildlife fields. In addition, as in human pathology, sub-specialisation into the different organ systems, such as the skin or nervous system, is emerging. However, despite the foregoing, the dominant emphasis will remain on the familiar production, performance and companion animal species traditional in our culture.

A veterinary anatomic pathologist can thus be defined as:

"an individual with a basic veterinary degree and a distinct area of specialist expertise gained by intensive prescribed post graduate training in the recognition and interpretation of animal disease at the level of cells, tissues and organs."

The anatomic pathologist plays a crucial role in the diagnostic process – modern anatomic pathology remains at the forefront in diagnosis and research, and will continue to do so.

This is not to diminish the importance of the specialists in the allied fields, and the society recognises this by welcoming and seeking their membership.

Training

The traditional international benchmark specialist qualification in veterinary anatomic pathology is that offered by the American College of Veterinary Pathologists (ACVP). Candidates prepare for examination in residence programs in pathology provided at various major North American universities, usually over two years. At institutions such as the New York State College of Veterinary Medicine at Cornell University, eight to ten residents are supported each year on stipends provided by the Pathology Department. However, such stipends are not available at all places of training and can be expected to become more restricted in availability. Residencies are open to non-US citizens, some of whom receive support from their home countries.

At the end of the training period the candidates take examinations set by the ACVP and run at a specified location (Ames, Iowa). Successful candidates are awarded the status of "Diplomate of the ACVP". During the 1970's and early 1980's, numbers of Australians gained this qualification often being supported by State Governments during their training. This kind of government support is no longer available, and to the best of the Society's knowledge, only one Australian candidate has been sponsored by the private sector during the last decade.

A similar program modeled on the ACVP has been launched in the EEC. Formerly, citizens of the EEC routinely went to the US for their training.

In Australia, a two-tiered training format is offered through the Australian College of Veterinary Scientists (ACVSc) via its Pathobiology Chapter. Candidates may gain a preliminary Membership qualification (MACVSc) for which they are given recognition as "having superior knowledge", but require the Fellowship qualification (FACVSc) in order to be able to register as specialists. Examination for Fellowship requires the holding of a Membership, which in turn requires a veterinary degree awarded at least three years previously.

The training programs must take place at an approved institution and be supervised by an approved person(s). In recent years, training programs have been provided by the universities, some state laboratories and some private sector laboratories.

The FACVSc is not currently recognised overseas. The issue of reciprocal international recognition is a difficult one and will no doubt be coming under examination as the EEC builds its specialist system. To date it has been routine that US qualifications are accepted universally, but there is likely to be pressure on this should the US continue to accept equivalent foreign qualifications.

The ACVSc sponsors similar training programs in certain of the allied disciplines, such as clinical pathology.

A National Perspective on Veterinary Anatomic Pathology

It can reasonably be said that the state of veterinary anatomic pathology in Australia in 1988 is not robust for the following reasons.

First, the demographics show an aging professional population many of whom will make their exit during the next decade. Given that there are a few young people entering this population, a crisis of supply can be predicted. This situation will be exacerbated by its impact both on the maintenance of expertise in the "frontline" and on the number of registered specialists available to supervise training.

Second, the opportunities for training to specialist level are limited and diminishing. University veterinary pathology departments have traditionally been centres for discipline training but in all five universities with such departments, attrition of resources and personnel have become acute within the general budgetary strictures imposed on the tertiary sector. Veterinary Schools general budgetary strictures imposed on the tertiary sector. Veterinary Schools are small but very expensive units within universities and are also under great intra-institutional budgetary pressure.

The dismantling of state veterinary diagnostic structures across the country has removed most of the ability of the surviving elements to provide a training environment and as previously stated, support for candidates for overseas training has evaporated completely.

Private sector veterinary laboratories are a relatively new and expanding addition to the scene in Australia. The bulk of service is presently provided by five companies but one of these has recently been taken over by a major US operator (introducing "transnationalism" as yet another dimension). One of the companies

has supported one individual for the ACVP qualification, and provided training to MACVSc level for one candidate in 1998. This type of support may continue but to an unknown extent. However, it is doubtful if the local private sector could provide programs for FACVSc training which would be approved by the ACVSc. The attitude of transnational operators to training in Australia in unknown.

Third, while private sector employers give due recognition and reward to individuals with specialist qualifications this has never been the case in the public sector. There remains little incentive or support for any individual to pursue specialist training other than personal pride and professional satisfaction.

STATE REPRESENTATIVES

Queensland	Jim Taylor Toowoomba Vet Lab, QDPI PO Box 102 TOOWOOMBA QLD 4350	 07 4688 1351
Victoria	Malcolm Lancaster VIAS 475 Mickleham Road ATTWOOD VIC 3049	 03 9217 4200
South Australia	Ruth Reuter VPS, PO Box 445 GLENSIDE SA 5065	 08 8372 3700
New South Wales	Paul Gill, RVL WOLLONGBAR NSW 2480	 02 6626 1261
Northern Territory	Anton Janmaat PO Box 990 DARWIN NT 0801`	 08 8999 2240
Tasmania	Roy Mason, TAWQDS PO Box 46 KINGS MEADOWS TAS 7249	 03 6336 5216