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VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
Queensland Department of Primary Industries
Toowoomba Veterinary Laboratory
PO Box 102
Toowoomba Qld 4350
Ph. (076) 314365 Fax (076) 331918

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EDITOR: Jim Taylor

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DEADLINE FOR NEXT VET. PATH REPORT IS JULY 31, 1995

SECRETARIAT

PO Box 114 Walkerville SA 5081
Phone: 08 3446337 (Pat Bosence) Fax: OB 3449227

ASVP EXECUTIVE 1991-1993

President	John Gibson	Toowoomba Vet. Lab, PO Box 102 Toowoomba Q 4350	076 314352
Secretary	Jim Taylor	Toowoomba Vet. Lab, PO Box 102 Toowoomba Q 4350	076 315365
Treasurer	Grant Campbell	Toowoomba Vet. Lab, PO Box 102 Toowoomba Q 4350	076 314410
Committee Members			
	Ross McKenzie	Yeerongpilly Vet. Lab, 665 Fairfield Road Yeerongpilly Q 4065	07 3629432
	Geoff Mitchell	VPS, PO Box 1119, Coorparoo DC Q 4151	07 3918500
	Dick Sutton	Dept. Vet. Path., University of Queensland St Lucia Q 4067	07 3772565

APPOINTMENTS

Chairperson (Registry of Domestic Animal Pathology)	Tony Ross
Newsletter Editor	Jim Taylor
Coordinator (Training Committee)	Robin Giesecke

CONVENOR - SLIDE OF THE MONTH

Rod Reece **National Registry of Animal Pathology, EMAI, Private Mail Bag 8, CAMDEN NSW 2570**

STATE REPRESENTATIVES

Queensland	Greg Storie, Yeerongpilly Vet Lab, 665 Fairfield Road, Yeerongpilly Q 4065	07 3629555
Victoria	Malcolm Lancaster, Dept. Agric. PO Box 388, Benalla Vic 3672	057 611100
South Australia	Ruth Renter, VPS, PO Box 96, Plympton SA 5038	08 3623544
New South Wales	Paul Gill, UVL Wollongbar 2480	066 240298
Western Australia	David Forshaw, Regional Office, WA Dept Ag, Albany 6330	
Northern Territory	Anton Janmaat, PO Box 79, Berrimah 0828	089 895511
Tasmania	Barry Munday, Uni Tasmania, PO Box 1214 Launceston 7250	003 243812

EDITORIAL

With this edition of the "Vet Path Report" you should receive at least three loose sheets. One for nominations for positions on the new executive and a second for registration for this year's conference. Both of these are to be returned to me by the due date. The third is the postal vote on the issue of affiliation with the AVA. This is to be returned to Pat Bosence at the Secretariat in South Australia for counting.

In accordance with the resolution passed at the last AGM we are now putting a postal vote to all members. The following points of clarification were recently obtained from Dennis Eager, Chief Executive of the AVA, **and all members should be clear on these two points**.

1. All full members of the special interest group (ASVP) would be required to be members of the AVA. Ordinary membership fees of the AVA range from $310-$390 depending on your State. **Associate membership is of the special interest group only**, not of the AVA, and as associate members of the ASVP you cannot hold office in the Executive and you do not have voting rights. The current AVA Executive has adopted a position of inaction against veterinarians who are associate members of special interest groups, however, we have no assurances this will be the attitude of future Executives.

2. As a special interest group we could still hold our own conference, independent of the AVA without having to pay fees to the AVA and the AVA is happy to promote the conference through mediums such as the AVA News.

I also again appeal to Victorian members to consider nominating an Executive by convention. Victoria is the next state for the Executive, but I would encourage any members from any state to consider positions on the Executive.

Our Tasmania correspondent, Barry Munday, has requested case material of Sarcocystis infection in mainland macropods. If you have any material could you contact Barry at the address on the inside cover of this report.

I look forward to seeing you at this years conference, May 20-21 at VIAS Attwood.

Jim Taylor
Honorary Secretary.

NOTICE OF THE ANNUAL CONFERENCE AND ANNUAL GENERAL MEETING.

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY.

DATE: MAY 20th and 21st.

VENUE: Victorian Institute of Animal Science,
Mickleham Road, Attwood
Melbourne, Victoria.

CONFERENCE THEME:
Dermatopathology

GUEST SPEAKER:
Dr Kenneth Mason B.Sc., M.V.Sc., F.A.C.V.Sc.
Animal Allergy @ Dermatology Service
Albert Animal Hospital
Springwood, Queensland 4127

CALL FOR MEMBER PRESENTATIONS.

Any member wishing to make a presentation at this years conference on the theme of dermatopathology or any other topic please submit an abstract or copy of the presentation to the Secretary at Toowoomba Vet Lab PO Box 102 Toowoomba no later than **MAY 1, 1995**.

Papers should be submitted on A4 paper on computer disc (WP 5.1). No facsimile transmissions please.

Agenda items and any relevant documents for the annual general meeting should also be submitted by **MAY 1, 1995**. The Annual General Meeting will commence at 3:30 pm Saturday the 20th.

Presentations should be 15-20 minutes duration.

Jim Taylor
Honorary Secretary
Australian Society for Veterinary Pathology Inc

QUEENSLAND - Greg Storie

Avian Influenza - Jim Taylor (Toowoomba Vet Lab), Laurie Gleeson (AAHL)

In December 1994, a producer at Coominya in the Lockyer Valley observed increasing mortalities in his layer flock of 16000 birds. Mortalities increased to 80-100 birds a day with signs of swollen dark combs and wattles, green loose droppings and "hot" legs, feet and joints. Fowl cholera was suspected and 4 live birds were submitted to the Toowoomba Veterinary Laboratory for examination.

Gross lesions were seen in two of the birds. The affected birds had mild to moderate swelling of the combs and wattles with cyanosis and haemorrhage at their tips. Scattered subcutaneous ecchymoses were also present around the hocks. The head swelling was due to locally extensive subcutaneous oedema. Both birds had a mild suppurative exudate in the peritoneal cavity and mild opacity of the airsacs. One of the lesioned birds also had a small foci of pallor in the breast muscle and focal haemorrhage in the crop wall.

After 24 hours, aerobic bacteriological culture from numerous tissues had no significant growth. Samples from the two lesioned birds and one of the non-lesioned birds were despatched to the Australian Animal Health Laboratory at Geelong for exclusion of Avian Influenza.

At AAHL impression smears of pancreas and kidney were stained using influenza A and NDV monoclonal antibodies. Pancreatic and kidney impression smears were strongly positive for avian influenza virus antigen, and this provided early presumptive evidence that avian influenza virus was the cause of the disease. All samples were negative for NDV. Histopathologically the sections of comb and wattle had marked diffuse oedema with multifocal to locally extensive areas of coagulative necrosis and haemorrhage associated with mononuclear perivasculitis and vasculitis. The lesion in the breast muscle was a focal area of acute myonecrosis. Both birds also had a mild diffuse peritonitis. The muscle, comb, wattle and peritonitis lesions were strongly positive for influenza virus on immunoperoxidase staining.

Twenty hours after egg inoculation haemagglutinating activity and classical orthomyxovirus particles were detected in allantoic fluid, and the virus identified as avian influenza virus H7N3. The nucleic acid sequence of the haemagglutinin gene was different from the H7N3 isolated from Bendigo in 1992.

Perennial Ryegrass Staggers - Jim Taylor, Grant Campbell (Toowoomba Vet Lab)

During early summer on two dairy farms in the Darling Downs region, veterinarians investigated a neurological syndrome in milking cows. Five cows were affected out of 120 on one farm and 15 of 60 cows were affected on the other. Clinically the animals were agitated, had muscle tremors and would stagger and collapse if exerted. If left alone affected animals appeared to recover. Both herds were grazing pasture containing perennial rye-grass (Lolium perenne). There were no significant abnormalities in the haematological or biochemical profiles of the cows tested. Samples of rye-grass submitted from both properties were heavily infected with the endophyte Acremonium loliae. One sample was sent to Centaur International Hamilton for lolitrem assay. The sample had 7.1mg/kg of lolitrem B. This is believed to be the first confirmed case of Perennial Rye-grass Staggers in Queensland.

Cyanosis in piglets - Jim Taylor

One litter of pigs in an 80 sow herd had two ten day old piglets with marked diffuse purple discolouration of the skin. The discolouration was most noticeable in the ventral skin of the carcass. Both pigs also had mild subcutaneous oedema in the inguinal area and marked oedema of the mesocolon and the retroperitoneum. Histologically the skin had diffuse superficial congestion and haemorrhage. Vessels in the skin and oedematous areas had swelling of the endothelium with occasional endothelial necrosis and perivasculitis. Many endothelial cells in these and other locations contained large basophilic intranuclear inclusions suggestive of adenovirus infection.

Papovavirus Disease in Peach Face - Yeerongpilly Vet Lab

Two birds died in an aviary at Lowood which contained 19 peach face lovebirds. A male bird (8 mo) examined on the day of death was in good body condition with areas of subcutaneous haemorrhage on the ventral neck and posterior abdomen; the liver showed patchy congestion and the spleen appeared small. Histological findings were: areas of necrosis and haemorrhage in the liver with irregular distribution; lymphocyte depletion and single cell necrosis in the spleen with numerous macrophages(?) showing large vesicular nuclei; kidney glomerular tuft cells and feather shaft and follicle cells in the skin showing large vesicular nuclei.

The changes seen are consistent with papovavirus infection and the characteristic nuclear swelling has been shown to be associated with the presence of virions.

The aviary was in the Lowood avian influenza outbreak control area and pancreas smears were negative for AI in the FA test.

Alkaligenes denitrificans abortion in a horse - Yeerongpilly Vet Lab

Alkaligenes denitrificans was isolated in pure culture from the lung and stomach of an aborted foal with severe purulent pneumonia and in mixed culture from the placenta and cervix of the mare. The attending veterinarian reported subsequently that of 5 pregnant mares on the property, all had aborted. Only 1 of these abortions was investigated through this laboratory.

Syndrome of nephrosis and lymphocyte necrosis in cattle - Yeerongpilly Vet Lab

Three dead and 12 sick animals were reported in a beef herd of 120. The animals were penned and fed a proprietary ration. The water source was a stagnant watercourse. Affected animals were 3-6 months old. Anorexia, some scouring, incoordination and staggering were seen clinically. The body temperature was normal. Swollen liver, enlarged mesenteric lymph nodes and black ulceration of the abomasal mucosa were seen at necropsy.

Histology of samples from one animal showed the following:

abomasum - oedema of submucosa; oedema and segmental necrosis of mucosa.
large intestine - hyperplasia of the lymphocyte follicles and prolapse of glands into lymphocyte follicles.
ileum - complete loss of lymphocytes from Peyer's patches and replacement by macrophages.
mesenteric lymph node - follicles poorly defined with macrophages replacing them, prominent individual cell necrosis of paracortex.
kidney - tubular epithelial necrosis at the corticomedullary junction.
spleen - prominent individual cell necrosis of follicular lymphocytes.
lung, brain, heart, liver - no significant change.

Serum biochemical profiles were done on 4 animals and 3 of these showed evidence of liver and kidney damage - elevated GLDH, bilirubin and urea. Blue green algal toxins, mycotoxins and mucosal disease were included in the differential diagnosis. The disease problem ceased following 4 inches of rain which cleared the creek of stagnant water. The water was therefore thought to be the source of the toxin. The proprietary ration was used without incident on numerous other properties in the area.

Department of Biomedical and Tropical Veterinary Sciences, James Cook University, Townsville, 4811 - PW Ladds.

New (BSc. honours) research projects in pathology to be undertaken this year include skin lesions in young crocodiles, and cell mediated immunity in ruminant genitalia. Interesting diagnostic cases seen recently were:

Canine: Further cases of babesiosis in young pups; disseminated aspergillosis in a German Shepherd (presented for veterinary examination because of anorexia and weight loss, and found clinically to have lymphadenopathy and a large abdominal tumour); multifocal subcutaneous botryomycosis; fulminating acute Streptococcus G cellulitis with extensive skin necrosis in a greyhound from a kennel in Darwin where 8 other dogs were similarly affected and did not respond to treatment; disseminated focal meningoencephalitis, myelitis, neuritis and myositis caused by Neospora common; several unusual neoplasms including a probable subcutaneous liposarcoma and a very large and aggressive pulmonary adenocarcinoma - considered to be primary in that location.
Feline: Osteosarcoma was diagnosed in an 11 year old cat which had been depressed for a few days prior to examination - which revealed a hard tumour "on top of the head". At necropsy firm tumour tissue extended in a wedge-shaped fashion from the skull deep into both cerebral hemispheres.
Other: Granulomatous meningoencephalitis caused by blood flukes and especially their eggs, resulted in hydrocephalus and lesions targeting blood vessels in a Green Sea turtle (*Chelonia mydas*) which was found disoriented and unable to feed; scoliosis in several *Crocodylus porosus* hatchlings in a wildlife park was considered to be a consequence of osteomalacia due to inappropriate diet; severe diffuse mycotic airsacculitis and pneumonia was the cause of death of a 20 month old ostrich.

VICTORIA - Malcolm Lancaster

CENTAUR INTERNATIONAL (Benalla)

Listeria myelitis - Judith Nimmo Wilkie

Twenty adult sheep, in two mixed sex mobs of about 500 animals became paralysed in the hindquarters. The paresis was usually bilateral but some cases were unilateral. Affected animals usually remained bright and alert. There was no recent dipping, shearing, vaccination, hay or silage feeding. The sheep were on native pasture or lucerne. The outbreak occurred in spring after a period of cold weather. Three sheep were necropsied and there was a mixed, principally mononuclear, meningomyelitis with microabscesses typical of encephalitic listeriosis restricted to the lumbar cords. Intracellular Gram positive bacilli were present within the microabscesses. Listeria monocytogenes was isolated. No brain lesions were seen.

Prototheca mastitis - Judith Nimmo Wilkie

Prototheca sp., a yeast-like achlorellic alga, was isolated from the milk of a cow with a persistently high milk cell count. This environmental organism is a facultative pathogen of the mammary gland. Host cases of protothecal mastitis are sporadic but outbreaks may occur.

VICTORIAN INSTITUTE OF ANIMAL SCIENCE (Attwood)

Enteritis in ostrich chicks - Kit Button, Grant Rawlin

Approximately 170 of 280 five day to five week old ostrich chicks died on a Victorian farm after a short clinical course (often less than 12 hours) of mucoid to watery diarrhoea. Four waves of mortality occurred over a period of 3 months. In two of the waves, mortality (death or euthanasia in extremis) was 100%.

Initially, one live affected chick and 2 composite faecal samples were submitted to VIAS Attwood. Gross findings in the chick were limited to the intestines; SI, caecal and LI contents were fluid and mucoid. Further samples have been received and are being processed at present.

Histological lesions in the SI and LI were of a patchy distribution. The lamina propria appeared slightly hypercellular with increased numbers of mononuclear cells and a few scattered heterophils. Numerous enterocytes were degenerate or necrotic; the process resulted in rounding of enterocytes with cytoplasmic eosinophilia and sometimes clear cytoplasmic vacuoles. Some otherwise normal enterocytes still attached to the basal layer had circular eosinophilic bodies in their apical cytoplasm.

No Salmonella, Yersinia or Campylobacter sp. was isolated from composite faecal samples nor from a colon swab of the autopsied chick. No cryptosporidial oocysts were seen in any faecal smears. E.coli, typed as 0 25 was isolated from the faeces and intestine.

No haemagglutinating agents were detected after 2 passages of faeces, intestinal and liver homogenates through the allantoic sacs of 9 day old SPF embryonated eggs and no CPE was seen on monolayers of primary chicken kidney cells.

Negatively stained intestinal material revealed clubbed bodies of approximately 50 nanometres diameter consistent in morphology with coronavirus particles.

The above findings are consistent with a single case report of coronaviral enteritis in an ostrich chick (Frank et al, Journal of Zoo and Wildlife Medicine, **23**:103-107, 1992). Outbreaks of lethal diarrhoea in ostrich chicks have been occurring in most Australian states over the past few years.

Staff at VIAS are attempting to culture cell lines from ostrich embryos to facilitate future virological examinations. The source of the infection is not known; however, some ostrich farmers are now taking steps to keep wild birds out of their chick runs.

Perkinsosis in abalone - Grant Rawlin

Six abalone sourced from Port Lincoln, South Australia were submitted from the processor after gutting and washing. They all presented with multiple small pale brown circular lesions in all areas of musculature.

Histopathology of the lesions showed minor mononuclear inflammatory cell infiltration along with unicellular protozoan cells with comma-shaped nuclei. Signet ring-shaped cells typical of Perkinsus were seen. After several days in anaerobic media, samples of muscle were stained with Lugol's iodine and many spores were seen. Perkinsosis is an ulcerative syndrome of abalone caused by Perkinsus olseni. This syndrome has been reported in wild caught abalone in South Australia in the early 1980's. The syndrome cited by the OIE as "Perkinsosis" in oysters refers to one caused by a related organism, Perkinsus marinus.

CENTAUR INTERNATIONAL (Hamilton)

Lymphoid tumour in a koala - Janeen Samuel

A mature female koala was found on a roadside and taken to a private veterinary practitioner. It was emaciated and had pale mucous membranes. Haematological examination at this laboratory showed anaemia (PCV 16%, haemoglobin 51g/L) and normal white cell count (3.5 x 10^9/L) with 12% neutrophils and 84% lymphocytes.

The koala died in spite of supportive therapy. At post mortem exam there was excess fluid in the abdomen; it was cloudy but the cloudiness settled out on standing. The omentum was covered in circular thickened areas, 1 to 5 mm in diameter, some of which were attached to the serosa of the stomach and spleen. Similar lumps were present on the visceral surface of the diaphragm. The mesenteric lymph node appeared enlarged and lobulated. There were no other abnormalities.

Histologically these lumps consisted of small cells resembling lymphocytes, in a loose matrix of fibroblastic cells. The cells were infiltrating between muscle fibres in the diaphragm and the stomach wall. Infiltrates of similar cells were present in other organs including the liver and kidney and pharyngeal mucosa. At all these sites, the majority of the cells were showing karyorrhexis and cytoplasmic vacuolation, which made it difficult to be certain of their identification. Lymphocytes in the spleen and lymph node showed similar degeneration. The only other finding was an inhalation pneumonia and pigmentation of hepatocytes.

Sections were examined by Peter Mitchell (at the time at our Bairnsdale Laboratory), and by Paul Canfield of Sydney University. Both agreed with the diagnosis of a lymphoid tumour.

The cause of the generalised lymphocytic necrosis, involving both tumour and normal tissue, was not clear: possibly acute stress. The practitioner had not treated the animal with corticosteroids.

OUTSTANDING SUBSCRIPTIONS 1993 & 1994

ACKLAND HM	McEWAN DR	MARSHAL DJ
MUNTZ F	STAPLES P	VANSELOW BA

OUTSTANDING SUBSCRIPTIONS 1994 ONLY

ALLISON JF	BAXENDELL S	CAR B
CHARLES JA	CHICK B	CONDRON R
CROWLEY AM	DUFF BC	ELLIS TM
FRANCE MP	FRASER G	GLAZEBROOK JS
GODWIN J	GRAYDON RJ	HANDLINGER JH
HUMPHREY J	JACKSON C	JONES R
KELLY R	KING J	LOVE SC
MASON RW	McCORIST S	MILLER RI
MORRISON J	PHILBEY AW	PRITCHARD DH
ROGERS RJ	SAMUELS J	SEAWRIGHT AA
SIMS LD	SMALL AC	SMITS B
STEWARD DJ		

REMOVED FROM LIST FOR LAST PAYMENT BEING 1991

CROSS GM	HINDMARSH M	LATTER M

WHERE ARE YOU? - Mail Returned.

MAIN DC - 1994 outstanding. PEET R - 1993 & 1994 outstanding.

WESTERN AUSTRALIA - David Forshaw

Chronic ulcerative typhlocolitis in nude rats - Darren Thomas and David Pass, Animal Resources Centre Murdoch

Nude rats were maintained in a flexible film isolator until August 1993 when they were caesarian-derived into an SPF barrier unit. In this unit the animals are handled, cleaned, fed and watered before other animals and production meets expectations.

Disease had never been noted in rats when isolator reared, but in January 1994, 6 months after their introduction to the SPF unit, some rats were noticed to be producing unformed droppings. Preliminary investigations revealed that these animals had ulcerative typhlocolitis. The disease only occurs in homozygous animals, with no sex predisposition (53% females and 47% males). The earliest age at onset (first identified by presence of wet bedding) is 8 weeks with a peak onset at 10-12 weeks of age. A survey of all homozygous offspring indicated a prevalence of 20% of the disease within the colony. The rats produce pale green pasty to "pelletoid" droppings often with a mucoid coating. They fail to grow as well as unaffected animals and are approximately 18% lighter in weight than unaffected homozygous rats of the same age. In chronically affected cases there is perineal and anal ulceration. Pathological examination reveals changes to the lower gastrointestinal tract. In most cases gross abnormalities are not present. In the most severe cases the colon and caecum are enlarged due to thickening of the wall and dilation of the viscus with gas. The lumenal contents are pasty or in the form of loose pellets with a mucoid coating, and pale green in colour. The ileocaecal lymph node may be hyperaemic and enlarged in the most severely affected cases. Lesions are present in the rectum, colon, and caecum. In all cases there is depletion of mucus from goblet cells and dilation of the crypts of Lieberkuhn. Ulceration of the epithelium is present in 47% of cases examined. Of these, ulceration extended into the submucosa in 43% of cases. Chronic inflammation of variable degree and cuboidal metaplasia of the enterocytes on the tips of villi was present in 86% of cases.

"Crypt abscesses" and extension of crypts into the submucosa is common. Sections stained with Warthin-Starry, Gram, acid-fast and by the periodic acid Schiff reaction did not reveal microorganisms associated with the lesions. Routine bacterial culture and smears failed to reveal recognised pathogens. Sera from heterozygous in contact nude rats were negative for known rat pathogens including Bacillus piliformis. The pathogenesis of the condition in CBH rnu/rnu is not known. In the absence of known pathogens it is likely that the pathogenesis is related to the immunological status of the nude rat. Prior to caesarian derivation into the barrier unit, there was no evidence of this condition, and it is unlikely that a change has occurred due to caesarian derivation. It is possible, however, that in a barrier environment rats are exposed to antigens not encountered in an isolator.

The lesions have some similarity to ulcerative colitis in man and lesions reported in "cytokine knockout", T-cell receptor (a, b, b x d) mutant mice and class II MHC complex mutant mice.

Necrotic enteritis in an Ostrich - Marc Kabay, Department of Agriculture South Perth.

Ostrich farming has only recently started in W.A. and so our exposure to diagnostic submissions has been limited. To date we have received only 25 ostrich accessions. This report describes an outbreak of necrotic enteritis in a group of 10 farmed ostriches on a property 300 km north of Perth. The features of the disease were very similar to those described for necrotic enteritis in poultry.

The birds were 9 weeks of age when they developed a fluid diarrhoea containing blood. Six birds were affected and three died. On necropsy, the proventriculus was impacted with fibrous plant material including pieces of bamboo. Petechial haemorrhages were present on the serosal surface of the jejunum and caecum and the mucosal surface was covered with a necrotic cellular debris.

Histological examination of the jejunum and caecum showed a segmental necrotising enteritis and typhlitis with the presence of many intralesional gram positive rods. *Clostridium perfringens* was recovered in heavy growth from both sites. Salmonella and Campylobacter culture was negative.

The condition may have been predisposed by impaction of the proventriculus. Dietary factors and concurrent parasitic disease (coccidiosis) are important predisposing factors for necrotic enteritis in poultry.

Black Soil Blindness - a new disease of cattle - Cleve Main, Department of Agriculture South Perth.

A new disease of cattle has been recognised in the Kimberley region of Western Australia and adjacent parts of the Northern Territory. It is known locally as Black Soil Blindness because of its link with Mitchell grass pastures growing on the "black soil" areas in that region, and the at least partial blindness apparent in many of the cattle affected with the disease. It is caused by a previously unknown fungus which is believed to be a Corallocytostroma sp., that produces conidial stromata (referred to as corals) on the growing points of the Mitchell grass.

The disease is confined to 2 adjacent stations in the south east Kimberleys as well as 2 stations in the Victoria River Region of the Northern Territory. (K de Witte and A Yanmat, pers comm.)

In Western Australia at least 500 cattle have died over the April to November dry season. Clinical signs include apparent loss of sight, depression, hind limb weakness, recumbency and almost invariably death within 1-7 days.

Post mortem findings vary. The carcase is very often dehydrated. The liver can be a yellow orange colour with large irregular red blotches over the surface. Sometimes there is severe perirenal oedema and the kidneys appear enlarged and pale coloured. In some cattle no macroscopic lesions are evident. In nearly every instance numerous hard rubbery white fragments of fungal corals can be found in the rumen and reticulum contents.

Histopathological lesions are characterised by severe cortical tubular nephropathy and less frequently by concurrent hepatic lesions of diffuse fatty change often more severe in the centrilobular areas. In some animals there is also necrosis of individual hepatocytes in the mid and centrilobular zones. Islets of hyperplastic bile ductules are occasionally present. Many cattle have severe focal epithelial necrosis of the reticular mucosa, sometimes also involving the omasum and occasionally the rumen. No recognizable histopathological lesions have been found in the eyes, optic nerves, optic tracts or other areas of the brain.

The disease has been reproduced in cattle and sheep by feeding Corallocytostroma collected from the properties involved. Ovine lesions are consistent with those seen in cattle, but the epithelial lesions produced were far more extensive, involving the mucosa of all three forestomachs.

Thrombotic Meningo Encephalitis - Cleve Main

TME has been diagnosed in feedlot cattle for the first time in this State. 10/1200 cattle have been affected. Clinical signs include respiratory distress and collapse, or death without premonitory signs. Rectal temperatures of 2 affected cattle were 41.8^{o}C and 40^{o}C. In one of the three animals necropsied there was severe fibrinous pleuropneumonia, which was not evident in the other 2 cattle. Of 2 brains examined, macroscopic lesions consisting of multifocal meningeal haemorrhage and 1-2mm dark red foci in the thalamus and cerbellum were found in one but not the other.

Histopathological lesions in both brains were characterised by multifocal purulent meningitis and encephalitis with vascular necrosis, thrombosis, multifocal perivascular and focal parenchymal haemorrhage with multifocal micro-abscessation. Lesions were widely dispersed throughout the thalamus, cerebral cortex, medulla and cerebellar folia. Lung tissue from the animal with severe pneumonia was not submitted. However, necrotising vasculitis was evident in a small sample of mediastinal fatty tissue that was submitted. Pulmonary lesions in the other cattle were characterised by focal interstitial pneumonia. Gram negative bacterial rods were evident within some micro abscesses and thrombi. Haemophilus somnus was isolated from the brain, lung and joint fluid.

Control of the problem was achieved by adding tetracyclines to the ration of introduced cattle. The likelihood of successful long term control without antibiotic feed supplementation is as yet uncertain.

"Post weaning enteritis" in sucker pigs - David Forshaw, Department of Agriculture Albany.

A large extensive piggery developed a problem with piglets dying at the point of weaning. The piglets were sometimes seen to be ill but generally they were found dead. Diarrhoea was not a feature of the disease. Consistent gross lesions were seen in a number of piglets necropsied; patchy skin congestion, mild dehydration and gastric mucosal congestion/infarction. Most piglets necropsied had pasty small intestinal contents but a few had liquid diarrhoea.

A haemolytic E.coli was cultured from all gut samples and from the liver and brain of one animal. This was serotyped at Bendigo as K88, O149.

Histopathology findings were multifocal acute necrotising enteritis with fibrin thrombi in blood vessels of the lamina propria and submucosa in the sites of inflammation. Infarction of both the gastric and small intestinal mucosa was seen. Patchy adherence to the epithelium of E.coli-like organisms was seen.

The haemolysis seen on blood agar, the serotypes present and the pathology is consistent with "post weaning enteritis" however, the age of the piglets is not. Talking to the pig experts at Bendigo, they see this occasionally but it doesn't seem to have made it into the books. Control involves making sure that the sows are vaccinated with the appropriate serotypes of E.coli.

Possible copper deficiency in an emu - David Forshaw

A four month old male emu A94626 had a two week history of slowly deteriorating ataxia with frequent stumbling. No other birds were affected. The neck was held slightly to the left side. Otherwise the bird was alert and eating although it was underweight.

On necropsy, no gross lesions were seen.

Numerous chromatolytic neurones were seen in various nuclei in the medulla and midbrain. Rare digestion chambers were seen in the white matter at various levels of the spinal cord with no particular pattern.

Liver copper level was 22mg/kg.

A slide of the month contribution in September 1993 from Victoria quoted liver copper levels of 20.3ug/g in a clinically similar case. In the Victorian case there were no neuronal cell body changes but very subtle axonal oedema. Similar but more marked axonal changes have been seen in emus necropsied at South Perth but in the Perth birds, their liver copper levels were much higher.

NEW SOUTH WALES - Paul Gill

Collaborative research - Aspergillosis in ostriches - Stephen Love

RVL Armidale is collaborating with The University of New England (Dr Harry Gill, Senior Research Fellow) on RIRDC supported investigations into "The epidemiology and diagnosis of aspergillosis in ostriches". Despite an unfortunate setback towards the end of last year, good progress was made in the first year of this three year project.

An ELISA has been developed and early indications are that this test performs much better than the AGIO, the test usually employed. Further development on this and other tests is planned. A survey of Australian ostrich farmers to collect basic epidemic logical data is in the analysis stage. We have also been supplying samples and information for a complementary project (Drs Katz and Cheetham) which is using the polymerase chain reaction to study aspergillosis in ostriches.

We now have sufficient *Aspergillus* sp isolates from around Australia, but would like to thank those colleagues and labs who have gone to the trouble of supplying us with isolates from cases of aspergillosis.

Crystal-associated cholangiohepatopathy - Stephen Love & Steven Hum

Three weeks after being moved to a paddock with an abundance of "cathead" (*Tribulus* sp), ten sheep had died and a further ten were sick out of a mob of 400 Merinos. Clinical signs were those of photosensitisation.

Fixed liver from one case was submitted and histologic examination revealed fine flat crystals in the bile ducts, peribiliary fibrosis, bile pigment accumulation (mostly within Kuppfer cells) and variable hepatocellular damage. (AN95/0118/SH). Such liver pathology is rarely if ever seen at this lab; perhaps it is more common at labs whose service area includes more "*Tribulus* country" i.e. slopes and plains.

Crystal-associated cholangiohepatopathy is said to occur not only with *Tribulus* plus sporidesmin intake, but with the ingestion of other plants (*Agave* for example) as well. (Ref Jubb & Kennedy).

Regional Veterinary Laboratory Menangle - Jim Rothwell.

Congenital Epidermolysis bullosa in a Friesian calf.

A premature calf was presented to a practitioner and the district veterinarian at Bega. The skin was abnormally thin, was absent over 20% of the body and there was incomplete hair cover. There were craniofacial abnormalities and fixation of the fetlock, hock, metacarpus, elbow and fetlocks. No other similarly affected calves have been reported in the herd or local district.

Pericardial fluid contained 5780 µg/ml immunoglobulin (greater than 80 µg/ml suggests an in-utero antigenic challenge) but was negative for Bluetongue, EHD, Akabane, Palyam or Pestivirus on serology.

Histology of the skin revealed a cleft between the dermis and epidermis with the basement membrane remaining on the base of the cleft. In skin adjacent to the cleft there was hydropic degeneration of basal epithelial cells. There were no other significant histological findings. Dr Julie Yager of Guelph University agreed that the skin lesion was consistent with epidermolysis bullosa. Whether the condition is a true

hereditary form of epidermolysis bullosa or whether the skin lesion is one of several manifestations of some in utero insult is unclear.

Presumed porto-systemic shunt in a calf.

A 3 month old female Hereford calf at Bombala was presented with neurological signs that came and went. Ten days later the calf died and was necropsied. The liver appeared fibrous, but no shunt was seen grossly! Histopathology showed severe cerebral oedema/status spongiosis. The liver appeared to contain more portal triads per field than usual. These triads contained 2 or more arterioles, few portal veins and an apparent increase in biliary ducts. Dr Roger Kelly agreed that this liver lesion was probably due to a congenital portosystemic shunt and is the second he has seen in cattle. The brain lesion is hepatio-encephalopathy.

Hypocalcaemia in weaner sheep following yarding.

A group of 150 recently weaned sheep at Yass in September were yarded for a day, drenched the next day, held in yards for another day then released onto oats. This was done as a modification of recent work by CSIRO that has shown increased efficacy of benzimidazole anthelmintics if animals are fasted prior to treatment. Unfortunately a number of sheep went down and 5/5 sera had very deficient calcium levels (0.93 - 1.43 mmol/L) . Marginal blood selenium and high CK were also seen. This case illustrates nicely the folly of applying some new management practice without considering it's effect in context. Recently weaned sheep, grazing drought affected pastures in winter, starved for 3 days then released onto oats is a recipe for disaster. Fortunately only 1 sheep died.

Regional Veterinary Laboratory. Wollongbar

Cardiomyopathv in Wagyu calves.

We have seen two cases of suspect cardiomyopathy in young Wagyu calves. Both 4 day-old animals died unexpectedly. Necropsy findings included ascites and oedema of the thymus, gall bladder and pancreas. The right ventricle of the heart was dilated but the wall seemed of normal thickness. Several foci of ventricular myocardium seemed soft and darker in colour than normal. Histological findings included interfascicular fibrosis mainly affecting the interventricular septum including the a/V node and Purkinje fibre bundles and the wall of the left ventricle towards the apex of the heart. These heart lesions are similar to those of the congenital cardiomyopathy of Polled Hereford cattle. A congenital cardiomyopathy was described in 1979 as an autosomal recessive in Japanese black cattle, a closely related breed to the Wagyu. We are keen to hear from anyone who has seen similar oases in Wagyu calves.

Distemper in foxes - Terry Rothwell

The CSIRO Division of Wildlife and Ecology is engaged in a major research program into fox biology. A component of the program is collection of young feral foxes by contractors who hold them until collection by CSIRO and transportation to Canberra. Here they are vaccinated against distemper and treated with an anthelmintic. Recently, many deaths occurred in foxes after arrival in Canberra.

Post mortem examinations were performed by a veterinarian participating in the project and fixed tissues sent to the Department of Veterinary Pathology. Almost all the 9 foxes examined had lung lesions. These ranged from hyperaemia and oedema, to well developed interstitial pneumonia and in a few cases bronchopneumonia. Large intracytoplasmic eosinophilic inclusion bodies were present in the airway epithelium of 5 animals with pneumonia. Other common changes were depletion and necrosis of splenic white pulp lymphocytes and necrosis of small intestine crypt epithelial cells. Most foxes had an eosinophilic enteritis with numerous coccidia present. Nematodes and cestodes were found at autopsy and worm profiles were present in some intestinal sections. No brain lesions were observed.

Foxes are particularly susceptible to distemper and attenuated vaccines, safe for dogs, can cause disease in foxes. In this outbreak, the vaccines used had not caused problems previously and one of the affected animals died before being vaccinated.

Mouse urological syndrome - Malcolm France.

Three laboratory mice were submitted in poor bodily condition, one of which was moribund. All had palpable abdominal masses which on post mortem examination were found to be massively distended seminal vesicles and coagulation glands. In the moribund mouse, the pelvic urethra was thickened and the bladder was distended suggesting urethra1 obstruction; histological examination of this mouse revealed a proteinaceous plug occupying the entire urethra and surrounded by necrotic tissue and suppuration. Apart from distension of the accessory sex glands, there were no significant abnormalities in the other 2 mice.

This condition is termed ‘mouse urological syndrome’ or ‘obstructive uropathy’ - both misleading terms since the primary problem seems to lie in the accessory sex glands with urinary tract obstruction usually occurring late in the disease. The cause is unknown but the incidence increases with age and varies between strains.

Hyperplastic typhlocolitis in laboratory mice - Malcolm France

Two C57BL/10 mice from a large colony were passing soft, slightly mucoid faeces. On post mortem examination, the caecum and colon of both mice were thickened, firm and grey. Histological examination revealed markedly hyperplastic caeca1 and colonic mucosae in which the glandular epithelium displayed pseudostratification and mitotic figures near the gland orifices. In the same sections there was also dilation of glands, occasional ulceration, luminal exudate and scattered neutrophils within the lamina propria.

These findings are consistent with murine transmissible colonic hyperplasia, the cause of which is thought to be *Citrobacter freundii* (biotype 4280). Failure to isolate the organism from these cases may reflect the finding that lesions often persist for some time after the infection has been eliminated by the host. No treatment was undertaken, and no further cases have been recorded.

REGIONAL VETERINARY LABORATORY WAGGA WAGGA - JOHN GLASTONBURY

Sadly we farewelled Jocelyn Godwin on 25 November 1994. Jocelyn and James are expecting their first child in February 1995, and have returned home to Western Australia. Her smiling face and conscientious approach to pathology will be greatly missed at this laboratory.

Canola Toxicity

During our dry spring, the grazing of failed canola (*Brassica napus*) crops was associated with mortalities in sheep on 8 farms. Of the 6,280 animals at risk, 3.5% died.

Clinical signs, which included respiratory distress and nervous derangement, occurred within 48 hours of gaining access to the canola. The latter included staggering, hyperaesthesia, recumbency and convulsions.

Excessive volumes of serous fluids, generalised petechial and/or ecchymotic haemorrhages and pulmonary congestion as well as emphysema were the most commonly reported gross pathological findings.

Histological examinations confirmed the gross observations. Mild to moderate acute periacinar hepatic necrosis in samples from three farms, splenic haemosiderosis (one farm) and pulmonary oedema were also detected.

Tests for nitrate/nitrite (four farms) and prussic acid (two) were negative.

The pathogenesis of the mortalities is uncertain. However, there was evidence of intraphagocytic haemolytic anaemia in material from three farms.

Strangles

The spectre of the acute equine respiratory syndrome was raised during October 1994 when horses grazing on neighbouring farms in the Young district displayed respiratory signs. Affected horses were pyrexic and had variable nasal discharges and wheezing respiration. Serology on four animals, performed at the Australian Animal Health Laboratory (AAHL), Geelong, was negative for equine morbillivirus.

One of the lesser affected horses had a recurrence of respiratory disease in January 1995. It died after a short illness.

Fortunately the field veterinarian submitted the larynx, trachea, lungs and heart to the laboratory. Severe diffuse pulmonary congestion and oedema heightened fears of the acute equine respiratory syndrome. However, closer inspection found a spherical abscess, 13cm in diameter, compressing the trachea, 35cm distal to the larynx. The abscess contained a copious quantity of lemon-coloured pus, which yielded a heavy pure growth of *Streptococcus equi* subsp *equi*.

Ovine Interstitial Pneumonia

As part of a routine mortality investigation, fixed lung was submitted from an eight-month-oId Texel sheep.

Histological findings were chronic multifocal interstitial pneumonia, acute diffuse pulmonary congestion and very mild acute suppurative bronchopneumonia. The interstitial pneumonia was characterised by adenomatoid epithelialisation, fibrosis of alveolar walls, fibromuscular hypertrophy and virtually no leucocytic response.

Because of the possibility of Jaagsiekte or pulmonary adenomatosis, material was sent to AAHL for Peter Hooper's opinion. Peter commented that the degree of fibrosis favoured inflammation, the epithelialisation was not adenomatoid enough and the age of the animal made Jaagsiekte unlikely. Confirmation of a diagnosis of Jaagsiekte does present certain problems, but these pale into insignificance in comparison to those which a positive diagnosis would cause the regulatory authorities.

REGIONAL VETERINARY LABORATORY (ORANGE) - Jeff Marshall

Nephrosis following ingestion of Amaranthus retroflexus

Case 1

History: A mob of 600 merino wethers, 18 months of age and in good condition were introduced to a fallow paddock dominated by an even growth of Amaranthus retroflexus (redroot, pigweed, Prince of Wales' feather) approximately 5 to 7 cm in height. One week later when the sheep were moved from the paddock, 3 sheep had died, two were noticeably sick.

Gross Pathology: Two sheep necropsied had white mottled kidneys, marked perirenal oedema and a large quantity of fluid in both the abdominal and thoracic cavities.

Biochemistry: The kidney contained 0.03% oxalate on a wet weight basis.

Histopathology: Diffuse degeneration and attenuation of the epithelium of proximal and distal convoluted tubules. Distal and collecting tubules were dilated and contained casts of proteinaceous material and necrotic cellular debris. There was interstitial oedema and moderate interstitial fibrosis. Small numbers of lymphocytes and plasma cells were present at multiple foci within the interstitium.

Case 2

History: A mob of 1500 crossbred lambs of mixed sexes and 6 months of age were introduced into a paddock containing little apparent feed where they were maintained on a diet of grain and lucerne hay. Fifteen of the lambs were found dead within 7 days of introduction and a further 15 appeared sick. Closer inspection of the botanical composition of the paddock revealed the presence of abundant Amaranthus retroflexus plants located at the sheep camp sites. The top of the plant had been eaten leaving a short stem and characteristic red taproot.

Gross Pathology: Two animals necropsied revealed marked perirenal oedema and abundant fluid in the thoracic and abdominal cavities. Petechial haemorrhages were present beneath the capsule of the pale kidneys of enlarged size.

Biochemistry: The kidney was shown to contain low levels of oxalate (<0.03% on a wet basis).

Histopathology: Severe diffuse subacute to chronic toxic tubular nephrosis with focal necrosis of the renal tubule epithelium throughout the cortex and extensive casting of the tubular lumens with proteinaceous material. Minimal inflammatory cell infiltration and early tubule regeneration with some focal interstitial fibrosis.

Comment: Reports of the toxic effects following ingestion of the plant Amaranthus retroflexus are confined to cattle and pigs, not sheep. The nephrotoxic principle has not yet been described. The plant often contains high levels of oxalate and nitrate; but these agents usually cannot be implicated. Oak shrubs and trees (Quercus spp.) cause a similar condition in ruminants and horses in which the toxic principle has been shown to be tannins. Phenolic compounds which have been identified in the leaves of Amaranthus retroflexus may be the causative toxin.

References: Maxie MG (1993) The urinary system. In Pathology of Domestic Animals (Vol 2), ed Jubb KVF, Kennedy PC and Palmer N, 4th Edition. Academic Press, Harcourt Brace Jovanovich, New York, NY, pp 487-495

Everest SL (1981) Poisonous Plants of Australia, Angus and Robertson, Sydney, pp 69-71

Toxicosis associated with sheep grazing failed canola crops

Case 1

History: A mob of 870 mixed age and mixed sex Merino sheep, had been grazing a 40 acre paddock of canola (*Brassica napus*). The crop was described as "failing" due to prolonged dry seasonal conditions and was in flower. The mob was yarded for a period up to 42 hours for crutching. Forty sheep were found dead the following morning less than 24 hours after being reintroduced to the crop and a further 8 were sick. Dead sheep were found in lateral recumbency with distended abdomens and evidence of considerable paddling prior to death. Many of these sheep froth at the mouth or the soil adjacent to their nose and mouth was wet.

Necropsy: Rumen contents were mainly stems and leaves of canola plants. Significant findings were limited to petechial haemorrhages on the coronary grove of the heart.

Biochemistry: Canola plant specimens collected from the paddock were positive in the Diphenylamine blue test (DPB) for nitrites and nitrates, but not samples of rumen contents and aqueous humour from one of the dead animals. One of two serum samples presented from sick animals was hypocalcaemic (1.54 mM/L).

Histopathology: The lung was diffusely congested and the alveoli contained a serofibrinous exudate accompanied by small numbers of macrophages, neutrophils and lymphocytes. Alveolar septae were oedematous and populated with increased numbers of leucocytes of mixed morphology.

Case 2

History: A mob of 500 adult Merino wethers had been grazing a failed canola crop for a week when the owner found 1 dead and 2 frothing at the mouth and ataxic.

Necropsy: Petechial haemorrhage in cardiac muscle and pericardium, lungs dark red/purple, froth in airways, liver mosaic appearance.

Histopathology: Mild periacinar necrosis of the liver with diffuse haemosiderosis, myocardial haemorrhage and mild diffuse subacute interstitial pneumonia.

Comments: The lung changes are suggestive of acute indole glucosinolate toxicity which has been reported for cattle on Brassica crops (Seawright 1989), including canola. Cruciferous plants contain high concentrations of indole glucosinolates which are hydrolysed in the alimentary tract to form 3 hydroxymethylindole. The lesions are similar to those reported for bovine atypical interstitial pneumonia induced experimentally with 3-methylindole (Gonzalez *et al* 1986). Some of the findings in case 2 were suggestive of a previous haemolytic crisis. Further samples were not available to confirm this suspicion. Interestingly the amino acid S-methylcysteine sulphoxide has also been isolated from cruciferous plants and is thought to cause haemolytic anaemia.

References: Seawright AA (1989)- Cruciferae glucosinolates. In "Animal Health in Australia, Volume 2, Chemical and Plant Toxins". Australian Bureau of Animal Health, Canberra, p38-40.

Gonzalez JM, Yusta B, Garcia C and Corpio M (1986)- Pulmonary and hepatic lesions in experimental 3-hydroxymethylindole intoxication. Veterinary and Human Toxicology 28:418-420.

SOUTH AUSTRALIA - Ruth Reuter

VETERINARY PATHOLOGY SERVICES (Adelaide) - Ruth Reuter

"New Pathologist in Adelaide"

Like many groups in diagnostic pathology, VPS has undergone some changes during the past year. There have been movements of staff in all of our laboratories. However, the most significant one to occur in Adelaide has been the addition of another pathologist to the group here.

In January we welcomed Martin Copland to our organisation. Many of you will know Martin from his previous position with VETLAB in Adelaide. His interests are primarily in the area of clinical pathology, and his expertise is a great asset the practice.

Fish Kill Investigation Workshop

From the report in this newsletter by Peter Durham and associates, you may gain the impression that fish diseases are "in vogue" this month in South Australia! This is an area of pathology which tends to be overlooked or even avoided for a variety of reasons. However, I believe there is a great potential for involvement of pathologist in this area. I was able to attend the recent Fish Kill Workshop which was advertised in the November 1994 Veterinary Pathology Report and felt it was well worth attending.

The sessions were very capably conducted by Judith Handlinger and Barbara Nowak and were attended by 38 people, primarily Fisheries and Environmental Control officers. There were 2 other veterinarians present; one who is farming silver perch with her husband and one who is on a three month stint with the DPI in Tasmania.

The program was broad-ranging, from the legal aspects of fish kills to case discussions. There was significant mention of the role of pathology by many of the participants, some of which was not particularly complimentary! However in informal discussions I gained the impression that many of the people there were very interested in co-operating with pathologists. I believe that the more our profession can become involved in this area the better. There is unlimited scope for involvement with both wild fish and aquarium species. If we don't take advantage of this the opportunity will be taken by others with lesser expertise in the area of health and management than we have.

Serial Sampling - M Copland

A frustrating aspect of veterinary medicine is that frequently "economic" and perhaps traditional reasons constrains ones examination to a mere snapshot of the disease. This denies the opportunity for a reasoned analysis of what is essentially a dynamic process. Diagnostic and prognostic ability suffer as does an understanding of disease progression and remission. The following is an example:

The owner of a l0yo Keeshond noticed her dog stumbling prior to becoming recumbent. She presented the animal to her veterinarian who reported it to be 'flat' and anaemic. Blood samples were collected at the initial presentation, and two days later. Here are some of the results.

	Initial Presentation	2 Days Later
RBC	2.5	6.5
Hb	59	138
PCV	0.17	.42
MCV	68	69
MCH	23.2	22.6
MCHC	342	329
nRBC'S	0	2
Plasma Protein	57	
Serum Protein		78
Reticulocytes	25	427
	1+ polychromasia	2+ polychromasia
	1+ poikilocytosis	2+ poikilocytosis

If it were not for the plasma protein value in the initial tests, one might suggest a non-regenerative anaemia such as seen in chronic disease. However, in spectacular fashion, the follow up results show a highly responsive bone marrow and offer a likely explanation of haemorrhagic anaemia with post haemorrhagic haemodilution.

VETLAB (ADELAIDE) - John Finnie

Immunohistochemical marker of axonal injury in brain.

VETLAB has recently been investigating the detection of injured axons in ovine brains using amyloid precursor protein (APP) as a marker. APP is transported by fast axoplasmic transport and is believed to accumulate when the cytoskeleton breaks down. Sections are incubated with a monoclonal antibody to APP, stained with 3,3-diaminobenzidine tetrahydrochloride with avidin-biotin peroxidase, and counterstained with haematoxylin.

This technique has the advantage that in contrast to more traditional silver stains, only damaged axons are marked, with no background staining due to normal axons. Furthermore, formalin fixed, paraffin embedded brain can be used, injured axons are visible well before axonal spheroids (retraction balls) are evident in conventionally stained sections (as early as 1 hour after axonal damage) and, since APP accumulation is an energy requiring process, its presence indicates injury to axons during life.

FISH HEALTH UNIT - Peter Durham

Deaths in Fur seals - PJ Durham, I Queale

Samples of fixed lung from two fur seals were supplied from the SA Museum. The animals were washed ashore dead on the SA coast.

One showed severe mixed pulmonary oedema, emphysema and collapse of recent origin. Moderate numbers of diatoms were present in the affected lung tissue. It was concluded that the animal had probably died from drowning, possibly due to fighting or storms.

The other animal contained numerous well circumscribed spherical lesions up to 2.0 cm in diameter which were distributed throughout the lung. Similar lesions were reported in the other body organs such as mesentery. Histologically the lesions resembled a well differentiated leiomyoma with very low mitotic activity and no evidence of invasion. However, the multiple distribution of the lesions suggests that they were metastatic and were therefore malignant.

Squamous cell carcinoma in a Rainbow fish - PJ Durham, A Spanner

Organ samples of lesions from a rainbow fish were submitted from the Adelaide Zoo, with a history of red lesions on the lower jaw and abscess-like lesions in the spleen and kidneys. Histologically the spleen and kidney contained well circumscribed squamous cell carcinoma with prominent epithelial pearls. Similar but less circumscribed lesions were seen in the skin of the lower jaw, accompanied by secondary infection and inflammation. It was concluded that the tumour originated in the skin of the jaw with metastasis to other organs.

Lymphocystis disease in Snapper - PJ Durham, P Hone, M Allanson

Live juvenile snapper were submitted from the SARDI laboratories with a history of recent development of lesions on the fins while in holding tanks. The fish were part of an experimental study and had been wild caught off the SA coast. Small off-white lesions ranging from 0.5-4.0mm in diameter were noted on all fins . Histologically the lesions were typical of lymphocystis disease, with massive enlargement of dermal cells. Electron microscopic examination of ultrathin sections of fins revealed very numerous iridovirus particles in affected cells. Attempts to grow the virus in fish cell cultures (RTC, BF2 and CHSE24) were unsuccessful, but this is not surprising as the iridovirus is known to be very selective in its host cell range.

The infection probably originated in the wild-caught stock and has not been previously recorded on the southern Australian coast.

Picornavirus infection in Barramundi - PJ Durham

Two outbreaks of picornavirus infection were recently recorded in farmed barramundi. The disease caused substantial mortalities in barramundi larvae aged between 10 and 21 days. Histologically vacuolar lesions typical of picornavirus infection were detected in the brain, spinal cord and retina of the eye. Electron microscopic examination of brain and eye tissues confirmed the presence of large numbers of typical picornavirus particles. The origin of the infection is probably from source stock in north Queensland, where the disease is endemic.

Perkinsus infection in SA abalone - PJ Durham, I Carmichael

Samples of dead South Australian abalone were submitted with complaints of muscle lesions. Numerous off-white to brown coloured lesions (l-5mm diameter) and large abscesses containing salmon pink coloured pus were located in the muscle. Histological examination showed the lesions consisted of various sized abscesses enclosing protozoan parasites typical of Perkinsus species (which were also detected in squash preparations). These parasites have previously been recorded in South Australia

JOBLINE

The following was received from Dr Phil Ladds at James Cook University, Townsville from Trevor Whitbread, Abbey Veterinary Services, 14 Oak Place, Newton Abbot, Devon TQ 122HW, England. Ph 0011 44 626 53598, Fax 0015 44 626 335135.

"I was wondering if you have come across anybody who may be interested in a job in pathology in the UK. I think the minimum requirement would be somebody who has done some pathology, not necessarily to MRCPath or American Board standard, who can work on their own, but in particular someone who knows when they don't know. I have had two assistants so far who have had about a year of experience and have then come to us and they have both worked out extremely well, but obviously if there is somebody with more experience than this, this would be fine. I think the experience would need to be substantially in small animal pathology. The majority of work we do is small animals, exotics and some birds. Most of the meat animal pathology is carried out by the ministry in the UK. If you know of anybody who may be interested, then please ask them to contact me."

The College of Veterinary Medicine, North Carolina State University, announces a residency position in veterinary clinical pathology beginning July 1, 1995, or as soon thereafter as possible. The program will emphasize competence in clinical pathology to prepare the trainee for certification by the ACVP. Applicants must possess a D.V.M. or equivalent degree; previous clinical experience desirable but not mandatory. Individuals successfully completing the training program will be encouraged to continue their training in a research program leading to the Ph.D. degree. Stipend is competitive. Applicants should send a curriculum vitae, a statement of goals and interests, complete transcripts, and three letters of recommendation.

Closing date for applications is March 15, 1995, or until a suitable candidate is found. Send communications and all application materials to the Office of Associate Dean for Services, Director of Internship and Residency Programs, North Carolina State University, College of Veterinary Medicine, 4700 Hillsborough Street, Raleigh, NC 27606.

For details about the program call Drs. J.R Stevens/Carol Grindem, Clinical Pathology Residency Coordinators, at (919) 829-4488/829-4277.

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NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY

The NRDAP and Taronga Zoo Pathology are now separate.

FUTURE Funding for the NRDAP has been obtained for the next 4 years, and the Standing Committee on Animal Health Laboratory Services (SCAHLS) is looking at the possibility of establishing permanent funding.

NEW TECHNOLOGIES At the 1994 ASVP Meeting , Rod was asked to investigate the possibilities of some of the new technologies:

LASER DISC. The NRDAP has a copy of the 6th edition, 1993 NOAH'S ARK laser videodisc from the USA (Georgia). It has approximately 25000 transparencies of pathological conditions from many species around the world (plus cat and dog neurological conditions) with an interactive interrogation program. To utilise this we would need about $2500 for a Pioneer LD-V4300 or similar player as the laserdisc is NSTC format and not compatible with standard Australian videodisc players. The NRDAP has a high quality display screen which would be suitable, and the computer in the registry is sufficiently powerful to handle the programs. Melbourne University, Department of Veterinary Pathology has an earlier edition of this videodisc and the wherewithal to run it. The questions, answers, notes etc are portrayed on the computer screen and the figures are on the other screen. It would be useful for teaching and training and would give access to material not readily available in Australia. It would not be readily transportable and would be dedicated to one disc or its replacement. Alternatively, the disc can be used as an expensive frisbee!

CD. ROM and PHOTO.CD is a convenient way to store and retrieve transparencies. We have had a batch of transparencies copied onto CD at a cost of approx. $100 for 60. They are of very high quality but can only be read by computer with a CD reader and a suitable program such as CORRAL DRAW. There are actually 7 copies of each figure but at different resolutions: at the highest resolution it takes a long time to bring up a frame. Such figures would be useful if used in conjunction with written notes. If used with a text on a floppy disc it would be slow to move between the text and figures. Up to 10 000 transparencies could be copied onto one CD.ROM using one resolution.

INTERACTIVE CD This appears to be the new technology and suitable units should be available readily in Australia shortly - these will cost over $1 000. Touch screens will be more expensive. If we were to develop a training set of transparencies and notes with interactive questions and answers along the lines of NOAH'S ARK it would be possible to have a master interactive CD produced for $25 000 for 30 min!! Copies are cheap but costed to recoup outlay. A shorter prototype could be developed for about $12 000!!!.

FLOPPY DISC It is possible to have transparencies copied onto floppy disc (3.5 in) for use with an ordinary computer at $1 per slide (100 per HD). The quality of the image is not as good as with CD. Can also include an indexing text with the figure so it can be retrieved and sorted - unlike the photo.CD. This gives a split screen of Figure with text (sorting codes, case signalment, lesions etc) underneath. It may have some value in distribution of case material but questions and answers, interactive text etc would still have to be hard copy. Each user would need to purchase the program which runs it if we were to distribute courses in such a manner.

PERHAPS THE BEST SOLUTION FOR PROVIDING **CONTINUING PROFESSIONAL EDUCATION AND TRAINING** would be to send out a set of notes in the form of a review written by a knowledgeable colleague, with questions, on a quarterly basis. The answers would be sent in to be marked. We could include transparencies of gross or histological specimens on a rotating basis much the same as the slide of the month.

THE REGISTRY STILL LACKS TRANSPARENCIES OF GROSS PATHOLOGY!! PLEASE SEND!

AFIP TRAINING SETS can be obtained but we would have to undertake to replace any sets which were lost or damaged. Many of the sets are valued at more than $1 000 and the registry does not have funds to cover such losses.

CL DAVIS FOUNDATION FOR COMPARATIVE PATHOLOGY. My understanding of the correspondence I have received is that, to have ready access to their material we would have to establish an Australian division of CL Davis, about $50 each person or $250 for an institute. We would then distribute their newsletters with an Australian section. I take it that this would not replace ASVP but run parallel to it. Perhaps we could persuade the OIC of each vet lab, and Heads of University Depts to become members and obtain material through them?

PRELIMINARY REPORT ON TRAINING NEEDS SURVEY

Roger Kelly joins the Training Committee:
Roger has accepted an invitation to join the training committee - and will bring to it his experience regarding university based education.

Training Survey:
In January questionnaires were mailed directly to 163 members., including a small number in New Zealand, Canada, United Kingdom, Asia and America, based on the mailing list as at December 1994.

An invitation extended to the New Zealand Society for Comparative and Veterinary Pathology to participate in this survey was not taken up so only those ASVP members in New Zealand have had the opportunity to participate so far.

A separate questionnaire was sent directly to each member's managers - involving 63 laboratory or department managers and 22 policy making personnel in Government departments, CSIRO, universities, private pathology organisations, the pathology registries and members of SCAHLS in Australia and New Zealand.

The ability to mail questionnaires directly to these managers was made possible with the help of state representatives and members of the training committee in verifying the names of the relevant managers in each state. This help is gratefully acknowledged.

Up to the end of February 75 questionnaires had been returned from pathologists (45% response and 19 from mangers and policy makers (22.3% response) . The analysis of replies will be available in a full report at the AGM.

If any current member has not yet received a survey form please send Pat Boscence at PO Box 114 Walkerville SA 5081 a note with your current address and a form will be sent to you. From the replies received to date a number of changes have had to be made to members addresses and an updated membership list should be one of the spin-offs from this exercise.

Scholarship for intending ACVSc Fellowship candidates:

It will be of interest to members considering Fellowship that the Australian College has created a scholarship to assist College members enrolled as candidates for the Fellowship examination. To obtain the scholarship their training program and credentials must be approved by the Chief Examiner . The aim of the scholarship is to help meet the costs of preparation by candidates attached to an institution where there is an approved supervisor of the candidate's training program. In 1994 this scholarship was worth $ 5000, of which 75% was paid to the candidate in three quarterly payments and 25% to the supervisor's institution.

Robin Giesecke
Coordinator, Training Committee

OBITUARY:

DR RATIMIR RAC

Ratimir Rac.VS. Dr. med. vet., MACVSc, a veterinary pathologist at the Institute of Medical and Veterinary Science from 1954 to 1982, died peacefully in Adelaide on 3 December 1994.

He was born at Udbina, Croatia on 12 October 1917, graduated from the Veterinary School at Zagreb in 1942 and worked at the State veterinary diagnostic laboratory until the end of the war . While at the laboratory, he gained his doctorate, in 1944, for work on anthrax. Immediately after the war he fled to Austria, worked for a short while with the International Refugee Organisation and emigrated to Australia, on the promise of a veterinary posting within six months, in 1949.

In August of that year he obtained a temporary position as laboratory technician in the Veterinary Pathology Division of IMVS. Although upgraded to the status of graduate assistant he was unable to take responsibility for any veterinary work until 1954, when he successfully passed the Australian qualifying examination for veterinarians with European degrees. In 1956 Ratimir took out Australian citizenship.

With his degree now recognised, he was reappointed to the IMVS staff as Veterinary Pathologist and placed in charge of routine diagnostic services. In 1959 he was asked to form a veterinary histopathology unit and despite several internal moves for the unit, remained its head until his retirement. His early research centred on establishing that Johnes Disease of cattle, if introduced, would not spread under the conditions then prevailing around Adelaide. In 1959 he made the first diagnosis in Australia of Inclusion Body Rhinitis in pigs and was the first to describe congenital goitre in stud Merino sheep - which became a successful animal model for congenital thyroxine deficiency in humans. In 1971 he became a foundation member of the Australian College of Veterinary Scientists. Histopathology remained his joy. He made a study of tumours of animal origin, collecting over 2000 tumours and from 1960 until his retirement taught histology and elements of histopathology to students at the Waite Institute. During his 23 years as a veterinary pathologist at IMVS Ratimir collaborated with medical and veterinary colleagues in numerous investigations and reports of diseases which resulted in more than fifteen publications.

"Doc Rac" as he was known to many, contributed much to the pathology services provided by IMVS despite his deafness - legacy of a childhood illness. Although an exacting taskmaster, he won the respect of his colleagues for his skills in histopathology, and his interest in their problem cases.

Ratimir retired on 29 June 1982, ending a 32 year career at IMVS in which he rose to the position of a Senior Specialist Veterinary Pathologist. Since retirement he spent more time on philately, an avid interest since childhood, more time with his family and more walking. He was predeceased by his wife, Gabriela, and is survived, in Australia, by his children Ivan, Maria and Marko, their partners and his grandson Luke.

R.G.