



VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
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STATE REPORTS

10	Queensland - Fraser Trueman. ARJ, Yeerongpilly, QLD, 4105.	07 3629400
15	Victoria - John Mackie. RVL, PO Box 388, Benalla, VIC, 3672.	057 622933
19	S. Australia - Vui Ling Tham. CVL.GPO Box 1671, Adelaide SA,5001.	08 2287390
21	New South Wales - Paul Gill. RVL, Wollongbar, NSW, 2477.	066 240298
24	Western Australia - Ron Peet. AHL, South Perth, WA, 6151.	09 3683333
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	Tasmania (to be advised)	

DEADLINE FOR NEXT VET. PATH. REPORT IS JANUARY 15, 1992

1.

From the President

Welcome to VPR Issue No.32 and the second for our 1991-92 term of office. The EMAI executive trust you find it informative and encouraging.

The good news is that the National Registry of pathology will continue for at least another two years until December 1993 with its present structure intact (see separate report by Dr. T. Ross).

In the light of the above encouraging National support, the ASVP is planning for you a golden 'get-together' opportunity for the Adelaide AGM.

Date:	9 - 10th May 1992	(immediately preceding AVA Conference 11-15 May)
Venue:	Verco Theatre, IMVS Adelaide	
Accommodation:	Residential Wing, Royal Adelaide Hospital or alternatives close by of your choice	
Training Theme:	Bone & Joint pathology, including in-house tour of the I.M.V.S. specialist bone pathology laboratory.	

Local and interstate speakers are being arranged and members invited contributions on the training theme will complement the format. The executive intends to limit the formal (training) agenda so that a wide range of members' papers on other subjects can be accepted. This should assist in obtaining travel support from employer organizations.

Plan now to join us for ASVP in Adelaide in May 1992.

The bad news is that realistically veterinary pathology positions and services, especially in Government employ, are under economic pressures and serious review in all States. The executive is contributing to a draft AVA policy on laboratory charges being developed within the Sheep Veterinary Society to help enhance our member's contributions to and rewards from the Australian community in the medium term. I commend the ASVP to all its members as a suitable voice for representations to Government, employing organizations or industry as appropriate. Please provide the Executive with a well documented summary if you feel the Society could weigh in behind you on any particular matter.

May your sanity survive the 37 or less shopping days to Christmas 1991 and may the promises inherent in Christ's birth bring you genuine peace on earth this Festive Season.

Best wishes to all members for the New Year of 1992.

Keith Walker
ASVP President, 1991-2

Further Funding for The Registry

The Registry management committee was delighted to learn that its submission for a further two years funding covering the period January 1992 to December 1993 was successful.

Animal Health Committee - which consists of the Chief Veterinary Officers of each State and Territory, CSIRO and the Commonwealth approved the funding package during their meeting in October.

This will ensure that each State and Territory is entitled to a training course of up to a week's duration in each State in each year. The Registrar's travel and living expenses are covered by the grant.

Special thanks go to members of ASVP who lobbied successfully for the Registry to continue its valuable work.

Dr. Bill Hartley has been invited to continue in the position of Registrar but has not yet confirmed his availability.

Should Bill decide not to continue, then the position will be advertised by the ASVP in February 1992. It is the committee's view that the Registry should be located wherever the best Registrar is located - provided adequate facilities are available to house it and that the location is easily accessible.

Tony Ross
Chairperson
Management Committee

From the Editor

Despite the short time between this and the previous report, we have received an interesting variety of material. We are now almost back on schedule. The June report became the August report and was finally distributed in September, this October report has become the November issue and will hopefully reach you this month, the next report is planned for late January/February, and the deadline for material will be January 20. If you wish to send material to your state representatives earlier to avoid the Christmas period, by all means do so.

We have included in this report our most up-to-date membership list. Please check not only the details of your own membership, but of any other members who may have moved etc. and advise us of any changes and additions. We are keen to update our records, including phone numbers and will publish the updated list in the interests of better communication amongst us all next year.

Gary Reddacliff
Honorary Editor

3.

NAME	ADDRESS	90	91	NOTES	PHONE
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BOULTON, JC	REGIONAL VETERINARY LABORATORY, WOLLONGBAR NSW 2480	Y	Y	.	.
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SIMS, LD	PO BOX 125, BENDIGO VICTORIA 3550	Y	Y	.	.
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SMALL, AC	REGIONAL VETERINARY LABORATORY, DEPT. OF AGRICULTURE, FOREST RD, ORANGE NSW	Y	Y	.	.
SMITH, BL	RUAKURA AGRICULTURE CENTRE, PRIVATE BAG HAMILTON, NEW ZEALAND	.	Y	.	.
SMITH, HV	10 MANICA ST. WEST BRUNSWICK, VICTORIA 3055	Y	Y	.	.
SMITS, B	C/O 57 MURRAY STREET, LANE COVE NSW 2066	Y	Y	.	.
STAPLES, P	ELIZABETH MACARTHUR AGRIC INST, PMB 8, CAMDEN NSW 2570	Y	Y	.	.
STEPHENS, L	VET. RESEARCH INSTITUTE, ATTWOOD, MICKLEHAM RD WEST MEADOWS VIC 3049	Y	.	.	.
STEWART, DJ	8/63 THE ESPLANADE, CRONULLA, NSW 2230	.	Y	NEW 91	.
STORIE, GJ	ANIMAL RESEARCH INSTITUTE, 665 FAIRFIELD RD, YEERONGPILLY, QUEENSLAND 4105	Y	.	.	.
SULLIVAN, N	UNIVERSITY OF MELBOURNE, VET. CLINICAL CENTRE, PRINCES HIGHWAY, WERRIBEE VIC.	.	.	NOT SINCE 88/89	03 7413500
SUMMERS, BA	NYS COLLEGE OF VET. MEDICINE, CORNELL UNIVERSITY, ITHACA NY 14853	Y	Y	.	.
SUTTON, RH	DEPT. OF VETERINARY PATHOLOGY, UNIVERSITY OF QLD., ST LUCIA QUEENSLAND 4072	Y	Y	.	07 3772565
TAYLOR, JD	TOOWOOMBA VET. LAB., DEPT. OF PRIMARY INDUSTRIES, PO BOX TOOWOOMBA QLD 4350	Y	.	.	.
THACKER, L	ANIMAL DISEASE DIAGNOSTIC LAB., PURDUE UNIVERSITY, W. LAFAYETTE, INDIANNA	.	.	NOT SINCE 88/89	.

7.

NAME	ADDRESS	90	91	NOTES	PHONE
THAM, VL	CENTRAL VET. LABORATORY, DEPT. OF AGRICULTURE, GPO BOX 1671, ADELAIDE S.A.	Y	.	.	08 2287322
THOMAS, J	8 CARR STREET, WEST PERTH W.A. 6005	Y	.	.	.
THOMPSON, J	C/O BATCHELAR ANIMAL HEALTH, LAB., PO BOX 1654, PALMERSTON NORTH, NEW ZEALAND	.	.	NOT SINCE 89/90	.
THOMPSON, KG	GROVE RD 10 RD, PALMERSTON NORTH, NEW ZEALAND	.	.	NOT SINCE 89/90	.
TIMMINS, BJ	87 PINJARRA RD, PINJARRA HILLS, QLD 4069	.	.	NOT SINCE 89/90	07 8929555
TRUEMAN, KF	ANIMAL RESEARCH INSTITUTE, 655 FAIRFIELD ROAD, YEERONGPILLY, QUEENSLAND 4105	Y	Y	.	.
UAKABUA, JN	VETERINARY PATHOLOGY LAB., PO BOX 77, KORONIVIA, NAUSORI, FIJI	.	.	WARNED OWE \$57.50	.
UTTERIDGE, T	NO VET PATH, RESIGNED 1990, 100 WATERFALL GULLY RD, WATERFALL GULLY S.A. 5066	Y	N	RESIGNED SEPT 1990	.
VANSELOW, BA	REGIONAL VETERINARY LAB., ARMIDALE, NEW SOUTH WALES 2350	Y	.	.	.
WALKER, KH	ELIZABETH MACARTHUR AGRIC. INST. PRIVATE MAIL BAG 8, CAMDEN, NSW, 2570	Y	Y	.	046 293333
WATSON, JL	ANIMAL HEALTH LABORATORY, MT PLEASANT LABS., PO BOX 46, KINGS MEADOWS, TAS.	?	?	NO RECORD OF RECEIPT	003415217
WATT, DA	DEPT. VETERINARY PATHOLOGY & PUBLIC HEALTH, UNIVERSITY OF QLD, ST LUCIA QLD.	.	.	NOT SINCE 87/88	.
WEBB, R	AGRIC. RESEARCH & VETERINARY CENTRE, FOREST ROAD, ORANGE NSW 2800	.	.	NOT SINCE 87/88	.
WEBBER, JJ	REGIONAL VETERINARY LAB., PO BOX 406, HAMILTON, VICTORIA 3300	Y	Y	.	.
WHITE, WE	VETERINARY RESEARCH INSTITUTE, PARK DRIVE, PARKVILLE, VICTORIA 3052	.	.	NOT SINCE 87/88	.
WHITELY, P	DEPT. VETERINARY SCIENCE, 1655 LINDEN DRIVE WEST, UNI. OF WISCONSIN, MADISON	Y	.	.	.
WHITTINGTON, R	47 MENANGLE ROAD, CAMDEN, NSW 2570	Y	Y	.	046 293333
WICKHAM, N	16 MOONA PARADE, WAHROONGA, NEW SOUTH WALES, 2076	.	Y	.	.
WILKS, C	DEPT. OF VET. PATH & PUBLIC HEALTH, FACULTY OF VET SCIENCE , MASSEY UNI. PALM	.	.	WARNED OWES \$57.50	.
WILLIAMS, DM	P.O. BOX 729 HAMILTON, VICTORIA, 3300	Y	.	.	.
WILLIAMS OJ	83 CROMWELL DRIVE, ALICE SPRINGS, NORTHERN TERRITORY 0870	Y	Y	.	.

ASVP SLIDE-OF-THE-MONTH

The slide-of-the-month, as the name implies, is a monthly mailing of veterinary pathology cases from various contributing laboratories. "The slides are sent out in a "mystery slide" format so that recipients can try to arrive at their own diagnoses given the history, gross pathology and the slide. Two cases per month are sent, a mammalian case and a non-mammalian selection.

The convenor of the SOTM seeks contributions from well established laboratories by invitation annually, however, would be keen to hear from members of the ASVP who have not been asked to contribute and believe they have something to offer.

Only one set of slides is sent to recipient laboratories. These are sent to an individual at the establishment who must be a member of the ASVP. Of course non-members of the ASVP at these establishments also benefit by being able to view the slides.

The SOTM has been running for several years now, with Clive Huxtable as the initial convenor, and a computerised up-to-date list of cases is available from the current convenor.

Peter Phillips
Convenor, ASVP Slide-of-the-Month
Vetlab
GPO Box 1671
ADELAIDE SA 5001

LETTERS TO THE EDITOR

ERRATUM NOTICE

From Roger Kelly:

There is a sloppy error in the monograph: "Liver Diseases: Pathophysiology" by WR Kelly in the ASVP Annual Conference Proceedings, 1991.

Under "*Cestrum* Poisoning", lines 2 & 3 should read:

"... identified as an atractyloside, thus placing it with toxins from members of the Compositae such as *Xanthium* sp..."

(I'm glad Selwyn Everist was spared this shocker!)

Position Announcement

**Batchelar Animal Health Laboratory
Ministry of Agriculture and Fisheries
Palmerston North, New Zealand**

Position: Veterinary Pathologist

Available: 1st February 1991

Salary: \$NZ55,000 - \$58,108 pa

Duties and Responsibilities: The veterinary pathologist is responsible for necropsy and histopathological examinations on multispecies material, routine case management, client liaison; participation in special projects or commercial contracts either individually or as a member of a team; provision of surveillance or other disease information as required by the laboratory manager or chief veterinary officer.

Qualifications: A candidate must possess a BVSc degree or equivalent registerable veterinary qualification and formal training and experience in diagnostic anatomic pathology is essential, preferably with a post graduate qualification in this field.

Enquiries: Dr A C Johnstone
Batchelar Animal Health Laboratory
PO Box 1654
Palmerston North
New Zealand
Phone (06) 35 61911

QUEENSLAND - Fraser Trueman

TOOWOOMBA VETERINARY LABORATORY

Inherited Spinal Myelinopathy of Murray Grey cattle (John Gibson)

A suspected case of inherited progressive spinal myelinopathy of Murray Grey cattle was diagnosed in a Murray Grey steer from a small herd at Millmerran. The animal was bright, alert and in sternal recumbency and unable to rise when presented for necropsy. The owner reported the animal was able to drag itself around using only its forelimbs. Five similar cases had been seen by the owner over several years. At necropsy there was severe bruising and oedema around both hocks with extensive pressure necrosis of muscles in the right hindlimb. Histologically there was severe demyelination of the ventral and dorsolateral white matter tracts in the thoracic and lumbar spinal cord. The fasciculus gracilis and cuneatus were only mildly affected.

Stringhalt (John Gibson)

Private veterinarians at Stanthorpe have reported a sharp increase in the number of cases of stringhalt in the area. Following a very dry spring in 1990 cases began appearing in January 1991. All horses had access to Flatweed (*Hypochoeris radicata*) though many also had access to good quality pasture. At least 10 properties were known to have at least one horse affected and in most cases the horses were homebred. Some cases were so severe that the animal had to be destroyed because of massive trauma to the feet. Selenium and copper levels on affected horses were normal. No material for histology was received.

Subdural Haematoma (John Gibson)

A thoroughbred weanling colt suddenly collapsed while being haltered in its stall. It was semi-comatose with no pupillary reflex and only a sluggish corneal reflex. The motor reflexes in the hindlimbs were also greatly diminished. A brain lesion was suspected and the animal was destroyed. The head and neck were submitted for examination. Necropsy revealed a large 8cm by 4cm elliptical subdural haematoma on the left lateral aspect of the cranial cavity adjacent to the left cerebral hemisphere. There were no fractures of the skull or cervical vertebrae. Sections of brain, and especially the midbrain, had marked extravasation of erythrocytes around vessels. There was severe focal haemorrhage and malacia in the midbrain ventral to the mesencephalic aqueduct. The weanling was one of a group of 26 involved in a paddock accident 6 days prior to its collapse. The weanlings despite being in the same paddock for 6 weeks decided to stage their own version of a demolition derby. After breaking into two roughly equal groups they retreated to opposite corners of the paddock. With youthful enthusiasm they simultaneously wheeled and galloped to the centre of the paddock. The resultant collision left one with a broken pelvis and another with two fractured limbs. He can only assume that this incident predisposed the weanling we examined to a subdural haematoma.

Encephalomyocarditis Virus Infection (John Gibson)

A severe outbreak of encephalomyocarditis was diagnosed in a 200 sow piggery. Deaths occurred mainly in the weaners with some 50 pigs dying over a period of 3 weeks. Pigs died suddenly and no clinical signs were observed. Four pigs necropsied all had consistent gross pathology only varying in the degree of severity. All had fine fibrinous adhesions over the serosal surface of the intestines and liver. There was excess pericardial fluid, fine fibrinous adhesions and clots in the pericardial sac. Pale streaks and patches were evident on the epicardium and within the myocardium on cut section, histologically there was a severe acute myocarditis characterised by extensive myocardial necrosis and a diffuse mononuclear inflammatory cell infiltrate. There was moderate peri-acinar necrosis of hepatocytes no doubt due to terminal hypoxia. EMC virus was isolated from the heart of all pigs.

11.

Canine Babesiosis (John Gibson)

Babesiosis due to *B. canis* was diagnosed as the cause of death of two 5 week-old Kelpie pups recently introduced from interstate to a sheep property at Bollon. The property has a breeding kennel producing working sheep dogs from a group of 30 mature dogs. One week after their arrival the pups had a sudden onset of depression, anorexia and anaemia.

Gross pathology described at necropsy included extreme pallor of the carcass, excess pericardial fluid and hyperaemia of lymph nodes. Both pups had a heavy roundworm burden. Clinical pathology included severe anaemia (Hb 2g/L; PCV 7%) and elevated bilirubin (20 Um/L), AST (119 IU/L) and ALT (162 IU/L). The pups were also hypoproteinaemic (TP 32, Alb 19).

Histological changes were consistent in both pups and included severe diffuse periacinar hepatic necrosis with extramedullary haematopoiesis in the liver and spleen. There was erythrophagocytosis in the sinusoids of the liver and lymph node. The spleen was depleted of erythrocytes and there was occasional focal necrosis and karyorrhexis of lymphoid follicles. There was mild bile retention. Intraerythrocytic parasites consistent with *Babesia can* were readily demonstrated in the capillaries of most organs using Giemsa stain.

Salmonellosis (Jim Taylor)

Twenty one calves were purchased through the Toowoomba sale yards. The day after purchase 6 developed a white scour and started to lose condition, 3 subsequently died. One was autopsied by a local practitioner and samples submitted to the laboratory. Significant autopsy findings were a necrotizing stomatitis and excess greyish/yellow fluid contents in the large intestine. Laboratory findings include a neutrophilia with marked left shift, non-regenerative, normocytic, normochromic anaemia, hyperfibrinogenaemia and a mild elevation in GLDH. The liver had classic paratyphoid nodules in histologic sections and mild crypt abscessation in sections of small intestine. *Salmonella dublin* was isolated on primary, non-selective media from both the small and large intestine.

YEERONGPILLY VETERINARY LABORATORY

Pyrrrolizidine alkaloidosis (Laurie Dowling)

Pyrrrolizidine alkaloid poisoning was diagnosed in a 9 month old Friesian heifer at Beaudesert in June 1991. Three of a group of 28 were affected. One had died. Loss of condition, diarrhoea and submandibular oedema were reported. Typical liver lesions with megalocytosis, biliary ductular hyperplasia and fibrosis were seen histologically. Blood samples from the 2 survivors revealed mild anaemia, had reduced albumin concentrations (28, 25 g/l) but mild increases in total bilirubin concentrations (20, 25 umol/l) but other values were normal. A further heifer died after submission of specimens but was not necropsied. An inspection of the pasture revealed significant quantities of blue heliotrope (*Heliotropium amplexicaule*), a known pyrrrolizidine alkaloid container.

Lactation tetany (hypomagnesaemia) in dairy cows (Greg Storie, Ross McKenzie)

Lactation tetany (hypomagnesaemia) was diagnosed in a Friesian dairy herd in late May 1991. A herd of 180 cows had been milked, fed grain in the bails and let into a young ryegrass (*Lolium rigidum*) pasture (10cm high) at 11.00 am. By 1.00 pm, 2 were dead and 3 others were drooling saliva, frothing at the mouth and behaving in a demented manner, shivering/shaking and were very difficult to handle. Calcium borogluconate treatment by the farmer was ineffective. The herd was moved and no other cows were affected. The attending veterinarian discounted nitrate poisoning (mucous membranes were pink, not brownish) and administered monopropylene glycol therapy for suspected ketosis. One cow was noted to

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chew continuously at the bars of the race where she was being examined. Nervous signs abated by the next day. Affected cows were back in milk in 4 days after treatment for hypomagnesaemia. Necropsy of one cow revealed subendocardial haemorrhage, some pulmonary oedema and a large volume of ruminal fluid, only about one third of which was grass. Histopathology was not enlightening.

Laboratory findings in the 3 surviving affected cows were profound hypomagnesaemia of 0.06 and 0.15 mmol/l in 2 cows (normal 0.65-1.30) accompanied by hypocalcaemia of 0.97 and 1.31 (normal 2.1-2.8). This, the clinical signs and the association with lush pasture was consistent with a diagnosis of lactation tetany. Pasture nitrate concentration was potentially hazardous at 3.5% KN03 d.m., but not regarded as significant in this case. A complication was reduced activities of serum acetylcholinesterase of 14, 58 and 66 IU/L suggesting exposure to an inhibitor of the enzyme such as organophosphate or carbamate insecticides, but no known exposure could be established.

Nitrate-nitrite poisoning on ryegrass (Laurie Bowling, Ross McKenzie)

Nitrate-nitrite poisoning was diagnosed in 3 herds of Friesian dairy cattle grazing ryegrass (*Lolium* sp.) at Kerry near Beaudesert in mid June 1991. Two cows died and one other was affected of 144 at risk in one herd and one cow died and 3 others were affected of 100 at risk in the second herd. No epidemiological data were available from the third herd. Signs described were staggering, tachycardia, polypnoea, brown-tinged mucous membranes, decreased rumen motility, excessive salivation and normal rectal temperature. Blood was brown at necropsy. Concentrations of nitrate in aqueous humour from 3 dead cows were 50, 25 & 25 mg/l. The last 2 of these cows had been dead for about 20 hours. Normal cattle can be expected to have about 5 mg nitrate/l aqueous humour. Nitrite was detected in the first sample, but not in the others. Nitrite is not normally found in aqueous humour 3 hours after death. Pasture nitrate concentrations at the time of the incidents were 3.3 & 5.8% KN03 in dry matter. A pasture sample taken 14 days later on a warm sunny day after a period of fine weather contained 8.1% KNQ3 in dry matter.

The circumstances surrounding these incidents did not conform to the classical cloudy weather, high soil nitrogen, water stressed plants scenario. The weather was warm and sunny during the day, cold at night and there was good soil moisture and the plants appeared to be growing well. Only small amounts of fertilizer had been applied at sowing. Never-the-less, there had been a dry summer with little rain to leach out the build-up of soil nitrogen during this time. This may have been the underlying cause of toxic nitrate concentrations in the ryegrass.

A report from another veterinarian in the district revealed that nitrate-nitrite poisoning on ryegrass was common in the local dairy herds at that time. He reported treating numerous cattle.

Hydrocephalus, anencephaly and abortion/arboviruses (Ross McKenzie, Greg Storie)

Bilateral internal hydrocephalus of the lateral ventricles and involving the mesencephalic aqueduct and IVth ventricle was seen in an aborted foetus of crown-rump length 51cm submitted from a beef cattle herd at Yangan via Killarney in late June 1991. The cow had a mucoid discharge for a week before the abortion. The foetus presented with arthrogryposis secondary to the brain lesion. Serology on foetal blood revealed a 1/16 titre to Aino virus, the significance of which is unclear in the light of the pathology of the case. Serological tests for Akabane, Ephemeral fever, Bluetongue and Mucosal disease viruses and for *Leptospira hardjo* and *L.pomona* were negative.

A subsequent submission from the same herd had anencephaly with almost the entire cranial contents missing except for a lining of condensed dura. Serology on foetal fluids revealed a 1/16 titre to Akabane virus and no reaction to either Aino or Mucosal disease viruses.

13.

Diazinon poisoning in sacred ibis (Ross McKenzie)

Acute organophosphate poisoning was diagnosed in a flock of sacred or white ibis (*Threskiornis molucca*) at Caboolture in late June 1991. On 24th June a large flock of about 100 ibis was feeding near a dam and appeared normal. On 25th June, 14 ibis were found dead and the rest of the flock had gone. A range of water fowl of other species were on the dam and appeared normal. Horses in the same paddock were normal. Three dead female ibis were examined at necropsy. They were in moderate-to-good condition. There was some soiling of the vent area in 2 birds suggesting diarrhoea. All birds had large amounts of abdominal fat. Their proventriculi and gizzards were full of ingesta which had a pink colour in some. No significant changes could be seen grossly or histologically in viscera. A strong positive qualitative test was obtained for diazinon in stomach contents.

Reo-like virus isolation and liver and kidney lesions in imported guppies (*Poecilia reticulata*) (Peter Ketterer, Yung Chung)

Acute and subacute liver lesions were seen histologically in a batch of red tail guppies which had 28% mortality. They were treated with neugvon formalin and furabac while in quarantine. Virus isolation was not attempted. A further batch of female guppies from the same overseas supplier had 52% mortality and had similar but less severe liver lesions. Treatments used were formalin and malachite green. A reo-like virus was isolated in Australian bass kidney cell culture from a pooled liver sample of 11 fish. Isolation was unsuccessful in bluegill fry cell line. Two batches of blue neon guppies from the same supplier but a different importer had high mortalities in quarantine (72%, 100%). No treatments were used. Fatty infiltration was seen histologically in livers and progressive focal chronic granulomatous nephritis in kidneys (epithelioid granulomas derived from degenerating tubules with mineral content). A reo-like virus was isolated from a pooled liver sample of 8 fish, in Australian bass kidney cell culture and in bluegill fry cell line.

Although the reo-like virus could not be definitely associated with histological liver or kidney lesions seen in the guppies its isolation in Australian bass cells indicates it is potentially pathogenic to an important Australian native fish. The use of cell cultures and cell lines from Australian native fish is also seen to be important in this study.

TOOWOOMBA VETERINARY LABORATORY

(John Gibson)

IBR Encephalitis

Encephalitis due to bovine herpesvirus Type 1 was diagnosed in a group of Belgian Blue calves all derived by embryo transfer on a property at Warra. The calves were 2 weeks old and three of eight died after clinical signs of depression, reduced suckling reflex, incoordination, head nodding and terminal convulsions. The calves were delivered by elective caesarean section after induction of the recipient cows with corticosteroids and prostaglandin. Histologically there was a severe acute encephalitis characterised by a non-suppurative vasculitis, malacia and neuronal necrosis. Numerous eosinophilic intranuclear inclusion bodies were present within glial cells and neurones. There was an acute necrotising bronchiolitis with viral inclusions in the bronchiolar epithelium. IBR virus was isolated from swabs of the brain. It is likely the use of corticosteroids to induce parturition caused re-activation of latent IBR virus in the dams. Several workers have reported the re-activation of latent IBR infections in cattle using synthetic corticosteroids (Dennet *et al* 1973, AV.J. 49:594).

Veterinary School - The University of Queensland (Dick Sutton).

Ceroid - Lipofuscinosis in a Miniature Schnauzer.

This is a degenerative storage disease which has been recorded in many breeds of dogs (Chihuahua, Dachshund, English Setter, Cocker Spaniel), Siamese cats and some cattle and sheep breeds. The biochemical processes in the disease are poorly understood but like cost storage diseases the main effect is on cells of the central nervous system. This condition has not, to our knowledge, been previously recorded in the Miniature Schnauzer. The 4 year old female had sudden onset of blindness 7 months prior to necropsy. In the final 2 months there were CNS signs of aimless wandering and un-responsiveness.

Post-mortem findings included brownish-yellow pigment accumulation in neurones throughout the brain with highest involvement in the brain stem and post particularly, the Purkinje cells. Special staining with PAS and Sudan black confirmed this as a case of ceroid -lipofuscinosis. There was similar pigment storage in the Kupffer cells of the liver and also in the neurones of the ganglion cell layer of the retina. Involvement of the retina in such a storage disease has not been a feature of reports of the condition. It may be a possible explanation for some cases of sudden acquired retinal degeneration (SARD) which is a term used for acute onset of blindness with an ophthalmoscopically normal fundus.

Mast Cell Tumours in Cats.

There have been two cases encountered in the last two months, one of which was leukaemic.

The first case was a 10 year old domestic short-hair which had been vomiting for a week. It was polydipsic and inappetent with a palpable abdominal mass. The main feature of the peripheral blood sample was 14% mast cells in a count of 20×10^6 /litre. Post-mortem examination showed a very large pale fleshy spleen (Wt = 184g. Total BW = 4.5Kg), and a perforated pyloric ulcer with localised peritonitis. The spleen was virtually entirely replaced and distended by mast cells with a few remnants of normal splenic structure. The liver showed periportal infiltration but the bone marrow was not involved. The second case was a 15 year old tabby neutered male which had lost weight over several months and with a distended abdomen. There were no marked changes in the peripheral blood sample. The spleen was grossly and microscopically similar to the previous cat and there was similar infiltration of the liver.

Splenomegaly is the main feature of this disorder with bone marrow, liver and other organs such as lymph nodes and kidneys frequently involved. Gastric or duodenal ulceration, frequently with secondary peritonitis, occurs due to the histamine stimulated hypersecretion of acid.

Generalised Lymphadenopathy in a Horse.

A young (2 year old) thoroughbred was presented with loss of weight, nasal discharge and marked enlargement of many of the superficial nodes. An aspirate suggested a chronic inflammatory process but because of the generalised nature of the disease, palpable enlargement of visceral nodes by rectal examination were apparent, lymphoid neoplasia was suspected and the horse necropsied. The very enlarged nodes showed loss of normal architecture with a florid epithelioid cell and giant cell response against a background of fairly normal but non-structured lymphoid tissue. Similar reactions were present in the liver, lungs and visceral and parietal pleura. Culture and special stains, including ZN have failed to reveal a possible causative agent. Recent thinking includes an unusual form of lymphoreticular neoplasia. Further opinion is being sought and sections can be forwarded to anyone who may be interested.

Pneumonia in Pigs.

We have recently seen a couple of cases of severe fibrinous pleuropneumonia in 3-4 month old pigs. The main features were extensive fibrinous pleuritis, extensive lung consolidation which histologically varied from suppuration to severe intra-alveolar and septal oedema. There was also a mild fibrinous serositis of the intestinal serosa. Whilst viral and possibly mycoplasma agents are possible initiators of such a reaction the organism isolated was *Pasteurella multocida*. Culture was negative for *Haemophilus* (*Actinobacillus*).

VICTORIA - John Mackie**CENTRAL VETERINARY DIAGNOSTIC LABORATORY****Pancytopenia in a horse** (Peter Lording/Sue Friend)

A four year-old Thoroughbred gelding was seen by the referring veterinarian because of lethargy and anorexia. On examination there was fever (41°C) but no other clinical abnormality.

The horse was treated with penicillin, neomycin and phenylbutazone. Body temperature returned to normal and the horse regained his appetite. Two days after the completion of treatment, there was a further episode of fever and anorexia which was treated with tetracyclines and phenylbutazone. Treatment was stopped when the horse developed soft faeces. Since that time he has been subdued and eating only small amounts.

Blood was collected for haematology and the following results were obtained.

HAEMATOLOGY

		TEST RESULTS	NORMAL EQUINE VALUES
RBC x	10 ¹² /l	3.48	6.5-12.5
Hb	g/l	64	110-190
PCV	l/l	0.19	0.32-0.52
MCV	fl	57	34-58
MCH	pg	18	12-18
MCHC	g/l	320	310-370
T. Protein	g/l	80	60-80
WBC x	10 ⁹ /l	4.10	5.50-12.50
Seg Neutrophils		0.04	2.50-6.50
Lymphocytes		4.08	1.5-5.50
Poikilocytosis		+	
Rouleaux		++	
Lymphocyte Activation		++	
Platelets		decreased	

Many of the lymphocytes in the peripheral blood were large. The nuclei were occasionally irregular in shape with coarse nuclear chromatin and nucleoli. Some cells had a large volume of blue/grey cytoplasm which was vacuolated in some cases.

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The key findings in the haematology examination were anaemia, neutropaenia and thrombocytopaenia with numerous atypical lymphocytes present. The tentative diagnosis of pancytopenia and myelophthisis secondary to lymphosarcoma was made. Bone marrow was aspirated from the sternum and smears contained a large number of lymphoblastic cells. Cells of the erythroid, myeloid and megakaryocytic series were not detected.

REGIONAL VETERINARY LABORATORY, BAIRNSDALE

ARSENIC POISONING IN CALVES (Kit Button)

Nine of 35 Friesian heifer calves aged 3-6 weeks on a Phillip Island property died of arsenic poisoning. Some affected calves had shown nervous signs and others had been scouring. No diagnostic gross changes were seen during field necropsies. Thiamine deficiency and lead poisoning were ruled out on materials sent to the RVL. An old sheep dip was present in the calf paddock and subsequent tests on livers from several dead calves were positive for arsenic. Arsenic concentrations in soil from the run-off area of the old dip and in dip water were approximately 30 ppm and 2.5ppm respectively.

OVINE ABORTIONS (Kit Button)

Multiple abortions occurred on two properties in Gippsland. A cotyledon from one property showed necrosis of villi and mixed inflammatory cell infiltration. A heavy growth of *Campylobacter fetus* was isolated from the cotyledon and foetal stomach contents. A caruncle from the second property showed patchy necrosis and mineralisation with infiltration of neutrophils and colonies of gram positive coccobacilli. Large numbers of *Listeria monocytogenes* were isolated.

INTESTINAL TORSION AND LUCERNE (Peter Mitchell)

Three rams in a group of 35 died suddenly on a property near Stratford in July. The animals were grazing 15cm long lucerne. They had received a complete course of vaccinations and had been drenched with Ivomec. One ram submitted for post mortem examination had a 180° torsion at the root of the mesentery. No parasites, bacterial pathogens or clostridial toxins were found. A disease known as "red gut" was described in New Zealand in 1973 (NZ Vet J 21:178, 1973); deaths were caused by intestinal torsion in lambs grazing pure stands of lucerne; only one lamb died in a group grazing a weedy stand of lucerne. The pathogenesis of "red gut" was not determined but there appears to be an association between lucerne and intestinal torsion in the New Zealand report and also in the rams from Stratford.

LUNGWORM IN FALLOW DEER (Ian Jerrett)

Dictyocaulus infestation was diagnosed as the cause of death in deer on four Gippsland properties this winter. In all cases, fallow deer around 6 months of age were affected. Morbidity was estimated to be 15-30% and mortality was 5-10%. Prominent signs of respiratory distress were observed in animals on 2 of the 4 properties. Autopsies revealed mottled consolidated lungs, lesions being most severe in the middle and caudal lobes. A flushing technique applied to two sets of lungs recovered 470 and 340 mature *Dictyocaulus viviparus*. Massive abomasal worm burdens were also detected in deer from one property.

YERSINIOSIS IN RED DEER (Ian Jerrett)

Yersinia pseudotuberculosis was isolated from the intestine of deer submitted from 4 properties and from diarrhoeic deer faeces submitted from a fifth property. In all cases Red deer or crossbred Red deer were affected and animals ranged from 5 to 9 months of age. Animals were either found dead or showed diarrhoea, depression and dehydration. In cases in which tissues were not autolysed, a haemorrhagic enterocolitis characteristic of *Yersinia spp* infection was found.

OSTRICH AIR SACCUKITIS (Kit Button)

Three cases of air sacculitis were diagnosed recently. A three year old cock bird suffered severe dyspnoea with no response to courses of antibacterial and antifungal therapy. A stunted 6 month old male chick exhibited dyspnoea after exercise, and was "wheezy"; no treatments were given. A 5 month old male chick was stunted and showed no response to antimicrobial therapy, vitamins and anabolics. All 3 birds had air sacculitis. No bacterial or fungal pathogens were isolated from the first 2 birds; *E coli* was isolated from the air sac membrane, liver, spleen and lung in the third bird.

Inflammatory changes were chronic and pyogranulomatous in the first two cases, with vacuoles present in giant cell and macrophage cytoplasm. In the first case, fat stains indicated that the vacuoles contained a lipophilic material. There is a developing suspicion that many cases of air sacculitis in ostriches may have a foreign-body aetiology. Liquid paraffin (mineral oil) is a possible candidate substance. Proventricular impaction is a well-recognised problem in ostrich chicks, and many owners administer liquid paraffin, sometimes without the use of a stomach tube.

In the third case, air sacculitis was part of a disseminated bacterial infection affecting multiple tissues/organs.

ENCEPHALOMALACIA IN A GREY KANGAROO (Peter Mitchell)

Ataxia, weakness and lack of balance developed in a fifteen month old eastern grey kangaroo developed. A previous similar case recovered; this animal worsened over five months and was euthanased. Histology of the brain revealed bilaterally, symmetrical areas of malacia in the thalamus, the central grey matter of the mid brain and the cerebellar nuclei, characterised by large numbers of swollen axons and large foamy cells and by marked vascular proliferation. Nerve cell bodies appeared normal. In addition, groups of macrophages (mainly perivascular) containing haemosiderin were present in an area of the medulla, probably the result of trauma. The vascular proliferation and large number of foamy macrophages in the malacic areas were unusual (in my experience and that of WJH) but may be the normal reaction of a macropod brain to disease such as FSE or thiamine deficiency. The cause of the malacia remains unknown. Has anyone seen a similar case?

GIANT CELL TUMOUR OF SOFT TISSUES IN A COW (Peter Mitchell)

A five year old cow developed a 15cm diameter ulcerated mass on the stifle and generalised enlargement of lymph nodes. Lymph nodes contained pale, nodular circumscribed masses, with areas of necrosis in the centre of the larger lesions. Histologically, the stifle lesion and lymph nodes contained sheets of tumour cells characterised by cells with large nuclei and abundant, slightly granular cytoplasm by fusiform cells associated with strands of collagen, and by multinucleate cells with up to 15 nuclei and abundant cytoplasm. Mitoses were rare. In the lymph nodes, fibrous tissue separated the tumour from normal tissue. The tumour was diagnosed (for want of suitable alternatives) as a giant cell tumour of soft tissues. A hunt through Veterinary Bulletins uncovered two reports of similar tumours, one in rats (Majed et al 1988. J Comp Path 99:235) and one in horses (Render et al 1983. JAVMA 183:790).

RYEGRASS STAGGERS AND CLOSTRIDIAL ENTEROTOXAEMIA (Peter Mitchell)

A group of 470 sheep developed ryegrass staggers and were moved to a new paddock on Thursday May 2nd and supplemented with barley. Ten died over the next three days. Two live sheep presented on Tuesday 7th were blind, one was head pressing and the other was in sternal recumbency. Areas of haemorrhage and malacia in the brain were typical of focal symmetrical encephalomalacia. Swollen axons in the granular layer of the cerebellar cortex were consistent with ryegrass staggers. Did the ryegrass staggers initiate the enterotoxaemia, or was it the change of diet or paddock? '

RVL BENALLA (John Mackie)

ACTINOBACILLOSIS IN SHEEP

Discharging subcutaneous abscesses and scabs developed on the faces of mixed sex, mature, fine wool Merino sheep. Other clinical signs included depression, apparent blindness and recumbency. The mortality over 6 months was approximately 50 of 4,000. Necropsy of one sheep revealed abscessation of subcutaneous tissues and lymph nodes of the head with extension into the cranio-ventral region of the brain. Histologically there was pyogranulomatous lymphadenitis with abscessation and a localised pyogranulomatous encephalitis, associated with club colonies containing gram-negative bacteria. *Actinobacillus lignieresii* was isolated.

Actinobacillosis in sheep is reported to occur usually sporadically and occasionally in outbreak form. Characteristic lesions occur in the subcutaneous tissues of the head, especially of the cheeks, lips, nose, throat and in the nasal cavities. The frequency of lip involvement in sheep has led to the disease being called "learner lips" in some areas, though in this case lip involvement was not a feature either in the sheep necropsied or in the others according to owner observations.

Much dry, spikey plant material, including thistles, was present in the paddocks over summer and autumn following the dry seasonal conditions, and may have predisposed the infection. The sheep had been given supplementary feed from mid-summer onwards.

SOUTH AUSTRALIA - Vui Ling Tham

VETLAB, DEPARTMENT OF AGRICULTURE, ADELAIDE

WEIGHING OF ANIMALS IN A POST-MORTEM ROOM (Pathology Section)

The weighing of large animals in our post-mortem room has always been a problem with the facilities we have. This problem has been overcome recently with the purchase of an electric weighing platform as shown in the following figure.

The platform scale has a capacity to weigh up to 2 tonnes with an accuracy to the nearest 100 grams. This platform has been built into one of our post-mortem tables so that animals can be weighed automatically when placed on the table awaiting necropsy.

Post-mortem table with electric weighing platform

VETERINARY PATHOLOGY SERVICES PTY LTD.**LYMPHOSARCOMA IN A SUGAR GLIDER** (Ruth Reuter)

A 4-year-old male sugar glider was presented to the veterinary clinic with vesicular eruptions over the body, head and ears, and large ulcerative lesion on the hind leg. Treatment with Lincomycin, Amoxil, Flagyl and topical gentamycin at various times over an extended period had no effect. The animal was bright and apparently clinically normal otherwise. A small wedge biopsy of the ear tip was submitted in formalin to the laboratory.

Microscopic examination showed hyperkeratosis with collections of spores suggestive of dermatophyte infection on the surface of the skin and surrounding the hairshafts in the follicles. The dermis was infiltrated by large numbers of mononuclear cells, many with indented nuclei and moderately eosinophilic cytoplasm. Mitotic figures were present in some of these cells. The microscopic picture resembled "mycosis fungoides" of humans and a diagnosis of lymphosarcoma was made. The glider was euthanased and a complete post-mortem performed.

There was extensive involvement of the skin with nodules similar to that originally biopsied. The spleen exhibited a combination of extramedullary haematopoiesis and neoplastic invasion. Peripheral lymph nodes were obscured by neoplastic lymphoid cells extending out into the surrounding connective tissue. Small lymphoid foci were present in the renal cortex. Other organs systems were normal

MALIGNANT TESTICULAR TUMOUR IN A DOG (Ruth Reuter)

A 12-week-old male German Shepherd dog was presented to a veterinary clinic with an enlarged, nodular, non-painful left testicle. On clinical examination the nodules appeared to extend up the spermatic cord to the inguinal canal. The right testicle was small but normal in consistency. The dog was castrated and the affected left testicle and a section of the spermatic cord was submitted in formalin.

Microscopically the testicle contained a wide range of elements including areas of bone formation; acini lined by cuboidal cells with lumina distended by hyaline material, adipose and fibrous connective tissue, and cells with oval to round vesicular nuclei, basophilic cytoplasm and prominent nucleoli. Giant cells, foci of necrosis and numerous mitotic figures were also seen. Similar cells were seen in the nodules on the spermatic cord. A diagnosis of malignant neoplasm, possibly embryonal carcinoma or teratoma, was made. The puppy was euthanased.

At necropsy there was gross distension of the abdomen with blood-stained fluid. The mesenteric lymph nodes, alt lymphatics and large areas of peritoneum and diaphragm contained soft white nodules. Foci of implantation were also present on the gastric serosa. This case was seen by various veterinary and human pathologists in the country, and was also sent to the Arm Forces Institute of Pathology in Washington DC for comment. The Departments of Veterinary and Genitourinary Pathology there gave a differential diagnosis including carcinoma of the rete testis, embryonal carcinoma and mixed tumour of germ cell and stromal elements. They are pursuing the case further with wet tissue, and will let us know if they can make a definite diagnosis.

NEW SOUTH WALES - Paul Gill

Regional Veterinary Laboratory, Wagga Wagga (John Glastonbury)

Bovine congenital dyschondroplasia (Jocelyn Godwin)

Following on our item in the previous edition of the "Veterinary Pathology Report", there have now been 23 cases of dwarf calves submitted to the laboratory from 13 different farms in the Gundagai, Wagga Wagga and Urana Rural Lands' Protection Boards over the last 2 months. This represents a total of 99 affected calves out of a combined herd size of 529 cows. The incidence on individual farms varies from <5% to >90% of calves born. All calves have been underweight (average about 24 to 26kg) and undersize (average, 65cm crown-rump length). They were stillborn or died within 2 to 3 days of birth. Those calves that were submitted live appeared bright and alert; however, they had severe ataxia and paresis in all 4 limbs. They had apparently normal vision. There were some reported cases of joint laxity in other calves from the affected herds. Affected calves hand reared by the owners showed improvement in the limb deformities, however, they remained stunted in body growth.

The calves are born with various degrees of bony deformities of the skull, long bones and vertebrae. Supination or pronation of both forelimbs and/or hindlimbs was often present with valgus or varus deviation from the carpal, tarsal, hock or knee joints in some cases. Arthrogryposis was present in some cases. Scoliosis, lordosis and/or kyphosis were present in a few cases. Superior brachygnathia was a common finding and resulted in a compact or shortened skull.

Grossly, the long bones appeared shortened with enlarged articular condyles. On sagittal section the physes appeared irregular and discontinuous. In some cases the articular-cartilage had an uneven surface.

Histological findings have included physes of irregular thickness with segmental premature closure. The hypertrophic zones of the physes appear disorganised and in many bones the physes have been completely disrupted leaving islands of cartilage separated by bridges of marrow. In many cases tongues of hyaline cartilage have extended into the metaphysis which in some contained horizontal secondary spongiosa indicative of growth arrest.

Studies are currently being carried out to identify a common factor linking the affected farms. The breeds affected include Hereford, Angus, Murray Grey, Shorthorn and Braford. Affected cows have produced normal calves from the same bulls in previous years which eliminates a genetic problem. The grazing and feed supplement histories of affected herds varies considerably as does the soil type of affected farms.

Laboratory investigations have shown all but 1 affected calf to have presuckled immunoglobulin levels of <80ug/ml indicating no immune response was mounted after the foetus had developed immunocompetency. Liver and kidney manganese levels of affected calves have been in the low to normal range. Vitamin A levels have not been determined, however, tissues have been held for further testing.

Suspected exotic disease - bovine papular stomatitis

A scare was raised when a 14-day-old Hereford bull calf was found to have shallow ulcers with raised borders in the tunica mucosa of the gums and buccal cavity. Within 24 hours of submission of the samples the Australian Animal Health Laboratory at Geelong was able to provide the alternative diagnosis of bovine papular stomatitis. Parapoxvirus particles were detected electromicroscopically and cell cultures on BTY, BHK, Vero and MDBK cell lines were negative as was the capture ELISA for pestivirus.

Yersiniosis in Sheep

Cold wet weather precipitated outbreaks of yersiniosis in 6 to 12-month-old weaners on 7 farms. Clinical signs were diarrhoea and death. Out of a total of 5,020 animals at risk the morbidity and case fatality rates were 15.3% and 13.6% respectively. In each case *Yersinia pseudotuberculosis* III was recovered. Histological confirmation was obtained in 3 outbreaks by the detection of acute segmental erosive enteritis in 2 and diffuse necrotising cholecystitis in 1.

Coccidiosis in Sheep

This protozoan made a major contribution to diarrhoea and death amongst 4 to 6 month-old weaners on 2 farms. The morbidity and case fatality rates were 9.5% and 23.2% respectively, amongst the total of 5,900 animals at risk. Moderate to severe necrotic enteritis was found histologically.

Regional Veterinary Laboratory, Armidale

Ketosis, hypomagnesaemia in cattle (Steven Hum)

Nutritional and metabolic problems were frequently encountered throughout the winter due to the dry conditions and subsequent poor pasture (AN91/1813, 1982, 2022, 2191, 2297, 2298, 2400). Clinical and subclinical ketoses were frequently diagnosed by demonstrating elevated serum beta hydroxy butyrate (BHBA). In severe cases readings were as high as 20-30 mmol/L (normal 0.38-0.96).

Hypomagnesaemia was also frequently diagnosed. In many instances, animals at pasture were found dead without illness having been observed. In these cases diagnoses were often made by detecting low Mg level in aqueous humour compared to reference ranges for these minerals established in our laboratory last year. The correlation between serum and aqueous humour Ca and Mg level was well illustrated in one case where serum and a subsequent aqueous humour sample were submitted.

	Units	Serum	Aq humour	Normal values	
				Serum	Aq humour
Ca	mmol/L	1.52	1.19	2.2-3.0	1.4-1.8
Mg	mmol/L	0.22	0.95	0.7-1.91	1.2-1.6

Intra-uterine Akabane Infection

Maternal blood and pleural and pericardial fluid collected from a full-term calf with "frozen" joints were submitted for arboviral investigation (AN91/2306). The samples were referred to EMAI where the immunoglobulin assay showed 260(µg/ml IgG in foetal fluids. This finding was consistent with in-utero antigen stimulation and suggested an infectious cause. The foetal fluids were negative for Pestivirus, Palyam and Aino viruses, however, both the Gel Diffusion Precipitin and Virus Neutralisation tests were positive for Akabane. The maternal serum sample was also positive for this virus by the virus neutralisation test.

Regional Veterinary Laboratory, Wollongbar

Nervous conditions in neonatal calves (Paul Gill)

We have diagnosed a number of nervous conditions in young calves over the last few months.

A 2-week-old Angus calf (1/500) from a closed herd in the Border Ranges of New South Wales had severe ataxia since birth. Clinical signs included pronounced hypermetria in all limbs, wide-based stance, poor visual response and slow, mild head tremor. The calf was normal in size with moderate brachygnathia, a dished face and slightly dished forehead. Microscopic examinations revealed axonal spheroids and foamy vacuolated neuronal cytoplasm in the basal ganglia, thalamus, tectum, tegmentum, pons, medulla, Purkinje cells and granular layer of the cerebellum, cervical, thoracic and lumbar spinal cord. Biochemical analyses confirmed the calf was homozygous recessive for alpha-mannosidosis,

A 1-week-old Braford was dull, disinclined to move and convulsed intermittently. Microscopically, there was extensive meningoencephalitis, particularly involving the cortex, hippocampus and medulla. There were perivascular cuffs of mononuclear cells, endothelial proliferation, foci of granulomatous inflammation and necrosis of neurones. A few smudgy intranuclear herpesvirus inclusions were evident in neurones and glia. The origin of the herpes virus is uncertain as the submitting veterinarian made no mention of concurrent IBR-like disease in the herd.

A 2-day-old Friesian was described as dopey since birth and apparently failed to suckle properly. Microscopic examination revealed axonal degeneration in the white matter of the cerebellar peduncles characterised by scattered ballooning of myelin with axonal loss and myelinophagia in digestion chambers. Sections of cervical, thoracic and lumbar cords revealed axonal degeneration in all funiculi with axonal loss and digestion chambers. Luxol fast blue stains revealed marked demyelination of all funiculi of white matter. A diagnosis of Friesian axonopathy was made on the basis of the above lesions.

Miniature Dwarfism in calves

We have received cases of miniature dwarfism from 2 Orara Valley herds: one has had 5 affected Jersey calves since December, 1990; the other has had 3 Angus-cross Poll Hereford calves affected. Affected calves are approximately 55cm crown to rump and weigh about 6kg. There were no lesions in the 2 calves examined at the laboratory. Investigations indicate no known teratogenic infections. Breeding studies are planned.

WESTERN AUSTRALIA - Ron Peet

ANIMAL HEALTH LABORATORIES

Plastic assumptions in fish pathology (Jeremy S Langdon)

Fish are reared in a bewildering variety of containers, yet rarely do we assess the toxicity of the component materials. Two examples of the need to do so came this way recently. First we saw several species of tropical aquarium fish dying with greater than usual vigour in newly built glass aquaria, composed only of glass (from which one drinks beer) and silicone (with which one seals and supports), yet no cause other than an exogenous toxin seemed tenable. The fish displayed acute necrosis of the gill epithelium and the renal tubular epithelium, within a few days of introduction. Replacement of the silicone sealant with a different brand solved the problem; you will appreciate I cannot name the culprit brand here. Beer is O.K.

We then saw similar signs in trout and goldfish held in blue plastic above-ground swimming pools; again, replacement of the plastic liner with a polythene type solved the problem. Swimming in plastic pools is O.K., I hope, unless you have gills. It would seem that certain components, types or batches of plastics and silicone-based sealants can be toxic to fin fish. Yet these same products are often used without apparent harm; my purpose here is to point out the need to include this differential, and look for the relevant pathology, when faced with perplexing fish deaths in plastic containers... and isn't that all the time?

Cryptosporidiosis in broiler poultry (M J Kabay)

Cryptosporidiosis was diagnosed following an investigation of mortalities with diarrhoea in 8 week old broiler chickens. Evaluation of HE sections showed large numbers of cyptosporidia, in various stages of the life cycle, adhered to the epithelium of the bursa of Fabricius. The organism was not present in sections of the lung.

Cryptosporidiosis has been reported to cause respiratory and enteric disease in chickens (Calnek *et al* 1991). I would be interested to hear from anyone with experience of this disease causing significant losses in chickens.

Reference: Calnek 1991 in Disease of Poultry 9th Ed. Iowa State University Press, p. 797.

ASVP CASE OF INTEREST FOR VETERINARY PATHOLOGY REPORT (Ron Peet, Animal Health Laboratories, South Perth)

Congestive heart failure and pulmonary arteritis in an orangutan

A 4-year-old female orangutan became anorexic over a few days and died on a Saturday morning. The animal had been described as "slow and retarded" by Zoo staff but clinical examination and pathology revealed nothing of significance apart from respiratory rates and slightly elevated liver enzymes.

Post mortem examination revealed approximately 100mls of clear fluid in the pleural and abdominal cavities and a markedly swollen pale yellow liver with rounded edges. The lungs were swollen and full of fluid but did not appear consolidated. The animal had very little subcutaneous or other fat reserves but these did appear to have a yellow/orange colour - initially thought to indicate jaundice, but subsequently thought to be the animal's natural pigmentation.

25.

No valvular abnormalities were detected in the bean or pulmonary/arterial valves, but the walls did appear to be pale and flabby.

Histopathology revealed severe oedema in the lungs with numerous macrophages containing yellow pigment ("heart failure cells?") in the alveoli. Arteries in the lung sections showed marked thickening with whorling fibroplasia of the vessel walls, fibrin deposition, plasma/lymphoid cell and macrophage infiltration and haemorrhage. Occasional vessels appeared almost thrombosed with attempted recanalisation. The liver was markedly oedematous, but otherwise unremarkable (thankfully no evidence of hepatitis!). The heart had occasional large elongate brick-shaped nuclei in some myocytes supposedly associated with attempted cardiac hypertrophy in humans (refer Barry Richards).

There were no visible lesions seen in the brain or other tissues examined which included kidneys, spleen, gastrointestinal tract and skeletal muscle.

The liver yielded no significant isolates in bacteriology.

The animal apparently died of circulatory problems but there is some debate as to the primary aetiology. The thrombosing arteritis seen in the lungs was obviously a chronic, but active lesion which may have produced cardiac myaesthesia, but the cause of the severe vasculitis is unknown - suggestions would be welcome.

MURDOCH VETERINARY SCHOOL

Malignant Catarrhal Fever in Rusa Deer (J Thomas)

Four Javan Rusa deer died over a period of one week. They were part of a research project investigating reproductive physiology. They were housed adjacent to sheep. Prior to death, two had scoured and were treated with "Scourban". Gross lesions included haemorrhagic gastroenteritis, corneal opacity, enlarged lymph nodes and haemal lymph nodes, multiple pale myocardial nodules, and acute fibrinous pneumonia secondary to aspiration of drench.

Bacterial culture from several sites was unrewarding. Histologically, a severe vasculitis, predominantly arteritis was present, with marked perivascular lymphoid cuffing throughout many organs. Lymphoid tissue was hyperplastic with cells being somewhat blastic and atypical in areas, a diagnosis of MCF was made. To our knowledge this is the first instance of MCF in Rusa deer in WA.

Salt poisoning in pigs (C Huxtable, R Buddle)

40 deaths (3%) occurred over a weekend in 9/10 week-old grower pigs which were "Wet fed".

Some were observed to show "incoordination" prior to death, and some were found dead. Five animals were necropsied and two were found to have *Actinobacillus pleuropneumoniae* pneumonia, which was known to be a problem in the herd. Two other pigs had no gross lesions and no bacteria were cultured from the CSF. Histologically there was extensive acute laminar cortical neuronal necrosis with capillary endothelial activation, and mild mononuclear perivascular cuffing. Eosinophils were not conspicuous. Serum sodium concentrations were 188 and 160mm, and CSF 203 and 167mm respectively.

A diagnosis of salt poisoning was made on this basis, noting that both the clinical and pathologic features were atypical