THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
the Department of Primary Industry, Tasmania,
Mt. Pleasant Laboratories,
P.O.Box 46, Kings Meadows, TAS. 7249.

Registered by Australia Post Publication No. VBG 6333

EDITOR: David Obendorf

Number 27 March 1990

PAGE CONTENTS

DEADLINE FOR NEXT VET. PATH. REPORT IS June 1

PRESIDENT'S PAGE

PRESIDENT'S REPORT FOR 1989-90
AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

It is fitting that the 1990 ASVP Scientific Meeting should be held in Tasmania. Tasmania has been successful in establishing cold water fish farming. Veterinary pathologists have played a key role in defining fish diseases and enabling the fledgeling industry to survive. In this respect, I would recognise Dr Barry Munday who has pioneered fish disease diagnosis in Australia.

We are fortunate also to have two other specialists fish pathologists with us at this meeting in Dr Jeremy Langdon, who the ASVP has bought over from Perth and Dr Judith Handlinger, our revered treasurer.

A very successful Scientific Meeting on the Pathology of Exotic Diseases was held at the Australian Animal Health Laboratory at Geelong in May. The meeting was part of the farewell functions to mark the retirement of Dr Bill Snowdon. A number of members stayed on to participate in the inaugural exotic disease training course at Geelong.

Following the Scientific Meeting in May, the Executive was transferred from South Australia to Mt Pleasant Laboratories, Launceston. A huge debt of gratitude is due to Peter Phillips and his team for running the Executive from 1987-1989. The present executive is running smoothly under the energetic secretaryship of Roy Mason. One of the most valuable functions the ASVP performs is the publication of the quarterly Veterinary Pathology Report. The journal is a very valuable informal contribution to knowledge of animal diseases in Australia. Editor, David Obendorf has produced a very well presented magazine. I know of no other source which can document animal disease occurrences in Australia so well.

On a bright note, the Animal Health Committee agreed to provide reading for the National Registry of Domestic Animal Pathology for two years from 1 January 1990. This will go a long way to ensuring the future of Registry. One way members can guarantee the existence of the Registry is to see that each state's laboratory service sets aside a budget allocation for the Registry.

One of the earliest tasks the present Executive undertook was to develop an opinion on accreditation of veterinary pathologists and veterinary pathology laboratories in Australia. Members were invited to comment on the opinion published in the most recent edition of the Veterinary Pathology Report. To date, only 3 responses have been received.

The ASVP looks forward to collaborating with the Pathobiology Chapter of the Australian College of Veterinary Scientists in developing a training program in Veterinary Pathology. The ASVP and the ACVSC can complement each other in fostering veterinary pathology.

On a final note, I would point out that the organisation of this year's scientific meeting has faced major difficulties with the pilot's strike and the uncertainty of air travel to Tasmania. May I congratulate Roy Mason for organising the meeting and welcome our delegates from interstate who are present today.

R Oliver

SECRETARY'S REPORT

ASVP LOGO

Most organisations today have an identifying logo. The Australian Veterinary Poultry Association has a chicken in a pecking position such that its outline represents the shape of Australia with Tasmania as the beak.

Peter Phillips has put forward the following idea for a logo. What do you think? Have you an idea of your own?

Put your creative talent to work. The entries will be displayed at the next ASVP AGM and a choice considered from these for adoption as the ASVP logo.

All entries must include Tasmania. No Queensland Commonwealth Games opening omissions. Motto's also welcome.

Roy Mason, Hon. Secretary

UNABLE TO ADD ASVP PROPOSED LOGO.

TREASURERS REPORT 1989 (MAY - DEC)

This report, like the financial year, is short. Due to the alteration of the society's financial year from commencement in May to coincide with the calendar year, it has not been a full year since the last Annual Report, nor has the society been through a full subscription year. Many members have not paid their part year subscriptions. Fixed deposits have not matured and interest has not yet been credited. For these reasons the income and expenditure does not accurately reflect the society's annual finances.

The balance sheet for this shortened year is attached, together with a financial analysis of the 1989 annual conference.

Expenses this year have been higher for postage and for assistance with printing the Vet Pathology Report, but these should be more than offset by the higher subscriptions from this year and charging for position advertisements.

Incidental postage (postage other than the Vet Pathology Report) has been higher due to the short time between takeover by the new executive and the organization of the annual conference, compounded by the pilot's strike which resulted in uncertainty and understandable hesitancy by members about commitment to travel under these circumstances. It is not anticipated that this situation (even without the pilot's strike), will recur frequently as the Annual Conference and Annual General Meeting is generally still likely to coincide with conferences later in the year, whereas the frequency of coincident conferences of veterinary pathology interest in Northern Tasmania is low.

Judith Handlinger (Hon. Treasurer)

STATEMENT OF INCOME AND EXPENDITURE OF ASVP 1/5/1989 TO 31/12/89

INCOME		
Opening balance		6,759.71
Membership subscriptions		638.63
1989 conference - registrations (net)	1,400.00	
- accom + meals etc(net)		822.50
1990 conference - registrations to date	140.00	
- accom + meals		272.00
Interest		354.00
		10,386.85
EXPENDITURE		
Corporate affairs Office		43.10
1989 Conference inc. accom + catering	3,603.00	
Postage for 1989 Conference Proceedings		210.50
Fed. taxes, stamp duty		6.93
Vet Path Report - stationary and printing		623.34
- postage	177.50	
- registration		21.50
Incidental Postage	143.36	
Bank cheques		2.75
		4.841.98
Closing balance		5,544.87
		10,386.85

1989 CONFERENCE

Income reported in 1988 report	- registrations		880.00
	- other		692.50
Income 1989 report	- registrations (net)	1,400.00	
	- accom + meals		822.50
Expenses reported in 1988 report			
	- deposit Deakin Uni		200.00
	- proceedings stationary		277.00
Deakin Uni (Accom + breakfasts)			1,203.00
CSIRO AAHL social club (catering)		2,400.00	
Postage of Conference Proceedings			210.50
			4,290.50
Loss on conference		495.50	

Number of registrants - 57

EDITORS REPORT

This autumn's issue of VPR is larger than normal. It includes some responses from members about accreditation of veterinary pathologists and vet. path, laboratories. Surprisingly, we didn't receive a lot of correspondence on this topic.

Thanks to Jim Rothwell and David Williams for providing state reports for NSW and Victoria respectively over the last few years. Tony Ross and Grant Rawlin have agreed take over as state reps.

Minutes of the Seventh ASVP AGM meeting
Held at the Tasmanian State Institute of Technology
28 March 1990.

PRESENT

Roger Kelly	Dick Sutton	Belinda Timmins	Neill Sullivan
Michael Pearce	Wayne Robinson	Bill Hartley	Barry Munday
Adrian Philbey	Tony Ross	Deborah Seward	David Obendorf
Rodney Oliver	Judith Handlinger	Roy Mason	Jeremy Langden

(Note this attendance did not constitute a quorum)

NEW MEMBERS
Grant Rawlin
John Glazebrook

OPENING
APOLOGIES

David Williams	Bob Jones	Keith Walker	Phil Ladds
Bill De Saram	Denise McEwan	John Humphries	Susan Friend
John Glastonbury	Ian Links	John Searson	John Seaman
Jenny Charles			

MINUTES OF THE 1989 AGM

Minutes of the sixth AGM held at AAHL, Geelong on Saturday 13 May 1989 circulated to attendees. Accepted as a true record Tony Ross/Adrian Philbey.
Correspondence/Fax

ASVP Logo Peter Phillips
Accreditation of Veterinary Pathologists and Laboratories R W Gee
Reply to above presented in October 1989 issue of ASVP
Pathology Report
Accreditation of Veterinary Pathologists and/or Veterinary
Pathology Laboratories Susan Friend

Moved that correspondence be received Bill Hartley/Jeremy Langford CARRIED

Presidents Report

Report tabled for discussion - none
Moved that the report be accepted Roger Kelly/Tony Ross CARRIED
Report to be published in ASVP Pathology Report.

Treasurers Report

Report tabled for discussion(see page 2)

It will be necessary to explain to ASVP members because of the change in the financial year to a calendar year as of 1 January 1990 members owe $12.50 for the period May to December 1989 inclusive and $20.00 for the period 1 January to December 1990.

Membership report

Note most members unfinancial for the period May to December 1989.
Australian membership (December 1989).

NSW 42	Victoria 39	Queensland 23
Western Aust. 11	South Aust 18	Nth Territory 6
Tasmania 5		

Overseas Membership December 1989.

New Zealand 5	USA 15	UK 1
Malaysia 2	Indonesia 2	PNG 2
France 1	Fiji 1	Canada 2
Switzerland 1		

Registry Report

The president acknowledged the work undertaken by Tony Ross and the rest of the committee in obtaining commitment for funding of the registry for 2 years. W. Hartley added that while there were now over 2000 cases in the domestic animal registry and about 4000 on native animals, the registries were still very open for sample submissions. W. Hartley also raised the need for discussion of the long term staffing of the registry.

Veterinary Pathology Report

The VPR continues to be well supported by the membership, mainly through the state representatives. The format has continued to reflect the interests of the membership in case reports, however, the editor is encouraging contributions on other issues which may be of interest to readers.

The Report is now being printed by a commercial printer in Launceston and the finished article (including the cost of envelopes, stickers and bulk postage) is approximately $2.30 per copy.

Election of Executive Office Bearers

Current executive office bearers retain their present portfolios.
Moved Bill Hartley/Dick Sutton CARRIED

State representatives

Victoria:	Grant Rawlin to replace David Williams
NSW:	Tony Ross offered to replace Jim Rothwell

Other state representatives remain the same - namely

Queensland:	Fraser Trueman
West Aust:	Ruth Reuter
Nth Territory:	Lorna Melville
Sth Aust:	Vui Ling Tham
Tasmania:	Judith Handlinger

General Business

Logo - Idea of a logo to be aired in the Veterinary Pathology Report. Designs, including past submissions, to be submitted and judged at the next AGM.

1991 AGM Venue and Theme - Strong feeling that a meeting in Sydney in May 1991 held in association with the AVA AGM, would be desirable. A possible venue to explore is the Elizabeth MacArthur Centre.

A good theme common to our day to day work rather than specific theme, was considered desirable, though a single session on a more specialised area, such as pathology of endangered species as suggested by R. Oliver, could also be incorporated if the opportunity arose, and that this might allow utilisation of overseas speakers present for AVA etc..

Possibilities - pick up on a theme run in the AVA AGM, an organ system, detection of aetiological agents etc. Suggest NSW and Pathobiology chapter to liaise on a theme or themes.

Bill Hartley suggested a registry session to follow the ASVP meeting.

1991 ASVF executive
Tony Ross and Bill Hartley to raise this with ASVP members at Glenfield Elizabeth McArthur Centre.

Pathobiology - ASVP Pathology Training Programme.
Meeting addressed by Wayne Robinson. A training programme could:

- Provide broad training in gross and microscopic pathology of farm animals, companion animals, microbiology, serology and chemistry
- be undertaken as an external course (e.g. External Course associated with a co-operating University)
- Provide flexible option of 2-4 years to complete
- Possibly combine external degree with ACVS fellowship examination
- Provide uniform standard for recruits to Veterinary Pathology to facilitate registration by state veterinary boards.

The veterinary Pathology training committee (Keith Walker/Tony Ross) to follow up on behalf of ASVP.

Tony Ross reported that responses to their questionnaire of members recorded an overwhelming yes to the need for a training program for new pathologists provided that it is relevant to registration.

Concern was expressed at the disparity between states in the application of the Grandfather Clause to Registration of Veterinary Pathologists as Specialists. Both the questionnaire and the meeting agreed standards between states should be uniform. There was also concern that current experienced pathologists should not have to start from scratch and undergo a full training program to prove their proficiency.

The State Veterinary Surgeons Boards are meeting in May. The Executive is to seek information on whether registration of Veterinary Pathologists is on their agenda; what is the thinking of the Boards on this issue and of a training programme for recruit veterinary pathologists to provide standardisation for registration purposes; and whether the ASVP could and should be represented at this meeting. This was considered desirable so that training programs under discussion are not pre-empted, and especially as ASVP agrees that uniform standards be developed.

The Executive is to prepare a working document on accreditation of Veterinary Pathologists and Registration of Veterinary Pathology Laboratories for discussion through the Vet. Pathology Report, discussion with the Veterinary Surgeons' Boards if appropriate, and presentation to the next Annual General Meeting.

Registration of Veterinary Pathology laboratories:

Contentious issue, some comments made include;

- Diagnosis and data interpretation should be made by qualified veterinarians
- Courier services of one sort or another put veterinary practices within reach of veterinary laboratories
- There is no adequate quality control in place of laboratory animals if in medical laboratories
- A co-operative rapport has developed in many locations between the medical and veterinary professions. This relationship could be jeopardised.
- Effective marketing of services together with interpretive veterinary laboratory.
- Medical clinical laboratories and many private veterinary laboratories do not have Post Mortem examination facilities.
- Medical laboratories and private veterinary pathology laboratories do not have ready access to expertise in areas of animal husbandry/management, plant identification, soil etc that is available to Dept of Agriculture Veterinary Diagnostic Laboratories.
- Veterinarians may wish to avail themselves of sophisticated procedures that can be undertaken by medical/hospital laboratories (certain microbiological toxicology, hormonal etc) but for which veterinary laboratories are not technically or physically equipped to undertake.
- The correct sequence should have been for the training program for veterinary pathologists to have been instituted first, the Registration as Veterinary Specialists second and the Registration of Laboratories to follow that, with the laboratory registration criteria carefully considered so as to not unduly favour one type of laboratory (e.g. clinical pathology laboratories) over other more comprehensive services. Given that this sequence has not occurred and the disparity between states in registration of Veterinary Pathologists as Specialists, there was a need for ASVP input. (Hence the Working Document)

Subscriptions overseas members - Judith Handlinger raised the question of overseas membership fees which were not apparently considered at the last Annual General Meeting when general subscriptions were raised. As the meeting did not constitute a quorum, this issue is to be raised at the next Annual General Meeting.

ACCREDITATION OF VETERINARY PATHOLOGISTS AND/OR VETERINARY PATHOLOGY LABORATORIES

A letter from Richard Miller, Geoff Mitchell, Chris Belford and Robert Rahaley.

We refer to the paper "Accreditation of Veterinary Pathologists and/or Veterinary Pathology Laboratories", published in the October issue of The Veterinary Pathology Report.

We are dismayed by the contents of this paper and the fact that it should be submitted to a State Veterinary Board from the Executive of a Society who's express aims are to foster the development of veterinary pathology in Australia.

For such an important paper to be submitted without membership evaluation is most regrettable. In this age of electronic media transfer, the excuse of "time did not permit circulation of paper" is bunkum. We all have a stake in these most important issues and it is the responsibility of the current ASVP Executive to ensure that the express views of the Society reflect the majority opinion of all member veterinarians working in pathology.

We hope to offer constructive criticism of the paper. All the issues raised have a significant impact on us as pathologists and they are issues we have already considered in some depth.

First, on the question of qualifications of veterinary pathologists:

It is unclear whether the ASVP Executive is attempting to set a new definition of "Veterinary Pathologist" in parallel with the ACVSc Advisory Committee on Specialist Registration or wants this published definition to replace the Advisory Committee. In either case, the self-protective, attitudes expressed in this paper could well retard the coming of age of veterinary pathology in the country for yet another decade.

Specifically, does the Executive really believe "at least 3 years practising veterinary pathology and demonstrated ability" is a satisfactory definition of a pathologist to submit to a Veterinary Board? What is "demonstrated ability"? The various State Board should be given firm guidelines from the ASVP as to what we consider is an appropriate measure of competence.

Does the Executive really believe that State Veterinary Boards (essentially composed of non-pathologists and, in some cases, non-veterinarians) are the best people to decide who is qualified to be accredited as a veterinary pathologist? This concept, and the suggestion that the definition of a pathologist should somehow vary from State to State depending on the "needs of the community", are ludicrous in the extreme. Does a veterinary pathologist in Victoria or NSW have to be better trained than one in Tasmania or the NT because there are more pathologists in Victoria and NSW? Should the communities in Tasmania and the NT expect less of their specialist professionals because there are fewer of them?

We agree MEMBERSHIP of the College should not be "the only acceptable qualification" since by definition (70% of undergraduate level); it's not really much of a qualification at all, merely a stepping-stone to FELLOWSHIP.

For Veterinary Pathologists in Australia to attain a professional standing and remuneration level commensurate with their training and skills, there must be a credible accreditation system whereby individuals can irrefutably demonstrate that they have achieved an .appropriate level of knowledge and competence.

We submit that the ONLY people qualified to judge who has attained competence in veterinary pathology are other veterinary pathologists. There should be only ONE criterion for registration as a specialist veterinary pathologist and that is a suitable qualification obtained by EXAMINATION. The examination should be set at an appropriate level of difficulty, cover all animal species, and be administered by qualified veterinary pathologists. In Australia, this means FELLOWSHIP of the ACVSc or an equivalent overseas qualification.

For a credible specialist registration system, there can be no exceptions, no special cases, and no individual assessment. All veterinarians working in pathology should aim to sit the Fellowship examination. If they are not at that level then they should get into an appropriate training program.

There are two obvious advantages to a specialist registration system. From the pathologists' point of view, specialist registration should set a competence benchmark to enable salary negotiations with employer groups. This is most evident in the USA where salaries for ACVP accredited veterinary pathologists, relative to other veterinary graduates, are considerable higher than Australia.

The system should also ensure that when a veterinarian places the words "Veterinary Pathologist" after his or her name, the Australian community can be assured that the veterinarian has attained an appropriate level of knowledge and competence in the pathology of all animal species, regardless of where they live.

The paper prepared by the Executive of the ASVP does little to foster either of these advantages. Instead, the apparent thrust of the Executive's paper is to lower the standards for specialist registration or introduce some sort of geographically driven "sliding scale".

On the issue of facilities necessary for veterinary pathology and registration of facilities, it appears the Executive has taken the attitude that it's “too-hard” so we won't support it. There is no difference between the equipment needs of a private or government veterinary laboratory when one accepts the principle that no laboratory can, or should, perform every diagnostic test.

Standards for quality control and technology can be developed and we would support their introduction. Just as veterinary clinical premises should be registered, laboratories should meet minimum standards. One criterion we would deem essential is that veterinary pathology laboratories should be controlled by qualified veterinary pathologists! This issue is closely allied to the question of non-veterinary pathologists offering veterinary pathology services.

On the non-veterinary pathologist’s issues, the Executive again fails to adopt a stance, waffling ad nauseam about why veterinarians use one service as opposed to the other. In the final paragraph of the paper there is a hint of opinion!

The ASVP should be STRONGLY OPPOSED to non-veterinarians performing pathology tests on animal specimens. If we have only one unanimous opinion as a Society this must surely be it. This stance is so fundamentally in the interests of the ASVP members we are astounded at the lack of commitment in the Executive paper. Consider the likely reaction from our medical colleagues if veterinary laboratories offered human diagnostic services.

As we stated at the outset, the ASVP was formed to foster the development of veterinary pathology in Australia. We have come some distance towards gaining professional and community recognition of our expertise and services but we have a long way to go. The adoption of the specialist registration category “Pathobiology” is an example of how our clinical colleagues consider all “laboratory types” to be pretty much the same.

Better employment conditions for pathologists will only come if we set ourselves apart from other “laboratory types”. We must have a rigorous accreditation system and we must preserve our work area from non-pathologist encroachment. We would hope that the ASVP might share these ideals.

A letter from Sue Friend:

I would like to express my concern at some aspects of the statement promulgated by the executive of the Australian Society for Veterinary Pathology (ASVP) on accreditation of Veterinary Pathologists in the Veterinary Pathology Report #26, of October 1989.

I do not agree that the Veterinary Surgeons' Board of each state is best placed to decide if a person is qualified as a Veterinary Pathologist. Surely the Australian Society for Veterinary Pathology and the Australian College of Veterinary Scientists (ASVSc) Pathobiology chapter would be the appropriate bodies to make such a decision. The Board may be involved in the final decisions, but only after seeking advice from the ASVP and ACVSc as to whether a person is suitably qualified.

Certainly the essential qualifications should include a degree in Veterinary Science/Veterinary Medicine, recognised by the Veterinary Surgeons' Board and post graduate qualifications such as fellowship of the SCVSc in pathology or equivalent i.e. diplomate of the American College of Veterinary Pathologists. In my opinion, membership of the ACVSc in Pathobiology is not an adequate basis on which to be called a Veterinary Pathologist.

Section (ii) which states that a person should have demonstrated ability and at least 3 years practising Veterinary Pathology is not specific enough. There must be adequate supervision by appropriately trained individuals in suitably equipped training centres. There should be critical appraisal of the progress of the trainee and of the credentials of the training programme, personnel and centre. Who is going to do the training and who is going to critically evaluate the trainee and the programme? This is where a National Training Programme, under the auspices of the ACVSc, would be of great value.

STATE REPORTS

VICTORIA - Grant Rawlin

REGIONAL VETERINARY LABORATORY BAIRNSDALE

Apparent Clostridial Enteritis in a Foal

A two day old Thoroughbred foal presented with severe abdominal pain and haemorrhagic diarrhoea. A laparotomy was carried out and the foal died during surgery. A gross diagnosis of haemorrhagic enteritis was made by the surgeons. Histologically, there was a haemorrhagic, necrotising enteritis. Necrotic villi were covered by a layer of large, gram-positive bacilli morphologically consistent with Clostridium sp. No further deaths associated with enteritis have been reported on the stud in the current foaling season.

Clostridium perfringens Types A, B and C have been reported to cause haemorrhagic enteritis in foals. The first case involving Clostridium perfringens Type A was recently reported in Queensland (Dart et al 1988).

Reference: Dart AJ. Pascoe RR, Gibson JA and Harrower BJ (1988).
Australian Veterinary Journal 65: 330-331.

Bracken Fern Poisoning (Peter Mitchell)

Between August and September, bracken fern poisoning was diagnosed on ten properties in Gippsland, from Warragul to Bairnsdale. Up to five cattle died on some properties. Most animals exhibited typical signs of multiple haemorrhages on mucous membranes and epistaxis. One had small ulcers on the nose and in the mouth. Most were pyrexic. Haematology from these cattle typically showed low total white cell counts, consisting only of lymphocytes. No neutrophils were present and platelets were reduced. Most animals were also anaemic with haemoglobin levels down to 5.8 g/dl. One animal had a severe infection with Yersinia pseudotuberculosis. Post mortem examination of affected animals also showed haemorrhages through the body.

Post mortem examination of cattle from two other properties did not show any haemorrhages but large numbers of raised ulcers were present in the digestive tract, particularly in the pyloric part of the abomasum and the duodenum, and in the liver and lungs. Fungi were seen in sections of the ulcers and Aspergillus and Mucor spp were isolated from one. Fungal infections are associated with reduced immunity. The reduction in immunity at this time was attributed to bracken fern poisoning.

The toxic agent in bracken fern is thought to be a chemical known as ptaquiloside. This chemical has caused a reduction in neutrophils and platelets when administered to cattle. The reduction on platelets leads to increased capillary fragility and prolonged bleeding and hence the haemorrhages typical of bracken fern poisoning. The reduction in neutrophils results in a lowered immunity and hence secondary infections, sometimes with organisms that are not usually pathogenic.

The sudden appearance of many cases over a short period suggests a seasonal effect in the intake of bracken. Affected animals were in paddocks with mature bracken and with recently slashed bracken. At this time of the year, many young fronds are present. Cattle have been seen eating these fronds, but it is not known whether cattle actively select the fronds. The young fronds are reputed to be more toxic than mature fronds, but poisonings can occur even with old, dry fronds.

Exudative Diathesis-Like Disease in Ostrich Chicks

Three newly-hatched Ostrich chicks were submitted. Two were dead when assisted from their shells and one died within 24 hours of assisted hatching. Incubation period was 42 days. All 3 had severe generalised clear jelly-like subcutaneous oedema. No pathogenic bacteria were isolated from yolk sacs, livers or lungs. No gross muscle lesions were noted, however, patchy foci of unequivocal myonecrosis were evident in skeletal muscles. Many small blood vessels showed fibrinoid change or frank necrosis. Liver vitamins A and E and selenium assays are pending but results will be uninterpretable until we have access to results from normal chicks. These cases resemble those reported by RVL Wagga Wagga in the last Vet Path report No. 26, p. 23, October 1989.

REGIONAL VETERINARY LABORATORY HAMILTON

Deaths in Koalas (Cor Lenghaus)

We normally average about one koala annually, so it is rather unusual to report on four koalas received dead. They were all regarded as being in a thin, undernourished condition.

a) A young female with a freshly dead, unfurred young in its pouch had extensive skin lesions of the forepaws, face and belly. The affected skin was thickened and fissured over the joints, and there was considerable hair loss. Skin scrapings revealed large numbers of mites consistent with Sarcoptes scabiei. Histological lesions were characterised by marked hyperkeratosis and epidermitis, with only a mild to moderate, essentially mononuclear inflammatory cell response in the dermis. Mites were present in large numbers in the epidermal debris, and in burrows through the epidermis. Quite a number of them had internalised proteinaceous material tinctorally consistent with blood.

b) A juvenile male koala had a severe, chronic, granulomatous enteritis with ulceration through to the submucosa in some areas. Branching, septate, fungal hyphae consistent with Aspergillus sp were revealed in the granulomas using PAS stain. The limited inflammatory cell response which was present at the site of infection, suggested that this was an immuno-compromised animal.

c) A young female koala has marked bullous pulmonary emphysema at post mortem. Histologically there was marked hypertrophy of the smooth muscle media of many blood vessels in the lungs. Vessels in the liver were similarly affected and were also enveloped in a broad, dense, fibrous adventitia. Other tissues were unremarkable.

d) An aged male koala was admitted with severe, bilateral exophthalmos and associated keratoconjunctivitis. Superficially it resembled a form of the much reported Chlamydial-infection complex. Post mortem examination revealed soft, white fleshy masses extensively invading the periorbital tissues and temporal muscles. Similar tissue infiltrates were grossly obvious in cervical lymph nodes, in and around the spleen, pancreas, diaphragm, stomach, liver, mesentery and bladder, and in bone marrow of the femur. Histologically, neoplastic cells were considered to be of lymphocytic/lymphoblastic type. As well as the above sites, tumour cells were present in the myocardium, eyes, lungs, kidneys and the ventricles of the brain. Readers should be aware that there is considerable interest in establishing cell lines from various marsupials. Tumour cells are therefore a valuable resource. Dr Jenny Graves, Biology Dept. Latrobe University, Melbourne, would be very interested in liaising with anyone who has access to a terminally ill marsupial with neoplasia.

REGIONAL VETERINARY LABORATORY - BENDIGO

Amyloidosis in a Cat (R.T. Jones)

A 6-year-old male Abyssinian cat was presented to a veterinary clinic with a history of lethargy, polydipsia and vomiting. Clinically the cat was dehydrated, breathing heavily and had pale mucous membranes. A lastix test on the urine revealed increased bilirubin and a blood azo stix was negative for urea. Haematology and a biochemical profile were performed at RVL Bendigo.

RESULTS

Albumin	21	PVC	32
Albumin/Globulin	0.4	Haemoglobin	9
Amylase	755	RCC	6.6
		MCV	48.5
ALT	104	MCHC	28.1
Ap	7	MCH	13.6
AST	44	Plasma Protein	7.4
Total Bilirubin	31.7	WCC	7600
Conjugated Bilirubin	22.6	SN	50% (3800)
Creatinine	0.15	L	29% (2200)
Urea	14.1	M	6% (450)
Globulin	51	E	15% (1150)
Glucose	10.1		
Protein	72		

FTV Serology - negative
H.felis - negative

The cat was euthanased. Liver and pancreas were submitted in formalin.

Histopathology

Liver sinusoids were dilated and sinusoidal endothelium was separated from the hepatocytes by amorphous pale pink material which stained positive for amyloid with Congo Red and Thioflavin T and negative for collagen (van Giesen). The hepatocytes and biliary tract were not remarkable. The pancreas showed slight amyloid staining around the Islets of Langerhans. A diagnosis of hepatic amyloidosis was made. Has anyone else seen this condition in cats; could it be genetic in the Abyssinian cat?

Veterinary Staff - R.T. Jones

(a) There are five veterinarians employed at RVL Bendigo: Bob Jones (Director), Tony Fohy (Research), Rod Badman (Diagnostic Pathology), Frank Allison (Diagnostic Pathology) and Les Sims (Diagnostic Pathology).

(b) Les Sims is on secondment to the Papuan New Guinea Government for 3 years until March 1990.

(c) In Les' absence, Jonathan Lee was employed in a temporary position. Jonathan resigned on 30 August 1989 and is currently working at the Arid Zone Research Station in Alice Springs.

(d) From 30 October 1989 until Les returns in March 1990, Peter Carbonell will be working on Mondays and Wednesdays.

VETERINARY RESEARCH INSTITUTE, PARKVILLE

Swine Dysentery and Bordetella bronchoseptica pneumonia in weaner pigs (Mike Forsyth)

An 80 sow piggery was suffering 9% mortality in weaner piglets. The piglets were being weaned at 4 weeks and were usually in excellent health. By the time they reached 6-7 weeks of age, there had been deaths, scouring and the surviving piglets appeared to be in poor condition. A killed E. coli vaccine (Ausvac) was being used on the sows and suckers at 3 weeks of age. The pens were overstocked but hygiene standards appeared adequate. Lincospectin had been used for 8 months in the weaner ratio.

Three 6 week old piglets were presented for necropsy. The gross findings were poor body condition in all three. One piglet had chronic adhesions of the right lung (lung 1) to the parietal pleura. The second piglet had sub-acute consolidation of the anterior and ventral portions of both lungs (lung 2). The third piglet had a diphtheritic colitis of the apex of the colon.

Bacteriological examination of the piglets revealed that 2 out of the 3 lungs contained moderate, almost pure growths of Bortetella bronchoseptica on routine culture under aerobic conditions. The sensitivity pattern showed sensitivity to ampicillin, erythromycin, neomycin and tetracycline and resistance to penicillin G, streptomycin and sulphatrimethoprim.

Histopathological examination of lung 1 showed thickening of the pleura and fibrous tags, relatively normal lung architecture but with nodules of mononuclear cells associated with small bronchi and respiratory bronchioles. Lung 2 showed sub-acute pneumonia with areas of abscessation and associated fibrous proliferation and sub-acute pleuritis.

Examination of the colon of piglet 3 showed a necrotic and diphtheritic colitis in which Treponema-like organisms appeared adjacent to necrotic tissues on staining by Warthin-Starry method.

The opinion was given that the Swine Dysentery was the primary cause of the debility and the Brodetella bronchoseptica was of secondary importance. Incidentally, there did not appear to be an atrophic rhinitis problem in this herd.

NEW SOUTH WALES - Tony Ross

Palyam Virus Infection
(PAW Harper, P Kirkland)
Regional Veterinary Laboratories, Glenfield

A neonatal Friesian calf from a herd in the Hunter Valley was examined, in which four of ten calves born recently displayed posterior ataxia from birth. Severe hydranencephaly with cerebellar hypoplasia was observed, in addition to pulmonary artery haemorrhage. Serological examination displayed a strong antibody reaction to Palyam virus in sera from the foetus, however, no antibody to Akabane virus or pestivirus was demonstrated.

The serological results, the presence of CNS pathology, and possibly the vascular pathology, suggests that Palyam virus may have contributed to this calf's disease.

In 1989, there was extensive seroconversion to Palyam viruses; first observed on the North Coast of NSW with a high incidence in Hunter Valley and coastal herds north of Nowra. There have been occasional reports of disease associated with Palyam virus infection with 7 confirmed cases, 4 of which were cases of cerebellar hypoplasia. Encephalitis and an aborted mummified foetus were also associated with the virus infection. A further 6 cases of in-utero Palyam infection have been identified following retrospective testing of stored sera from cases of undiagnosed congenital abnormalities, characterised mainly by cerebellar hypoplasia (1977, 1983, 1984, 1987). These findings suggest that Palyam virus infection should be considered as a cause of bovine congenital deformity.

WOOLONGBAR RVL

CUMULATIVE COPPER POISONING IN CALVES (P.A. Gill and R.W.Cook)

Cumulative copper poisoning caused death of 20 out of 100 calves aged 8 to 16 weeks old over an eight week period in a calf rearing unit. Affected animals were markedly icteric (bilirubin to 254, GGT to 109), illthrift, ataxic and terminally were depressed and appeared blind. Anaemia was not a prominent feature in affected animals, although haemoglobinuria occurred consistently. Histopathological changes were subacute to chronic hepatopathy with marked portal fibrosis and biliary hyperplasia.

Reconstituted whey, supplemented with safflower oil was fed to these calves, with some cracked corn. A vitamin/mineral premix was formulated to give a final concentration in the whey of 30 mg Cu/lOL. However, more premix was added to the whey giving a final concentration of 50 mg Cu/lOL. The majority of calves affected were among those fed this high concentration of copper immediately they entered the unit during the first week of life. Losses started after 8 weeks on this feed. Calves consumed 7-14 L of reconstituted whey per day.

REGIONAL VETERINARY LABORATORY WAGGA WAGGA (John Glastonbury)

Since 3 September 1989 it has been our pleasure to have Parkash Dewani, a Pakistani Veterinarian, obtain experience in pathology at our laboratory. Parkash's visit was funded by the Food & Agricultural Organisation of the United Nations and he is one of 5 Veterinarians presently obtaining training and experience throughout the world. Upon his return to Pakistan, Parkash will be in charge of a veterinary diagnostic laboratory at Tando Jam in Sindh Province of Pakistan.

Since 1 September 1989 NSW Agriculture & Fisheries has introduced charging for testing which is deemed to be of private benefit to that particular owner. We are quietly pleased with the initial success of this venture and our submission rate has actually increased since the introduction of charges. During the period since charges were introduced we have performed 62 faecal egg count reduction trials and 35 virulence determinations on isolates of Bacteroides nodosus.

CATTLE

Rejection of Export Livers

A number of livers were submitted from a local abattoir following condemnation of the previous batch for black discoloration upon thawing in the United States of America. After thawing the submitted livers were found to have variable sized foci of brown to black discoloration, up to 4cm in diameter, in their capsules. The lesions did not appear to be due to haemorrhage, melanosis, telangiectasis, inflammation or necrosis. They may have been due to mild compression of hepatocytes beneath the capsule following packaging for freezing.

SHEEP

Eperythrozoonosis

During the spring period we diagnosed 10 outbreaks of Eperythrozoonosis mostly in Merino sheep of 3 to 6 months of age. The morbidity and case fatality rates in the most severe outbreak were 11.9 and 68.4%, respectively. In most instances the sheep had been mulesed in the previous 6 weeks and clinical signs included anaemia, jaundice and death. The diagnoses were confirmed by the detection of Eperythrozoon ovis organisms in Giemsa stained smears of peripheral blood and/or the histological observations of hepatic and splenic haemosiderosis, periacinar hepatocyte necrosis and splenic follicular hyperplasia.

Chronic Copper Poisoning

Following from our previous contribution for "Veterinary Pathology Report" a further 10 outbreaks of this condition were diagnosed during spring. For the 10 farms the overall mortality rate was 5.0% of 4670 sheep. The hepatogenous form of the disease associated with evidence of the ingestion of pyrrolizidine alkaloids was diagnosed on 9 farms while on the remaining farm the syndrome seemed to be phytogenous in origin. Histological examination revealed severe degenerative hepatopathy and marked evidence of a haemolytic crisis.

Sarcocystis Myelitis

An unusual case of ataxia in the pelvic limbs of a Poll Dorset ram lamb was investigated. Multifocal granulomatous encephalomyelitis associated with characteristic cysts of Sarcocystis spp was observed microscopically.

HORSES

Strangles

The isolation of heavy pure growth of Streptococcus equi from submandibular abscesses confirmed this diagnosis in 3 animals from different farms.

BIRDS

Tuberculosis

Following the detection of tuberculosis in pigs at an abattoir, trace back to the farm of origin found the disease in 3-year-old White Leghorns. Pathologically there was multifocal granulomatous hepatitis and splenitis and Ziehl Neelsen stained sections showed numerous organisms with a morphology consistent with Mycobacterium avium.

Glenfield

Veterinary Laboratories staff at Glenfield are spring cleaning in autumn prior to moving to their new $34 million laboratory complex at Menangle in March.

Jim Rothwell has been granted three years study leave for a Ph.D. at Sydney University commencing in 1990. This has created a vacancy for a pathologist in the Regional Veterinary Laboratory. Other structural changes are likely to produce a second vacancy from March 1990. Anyone interested in working in new laboratories in the countryside out-side Sydney should contact Peter Harper Officer-in-Charge (02 6051511).

QUEENSLAND - Fraser Trueman

ANIMAL RESEARCH INSTITUTE YEERONGPILLY

Dr Bruce Hill has returned from Ph.D studies at Edinburgh, and has been appointed Officer-in-Charge at the Rockhampton Veterinary Laboratory. His Ph.D involved a study of cryptosporidiosis in lambs.

TOXOPLASMA ABORTIONS IN SHEEP AND GOATS (G. Storie)

More than 12 lambs were stillborn or aborted in a small mixed breed flock. Two full tern foetuses with their respective foetal membranes were examined at the laboratory. Cotyledons of both foetuses were swollen and contained numerous white foci. Both foetuses had blood stained subcutaneous fluid, a large volume of blood stained thoracic fluid and congested non-inflated lungs. On histology large areas of coagulation necrosis of villi were present in cotyledons of both placentas and suspect Toxoplasma cysts were seen in both instances in very low numbers. No significant changes were seen in foetal tissues which included the brain of one foetus only since the other was absent due to predation. Serum samples were collected from 10 ewes and a kit indirect haemagglutination test for antibodies to Toxoplasma gondii gave the following results:

4 x <64, 2 x 64, I x 512, 1 x 1024, 1 x 2048, 1 x 8192

AGID test for bluetongue virus on the same sera gave negative results. Numerous cats were present on a neighbouring property and were considered to be the source of the infection.

A Cashmere doe from a group of 300 aborted a 4 month old foetus. Histological examination of foetal tissues revealed occasional foci of necrosis with associated gliosis and mineralisation in the brain, and multiple foci of necrosis and mineralisation with a very mild inflammatory response in the placenta. Kidney, liver, heart and lung were normal. These changes were considered consistent with a protozoal aetiology.

Over the next 3 weeks another 5 does aborted and sera were submitted for a Toxoplasma Indirect Haemagglutination Test. Titres obtained from these five does were 1/64, 1/128, 2 x 1/512 and 1/1024.

The shed which housed the animals feed was infected with rodents and several cats had taken up residence as a consequence.

The absence of lesions in tissues other than placenta and brain demonstrates the need for these tissues to be routinely submitted in cases of ovine and caprine abortion. The tissue prevalence of lesions in cases of ovine Toxoplasma abortion has been reported to be:

Placenta (63%); brain (27%); heart (25%); lung (15%) and liver (8%).

CARDIOMYOPATHY AND WOOLLY HAIRCOAT IN POLL HEREFORDS (G. Storie}

Five of twenty calves were reported dead. Calves were running in open forest and all appeared in fairly good condition prior to death. The owner reported that the five dead calves had been born with tightly curled woolly hair coats and had been sired by the sane newly introduced bull.

Specimens from the last calf to die were submitted to the laboratory. Fixed heart muscle could be seen to contain extensive areas of white streaking. Histologically there was severe chronic multifocal to coalescing interstitial fibrosis with widespread effacement of myocytes and focal mineralisation. The liver showed severe chronic venous congestion with both periacinar and portal fibrosis and atrophy of hepatic chords.

Previous surveys of GSH-Px levels on this property suggested adequate selenium nutrition. Also such extensive fibrosis not typical of white muscle disease in which damage is primarily to the myocyte leaving the basal lamina and satellite cells intact, thereby allowing almost complete regeneration in animals that survive.

Cardiomyopathy associated with a curly hair coat is seen only in Poll Herefords. All calves with this phenotype die before reaching 6 months of age (range 1 to 180 days). A simple autosomal node of inheritance has been suggested. The close association of cardiomyopathy and curly hair coat in all the observed cases so far suggests that the inheritance of these conditions is controlled at one locus or two closely associated loci. Heterozygous animals night be detected by the partial presence of one or both of these characteristics.

CONVULSIONS AND PULMONARY OEDEMA IN MACROPODS (L.. Dowling, R. McKenzie)

A male swamp wallaby (Wallabia bicolor), a. female and a male agile wallaby (Macropus parryi) and a castrated male eastern grey kangaroo (Macropus giganteus) were submitted during mid April, late May and the first half of June from a Zoological collection in Brisbane. These animals had all salivated excessively, convulsed and were killed for necropsy in extremis. Similar cases had occurred previously on the property. All occurred after periods of rain. All animals had bilateral pulmonary congestion and oedema and were dehydrated. The swamp wallaby and the kangaroo had microthrombi in pulmonary vessels and depleted red pulp in their spleens. No other noteworthy lesions were detected besides congestion of various organs. No significant bacteria were isolated. No lead was detected in liver samples. During late July and early August, two adult male eastern grey kangaroos (M. giganteus) and a female swamp wallaby (W.bicolor) were submitted after displaying similar signs. This occurrence did not seem to coincide with wet weather as the previous ones had done. One M.giganteus developed the syndrome after fighting with other males. Necropsy findings similar to those in the previous cases were seen. Tests for enterotoxins were negative. In early September, an adult male wallaroo (Macropus robustus) was submitted with the same syndrome, which still awaits a satisfactory diagnosis.

BRYOPHYLLUM SP. (MOTHER -OF-MILLIONS) (P. Ketterer)

Twenty-nine yearling Friesian heifer cattle died over a period of 4 days on a Nanango property. Their value was estimated at $7,500-$8,000. The herd had broken into a neighbouring paddock and eaten mother-of-millions on the day before deaths started. This was not known until some time after the incident was investigated. Myocardial degeneration and necrosis were seen in some specimens.

CASSIA OCCIDENTALIS (COFFEE SENNA) (P. Ketterer)

A high incidence of muscle lesions resulting in partial carcase condemnations at a Mackay meatworks occurred in cattle from a single property. A sample of neck muscle examined histologically had extensive degenerative changes possibly caused by C. occidentalis poisoning. Other possible causes included selenium/vitamin E deficiency and exertional rhabdomyolysis.

MYXOSPORIDIOSIS IN CALICO GOLDFISH (B. Timmins, I. Anderson)

Animal quarantine submitted five goldfish, carcassius auratus L. from a batch that had suffered one to five deaths daily during a month in quarantine. One of these fish was found to have white, nodular, .5cm diameter gill lesions.

Histologically, all fish revealed the presence of unidentified myxosporidia, (Myxobolus sp.-like); in skeletal muscle and in two fish they had invaded spinal cord cartilage. There were varying degrees of accompanying granulomatous reaction and myositis.

Encysted myxosporidia of the Genus Henneyuya were also present in the gill filaments of two of the fish.

NORTHERN TERRITORY - Lorna Melville

BERRIMAH AGRICULTURAL RESEARCH CENTRE

1989 ARBOVIRUS SENTINEL PROGRAM (L. Melville)

The 1989 arbovirus sentinel program has been particularly productive. A total of 275 viruses were isolated from livestock (cattle and sheep) and 79 from mosquito pools.

The cattle isolates have been identified as blue tongue types 1, 3 and 23; EHD 2; Simbu groups (Peaton and Tinaroo); and Palyan group (Bunyip Creek). The sheep isolates have been identified as bluetongue types 3 and 23. Mosquito isolates so far identified include Ross River virus; Murray Valley Encephalitis; Kunjin; Sindbis; and Kokobera.

Apart from the case of acute bluetongue in a sheep seen earlier in the year, there have been a number of cases of clinical bovine ephemeral fever, although no corresponding virus was isolated. This is a reflection of the relatively short period of viraemia which occurs with this virus in cattle where both BLU 3 and 23 were recovered for up to 5 weeks.

An interesting clinical syndrome was also seen this year in calves born to cows in the sentinel program. Of 19 pregnant cows, 8 calves were stillborn or were weak at birth, unable to suckle and died within a few days. Pre-suckle blood samples collected from these calves have been negative for the virus groups routinely monitored in this laboratory. They were also all pestivirus antibody and virus negative. The cause of the syndrome remains unknown at present.

ARID ZONE RESEARCH INSTITUTE ALICE SPRINGS

BOTULISM IN THE ALICE SPRINGS REGION (J. Lee)

HISTORY.

Several properties have recently experienced outbreaks of mortality tentatively due to Botulism.

All properties have had 1-5% mortality over 2-6 weeks. Mortalities are often regional with only some paddocks being affected. All affected areas reported osteophagia (bone chewing) in cattle was common. The season has been good and there is an extensive body of feed available and no increase in toxic plants detected.

CLINICAL SIGNS

Animals involved are all ages and mixed sexes. Initial signs include weight loss and lagging behind the mob. Animals begin to show ataxia and weakness. They have trouble gaining their feet. Many are agitated and aggressive. Many have a fine tremor of the head. Bilateral mucoid nasal discharge is a common finding. Terminally some show complete flaccid paresis, dyspnoea and tachypnoea. Death usually occurs in 3-7 days. Some were observed to have the tongue protruding and being unable to drink.

POST MORTEM EXAMINATION

No abnormalities were detected in any animal autopsied, except for relatively dry rumen and omasum contents. One cow had multiple small lacerations and ulcers of the abomasal mucosa, which may have been due to the ingestion of foreign objects e.g. bones. However, no bones were found in any alimentary tract. No evidence of toxic plants were found in the ingesta.

HISTOPATHOLOGY

The only charges observed were some perivascular haemorrhage and venous congestion in the CNS. These changes are, however, non-specific.

DIAGNOSIS

History and clinical signs together with the absence of any pathology seen had led to a diagnosis of Botulism on all of the above properties.

Confirmation of Botulism may be possible by submitting faecal or rumen samples to the lab for bacteriology. Here the samples are cultured in enrichment media in an attempt to grow the Clostridium botulism bacteria which produce toxin. The solution is filtered and injected into mice. If unprotected mice die, but mice protected by specific anti-toxin do not, this indicates that Botulinum toxin of a specific type (C or D) is present. Control mice are injected with heat treated solution (100 degrees Celsius), which inactivates the themo-labile toxin.

CONTROL

The disease can be prevented in susceptible herds by annual vaccination with specific vaccine (Type C or D) if the toxin type has been identified or with both vaccines where it has not. Type C is believed to be the main type in the Northern Territory.

ST GEORGE DISEASE IN THE NT (J Lee)

HISTORY

Mortalities occurred in a herd in the Alice Springs region, of approximately 230 beef Shorthorn cattle. Over a 3-4 week period there were more than 10 mortalities and 40 affected cattle. Animals affected were all age groups and mixed sex. Disease course was approximately 10-14 days in animals which died. Several animals were chronically affected but recovered slowly when removed from the affected pasture.

CLINICAL SIGNS

These included:

- rapid loss of condition from score 4 to score 1
- dependent submandibular and ventral oedema
- bilateral nasal discharge
- severe dyspnoea with head extended, elbows abducted and panting
- fetid scours.

One bull died of asphyxia during yarding.

HAEMATOLOGY

Most animals were slightly anaemic, hypoproteinaemic and had depressed WCCS.

BIOCHEMISTRY

Blood levels of LDH, AST and GGT were moderately elevated in one affected animal.

POST MORTEM FINDINGS

Three animals were autopsied. All showed similar lesions. There was serious atrophy of adipose tissue and extensive subcutaneous oedema particularly in the submandibular and antero-ventral regions. There was a serosanguinous hydrothorax. The lungs showed ventral atelectasis with a marked horizontal fluid line and compensatory dorsal emphysema. The interlobular septae were dilated with oedema fluid. The heart showed significant dilation of the right ventricle with a prominent apical notch and atrophy of the right ventricular wall. Liver showed multiple small blood-filled areas and was congested. There was a haemorrhagic enteritis.

HISTOPATHOLOGY

The lungs showed areas of atelectasis and marked interlobular septal oedema. There was perivascular infiltration of lymphocytes. The liver of some animals showed periacinar fatty degeneration and necrosis with associated haemorrhage and neutrophilic infiltration. One animal showed multiple large blood-filled cavities throughout the parenchyma. These cavities had no endothelial lining and were consistent with the condition of peliosis hepatis. There were many mononuclear inflammatory cells associated with sinusoids in the animals. Myocardium showed a diffuse myositis.

MISCELLANEOUS FINDINGS

The station has had a good year with heavy rain in April but has been dry since then. The property was heavily grazed. Both Pimelea trichostachya and Pimelea simplex were found on the property.

CONCLUSION

The typical clinical and post mortem findings associated with the presence of Pimelea spp support the diagnosis of St George Disease.

SOUTH AUSTRALIA - Vui Ling Tham

VET LAB. ADELAIDE

PINNAL VASCULAR DISEASE

The subject, "Maverick" was a two year old short haired miniature Dachshund. He was presented with small crescent-shaped lesions along the edges of both pinna. The lesions were dry and crusty and bled profusely if traumatized. They gave the appearance that mice had nibbled away portions of the edges of the ears. Treatment with topical cortisone had not been successful.

Two biopsies were submitted on separate occasions and revealed only necrotic epidermis and dermis overlying healthy tissues. Electron microscopy showed only necrotic collagen. Some superficial purulent exudate was present.

Meanwhile the submitting clinician laid up in bed with the flu, decided to catch up with some professional literature and happened upon a reference***** to a condition called pinnal vascular disease occurring particularly in Dachshunds, but also in Chihuahuas, Terrier crosses, Labrador Retrievers and Rhodesian (Zimbawean or Zambian?) Ridgebacks. The description matched Maverick's condition exactly. The lesion described in the reference is one of vascular hypertrophy, degeneration and thrombosis along a leading edge with coagulative necrosis of overlying epidermis and dermis. We did not see the vascular pathology but this may have been through not looking at a leading edge of the lesion.

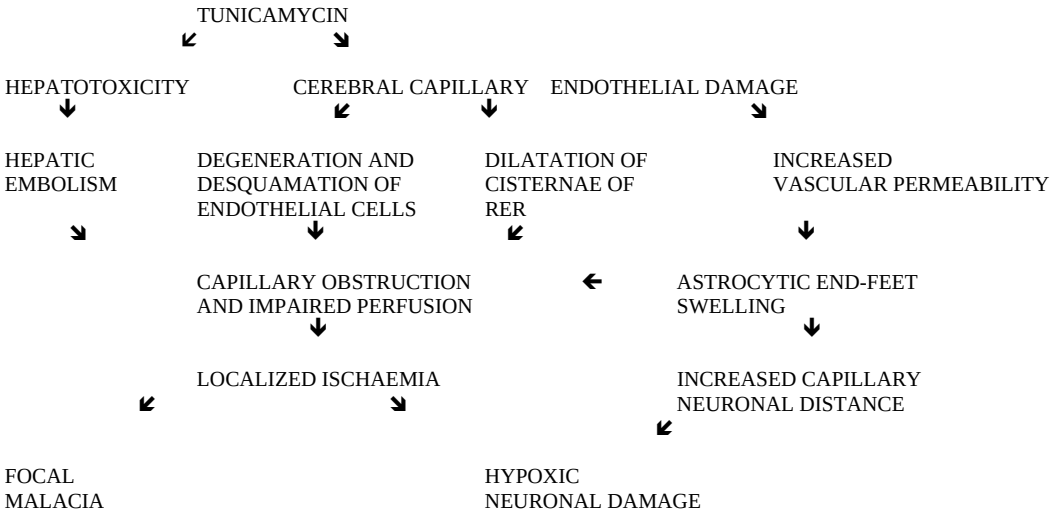
Treatment of choice is partial pinnectomy, with too conservative an approach frequently leading to relapse. Topical and/or systemic steroids and dapsone are reported not to be of benefit; however, the owner reports that Maverick has improved and his ears are not constantly bleeding.

*****REFERENCE. Scientific Proc. Of 56th Annual Meeting of AAHA, St. Louis (USA) April 1989: pp.305-306.

SOUTH EAST REGIONAL VETERINARY LABORATORY (J. Finnie)

Pathogenesis of annual ryegrass toxicity

The pathology of the neurological disease, annual ryegrass toxicity (ARGT), caused by corynetoxins (members of the tunicamycin group of antibiotics), has recently been investigated by Finnie and O'Shea (1988, 1989) and the following is the proposed pathogenesis of the cerebral lesions in this condition.



References: Finnie, J.W. & O'Shea, J.D. (1988) Acta Neuropath. (Berl). 75:411-421.; Finnie, J.W. & O'Shea, J.D. (1989) Pathology 21:194-199.;

BOOK REVIEW

Title: PLANT POISONINGS AND MYCOTOXICOSES OF LIVESTOCK IN SOUTHERN AFRICA.

Authors: S. Kellerman, J.A.W. Coetzer and T.W. Naude.
Publishers: Oxford University Press, 1988 Cape Town, South Africa

This hardbound textbook is a comprehensive manual which updates and reviews the available information on toxicology and pathology of poisonous plants and fungi occurring in Southern Africa. For ease of consultation, it is arranged in chapters according to the organ or body system which the toxic plants predominantly affect (e.g., the liver, heart, central nervous system, respiratory system, gastro-intestinal tract, urogenital system, haemopoietic system, the skin and adnexa). The whole book is well illustrated with 350 colour and 250 monochrome pictures, each selected carefully and of exceptional quality. Each chapter is subdivided into sections which assist the user in consulting the relevant sections on the basis of principal clinical and pathological features. The toxic plants are arranged within genus and species groupings with each plant described botanically and illustrated. Distribution maps across Southern Africa are given, along with detailed information on the toxicology, pharmacology, chemistry and mode of action of the toxic principle. Comprehensive accounts of the clinical, features of the intoxication in the live animal and the pathological findings at necropsy are given for diagnostic purposes. At the end of each chapter there are discussion sections which consolidate the information, drawing parallels between the various related toxic plants and eluding to other infectious or nutritional conditions which could be confused with specific plant toxicoses. Throughout the book the scientific literature on plant poisoning and mycotoxicoses has been extensively used by the authors; this helps to make the text very authoritative and soundly based.

Although the book is detailed, it is written in an interesting and refreshing way, which makes it useful as a reference for veterinary students, practitioners and specialists {e.g. veterinary pathologists, toxicologists and clinicians). The book is obviously written with South Africa specifically in mind; nevertheless, many of the toxic plants discussed are not restricted to South Africa. The book has relevance to other parts of Africa and indeed other continents like Australia, North America and South America. This textbook has relevance for the library services of veterinary faculties, agriculture departments and veterinary research organisations (David L Obendorf).

TASMANIA - Judith Handlinger

PARASITES AND BEACHED FISH - a non-pathology report (J. Handlinger, D. Obendorf and A. Clarke)

A large number of peritoneal parasites were the only finding in fish submitted from several fish kills on beaches around the state. Although considerable tissue reaction was often present, the findings did not account for the deaths. One episode was eventually closely observed when two large schools beached in the same area on successive tides. Subsequent investigation uncovered similar observations in other instances, namely that the fish were seen alive and actively swimming to the beach, indeed reportedly charging for the beach with heads protruding from the water. Live fish on the beach which were returned to the water promptly beached again. Other relevant observations were that this had not been seen previously in the area; that the fish were not spawning (occasional species spawn on beaches); and in all cases only one species was involved though other species of fish were seen in the area. The largest teachings were seen with Redbait, but there were similar findings with Mackerel and Jack Mackerel, and similar parasites from other species.

The cause of the beachings was considered to be chasing by predators. This pattern has been recognized in several parts of the world when large predators school large numbers of small fish, driving them from the deeper waters and well out of their natural habitat. (Redbait are considered a mid-water fish). The fear by the pursued of the predators outweighs their self preservation and they commit mass suicide. If the initial beaching is not observed (and even if it is) the cause of the kill may be difficult to determine and a pathologists dilemma. The predators may not be visible from the shore.

The recent pattern of fish distribution was unusual with Jack Mackerel, which may be a more common prey for the larger fish, being absent from many areas for much of the season.

The parasites from all the fish species were identified as Ascaradoid larvae, mostly of Contracaecum and Thymnascaris species which cannot be distinguished easily in larval stages. They are rarely pathogenic to the fish. Definitive hosts are usually seabirds, predatory fish or marine mammals. All have the potential to be zoonotic, but the true Anasakis has much greater potential to migrate into fish muscle after death and be commonly consumed. Subsequent investigations have shown that Asceradoid larvae appear to be much more common in this area than previously thought, involving a number of fish and parasite species, including Anasakis.

CANINE PARVO VIRUS MYOCARDITIS DEATHS PRECIPITATED BY VACCINATION?
(D. Obendorf and J. Handlinger)

Myocardial parvovirus inclusion bodies were found in samples received from a litter of nine 4-week old German Shepherd pups in which deaths commenced approximately 36 hours after combined tissue culture vaccine containing live attenuated parvovirus vaccination. All pups died within 5 days (7 days post-vaccination); the majority on days 2 and 3 post-vaccination. Vaccination was given early because parvo-virus infection had been diagnosed in another bitch from the kennel on the day the pups were born. Infection occurred despite the fact that the bitch had live parvovirus vaccination two weeks before mating.

All deaths were either sudden, sometimes observed as a single scream after abruptly ceasing apparently normal play; or following a few hours dyspnoea and hyperpnoea. Lesions were gross and there was histological evidence of pulmonary oedema in all pups; cardiac pallor or haemorrhages in some; abundant parvovirus type inclusions in myocardial nuclei; and small mononuclear reactive foci, especially in the later cases. There were no lesions in other internal organs. Parvovirus type particles within intra-nuclear inclusions were seen by electron microscopy.

Given the time scale of the deaths, the infection is clearly perinatal, but what part, if any, did the vaccine play in precipitating the deaths? If indeed this did precipitate destruction of infected myocardial cells, was the result merely to accelerate virtual inevitable deaths? Why didn't maternal vaccination protect the pups

(maternal serology samples have been taken but as yet no results of antibody levels)? Is vaccination recommended in these circumstances? Does anyone have any answers, comments, similar reports or pertinent references, please?

REGISTRY REPORT Tony Ross

OPEN FOR BUSINESS

The Chief Veterinary Officers of Australia have agreed to financially back the National Registry of Domestic Animal Pathology for 2 years from January 1990. Thank you to all ASVF members who lobbied for this outcome.

It is now up to us all to make effective use of the Registry by:

- add case material to the collection.
- submit cases for second opinion.
- plan training program in your home state,
- visit the Registry for 1 to 1 training.

LOCATION OF THE REGISTRY

Jan-March 1990 c/- Veterinary Laboratories, Roy Watts Road, Glenfield 2167. Phone (02) 605 1511 Fax (02) 605 2282

From April 1990 c/- Elizabeth Macarthur Agricultural Institute, Woodbridge Road, Menangle 2568. (Phone and Fax to be confirmed).

Menangle is located 2 stations south of Campbelltown on the main southern railway line.

REQUEST FOR MATERIAL

Thank you to the few who are regularly submitting material to the Registry. For those with unfulfilled good intentions please nominate someone in your lab to send each up to 6 of the most interesting current cases (slides, paperwork and blocks if available).

The latest wanted list includes:

Cattle	Pigs	Sheep
IBR	Erysipelas	B. ovis epididymitis
BVD	Salmonellosis	Skin conditions
MCF	Mulberry Heart Disease	
Lepto Hburia		

Goats	Dogs & Cats
Placenta1 conditions	Skin conditions
Foetal conditions	Tumours

NATIONAL REGISTRY OF NON-DOMESTIC ANIMAL PATHOLOGY

Bill will continue half time at each of the two Registries. The Non-Domestic Animal Registry will continue to be sponsored by and located at:

Taronga Zoo, PO Box 20, Mosman. NSW 2066. Phone (02) 969 2777 Fax (02) 969 7515

JOBLINE

The University of Sydney
Lecturer/Senior Lecturer
Department of Animal Health
Reference No. 48/28

Applications are sought from suitably qualified veterinary graduates for a position which entails responsibility for teaching, research and service in veterinary laboratory medicine at the department's Rural Veterinary Centre. The appointee, who will have qualifications equivalent to those required for registration as a specialist veterinary pathobiologist in New South Wales, will be familiar with diagnostic gross and histopathology, microbiology and clinical biochemistry and preferably will have a strong research interest in one of these disciplines.

Appointments to lectureships/senior lectureships have the potential to lead to tenure and such appointments are usually probationary for three years. Applicants should indicate if they wish to be considered for the senior lectureship only.

The successful applicant will be expected to begin work early in 1990. Further information from Professor J. R. Egerton (046) 552 300.

Salary: Senior Lecturer $41,459-$48,086 per annum. Lecturer $31,259-$40,622 per annum.

METHOD OF APPLICATION: Applications, quoting reference no., including curriculum vitae, list of publications and the names, addresses and FAX numbers of three referees to the Registrar, Staff Office, University of Sydney, NSW 2006 Australia. FAX + 612 692 4316.

Closing: 31st March 1990

The University reserves the right not to proceed with any appointment for financial or other reasons.

Equal opportunity employment is University policy.

TRAINING AND EMPLOYMENT OPPORTUNITIES FOR AUSTRALIAN PATHOLOGISTS IN USA

Donald McGavin, Editor of Veterinary Pathology has written to the VPR requesting that Australian pathologists (both budding and experienced) be aware of the range of clinical, anatomic and histopathology training and employment opportunities available in several US veterinary schools. Unfortunately the number of positions and the detailed position descriptions prevent me publishing them in the issue. I have photocopies of the positions for anyone interested in calling me on (003) 415218. I understand the positions are advertised in a recent American College of Veterinary Pathologists newsletter. There are 17 A.C.V.P. diplomats in Australia, so if you know someone in this category, ask for the late 1989 newsletter.

(**VPR Editor**)

VETERINARY PATHOLOGY IN PAPUA NEW GUINEA

Two positions are currently available for veterinary pathologists at the National Veterinary Laboratory, Port Moresby.

These positions would be suitable for competent young pathologists with at least several years' diagnostic pathology experience looking to broaden their horizons in a developing tropical country.

Salary and other conditions of service are better than those on offer in Australia and include free housing, six weeks leave per annum, leave fares from home after 18 months, gratuity of 30% of gross salary for the first year rising to 40% in the third year (currently taxed at 2%). Salary is either K19950 (V02) or K21985 (V03). Airfares and school subsidies are paid for two children under 18. The current exchange rate is K1 = $1.47. Employment is by contract and would be for a three year period initially.

Expressions of interest are also sought for the position of Chief Veterinary Pathologist (Veterinary Officer Class 5) which becomes vacant in March 1990. The Chief Veterinary Pathologist is the officer in charge of the national Veterinary Laboratory, a salary of K29645 is offered for this position, plus gratuities and other benefits.

The National Veterinary Laboratory has been in operation for over 20 years and sees a wide range of specimens. There is a bias towards poultry pathology but mammalian and wildlife specimens (including farmed crocodile) are well represented. Approximately 1800 accessions are received at the laboratory per annum comprising more than 20,000 samples. Applied research is encouraged as is provision of advice to the livestock industries on disease management problems. The laboratory is well equipped and records are computerised. The scope of the work is much broader than that offered in equivalent jobs in Australia. Currently there are 3 senior expatriate scientists/veterinarians at the laboratory supported by a team of 14 national scientific, technical and ancillary staff.

Life in Papua New Guinea is not without its occasional problems but the experiences gained make it all worthwhile. If you are at all interested please contact Ivor Owen 0011 675 217005 (w) or Mike Nunn (217405 (w) 256769 (h) for further details, or send a fax on 0011 675 214630.

STOP PRESS!

ASVP SUBMISSION ON SPECIALIST REGISTRATION

Following the recommendations of the ASVP Annual General Meeting in January 1990 and the widespread concern at disparity between States towards Grandfather Clause applications for Specialist Registration and the non-representation of ASVP in this process, the following submission has been prepared by Tony Ross on behalf of ASVP for submission to the Australian Veterinary Board's Conference in May. A preliminary submission has already been made to the NSW Board.

With the Grandfather Clause applications closed with only reviews of recommendations still outstanding, there is little enough time for action, but any comments should be submitted immediately for finalisation of the May submission. In general the ASVP submission closely follows the General Criteria for Qualification as a Specialist Veterinarian (see final item).

Comments please to Tony Ross, Convener on the ASVP Training Committee, at the Elizabeth Macarthur Agricultural Information Centre in NSW.

SUBMISSION TO AUSTRALIAN VETERINARY BOARD'S CONFERENCE

FROM: AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

QUALIFICATIONS FOR REGISTRATION AS A SPECIALIST VETERINARY PATHOLOGIST

BACKGROUND

ASVP:

- is the only organisation in Australia which represents the majority of Veterinary Pathologists in Australia. Current membership is 178. By comparison there are Currently 6 Fellows in the Pathobiology chapter of the Australasian College of Veterinary Scientists. (4 in Queensland, 1 in Victoria, 1 in New South Wales).

- supports registration of suitably qualified persons as specialist veterinary pathologists.

- sees the need for Australia-wide guidelines for registration.

- suggests that any system for registration of specialist veterinary pathologists must include the large number of highly competent veterinary pathologists presently excluded from fellowship.

- considers that the only currently available pathway for registration as a specialist veterinary pathologist, i.e. Fellowship in the Pathobiology chapter of the ACTS, is too restrictive and does not provide a suitable entry pathway for the majority of veterinary pathologists who start out in state veterinary diagnostic laboratories.

- suggests that if criteria are too restrictive then the specialist registration system will not provide assurance to the public of accepted performance for veterinary pathologists as most veterinary pathologists will be outside the system.

- strongly believes that there are many suitably qualified practising veterinary pathologists of several years experience who, in some states, are ineligible through the grandfather clause and who it is unreasonable to expect to undertake the complete Membership/Fellowship training program.

RECOMMENDATIONS

ASVP recommends that the -

1. only qualifications presently available as a specialist veterinarian i.e. Fellowship of the Australian Chapter of Veterinary Scientists is too restrictive and should be replaced by broader guidelines.

2. Australian Veterinary Board's Conference adopts the revised general guidelines for suitable qualification as a specialist in any discipline attached to this document.

3. substantial number of currently practising competent veterinary pathologists who are excluded under the grandfather clause must be accommodated to ensure specialist registration in veterinary pathology provides adequate assurance of quality services to the community.

4. ASVP executive or a nominee be appointed to the existing advisory committee to review applications on a continuing basis.

R. E. Oliver
PRESIDENT, ASVP

General Criteria for Qualification as a Specialist Veterinarian (Draft, May 1990)

1.1 Minimum Time from Graduation to Submitting for Specialist Registration

An applicant must have practiced as a veterinary surgeon for at least five years before becoming eligible to be registered as a specialist.

1.2 Minimum Time Working in Specialty for Registration

An applicant must have worked for at least 4 years full time equivalent in his/her specialist area before being eligible to be registered as a specialist.

Centres for training in the specialty require:

adequate work load in the specialty
adequate facilities for the performance of the specialty
adequate access to other professional expertise

1.3 Post-graduate Qualifications

An applicant should hold formal post-graduate qualifications in the relevant specialist field. Acceptable qualifications may include: fellowship of the Australasian College of Veterinary Scientists, a post-graduate qualification or relevant overseas qualifications.

An applicant with an extensive publication list but without formal qualifications may be considered after a minimum of ten years full time work in the specialty.

1.4 The Period of Formal Post-graduate Training

The applicant's period of formal training should be equivalent of at least 12 months full time.

1.5 The Selection and Role of Supervisors

Approved supervisors are necessary where Australian post-graduate programs are to be assessed for specialist registration. In the case of overseas qualifications there can be no control as to the supervisors who are appointed.

Supervisors shall be responsible for both instruction and continuing assessment of candidates. Supervisors shall be recognised as specialists working in the specialty area in which the applicant is to be examined.

Recognised specialists shall be either those registered by the boards as such or those with qualifications considered equivalent to board requirements.

1.6 Case Reports, Dissertations and Publications

The applicant's case reports, dissertations and publications shall be assessed by the board of veterinary surgeons.