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.

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DEADLINE FOR NEXT VET. PATH. REPORT IS JUNE 1, 1991

ASVP PRESIDENT'S MESSAGE

As Tasmania's two year tenure for running the ASVP draws to a close, I would like to raise a few emerging issues for Veterinary Pathologists.

1. Quality Assurance

   Quality assurance (QA) programs are being widely implemented within laboratories, eg DARA's QA program for veterinary laboratories. Many disciplines, for example, chemistry and microbiology have adopted QA programs but pathology has been slower to move to adapt QA. Reliance on professionalism and individual responsibility have served veterinary pathologists well in the past, but pathologists will have to come to terms with embracing quality assurance philosophies and practices.

2. State Debt

   State debt levels in Western Australia, South Australia, Victoria and Tasmania due to activities outside our control, for example State Banks will impact on funding for public services and our members in the state laboratories will be affected.

   Pathologists are going to have to become increasingly resourceful and utilise their skills and expertise to become involved in peripheral activities where a need for pathology input exists. Pathologists will need to do a little lateral thinking and recognise their skills and understanding of disease processes may enable them to move into related fields.

3. Specialisation

   It is becoming increasingly difficult for the generalist veterinary pathologist. Companion and performance animal pathology is one specialist stream. In the food and farm animal sector, the trend to industry funding makes it very difficult to sustain pathology effort across all species. Industries are interested in funding solutions to their problems, making it difficult for the generalist.

4. Modus Operandi of the ASVP

   The ASVP executive operates on the basis of considerable voluntary input of its committee in particular the Secretary, the Treasurer and the Editor of the Veterinary Pathology Report. These resources and the unseen administrative support of the host organisation do not show in the cost of the operating of the ASVP. Additional inputs are needed to address specific issues, for example specialist registration.

   It is unlikely that the ASVP can continue to be sustained nationally in the longer term, by cheap subscriptions and 'donated' administrative support. As a first step, realistic subscription fees should be established.

   Last, but certainly not least, on behalf of the ASVP, I must express sincere appreciation to the Tasmanian executive - Roy Mason, Judith Handlinger, David Obendorf and Barry Munday. Well done. After two years, it is time to pass on the baton.

Rod Oliver
ASVP PRESIDENT

EDITOR'S REPORT

Welcome to **VPR** issue No. 30!

Commenced in August 1983, the Report has gone from strength to strength. Several North American subscribers have been moved to congratulate us on this publication. Indeed the well-established **Australian Society for Parasitology** has decided to follow suit by producing a similar information newsletter. For many pathologists I'm sure, the **VPR** is eagerly awaited. Thank you to all contributors. The value of **VPR** depends on us all.

This issue is full of new and unusual case studies, with a high proportion on wildlife and "exotic" animal themes. Lead poisoning in magpie geese, jellyfish-induced deaths in fanned salmon, tuberculosis in W.A. sea lion, and diseases of ostriches to name a few.

We have an artist amongst us! Adrian Philbey has continued the wildlife theme by sending two possible **ASVP** logos. He also gives his thoughts on what the logo needs to include (Letter to the Editor). We need more submissions, please.

It's still not too late to register for the 1991 **ASVP** Conference at EMAI in May. Roy Mason has drawn up the scientific timetable with information on travel and accommodation options.

Ruth Reuter, our very able W. A. state correspondent, is leaving RVL, Albany and moving to South Australia to join **Veterinary Pathology Services**. Thank you Ruth for your contributions and best wishes for the move into private veterinary pathology. Ron Peet from AHL, South Perth will take over as W.A. rep.

Dave Obendorf
Honorary Editor

ASVP SCIENTIFIC MEETING AND AGM
11 AND 12 MAY 1991

VENUE: ELIZABETH MACARTHUR AGRICULTURAL INSTITUTE CAMDEN NEW SOUTH WALES

SCIENTIFIC PROGRAMME

SATURDAY, 11 MAY 1991

8.30-8.50 am	Assembly at EMAI	
8.50-9.00 am	Welcome and in-house announcements	
9.00-9.05 am	Introduction to first session on oncology by Terry Rothwell.	
9.05-9.50 am	(i)	U.V. radiation-induced carcinogenesis in the hairless mouse model **V.E. Reeve** and **M.J. Matheson***
	(ii)	The Pathology of U.V. radiation induced carcinogenesis in a hairless mouse model **Paul Canfield*** and **Gavin Greenoak**
9.50-10.00 am	Discussion	
10.00-10.25 am	Diagnosis of common canine cutaneous neoplasms **Terry Rothwell**	
10.25-10.30 am	Discussion	
10.30-11.30am	MORNING TEA	
10.50-11.30am	(I)	Radiation therapy of small animal neoplasms **Graeme Allan**
	(ii)	Chemotherapy **Elizabeth Dill-Mackey**
11.30-11.40	Discussion	
11.40-12.00 noon	Neoplasia in Marsupials **Paul Canfield*** and **Bill Hartley**	
12.00-12.10pm	Discussion	* Presenter of paper
12.10-1.00pm	LUNCH	
1.00-1.40pm	Tumours of large domestic animals **Tony Ross**	
1.45-1.50pm	Discussion	
1.50-3.00pm	Case Reports (first session)	

* Multifocal Symmetrical Encephalopathy of Simmental Calves - **John Finnie** and Multifocal Symmetrical Encephalopathy in Angus Calves **Adrian Philbey** (A joint presentation)

* A Bovine Tumicamycin-like Syndrome (or Flood Plains Staggers) **Chris Bourke** and **M.J. Carrigan**

***** Pathogenesis of focal hepatic necrosis **Alan Seawright**

3.00-3.30pm	AFTERNOON TEA
3.30-	ASVP ANNUAL GENERAL MEETING
EVENING	SOCIAL FUNCTION

SUNDAY 12 MAY 1991

9.00-9.40 am	Liver Diseases: Pathophysiology **Roger Kelly**
9.40-9.50am	Discussion
9.50-10.30 am	The Diagnosis of Pyrrolizidine Alkaloidosis by the detection of tissue sulphur-bound metabolites **Alan Seawright**
10.30-10.40am	Discussion
10.40-11.00am	MORNING TEA
11.00-12.30pm	Case Reports (Second Session)

* Pathogenesis of focal hepatic necrosis. - **Alan Seawright**
* Bracken Fern Poisoning - **Barry Smith** (New Zealand)
* Anasarca and myopathy in hatchling ostriches. - **Adrian Philbey**
* Demyelinating myelopathy in sheep exposed to stagger week **Adrian Philbey**
* Lupinosis in cattle - **John Mackie**
* Nephrosis in sheep associated with the ingestion of Lythrum hyssopifolia L., "lesser loosestrife" **John Glastonbury** et al.
* Algal infection in a cat **Jonathan Webster** & **Deborah Seaward**

This is a full programme. It may not be possible to include any further case reports in the formal programme because of time. However, if you have material, bring it along with a type-written synopsis for distribution, and it may be possible to present it "informally" after the formal programme concludes.

HOW TO GET THERE

* Interstate Air/bus/train travellers.

* Catch an electric train from Central Railway on the East Hills line to Campbell Town.

* Tony Ross will arrange to pick up people from Campbell Town Station within a defined period, Friday afternoon and Saturday morning. You must give Tony prior information of your ETA at Campbell Town Station.

ACCOMMODATION

* Country Club Motel Hume Highway SOUTH CAMDEN Ph (046) 558 402	Picton Village Motel Ph (046) 772 121	Crown Hotel/Motel Argyle Street CAMDEN Ph (046) 552 200

Transport to and from EMAI will only be to these three places of accommodation. If you choose to stay elsewhere, you will have to make your own transport arrangements.

TRAVEL DIRECTIONS TO EMAI FROM MASCOT

* Leaving Mascot (Gregory's directory map references 85, 84 and 93), take Airport Drive over Cooks River into Marsh Street,

* Turn right at lights at Wickham Street which becomes Forest Road (major road - 55).

* Stick with Forest Road. As you enter the suburb of Hurstville, Forest Road swerves left at a set of lights. Do not swerve, continue straight into what becomes Henry Lawson Drive (Gregory's map 91).

* Henry Lawson Drive follows the line of the Georges River through suburbs like Picnic Point, etc.

* Turn left into Newbridge road. This is a major intersection across the river (Gregory's map 89).

* This brings you into the minor city of Liverpool directly onto the Hume Highway (Gregory's map 88).

* Follow the Hume Highway (it becomes the F5 Freeway) towards Campbelltown. DO NOT TAKE THE CAMPBELLTOWN EXITS.

* Take the Camden exit onto Narellan Road (major road - 56).

* Go left onto Camden Bypass (major road 89) where there is a large blue sign reading Elizabeth Macarthur Agricultural Institute.

* Approximately 10 kms down the road, turn left into Finns road (another blue sign).

* Approx 3kms along, veer left into Woodbridge Road.

* Approx 2kms along, go left up big hill into Institute.

* The trip should take you between 1.50-2 hours depending on traffic.

UNABLE TO ADD MAP

TRAIN TIMETABLE FROM CENTRAL SYDNEY TO CAMPBELLTOWN

CITY-TEMPE-EAST HILLS-MACARTHUR
MONDAYS TO FRIDAYS.

	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm
Central arr	3.19	3.34	3.49	4.04	4.19	4.34	4.49	5.04	5.19	5.34	6.04	6.19	6.49	7.49	8.19	8.49
										5.49						
dep	3.20	3.35	3.50	4.05	4.20	4.35	4.50	5.05	5.20	5.35	6.05	6.20	6.50	7.50	8.20	8.50
										5.50						
Campbelltown arr	4.12	4.21	4.42	4.51	5.12	6.21	5.42	5.51	6.13	6.21	6.51	7.09	7.39	8.39	9.09	9.40
										6.42						
Central arr	9.19	9.49	10.19	10.49												
dep	9.20	9.50	10.20	10.50												
Campbelltown arr	10.09	10.39	11.09	11.48												

SATURDAYS

	am		am		am		am									
Central arr	9.19		9.49		10.19		10.49									
dep	9.20		9.50		10.20		10.50									
Campbelltown arr	10.09		10.39		11.09		11.39									

SUNDAYS AND PUBLIC HOLIDAYS

	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm	pm
Campbelltown dep	1.29	1.59	2.29	2.59	3.29	3.59	4.29	4.59	5.29	5.59	6.29	6.59	7.29	7.59	8.29		
Central arr	2.17	2.47	3.17	3.47	4.17	4.47	5.17	5.47	6.17	6.47	7.17	7.47	8.17	8.47	9.17		
Circular Quay	2.25t	2.55	3.25	3.55	4.25	4.55	5.25	5.55	6.25	6.55	7.25	7.55	8.25	8.55	9.25		
Wynyard	2.23t	2.57	3.27	3.57	4.27	4.57	5.27	5.57	6.27	6.57	7.27	7.57	8.27	8.57	9.27		
Town Hall	2.20t	2.59	3.29	3.59	4.29	4.59	5.29	5.59	6.29	6.59	7.29	7.59	8.29	8.59	9.29		

LETTER TO THE EDITOR

Reply to ASVP President's Comments on Specialist Registration:

I feel I must respond to the article on specialist registration which appeared in the December “Veterinary Pathology Report”. I agree wholeheartedly with the majority of your comments. However I do not agree with your comments in Item 7 which imply that staff at Universities or private pathology laboratories are not competent to evaluate applicants for specialist registration.

Every broad generalisation is, of necessity, coloured by the experience of the person making the statement. Having been in the profession for the past 29 years, 26 or which have been spent in the practice of pathology, and having been associated with a wide range of organizations, both governmental and academic, here and overseas, I have met a large number of pathologists of all interests and on all levels. Many of these, regardless of the location in which they are employed, have a very broad background which includes commercial livestock, and are fully qualified to comment on the needs of the diagnostic laboratory. Indeed, here in Australia the number of people in the profession is so small that qualified pathologists seem to move freely among the various agencies. I think personally that this greatly improves the service and the pathologist, as well as providing communication links between all areas.

My definition of "qualification" of necessity includes certification by the American College of Veterinary Pathologists, which requires a broad knowledge of all species.

My strong belief is that in selecting people to evaluate applicants for specialist registration, the background and individual qualities of the evaluators should be considered, not the institution for which they work! As I am sure you are aware, several private laboratories are now staffed by pathologists who previously spent a considerable time working in state Animal Health Laboratories and include ACVP certification +/- membership in the Australian College. No one would seriously consider them unqualified I am sure!

From RUTH REUTER PO Box 1405 Albany WA 6330

LETTER TO THE EDITOR

ASVP LOGO

A logo would give the Australian Society for Veterinary Pathology more visual identity, but we should not rush into making a decision and I hope that more than one pathologist responds to your request for submissions. It would be disappointing if our choice was limited to one logo only. I would like to list some possible criteria which could be incorporated into an acceptable logo for the Society:

1. A clear indication that we are a Society of veterinary pathologists.
2. A clear indication that the Society is Australian.
3. Visual appeal, with depictions of cell, animals, tools of pathology and/or a map of Australia.
4. It may or may not include the letters **ASVP**.
5. A simple design.

Personally, I would like to see more than the letters ASVP fitted into an outline of Australia, which I feel lack visual appeal. Therefore, I submit a design for a logo, which while not satisfying all of the above criteria, to me, has some visual appeal and is clearly Australian. It comprises a platypus motif containing the letters **ASVP**.

IN addition, on a more light-hearted note, I also submit a logo containing several elements relating to a theme of veterinary pathology in Australia: dancing wombats, a neurone, a microscope, a map of Australia and the letters **ASVP**! The wombats are smiling because veterinary pathology is fun.* Perhaps this motif could be used to introduce kindergarten children to our profession.

The **ASVP** should not just vote among the logos presented to it by the time of the 1991 meeting. If none of them meet the required standards, then we need to wait until a satisfactory logo is submitted. From ADRIAN PHILBEY RVL Wagga Wagga NSW 2650.

* Adrian's other ASVP logo design is on page 10

STATE REPORTS

QUEENSLAND – Fraser Trueman

UNIVERSITY OF QUEENSLAND

Acute Non-Colitis-X-colitis? (Roger Kelly)

A two year old thoroughbred colt, in training but with no history of recent transport or therapy, developed severe diarrhoea, shock and dehydration overnight. Another similar horse was found dead in the same enclosure the same morning, having shown minimal illness the day before. The sick horse was killed for necropsy.

The large bowel, including caecum but not caudal small colon, was distended by watery green fluid with little solid content. The mucosa was patently covered by a delicate fibrinous membrane. This could be easily lifted from rather normal-looking epithelium which, however, showed severe catarrhal enteritis microscopically. Affected mucosa showed pronounced ballooning of nuclei of glandular epithelial cells; these and other changes were similar to those seen in some virus infections, and were particularly reminiscent of the histological appearance of an acute typhlocolitis we nave seen recently in laboratory rabbits (VPR, October 1990). The distal colon and the small bowel were spared. Bacterial culture of large colon mucosa grew heavy but non-specific growth of haemolytic E coli. There is a lot we do not know about acute typhlocolitis of horses.

Acute Equine Small Intestinal Villous Atrophy (Roger Kelly)

Another acute intestinal drama began with an apparent epidemic of colic, including naso-gastric reflux, among a mob of high-priced subjects of a high-profile yearling sale at the Gold Coast. Amid rumours of poisoning and other nefarious practices, one colt worsened to the point where it developed laminitis and peritonitis and had to be killed after about 4 days of illness. It turned out to have a small bowel perforation; possibly due to attempted diagnostic paracentesis a couple of days before. What was more interesting, however, was the spectacularly severe villous atrophy and fusion present throughout most of the small intestine, which was uniformly distended with watery, slightly mucoid content, but which showed little inflammation.

The picture histologically was very suggestive of the later stages of porcine TGE; certainly, there was florid regenerative activity in the crypts. The large bowel (for a change) was completely unaffected.

Coccidiosis in a Green Sea Turtle (Roger Kelly)

Most of the sea turtles that have so far come our way from the Moreton Bay morbidity/mortality study have had serious arguments with power boat propellers or crab-pot lines, etc. One recent accession, however, which was found dying in a local marine, had severe diffuse enteritis with a delicate fibrinous overlay. Histologically, there was heavy infection by an as yet unidentified coccidian; the severity seemed sufficient to have been responsible for death. Another, which had showed navigational difficulties clinically, was found to have a severe encephalitis histologically; again, the culprit seems to have been a protozoan.

10.

Nocardiosis in Homo sapiens (Dick Sutton)

(This is a follow-up to the story in the previous VP Report). Treatment with Bactrim was discontinued after 8 weeks and no relapse has occurred in this subsequent 2 months. The literature on *Nocardia brasiliensis* infection in humans is sparse. There have been 19 cases reported in the US literature, all lymphocutaneous, of which 14 primarily involved the forearm, 2 the face and 3 the leg. Only 3 had evidence of immunocompromisation; one having lymphoma and the other two being treated with prednisone. There was only one case without evidence of a wound or abrasion being the primary site of infection. Initial treatment in all cases was inappropriate because the clinical diagnosis was incorrect; sporotrichosis, mycobacteriosis, staph and streptococci infections being the favourite diseases. Despite this, all cases eventually recovered following prolonged courses of sulphonamides or sulphonamide-trimethoprim and surgical drainage and/or excision.

YEERONGPILLY VETERINARY LABORATORY

Babesia abortion (P. Ketterer)

Babesiosis was diagnosed in a full-term Hereford calf which was born dead. Four other recent abortions/stillbirths had been reported from this property. Necropsy revealed subcutaneous oedema of the head and neck indicating that the animal was alive at the onset of parturition. There was a large volume of blood-stained fluid in the thorax and pericardial sac. Histopathology showed extensive serofibrinous fluid within alveoli and marked congestion with focal thrombosis and necrosis of alveolar capillaries. Haemoglobin casts were present in renal tubules and *Babesia* sp. was present within many erythrocytes, although the species could not be identified. Babesial invasion of the foetus is an unusual event although affected pregnant cows sometimes abort, presumably due to severe foetal anoxia. There was no evidence of clinical disease in the cow in this case.

Ixodes holocyclus paralysis in bulls (R McKenzie)

One 10 year old Poll Hereford died and a two year old bull was affected in a herd in Nambour. Clinical signs were dragging the hind toes and sluggishness. The dead bull had 12 *Ixodes holocyclus* attachment sites on its scrotum and the sick bull had 8. Clinical chemistry and haematology values in the sick bull were normal.

Acute oxalate poisoning of cows by kikuyu grass (R McKenzie)

Acute oxalate poisoning was diagnosed as the cause of death in a five year old Friesian cow, and sickness in another cow. Both were seven months pregnant and became depressed and recumbent with hypocalcaemic signs after being let out into a paddock of poor pasture but with a lush patch of kikuyu (*Pennisetum clandestinum*) watered by a leaking water reservoir. The cows had previously been on a high calcium/low oxalate diet. A serum calcium concentration of 1.4 mmol/1 (normal 2.1-1.8) was measured in the surviving cow. The dead cow had a superficial rumenitis and calcium oxalate crystals in the kidney cortex. The surviving cow developed tenesmus and dysentery.

Analysis of kikuyu samples from the paddock revealed total oxalate content of 1.2% and soluble oxalate content of 0.3%. These concentrations would not normally be regarded as hazardous to ruminants (>2.0% soluble oxalate is hazardous). It is possible that the sample analysed was not representative of the pasture eaten.

Kikuyu has not been linked with acute oxalate poisoning before. The only other grasses to be so linked are (*Setaria sphacelata*) and buffel (*Cenchrus ciliaris*).

Combined sulphaquinoxalone and salinomycin poisoning in poultry (R McKenzie)

Six week old female replacement broiler breeder poultry on a Redland Bay farm were affected by the combined effects of sulphaquinoxalone and salinomycin toxicity when errors of dosage of both drugs occurred. **Nine thousand birds** in one shed were affected (100% morbidity) with known deaths totalling over 2500 (28% mortality) in the first six days.

The incident started when caecal coccidiosis was diagnosed by staff on the farm and treatment was started (Day 1) with Toltro (sulphaquinoxalone and diaveridine) in the drinking water. A double dose was accidentally given.

Simultaneously, an error in feed formulation through computer malfunction had led to the inclusion of 6-7 times the recommended concentration of salinomycin in the feed. The birds began to become ill and die on Day 2 (80 dead) with large mortalities following on Days 3 (500 dead), 4 (750 dead), 5 (750 dead) and 6 (450 dead).

Necropsies done on farm on Day 3 revealed poor clotting of blood and haemorrhages from the liver. Sulphaquinoxalone poisoning was suspected by the field veterinarian.

Necropsies at the laboratory on Day 3 revealed pale hearts, oedema of the lungs, small spleens and pale carcasses as well as prolonged clotting times. Histopathology revealed focal myocardial degeneration supporting ionophore toxicity. On Day 4, necropsies revealed subcutaneous oedema, excess pericardial fluid, pale heart, ascites and pulmonary oedema. Histopathology revealed skeletal muscle degeneration and necrosis of the hind limb muscles but not the pectoral muscle in addition to myocardial degeneration.

A sample of the water revealed 200 ppm sulphaquinoxalone (recommended 80 ppm) and feed had 397 ppm, salinomycin (recommended 60 ppm). Birds in a neighbouring shed received the same batch of feed without ill effect. The sulphaquinoxalone poisoning accounts for the haemorrhagic component and the salinomycin for the cardiac and skeletal muscle lesions.

Vibriosis in crayfish (P. Ketterer)

Deaths in 18 month old redclaw crayfish (*Cherax quadricarinatus*) occurred on an aquaculture farm where a systemic rickettsia-like infection had previously been diagnosed. Three live sick animals examined at the laboratory showed blisters and erosions on the telson and uropods. Histology showed aggregations of haemolymph cells in gills and hearts of all three animals and antennal gland of one animal. *Vibrio mimicus* was isolated in pure culture from the hearts of all three animals. Poor water quality was thought to predispose to the bacterial infection.

SOUTH AUSTRALIA - Vui Ling Tham

VET LAB. DEPARTMENT OF AGRICULTURE. SOUTH AUSTRALIA

Epizootic Haemopoietic Necrosis Virus (EHNV) in Redfin Perch in South Australia (Peter Phillips & John Humphrey [AAHL])

In January 1991 a massive kill of Redfin Perch (Perca fluviatilis L) occurred in Mt Bold reservoir in the Mt Lofty Ranges about 30 km south-east of Adelaide.

The fish were reported to be leaping out of the water, behaving in a frenzied manner and then floating apparently exhausted and dying. Local pelicans were reported to be so full as to be practically unable to get airborne! Several redfin of various sizes were caught in a moribund state and brought to Vet Lab for investigation.

All fish were in good condition but had empty gastro-intestinal tracts. All had some gill necrosis, pale, white-spotted livers and oedema of the cranial cavity. One had marked ascites.

Histopathology revealed severe focal necrosis and inflammation of secondary lamellae of the gills, focal necrosis of the gastric mucosa, marked necrosis of ventricular myocardium and focal perivascular hepatic necrosis. One small fish had focal necrosis of renal haemopoietic tissue. No viral inclusions were detected. A provisional diagnosis of Iridovirus (EHNV) was made.

Aeromonas sobria was cultured from many tissues and is thought to be a secondary invader. Tissue was sent to AAHL who cultured EHNV from them and detected EHNV by electron microscopy.

This is the first confirmed case of EHNV in South Australia. In retrospect a massive redfin kill in Lake Alexandrina a month earlier vas probably EHNV, however, suitable specimens were not received at the laboratory. It is interesting that the water authorities had begun pumping water from the Murray River to Mt Bold only a few days before fatalities began yet redfin near the pumping point in the river have not been reported to be affected.

VICTORIA - Grant Rawlin

VIAS. ATTWOOD

Infestation with Anchor worm in an Axolotl (Grant Rawlin)

Two Axolotls (*Ambystoma mexicanum*) were presented to a veterinarian showing agitated swimming habits. On closer examination 5 mm white parasites could be seen protruding from the gills and skin of the dorsal surface of the body and head of the amphibians.

One of the parasites had been detached by the axolotl and was sent for identification. The parasites were identified as anchorworms. The anchorworms (*Lernaea spp*.) are copepod parasites of fish. The parasite seen in the clinical syndrome is the adult female. Eggs are retained by female until hatching into free swimming young which feed on superficial mucus and debris causing no clinical problem. There are several moults during the free living stage. Mating takes place in the water and only the adult female burrows into the skin of the host to develop into the anchor worm.

Lernaea are usually parasites of fish but have been seen in axolotis. This amphibian was fed on occasional live goldfish which is a likely source of infection.

The axolotls were successfully treated with Trichlorofon 0.25 ppm over a period of 10 days.

Encapsulated Anasakis spp larvae found in wild caught sea fish (Grant Rawlins)

Nematode larvae were submitted from musculature of Orange Roughy caught in Victorian waters. They were numerous in several fish found in a wholesale market. The lesions presented as pale round 2mm fibrous lesions within the major muscles of the fish. On closer examinations, a 4-6mm nematode larva could be leased from each fibrous capsule. This was identified at Queensland Veterinary School as examples of *Anasakis* larvae Type 2.

The larvae found in fish are part of a cycle starting with me adult nematode which is found in the gastrointestinal tract of toothed whales. The developing larvae are thought to pass through a free-living stage and then to pass the other stages of their life cycle within the muscle of crustaceans, squid or fish and hence passed back to marine mammals and to other mammals, such as man, which ingest them. The human disease is associated with the consumption of raw, salted, or cold smoked fish. Infection in man causes two syndromes:

(a) Gastric anasakiasis which is characterised by acute stomach pains, nausea and vomiting 4-6 hours after ingestion as the larvae migrate through the stomach wall. These signs usually become chronic and if not diagnosed, last more than a year.

(b) Intestinal anasakiasis is seen as severe lower abdomen pain, vomiting, diarrhoea and fever a week after ingestion.

Stillbirths and Abortions Associated with *Leptospira pomona* Infection in Cattle (John Mackie)

In a mob of 80 Hereford cows, 16 gave birth to dead calves and 4 aborted in the last trimester of pregnancy. A number of the affected calves died during assisted birth. Most of the 20 affected cows were first calf heifers. M.A.T. titres to *Leptospira pomona* ranged from 1024 to $\geq$8192 (median = 4096, n = 8). M.A.T. ties to *Leptospira hardjo* ranged from 32 to 256. Leptospires were seen with the aid of immunogold staining of formalin-fixed foetal kidney. The cows had not received Leptospiral vaccination at any stage in their life. An interesting feature of the case was that stillbirth was the predominant clinical sign rather than late abortion.

Congenital Hypotrichosis in Poll Dorset Sheep (John Mackie)

Two 5 month old Poll Dorset weaner ewes in very good condition presented with absence of hair and wool from the anterior part of the face, the ears and the lower legs. Eyelashes were absent and there was increased lacrimation. Skin in affected areas was thickened, wrinkled, erythematous and greasy with mild scalding.

The histological features of affected skin were absence of fibres from follicles, dilation of follicles, follicular keratosis, hypertrophy and hyperplasia of sebaceous glands, mild acanthosis and mild hyperkeratosis. There was also focal crusting of the epidermis with accumulation of neutrophils in the superficial dermis and migration of neutrophils through the epidermis. Some papillae of follicles were widely separated from the base of the follicle, in contrast to skin section from aged-matched unaffected sheep where papillae were intimately associated with follicle bulbs.

The condition was diagnosed as congenital hypotrichosis and follicular dysplasia, of probable genetic aetiology. The owner of the stud reported that the occasional Iamb and weaner had been similarly affected over the last 10 years. Affected lambs were first noticed at marking time and destroyed before reaching 12 months of age, with no apparent improvement in their condition. There was no associated loss of body weight or other systemic effect.

The absence of fibres from follicles and the wide separation of papillae and follicle bulbs suggests an arrest in the normal cycle of follicular activity, apparently in the telogen stage, followed by shedding of old fibres, lack of formation of new fibres and accumulation of keratin and sebum in follicles, resulting in blocking of infundibular and dilation of follicles. A similar condition has been reported in Poll Dorsets in Queensland (Dolling & Brooker, 1966) where an autosomal recessive mode of inheritance was suspected.

Reference: Dolling CHS & Brooker MG (1966) J Hered **57**:87.

Fatal protozoal infection in an Emu (Chris Morrow, Catherine Hollywell, Helen McCracken and Bill Hartley, Victorian Institute of Animal Sciences, Attwood, Victoria 3049, Royal Melbourne Zoo and Taronga Zoo)

A sub-adult emu presented depressed and collapsed three hours later. Examination under anaesthesia revealed lacerations to the head and blood in the mouth and nasal cavity. The bird died one hour later vomiting fluid.

On post mortem the bird was found to be a male with black testes (1cm by 4cm). The liver and spleen were enlarged and rounded. The contusions to the head and upper neck were superficial and consistent with being attacked or self inflicted damage. No significant findings from bacteriological culture of the spleen or heart blood. Blood films had no specific changes. Some vacuoles were seen in lymphocytes. High levels of SGPT, SGOT, Alkaline phosphatase and CPK were observed.

Histologically in the spleen, liver and lung there were many roughly circular bodies (up to 50 µm in diameter) containing what were presumed to be various stages of multiple schizogony of an apicomplexan protozoan. A few similar bodies were seen in kidney and brain. In the lung many capillaries were blocked by the presumed schizogony and there was an associated marked pulmonary congestion. The massive destruction seen in these organs is consistent with the serum biochemistry.

Ultrastructurally, the bodies in the lung did not appear to be endothelial cells in the blood vessel lumen. The bodies were made up of various organelles including mitochondria. Morphology of these bodies was consistent with apicomplexans and similar to those seen in the schizogenic cycle of *Haemoproteus* infection in pigeons. Further ultrastructural investigations are under way as well as DNA analysis of the protozoa.

Possible pathogenesis: The protozoal infection could be from a *Leucocytozoon* or *Haemoproteus* or closely related genus. Death was probably due to pulmonary capillary blockage by protozoal thrombi. The failure to demonstrate gametogeny in the blood smear could indicate that the bird died from overwhelming asexual production (i.e. before red blood cell invasion occurred).

The bird had been bred at the zoo but was in contact with birds of various origins. It was not possible to sample in-contact emus at the zoo. This represents a new disease entity in emus.

REGIONAL VETERINARY LABORATORY, BAIRNSDALE

Fibrinohaemorrhagic Enteritis in a calf with presumptive Adenovirus Infection (Ian Jerrett)

A three week old Murray Grey calf was found dead in its paddock. Clinical illness had not been observed and the remainder of the mob appeared normal. The carcass was pale and the lower small intestine and large intestine was filled with blood stained fluid containing strands of fibrin. Patchy diphtheritic membrane formation was evident on the ileal mucosa. Histologically large basophilic intranuclear inclusion bodies were present in damaged capillary endothelium in the mucosa and mesenteric lymph nodes.

The pathology is characteristic of adenovirus enteritis, an uncommon enteric disease of calves. The epidemiology of the infection is not known. Overt disease is usually sporadic despite a reported high prevalence of antibodies in many areas. BVD virus, a known immunosuppressive agent, was isolated from the spleen of this calf.

Vegetative Valvular Endocarditis in an Ostrich (Kit Button)

A 10.5kg male Ostrich chick was presented to the RVL with a history of inappetance and lethargy. Proventricular impaction and/or infection were suspected and the bird was treated with mineral oil and tetracycline by mouth. The bird died after a few days. At necropsy, the proventriculus was grossly dilated with 557 g of grass, sand and cereal grains. The gizzard contained adequate pebbles, shell grit and sand (116g). There was extensive vegetative endocarditis of the aortic and mitral valves from which *Streptococcus bovis* was isolated.

Histologically thrombosis and bacterial colonies were present in the myocardium, spleen and kidney. Impaction of the proventriculus is frequently seen in young ostriches with infectious conditions.

Could proventricular impaction be a secondary effect, with infection or toxaemia, etc. causing intestinal ileus?

Yersiniosis in Birds (90/6746. 7541) (Kit Button)

A Peach-faced Lovebird and a Canary both died suddenly. Splenic enlargement was noted in both birds with multifocal colonies of ovoid bipolar bacteria and associated heterophils in spleen, lung and liver. *Yersinia pseudotuberculosis* serotype I was isolated in heavy pure growth from liver, lung and intestine of the Lovebird and *Y. pseudotuberculosis* serotype II was isolated from the same organs in the canary. Yersiniosis is a well-recognised cause of acute losses in birds.

REGIONAL VETERINARY LABORATORY. HAMILTON

Diseases of Ostriches (Cor Lenghaus, Joe Webber and George Riffkin)

In common with other laboratories, we are receiving an increasing number of Ostrich submissions.

Given the paucity of information that is available locally regarding husbandry, management and disease, it is inevitable that there have been problems. We have seen *E. coli* septicaemia in chicks a few days old, crop

and gizzard impaction with coarse plant fibre in birds a few weeks old, and other weak and lethargic chicks kept indoors on carpet in a room where the relative humidity was 90%! In growing birds, 2-3 months old we have investigated spontaneous, mid shaft femoral fractures in birds which seemingly had too little calcium in their diet, and limb weakness and outward rotation which seemed similar to perosis of domestic poultry.

Fixed and fresh tissues were received from a 5 months old ostrich which was noticed ill in the morning and died the same day. The owners had given the bird paraffin oil in an attempt to relieve an "obstruction" which they believed it had; the bird's neck had become noticeably swollen before it died. The referring veterinarian reported severe oedema and haemorrhage of the larynx, trachea and associated tissues at autopsy. Histologically there was an acute cellutitis of the larynx, pharynx and trachea. There was focal necrosis in the spleen and myocardium. There were thick plaques of fibrin and heterophils on the visceral pleura and air sacs. *Pseudomonas aeruginosa* was cultured in heavy, pure growth from the lungs and subcutaneous oedema fluid.

A mature male ostrich which had died after an illness of 3-4 weeks, was submitted for autopsy. There was hyperaemia and erosion of the epithelium of the pharynx, larynx and tongue. Air sacs were coated with a 5mm layer of white to grey green cheesy material. There were miliary abscesses in the lungs, with inspissated, cream coloured contents. A heavy growth of *Aspergillus* sp. was cultured from the lungs, air sacs, kidney, spleen and tongue.

Ketosis. Hypocalcaemia or Cerebral Oedema? (Deborah Seward)

January brought a week of heat wave conditions with temperatures soaring into the 40's. Two days following the cool change 16 of 50 pregnant Angus cows were reported dead. The animals had been deprived of water for approximately three days and pastured in paddocks offering no shade. The referring veterinarian was perplexed by the appearance of neurological signs in six of the surviving cows, a couple of days following access to water. These animals had varying degrees of posterior paresis, some were down. The hind limbs of one of the downer cows were splayed. The clinical signs and abnormal postures of the downer cows were assessed to be atypical of hypocalcaemia and ketosis. Post rehydration cerebral oedema with polioencephalomalacia due to sodium ion toxicity was the provisional diagnosis.

Euthanasia of the six animals was recommended based on clinical assessment and animal welfare considerations. Serum samples from two of the affected animals and brain and liver from a freshly dead cow were submitted. Serum analysis revealed evidence of partial rehydration; low calcium and elevated betahydroxybutyrate levels.

It was disappointing that no histological lesions were present in the brain sections examined. The short interval between rehydration and euthanasia may have been insufficient for recognisable lesions to develop.

Of the surviving cows, three more died over the subsequent week but interestingly, no abortions were reported over the following month.

Padovan D. (1980) Cornell Vet 70: 153-159
Riffkin G.G. *et al* (1981) AVJ 57: 532-533

Necrotic Glossitis in Fallow Deer (Cor Langhaus)

On two consecutive days in mid March 1990, we received almost identical specimens from two Fallow deer fawns. Given that the properties from which these samples originated were separated by at least 100km, and the scarcity of Cervid submissions, the coincidence was quite remarkable. The specimens consisted of the body of the tongue from each animal, the bulk of which had become completely necrotic. Both cases came from well-managed enterprises, where the first sign of disease that the farmers had noticed was a “swollen neck”. Multiple cases had occurred on both properties, all of which had been fatal.

Histologically, there were large areas of coagulative necrosis, with a reaction zone of leucocytes and early fibroplasia separating the necrotic from the viable tissue. The perception that the tissues had infarcted was reinforced by the presence of thrombosed blood vessels, some of which, at the perimeter of the lesion, had recanalised. There was a massive infiltration of mixed bacteria into the necrotic mass, including Gram positive cocci and bacilli, and Gram negative bacilli and filaments. *Streptococcus* and *Bacteroides* sp.were cultured.

Three weeks later we received a liver, 12 week old fawn from one of the above properties. This animal was bright, alert and had marked swelling of the intermandibular tissues. At autopsy, the only significant finding was an irregular epithelial defect over the right lateral body of the tongue. The lesion had a granulated base and measured 8 x 2cms. The ipsilateral retropharyngeal lymph node was enlarged.

Histologically, the tongue contained the only lesion of note. There was a deep ulcer present with significant bacterial and plant contamination present. In areas removed from the ulcer, there was vacuolation of epithelial cells in the stratum granulosum and a severe, resolving vasculitis in the sub-epithelial connective tissues. *Actinomyces pyogenes* and *Streptococcus* sp. were cultured from the tongue and affected lymph node.

It is tempting to think that the above represented no more than outbreaks of necrobacillosis and sequellae. However, weather conditions were mild and dry and overall management practices were good, as far as we were able to judge. Attempts were made at virus isolation from a range of tissues, at the CSIRO Animal Health Laboratory, Geelong, but without success. Follow-up blood samples for serology, showed reactions to Bluetongue virus, using the AGID test, although there were no reactions with a more specific ELISA test used. Possibly these results represent a cross reaction to another infectious agent, or a response peculiar to these deer.

NEW SOUTH WALES - Tony Ross

REGIONAL VETERINARY LABORATORY - MENANGLE

Hepatopathy in a Tasmanian Devil (Patrick Staples)

A six year old female Tasmanian Devil in a Wildlife Park, here in Sydney died suddenly. The animal was in very good nutritional condition and the only significant autopsy finding was that the liver appeared enlarged with a marked lobular pattern. Histopathology revealed a generalised acute periacinar hepatic necrosis typical of a toxic hepatopathy. Liver Vitamin E and selenium levels were 290 umol/kg and 29.8 umol/kg (dry wt.) respectively, which are well above deficient levels in domestic animals. The animal's diet consisted mainly of "spent layers" from a neighbouring poultry farm, and was supplemented by occasional wallaby heads from road kills. Taronga Zoo has had a number of Devils die with the same histological lesion, but the cause remains unknown. Suggestions are invited.

Ependymoma in a Cow (Patrick Staples)

Sudden onset of circling, blindness and disorientation was observed in a 10 year old Australian Illawarra Shorthorn cow. Autopsy revealed a large friable mass filling the ventricle of the rostral brain stem and compressing the surrounding brain parenchyma. There was a sharp demarcation line between the tumor and surrounding brain tissue. On histopathology the tumor consisted of branching papillary processes lined by ependymal-type cells. It was circumscribed by a light collagenous capsule which had areas of haemorrhage within it.

REGIONAL VETERINARY LABORATORY. WOLLONGBAR

Progressive ataxia in Charolais Cattle (Paul Gill)

A Charolais heifer developed progressive ataxia from 9 months of age. The disorder was characterised by a wide-based posture and a systemic gait with inco-ordination and misplacement of the hind limbs. Histological lesions were characterised by multiple, often confluent eosinophilic finely granular/fibrillar plaques through the white matter of the brain and spinal cord. Gliosis and vacuolation of the neuropil were frequently associated with the plaques. The basic effect is thought to be a failure of oligodendrocytes to maintain normal myelination at axonal internodes.

1080 poisoning in Santa Gertrudis calves (Paul Gill)

Two extensively managed Santa Gertrudis calves - an eight week old male, and a 12 week old female, fell dead after a short period of staggering and respiratory distress. The heart of the heifer appeared normal at necropsy but moderate multifocal coagulative necrosis of myocardial cells was evident in the left ventricular wall and inter-ventricular septum histologically. The heart of the male calf was enlarged and rounded. There was concentric hypertrophy of the left ventricular wall and septum and clots were present in the left ventricle. Histological lesions were limited to mild multifocal non-suppurative interstitial myocarditis in the left ventricle and minimal focal myocardial necrosis in the septum. Glutathione peroxidase activities in the blood of cohorts were normal. There was a history of recent use of 1080 on the property.

Lantana poisoning in introduced cattle (Paul Gill)

Lantana poisoning resulted in the deaths of 50/500 newly introduced adult Brahmans and illness in a further 30. Jaundice with a swollen liver was common necropsy lesion. Histological changes included diffuse fine vascular change in the cytoplasms of hepatocytes, periacinar to midzonal necrosis, cholestasis and severe acute nephrosis.

REGIONAL VETERINARY LABORATORY. WAGGA WAGGA

(John Glastonbury)

From 3 to 11 December 1990, Alit Ekaputra and Ketut Putra, of the Eastern Islands' Veterinary Services Project, Indonesia, gained experience at our laboratory in bacteriology and serology. If nothing else, Alit learnt a few of the finer points of golf.

"Lesser Loosestrife", *Lythrum hyssopifolia* Poisoning

Morbidity and case fatality rates of 2.5% (640) and 62.5% respectively were investigated in a 9-month old Merino weaner. The sheep were grazing an oat stubble which had been severely waterlogged during winter and contained considerable *L hyssopifolia*. Loss of weight and a "tucked up" appearance were the principal clinical findings. Chronic nephrotoxic nephrosis was detected histologically.

Suspected Toxicity in Sheep (Adrian Philbey)

Starting suddenly in early December, deaths in one paddock of a farm at June, containing 5-year old crossbred ewes, continued to late January, when 300 of 580 at risk had died and a further 100 displayed clinical signs. The sheep, which were in fat condition and non-pregnant or in early pregnancy were initially found dead, some with a fatty liver. Others were depressed for several days up to one week before dying. These affected ewes had neurological signs, characterised by mild ataxia, slightly wide-based stance, mild fore and hind limb proprioceptive deficits, reluctance to move and intermittent collapse when forced to exercise. Hepatic fatty change and variable fat necrosis were found at necropsy. Histologically, the most consistent finding was swelling of axons in the dorsolateral medulla oblongata, particularly the cuneate

nucleus, ranging from formation of spheroids to large irregular eosinophilic plaques. Spheroids also were scattered through the reticular formation. The significance of these histological findings is uncertain. Investigations are under way in an attempt to identify a toxic agent postulated to explain the mortality.

Algal Infection in Sheep (Adrian Philbey)

Systemic algal infection was found in two sheep from a mob of 300 2-year-old wethers in which one had died and 20 had diarrhoea and loss of weight over a period of 6 to 8 weeks. The wethers were in a grazing trial ("Supirrflock") on a superphosphate-fertilised, irrigated pasture, which had dried off in mid-December 1990. At necropsy, both sheep had generalised lymphadenomegaly, with green suffusion of lymph nodes, intestinal wall, liver and kidney. Histologically, there was necrotising lymphadenitis, chronic granulomatous enterocolitis and granulomatous portal hepatitis. Unicellular organisms were in subcapsular sinusoids of lymph nodes and associated with inflammatory foci.

Myelopathy in Sheep Exposed to Stagger Weed (*Siachvs arvensis*) (Adrian Philbey)

In two successive years, 20 to 80% of 1,000 7-month old mixed sex Merino wethers at Young developed a locomotory disorder while grazing pasture containing stagger weed that had been spray-topped one week previously to control grass seed. Affected sheep had a stilted gait, dropping of the hindquarters when in motion and knuckling of the hind feet. They would stumble and collapse when forced to exercise. Sheep often were found recumbent by the owner and would die of dehydration, misadventure or crow-pick unless the owner intervened. At necropsy, there were no gross lesions, but a demyelinating myelopathy, characterised by formation of phagocytic chambers and mild spheroid formation, was found histologically. Changes were more severe in the caudal portion of the spinal cord.

Salmonellosis (Adrian Philbey)

Linco-spectin is being promoted as an economic form of treatment for footrot. The dangers of the practice were demonstrated in a recent severe outbreak of salmonellosis associated with its use.

The linco-spectin was mixed on the farm and administered subcutaneously to 2500 6-month old Merino weaners. Within one week, 150 had died after brief periods of diarrhoea and depression. *Salmonella typhimurium* was recovered from material submitted to the laboratory and subacute typhlitis and enteritis as well as multifocal granulomatous hepatitis were found histologically. We postulated that linco-spectin broad spectrum activity destroyed the normal intestinal flora allowing *S typhimurium* to proliferate. The stress of yarding for treatment would have compounded the problem.

WESTERN AUSTRALIA - Ruth Reuter

ANIMAL HEALTH LABORATORIES, SOUTH PERTH

Giardia infection in a Straw Necked Ibis (Threskionis spinicollis) (David Forshaw)

During a banding programme, officers from the West Australian Department of Conservation and Land Management (CALM) noticed heavy mortalities in fledgling straw necked ibis at a colony south of Capel in the south west of Western Australia. One estimate was of 75% mortality of birds before leaving the nest. Chicks were either seen dead in the nest or weak and sometimes moribund.

Two moribund birds were examined and blood samples from 10 birds examined for titres to Newcastle Disease and Avian Influenza. No gross lesions were noted and no titres to either virus were detected. Histological changes were confined to the upper small intestine in both birds. These comprised mild infiltration of heterophils into the lamina propria of villi. No obvious change in length or morphology of the

villi was noted. Numerous protozoal organisms morphologically consistent with *Giardia* spp. were seen in the lumen of the bowel.

Examination of the faeces of five clinically normal birds from a different colony north of Perth revealed large numbers of *Giardia* trophozoites in one. Faeces were sent to the School of Veterinary Studies at Murdoch University W.A. and an attempt was made to culture the organism in vitro. The attempt failed but this together with a different morphology suggests that it is not the species which most commonly occurs in mammals. Histological examinations of the intestine from the five chicks failed to reveal any abnormalities apart from a trematode infection in one bird. No Giardia were seen.

Whether the *Giardia* are the cause of illness in the southern ibis colony is not clear. *Giardia* infections in humans, dogs and cats can be associated with no histopathological changes, however. The finding is interesting as to my knowledge *Giardia* have not been reported in birds in Australia. The breeding season of the birds is over this year and the birds in the affected colony have either left the nest or died. We hope to further investigate the problem next year if it recurs.

Tuberculosis (*M. bovis*) in 2 hyrax (*Procavia capensis*) (Ron Peet)

A routine post-mortem examination of an adult hyrax (*Procavia capensis*) from the South Perth zoo revealed a lung abscess approximately 1-2cm. in diameter. There were no other visible lesions but histopathology of this lesion revealed a necrotic centre surrounded by plasma/lymphoid cells, macrophages, fibroblasts and very occasional giant cells. Z-N stains revealed acid-fast organisms consistent with *Mycobacterium* spp. Fresh abscess material proved negative for bacterial growth on routine and initial mycobacteria culture but eventually (after approximately 7 weeks) mycobacteria did grow and was eventually typed as *M. bovis*. Three remaining animals in the colony were placed in quarantine and a decision eventually made to euthanase them. Post-mortem examination of these animals (one adult and 2 young males) revealed no gross lesions but histopathology on one hyrax (the old male) revealed numerous focal granulomas in the lung and liver. Z-N stains again revealed numerous acid-fast organisms in the lung granulomas, but they were rare in the liver lesions. Giant cells were noticeably absent from these granulomas which consisted mainly of large swollen macrophages surrounded by fibroblasts and plasma/lymphoid cells. Routine culture of the lung proved negative and mycobacterial culture is progressing. These animals originally came from Africa and after residing in England for some time, were imported into Australia. This exercise shows the value of full routine post-mortem examinations of zoo animals and classifying zoo premises as quarantine areas.

REGIONAL VETERINARY LABORATORY. ALBANY

Mycobacteriosis in an Australian Sea Lion (Ruth Reuter)

A small colony of Australian sea lions (*Neophoca cinerea*) is present off the southern coast of Western Australia. Members of this colony periodically visit the King George Sound and Princess Royal Harbour adjacent to the town of Albany, somewhat to the consternation of the local fishermen. In January a young male sea lion was seen in the harbour, apparently in some distress, with dyspnea and numerous lacerations on the skin. Since it was the breeding season, it was assumed that the young bull had been fighting. He was observed swimming in the harbour in the evenings and lying on the beach during the day apparently oblivious to his surroundings. Three days later he was found dead on the shore.

On post-mortem the lungs were consolidated and gray-white with purulent material in the airways and oozing from the cut surface. The bronchial lymph nodes were enlarged and moist. Gram stains of the purulent material did not show any bacteria, however large numbers of acid-fast bacilli were present on Ziehl-Neelsen stain. An organism isolated from the lung tissue appears to be one of the Mycobacterium tuberculosis complex on the basis of biochemical, drug sensitivity and pathogenicity tests. It is similar to one isolated from a colony of captive Australian sea lions and New Zealand fur seals at a marine park in

Western Australia in 1986. Although several reports have described such conditions in captive seals and sea lions, this appears to be the first instance of identification of this organism in a wild marine mammal.

Porcine Intestinal Adenomatosis (Ruth Renter)

Six, 3 month-old piglets from a group of 60 free range piglets died over a period of several weeks. The piglets were all from 2 sows in the 12-sow herd. Clinical signs described included wasting, illthrift, swelling around the jaw and "mucus" in the throat commencing at 6 weeks of age. Two animals which showed similar signs had some response to drenching for worms. One piglet was submitted for post-mortem examination.

The pig was in fair body condition, with scaly skin on the legs. Scrapings showed large numbers of *Sarcoptes* sp. mites present. There was subcutaneous oedema of the neck and face. The ileum and caecum were moderately thickened with ridging of the surface. The intestinal content was watery. The mesenteric lymph nodes were enlarged. On histology of the intestine, the mucosa was thrown into folds with hyperplasia of epithelial cells and a mononuclear inflammatory cell infiltrate in the lamina propria. Warthin-Starry stain showed large numbers of *Campylobacter* sp. in the apices of the crypt cells.

Suspect B-mannosidosis in Saler cattle (Ruth Reuter)

Frozen embryos imported from Saler cattle in Canada are being implanted into surrogate cows at an embryo transfer centre near Albany. In the latest of a series of transfers, 70 calves have been born to date from 100 implanted cows. Five calves from one particular mating have been lost. Three of these have been submitted to the laboratory for examination. The length of life of the calves was 8 hours, 2 and 7 days. Gross findings were unremarkable apart from a consistent mild hydrocephalus and some suggestion of doming of the cranium. One calf had a dislocated hock joint.

On histology the characteristic vacuolation of a lysosomal storage disease was seen in neurons throughout the brain, tubular epithelium of the kidney and macrophages in lymph nodes and thymus. B-mannosidosis has been identified in the Saler breed in North America, and members of the particular line used as the source of the frozen embryos imported have been identified as affected. Further studies are underway on this problem.

NEW WESTERN AUSTRALIAN CORRESPONDENT FOR THE A.S.V.P.

It is with some regret that I must relinquish my position of ASVP correspondent for Western Australia. I have resigned from the Department of Agriculture effective April 1st (This is not an April fool's Joke!) and will be moving to Adelaide in the near future. Although I am sad to be leaving Albany, (which I think is one of the most beautiful spots in the country!) and the friends and acquaintances I have made in the time I have been here, I am looking forward to the challenge of working with Rob Rahaley and the crew of Veterinary Pathology Services in South Australia.

I intend to keep in touch via the newsletter etc. so this is not the last word from me by any means! With my departure, Dr. Ron Feet of the Animal Health Laboratories in South Perth has "volunteered" to take over as correspondent for the ASVP. I'm sure he will do an excellent job.

NORTHERN TERRITORY - Lorna Melville

BERRIMAH AGRICULTURAL LABORATORY

A Case of Suspected Screwworm Fly in a Feral Pig (J Lee)

During a Northern Australian Quarantine Strategy (NAQS) survey in Coburg National Park, feral pigs were shot and examined for evidence of exotic diseases.

One aged boar was found to have a large suppurating wound on the point of the left shoulder. This was infested with live maggots. SWF was suspected.

On postmortem examination the lesion extended through the dermis into the subcutaneous tissues. A thick zone of granulation tissue surrounded a central mass of necrotic tissue which contained large numbers of live maggots.

The wound was filled with a thick creamy malodorous exudate. The regional lymph nodes were swollen and contained a similar exudate to that found in the primary lesion.

All maggots were collected into boiling water and then into 70% alcohol for identification. Adult flies were also collected from the large number of flies, which were present as soon as the pig was shot, which presumably bad been attracted by the odour of the suppurating wound.

Tissues were collected for histology and bacteriology. Identification of the adult flies revealed C. megacephala and C. saffranea but no C. bezziana. Examination of the maggots revealed no C. bezziana.

A diagnosis of SWF was ruled out. This appeared to be a case of secondary strike of an existing infected wound. However, SWF would probably first be detected in very similar circumstances if it gained entry to Australia.

Lead Poisoning in Magpie Geese (M Bell)

As part of a three year survey by Peter Whitehead and Kurt Tschirner of the Conservation Commission of the NT (CCNT), I was asked to examine kidneys from 24 Magpie Geese. The Magpie Geese were from a local reserve and were unable to fly. Histological examination showed acid-fast intranuclear inclusion bodies present in 19 of the 24 kidney samples.

A short history follows of Peter's and Kurts investigations with lead poisoned geese this year, leading up to the 24 samples submitted.

On the 16 August 1990 during a water fowl survey at Knuckeys Lagoon it was noted that there were a few geese reluctant or unable to fly. Three were caught by hand, killed and the subsequent examination of their gizzards revealed 1, 2 & 4 lead pellets respectively.

A later visit on 26 August 1990 showed that the number of sick geese present had increased significantly. Four more geese were caught and each was found to have one pellet in the gizzard. They all showed wasted muscle tissue and enlarged gall bladders.

This lead to a survey conducted on 11 November 1990 to catch as many sick geese as possible at Knuckeys in a two hour period to assess the extent of the problem. The gizzards of these 24 geese were examined for lead pellets and compared with the results of the inclusion body tests (see table). The only bird out of 24 not to show evidence of lead poisoning was #10, although its gizzard lining showed some deterioration and staining.

Apart from Knuckeys Lagoon, two lead poisoned geese were brought in from McMinns Lagoon on 5 September, 1990 and at Howard Swamps on the 1 September 1990. Four out of twenty five hunter killed geese had pellets present in the gizzard.

In response to this situation the use of lead shot has now been banned from the four CCNT shooting reserves starting next year.

The full report "Lead shot ingestion and Lead Poisoning of Magpie Geese foraging in a northern Australian hunting reserve" has very interesting details of lead shot concentration in soils of the affected reserves.

Magpie Geese Taken From Knuckevs Lagoon 11/9/90

	Yes	**No**
Inclusion bodies in Kidney	19	5
Lead in gizzard	17	7

1990 Arbovirus Sentinel Program (L Melville)

The very dry seasonal conditions during the 1990 wet season resulted in a significant reduction in the number of viruses isolated compared to other years. To June 1990, 124 confirmed isolates were made from 1096 bloods.

Identification of these showed most were EHD group viruses. Significantly no isolates were identified from the Bluetongue group. This is the first time in 10 years that no bluetongue activity was recorded. The absence of bluetongue was confirmed in sentinel herds at three separate locations in the northern part of the NT.

As part of the North Australian Quarantine Strategy insect collections are now being made in conjunction with sentinel bleeding. It is hoped information gained by this will aid in understanding the epidemiology of these viruses in the Northern Territory.

NAQS funding has also enabled the establishment of several sentinel poultry flocks in localities along the northern coastline. This project has had mixed success. In one settlement all the birds vanished. The locals claimed the dogs ate them all in one night. In other sites the pythons and native cats have found our birds a good dietary supplement.

Another NAQS project has involved regular bleeding of tagged feral pigs. These samples have been tested for antibody to a range of viruses. While they have all been negative to the arboviruses tested for a significant number have had antibody to porcine parvovirus.

ALICE SPRINGS VETERINARY LABORATORY (Denise McEwan)

Nitrate poisoning of cattle

Nitrate poisoning is suspected as the cause of stock losses on a station where cattle, mainly cows and young stock were found dead around a particular bore. A test of trough and tank water showed nitrate levels around 1250ppm (maximum recommended levels are 200ppm). No autopsies were performed because of the state of the carcases. Differential diagnosis includes, botulism.

A liver syndrome in weaner cattle

Approximately 25 out of a total of 5000 weaner cattle (approx. 9 months of age) died in one herd. The weaners were noted to be unthrifty for approximately one month. On post mortem 2/3 had quite marked liver pathology, comprising moderate portal mononuclear cell infiltrations, with peribile ductular oedema, fibrosis and bile duct proliferation. There was some resemblance to facial eczema - sporodesmin toxic lesions. The cause of the lesions was not determined. It is known that there was low level flooding of the region some three months earlier.

Nervous syndrome with cattle deaths

This section is investigating cattle losses (including bulls, cows and weaners - total of 20 deaths). Cattle appear to be hyperexcitable, show incoordination or swaying of the hindquarters, may go head-over heels and may pivot in a tight circle. One affected weaner tested had a high CPK (615-U/L) and high AST (167 U/L) but has slowly recovered after being removed from the affected paddock. The only known toxic plants in the area are Whitewood (Atalaya hemiglauca) and Indigofera linnaei.

TASMANIA - Judith Handlinger

ANIMAL HEALTH LABORATORY. LAUNCESTON

Anisakid Nematodes in Fish (Anne Clark)

Reports have been made for many years of anisakid nematodes, including *Anisakis simplex*, in visceral samples of wild fish such as blue grenadier, jack mackerel, gem fish and orange roughy, caught in many Australian coastal waters.

Recently, with the increase in local consumption of exotic cuisine using raw, lightly marinated or cold smoked fish. Attention has turned to the potential zoonotic risk if anisakid larvae occur in edible flesh. Human anisakidosis presents usually as an acute gastrointestinal disorder with symptoms suggestive of gastric ulcers or tumours, appendicitis, and Crohn's disease for example. A recent history of raw fish consumption assists in the differential diagnosis.

No confirmed cases of anisakidosis have been reported yet in Australia. Cases continue to be reported from countries overseas such as Japan, North America and parts of Europe.

In Tasmania, farmed salmonids (rainbow trout and Atlantic salmon), striped trumpeter, tuna, trevally, trevalla, boar fish, garfish, squid, and occasionally jack mackerel are preferred by the leading sushi chefs. Surveys of these species were commenced by Sea Fisheries Division personnel working with the Fish Health Unit of the Tasmanian Department of Primary Industry early in 1990.

Preliminary results are as follows: trevallas, 10 whole fish examined by pepsin-acid digest ,none infected; striped trumpeter, 20 whole fish and 48 visceral samples examined, 63 infected (mostly in the viscera 2 fish with infected fillets); farmed salmonids, 70 whole fish and 530 visceral samples examined, one fish infected (viscera and belly flap).

It should be noted that as well as farmed salmonids being a low-risk species due to their commercially prepared diet, Tasmanian cold smoked salmon is usually prepared from pre-frozen fish.

The following are guidelines to minimise any risk to humans:

1. For general use, fish should be properly cooked right through.

2. If fish is to be eaten raw, it needs to be frozen to a core temperature of -20°C within 24 hours of capture and held at that temperature for at least 24 hours to kill any larvae.

3. If fish is to be marinated, a mixture of 15% salt and 57% acetic acid will kill any larvae if used for at least one week, with a ratio of fish: brine of preferably 1: 1, but not more than 1.5:1.

4. Anisakid nematodes can be seen in thin fillets of white fleshed fish by candling prior to consumption.

5. Fish should always be gutted, cleaned and chilled soon after capture not only to impede possible nematode migration from viscera to edible fillets but also to ensure high quality product, especially important if it is to be consumed raw.

Jelly fish deaths in Atlantic salmon (Judith Handlinger & Anne Clark)

Focal gill necrosis, sometimes extending to the primary lamellar vessel, marked gill congestion, skin wheals, and lysis of underlying muscles was seen with jelly fish stings of Salmonids this summer.

The initial observation was with Portuguese Man-o'-war ("blue bottles", *Physalia physalis*) which had been concentrated by the prevailing wind into floating cages. Distressed and dead fish were also observed. "In bucket" observation showed death of smolts (approximately 200g) within 30 minutes. Older fish (2-4 kg) showed fewer deaths though many survivors showed linear wheals which persisted to harvest as depressed scars.

Similar gill lesions had been seen earlier in the summer, and close observation showed gill lesions of this type in moribund smolts associated with the presence of the large red jelly fish (*Cyanea capillata*), and swarms of a small as yet unidentified jelly fish.

Multiple red skin spots attributed to jelly fish have been seen in Tasmania once previously (S. Percival and C. Foster, SALTAS pers comm.)

The Portuguese Man-o'-war is well recognised as predator of small fish. There is one literature report of mortalities in farmed Atlantic Salmon associated with jellyfish (Bruno and Ellis, 1985). Deaths occurred along the coast of Scotland, some again associated with *Cyanea capillata*, others with large numbers of a small (9-llmm) jellyfish identified as *Phialella quadrata*.

Portuguese Man-o'-war is actually a colony of several types of symbiotic polyps, with a float polyp, and separate stinging, reproductive, and feeding polyps. The stinging polyps have long trailing tentacles up to 40ft long (12m), armed with single celled stings called nematocyst containing a coiled filament which penetrates the prey and injects a protein toxin. Small portions of the tentacles may break off, and like the small jelly fish listed above, are likely to be caught in gills and cause considerable damage without being readily noticed. They are usually present in waters too warm for salmonids. *Cyanea* prefers colder water.

Several discharged nematocysts were seen in epithelial sections from skin wheals, but their appearance and number was not sufficiently characteristic to suggest that they can be consistently identified in stings.

Ref: D.W. BRUNO, A.E. ELLIS, Bull.Eur.Ass. Fish Pathol. 5 (3), 64,1985

JOBLINE

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Dr. Bill Hartley
C/o Sydney Zoo
Sydney
Australia

I8th February 1991

Dear Dr. Hartley,

Stepher Duffell, who at present works for ICI as a Veterinary Pathologist suggested I might like to contact you concerning a problem that we are experiencing.

I set up this laboratory about 6 years ago, to take in Diagnostic Veterinary Histopathology from practices and also to sub-contract to the commercial Diagnostic laboratories. I am pleased to say that in that time we have experienced almost an exponential growth each year and on average, we see 1800-2000 samples per month. In fact we see more Diagnostic Veterinary Histopathology than any other laboratory in the country. Dr. Vanda Lucke, who has recently retired from Bristol University comes two days a week and I have recently taken on a full time assistant, but despite this help, we are sti11 fairly pushed and we are finding no time for the interesting parts of the work, which are the follow-ups, writing up of interesting cases and establishing a data base.

There is in this country a very severe shortage of Veterinary Pathologists and I suspect this is probably reflected in other areas around the world and, when I have advertised posts, 1 have had very few suitable candidates.

One possibility that occurred to me was that, in veterinary practice, Australian and New Zealand graduates often come to England for a year or so, during which time they see Europe and then return and take up their careers. I was wondering if there was perhaps anybody in the pathology field that may wish to do this, with the potential of having perhaps somebody new each year.

I would greatly appreciate your comments on these possibilities. I would also appreciate it if you could give me some idea how I go about attracting somebody of this type, who to contact and, whether you know of anybody who might be interested in this proposal.

Many thanks,

Yours sincerely