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

Print Post Approved Publication No. PP544059/00003

EDITOR: Jim Taylor

Number 38 NOVEMBER, 1993

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DEADLINE FOR NEXT VET. PATH REPORT IS FEBRUARY 28, 1994

SECRETARIAT

PO Box 114 Walkerville SA 5081

Phone: 08 3446337 (Pat Bosence) Fax: 08 3449227

ASVP EXECUTIVE 1991-1993

President John Gibson		Toowoomba Vet. Lab, PO Box 102 Toowoomba Q.4350	076314352
Secretary Jim Taylor		Toowoomba Vet. Lab, PO Box 102 Toowoomba Q.4350	076314365
Treasurer Russell Graydon		Toowoomba Vet. Lab, PO Box 102 Toowoomba Q.4350	076314410
Committee Members			
	Ross McKenzie	Yeerongpilly Vet. Lab, 665 Fairfield Road, Yeerongpilly Q 4068	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 3652565

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 Rd, Yeerongpilly, Q.4065	073629555
Victoria	Deb Seward PVIH, PO Box 406 Hamilton 3300		055 730700
South Australia	Ruth Reuter, VPS, PO Box 96, Plympton, SA. 5038		08 3623544
New South Wales	Paul Gill, RVL Wollongbar 2480		066 240298
Western Australia	David Forshaw, Regional Office, WA Dept Ag, Albany 6330		098 420536
Northern Territory	Anton Janmaat, PO Box 79, Berrimah 0828		089 892240
Tasmania	Barry Munday, Uni Tasmania, PO Box 1214 Launceston 7250		003 243232

EDITORIAL

Changes continue to impact on society members. The latest is the contracting of Government veterinary laboratory services in Victoria and Tasmania. The Executive has some concerns about this development, not solely for the continued employment of members, but also for Animal Health Services and Government's commitments to core activities of state laboratories. Hopefully these concerns will be raised nationally in the next edition of 'The Veterinarian'.

Next years AGM and conference is fast approaching due to the earlier date next year. Preliminary information and call for member presentation follow in this edition. I would also ask members to consider agenda items for the AGM. Further details will follow at a later date.

Les Sims has taken a position in Hong Kong and John Mackie recently returned from the United States, has offered his services as Victorian Public Officer for the Society, which the executive has accepted.

Members working as private veterinary pathologists, your attention is drawn to the letter from Ray Webb on behalf of SCAHLS regarding a representative of private pathologists on the subcommittee. Would any interested members please contact me or John Gibson.

I look forward to receiving your papers for the proceedings of the 1994 conference and hopefully catching up with you all in Canberra.

Jim Taylor
EDITOR

S E A S O N S G R E E T I N G S

FROM YOUR EXECUTIVE

REPORT OF THE NATIONAL PATHOLOGY REGISTRIES

Rod Reece has been the registrar of the two pathology registries for just over one year. The Comparative Animal Pathology Registry (**CAPR**) is housed at and funded by Taronga Zoo; whereas the National Registry of Domestic Animal Pathology (**NRDAP**) is housed at Elizabeth Macarthur Agricultural Institute, funded by contributions from State Departments of Agriculture, CSIRO and DPIE, and managed by a committee from within ASVP.

NRDAP TRAINING COURSES

During the last 6 months, histopathology training courses sponsored by NRDAP were held in each of the states and more than 50 veterinarians attended. The general format was that on Day 1 they studied the histopathology of avian species, mainly concentrating on the respiratory system and musculoskeletal disorders of poultry. I was able to negotiate a limited re-issue of the proceedings of the 1987 Australian Veterinary Poultry Association training course in avian histopathology and copies have been lodged with most veterinary pathology laboratories in Australia. Day 2 was spent reviewing new and emerging diseases, and the pathology of the respiratory system (mainly in domestic mammals). Multiheaded microscopes were used and this enabled attendees to observe the details of many sections which they would not otherwise have been able to examine. Attendees prepared sections for distribution and presentation, and gave reviews of a number of conditions.

In an attempt to defray the costs of producing material for distribution, a small fee was charged. A greater fee (or the provision of facilities etc) was introduced for non-sponsors of the registry. University facilities were utilised and attendees included university staff and post-graduate students, several specialist practitioners, practicing private veterinary pathologists, and others.

HELP!

The NRDAP has only a small collection of transparencies (although there is limited access to Bill Hartley's own collection and the CAPR collection at Taronga Zoo, and I have my own extensive collection of transparencies of avian conditions) and we are attempting to put together a more comprehensive set. There is a copy of all the transparencies used in Les Simm's forthcoming book on Pig Pathology. I am attempting to catalogue and collate all these.

Initially, NRDAP has some funds specifically for **SHEEP PATHOLOGY**. So please help by sending transparencies for duplication, or sending copies of transparencies depicting clinical, gross and/or histopathology of normal, common and unusual conditions. It would be useful to have examples of many of the poisonous plants and some of the basic husbandry practices and breeds as well. It is cheaper and easier to take two shots, and to send one to the registry anyway!

NRDAP HIGHLIGHTS. The NRDAP now contains an extensive range of sections depicting most conditions commonly encountered in Australian domestic animals. From time-to-time you may receive a note requesting sections of conditions mentioned in newsletters, or described in published articles. To-date the response to such requests has been excellent, but don't wait for me to write - send sections and a copy of the paperwork of anything interesting or typical! Also transparencies, especially of clinical signs and gross pathology would be welcome. I have been able to obtain sections from many conditions described in international journals.

Please send me copies of **NEWSLETTERS** which are sent out to your clients. This will enable me to be familiar with the type of material you handle.

CAPR. In case you were wondering, Bill Hartley has a part-time position at Taronga Zoo as a consultant pathologist: he has not retired!!

We are still after sections of normal tissues, and pathology of Australian native species and exotic animals. Please co-operate by sending sections. If you encounter anything unusual or interesting please hold the wet tissues! Bill is still making forays into many labs examining and collecting sections for the registry. It is sad to note that sections from native animals are being examined by non-veterinarians (i.e. medical pathologists) at a time when employment opportunities for veterinary pathologists are decreasing.

R.L.Reece, Registrar NRDAP, EMAI PMB1 Camden NSW 2570
Phone 046 293 333, Fax 046 293 400

SLIDE OF THE MONTH

Please note that the ASVP Slide of the Month is now being co-ordinated by Rod Reece, Registrar of the National Registry of Domestic Animal Pathology. Please direct any enquiries to:

R.L.REECE, Elizabeth Macarthur Agricultural Institute, PMB 8, Camden, NSW 2570.
Phone 046 293 333 on Thursdays or Fridays; Fax 046 293 400.

Peter Phillips (formerly of IMVS Adelaide) has had to stand down as Co-coordinator of the Slide of the Month, and we extend to him our appreciation for his efforts in making this a great success, and we wish him well in the future.

VICTORIA

RVL BAIRNSDALE

MEGAKARYOCYTE TUMOUR IN A DOG - Peter Mitchell

Blood samples were received from an 8-year-old greyhound with a history of bleeding from the gums over the past few days, anaemia and finally collapse. The dog had low numbers of red cells (Hb 48 g/1, PCV 0.16 1/1, RBC 2.2 10^{12}/1), very high total white cell count (150 10^{9}/1) and very high platelet count (2800 10^{9}/l). Blood smear contained numerous large platelets and blast cells. There was no evidence of red cell regeneration. We suspected a bone marrow tumour involving megakaryocytes. The dog died a few days later, and we were able to examine bone marrow from the femur. The bone marrow contained large numbers of megakaryocytes with anisokaryosis and unusual nuclear lobulation (even for megakaryocytes) and many large abnormal mitoses. There were some foci of granulopoiesis but no evidence of erythropoiesis. Megakaryocytic leukaemia was diagnosed.

SAND EATING IN OSTRICHES - Kit Button

Two ostrich chicks 5 months old died after a very short illness. Signs included a reluctance to rise, recumbency and pre-terminal dribbling fluid from the beak.

Pathology was similar in both birds. The proventriculus was grossly impacted by fine sand and coarse vegetable material. Proventricular content to body weight ratios was 8.6 and 8.3% (ostriches which die suddenly of trauma have had ratios of less than 5 and usually less than 4%). The gizzards contained large amounts of sand and few pebbles; pebble to body weight ratio was < 0.1% in one bird (ca 1% is usual in ostriches dying of unrelated conditions or trauma). Large amounts of sand were present in the **SI, LI** and faeces - for example we measured 26.9% sand (dry weight to wet weight) in the faeces of one of these chicks.

Both chicks had torsions of the mid and distal SI. The affected intestine had ruptured in one resulting in a diffuse peritonitis.

It seems that sand eating predisposes to intestinal torsion. The likely cause for sand eating in this case was a deficiency of grinding material. Cohorts of the dead birds avidly consumed pebbles provided to them when the problem was diagnosed. The owner was advised to provide pebbles sparingly at first to limit the chances of impaction on pebbles. Chicks given constant access to grinding material do not overindulge, but there is a danger that deprived birds would. Other causes of sand eating are not known but stress, boredom and overcrowding may be involved.

AAHL

SOME OBSERVATIONS ON THE CNS OF AGED SHEEP AND GOATS - Peter Hooper

One of the duties of the veterinary pathologist at AAHL is to examine numerous brains and spinal cords of sheep and goats from the quarantine stations to exclude scrapie.

Tissues are formalin-fixed half-brains and representative pieces of cervical, thoracic, and lumbar spinal cord from animals dying on the quarantine stations as well as aged sentinel, surrogate maternal and first generation (Fl) animals. It has been a good opportunity to see so many spinal cords as these structures are rarely collected in these sorts of numbers.

In a group of the latter, mature animals without apparent neurological signs, it was interesting to see the amount of neuropathology as in the following table.

Condition	Sheep	Goats
Total number in group	45	21
Demyelination in spinal cord	21	1
Occasional spheroids	2	2
Spheroids in lateral medulla	2	0
Perivascular cuffs	10	1
Vacuoles	13	4
“Negri bodies” in hippocampus	2	0
Sarcocystis (no reaction)	1	0
“Renaut body” in nerve root	1	0

Demyelination: This was probably the most remarkable finding of them all. Nearly half the "normal sheep" were affected some with quite substantial neuropathology. The lesions were invariably in the spinal white matter and were not mere vacuoles but were often accompanied by "Gitter cells". The large prevalence did not occur in the goats.

Spheroids: I have categorised these into those in the dorsolateral medulla, specifically in the cuneate and gracilis nuclei where spheroids or large degenerate eosinophilic neurones are common in "normal sheep", and those seen elsewhere. I doubt that these findings are of any significance. The number in the medulla was probably an underestimate as the tissue cut in that area was directed to a more distal nucleus.

Perivascular Cuffs: It would be reasonable to say the same about perivascular cuffs of lymphocytes i.e. of doubtful significance as these are extremely common in "normal" Australian sheep. It would be interesting to find out why this is so.

Vacuoles: These were of exaggerated importance in the context of excluding scrapie. Some patterns are easy to interpret, e.g. those of hepatic encephalopathy, but others are hard to understand such as the large vacuoles common in white matter such as in the optic tract, and/or adjacent to the corpus callosum, and/or even along the corticospinal tract.

"**Negri Bodies**" in the Hippocampus: These eosinophilic, mostly ovoid, cytoplasmic or extracellular, bodies occurred in the pyramidal cells of the hippocampus of 2 sheep. While there was nothing really to suggest rabies, e.g. inflammation, we still conducted an immunoperoxidase test with negative results. Both sheep came for the same property in Australia and may have been related genetically.

Sarcocystis: An occasional finding - as in muscles, mostly without inflammatory reaction.

Renaut Body: These strange entanglements are reasonably common in peripheral nerves, especially in horses. There may have been more in the subject sheep and goats as only few nerve roots were visible on the spinal cords.

Some other Neuropathology in Quarantine Stations

The previous section are only those findings from only one quarantine station at the completion or final few months of its scrapie-freedom assurance programme. There have been a large number of conditions whereby brains have been submitted as a routine for scrapie-exclusion from sheep and goats of all ages. One remarkable sequence from 8 goats, submitted not for diagnosis but for scrapie exclusion, had 7 with significant neuropathology viz. 3 with F.S.E. (or possibly rye grass staggers), 3 with bacterial meningoencephalitis, 1 with polioencephalomalacia, 1 with probable enzootic ataxia (copper deficiency), and only 1 **N.V.L**.

RVL HAMILTON

PHOMOPSIN TOXICITY FOLLOWING FEEDING WITH LUPIN GRAIN - Deb Seward

500 10-12 month old lambs grazing oat stubble were brought into a three acre feedlot and fed a ration of 90% triticale and oats with 10% lupin grain for two weeks. A new batch of lupin grain was then purchased and the feed changed to 75% triticale and oats and 25% lupin grain.

After two weeks of feeding on the second ration the lambs were observed to be losing condition. Deaths commenced after three weeks with up to 20 lambs a day being lost. In total 130 sheep were lost. During this period some of lambs were sent to a local abattoir for slaughter. The carcasses were observed to be severely jaundiced and fixed liver and other tissues were submitted to the Hamilton laboratory by the Senior Meat Inspector.

Examination of the liver sections revealed changes typical of Phomopsin toxicity. A sample of the lupin grain was obtained, noted to be markedly discoloured and sent for Phomopsin assay. A result of 5400 ppm Phomopsin A was obtained.

At the Hamilton laboratory Phomopsin toxicity is frequently seen in sheep grazing lupin stubbles containing in excess of 1000 ppm Phomopsin A. There is a perception amongst farmers that Phomopsin toxicity is not associated with feeding lupin grain, presumably as only a small volume per day is consumed. Following a favourable season for toxin production, as was the case in Victoria last summer, very high levels of Phomopsin may be reached in lupin grain samples as this case illustrates. Lupin grain should be carefully examined and suspect batches tested for Phomopsin content prior to feeding.

CYANIDE TOXICITY IN CALVES - Deb Seward

Six of 30 four to eight week old calves were found dead over a period of 10 days. The owner had been visiting the property every third day and had not noticed any of the calves or cows to be sick. There was no known source of arsenic but access to sugar gums was possible.

The field post mortem performed by the veterinary practitioner on one of the carcasses revealed little obvious post mortem degeneration and a swollen red abomasum. The small intestines were stated to contain a reddish liquid whilst the large intestinal contents were described as normal. The gall bladder bile was found to be thick and brown and the urinary bladder distended with clear urine.

Heart blood, rumen contents and fresh liver and skeletal muscle samples were submitted to the laboratory for testing for arsenic and cyanide. The blood had been collected in a plain tube and was observed to be bright red and only a small clot had formed.

The liver and rumen contents were negative for arsenic. A strong positive reaction for cyanide was obtained from the skeletal muscle sample.

A diagnosis of cyanide toxicity was made and the paddock in which the calves had been grazing was then searched by the referring veterinarian. A sugar gum tree which had fallen during recent storms and noted to be stripped of its leaves was found and removed. No further deaths were reported.

Sugar gums *Eucalyptus cladocalyx* are planted quite widely in the western district as ornamentals and as wind breaks. Animals are more prone to investigate fallen branches when pasture roughage is low.

Cyanide poisoning in association with ingestion of sugar gum leaves has previously been reported in goats. Webber et al AVJ 1985 **62: 28**

NEW SOUTH WALES

EMAI

CHRONIC DERMATITIS IN MICE, PRESUMABLY ASSOCIATED WITH TRAUMA FROM CAGE MATES - GL Reddacliff

This laboratory received a number of submissions of mice from a local research laboratory, with an ill-defined history of alopecia and focal, chronic skin ulceration. The mice had apparently been "examined" elsewhere, with some suggestion of a bacterial involvement, and were subsequently submitted to Menangle RVL, with a request for bacterial culture. (MN93/5600 & 5977)

Gross examination: Skin lesions varied from mild, diffuse alopecia, often with considerable scurfiness, of lateral and dorsal body surfaces; to severe alopecia, extending to the ventral surface, with marked skin thickening. All mice had focal lesions, many of which appeared to be healing wounds. These were mainly on dorsal and lateral body surfaces, ears and sometimes eyelids. Some larger, chronic ulcerative lesions were present on several mice with a crusty surface scab and slight purulent exudate. Whiskers were missing on some mice.

KOH preparations from deep and superficial skin scrapings were negative for mites and dermatophytes, and fungal cultures were negative. Bacteriologic examination yielded a mixed growth of *Staphylococcus aureus* and S. *epidemidis* from 2 of 6 mice.

Histologically, the skin lesions were characterised by marked thickening of the epidermis (mainly of the spinous and granular layers), fewer than expected hair follicles, poorly formed hairs containing clumped pigment in many follicles, focal hyperkeratosis, plugging of hair follicles by lamellated keratin debris, dermal fibrosis and multiple deep granulomas, centred on keratin debris of hair fragments. There was also a diffuse increase in melanin pigment in the dermis. In addition there were occasional focal areas of full thickness ulceration, with a crust of fibrinopurulent material sometimes containing bacterial colonies, and associated chronic active inflammation in the underlying dermis.

These changes are consistent with chronic hair chewing/pulling as described by Thornburg et al (1973), Lab. Animal Science **23**, 843-850. The bacterial infections are most likely secondary to traumatic lesions. It has been suggested that hair chewing and whisker eating is a multifactorial genetic behavioural trait, more common in black mice. This colony comprised many mixed mouse strains but the problem was seen most often in cages containing black mice. Moderately affected mice have now been housed individually to observe the expected recovery if, indeed, hair chewing by other mice is the underlying aetiology. Relative overcrowding of adult mice was a likely contributing factor.

MULTIPLE MYOCARDIAL ABSCESSES & VENTRICULAR SEPTAL DEFECT IN A CALF - GL Reddacliff

A 3 month old Friesian heifer died suddenly, with no observed previous illness. It was necropsied in the field, and multiple myocardial abscesses were reported. In addition there were abscesses in the spleen and lungs. Only the heart was submitted to the laboratory. (MN93/5454).

The heart was markedly enlarged, (about twice the expected dimensions) and almost spherical in shape, the enlargement due mainly to right ventricular hypertrophy. A ventricular septal defect 40mm in diameter was present, immediately below the level of the aortic valve. There was some endocardial thickening of the left ventricle. All valves were normal. Multiple, chronic, caseous centred, fibrous encapsulated abscesses from 5 to 20mm in diameter were present in the walls of all cardiac chambers, including many in the interventricular septum, but none appeared to encroach significantly on the cardiac lumen.

Histologically some abscesses had a liquefactive core, surrounded by a zone of caseous material containing scattered necrotic muscle fibres, granulocytes and debris, which merged into a granulomatous capsule with abundant macrophages and proliferative fibrous tissue. Others were centred on a large area of necrotic heart muscle, surrounded by a zone of granulocytes, often with calcification, and then a granulomatous capsule. The encapsulating fibrous response was severe, and extended considerable distances from the abscess cores, causing marked disruption of the myocardial architecture.

Gram stains revealed large numbers of pale-staining, filamentous, gram-negative bacilli, most prominent in the necrotic myocardium adjacent to the layer of granulocytes. Anaerobic culture on sheep blood agar yielded a sparse pure growth of a *Fusobacterium* sp., which was not further identified. There was no growth on aerobic culture.

It seems that death in this animal was due to heart failure, associated both with a severe septal defect and the effect of multiple chronic abscesses. In places the fibrous abscess capsules had surrounded and disorganised the Purkinje fibres, so the possibility of conduction abnormalities existed. The sheer number of myocardial abscesses was spectacular, and one can speculate whether the haemodynamic effects of the septal defect predisposed the myocardium to lodgement and growth of emboli containing *Fusobacterium*. One has to assume that in this case the farmer was not particularly observant!

PRESUMED VIRAL HEPATITIS IN OUTBRED SWISS NUDE MICE - G.L. Reddacliff

Eighteen of 20 laboratory maintained 6 month old, female, outbred Swiss nude mice died over a period of several days. The 2 surviving mice were euthanased 8 days after the first deaths. (MN93/4220, 4251 & 4371)

Grossly, the mice that died had swollen, pale and mottled livers. There was splenomegaly, with miliary pale foci, and miliary haemorrhagic foci in the lungs. In the 2 euthanased mice, livers were pale and slightly mottled, but not enlarged, and there was mild splenomegaly.

Liver, lung and spleen from 4 dead mice were cultured aerobically on sheep blood and MacConkey agar. There were no consistent or significant isolates. One mouse yielded a predominant growth of an untypable *Streptococcus* sp, and another a *Lactobacillus* sp.

Histological lesions in the dead mice were as follows. Livers had severe, multifocal, tending to midzonal, coalescing areas of acute, coagulative necrosis, with associated haemorrhage and granulocytic infiltration. Surviving hepatocytes often had intranuclear inclusions. These varied from discrete, eosinophilic, round to oval inclusions, surrounded by a clear halo (typical of Herpes viruses) to larger, irregular, often basophilic inclusions which took up the whole nucleus and distorted the cell shape (similar in appearance to the inclusions of Fowl Adenovirus Infection). There were also moderate numbers of multinucleate syncytial cells, which along with other swollen hepatocytes often contained multiple, eosinophilic, variably sized, round to oval, intracytoplasmic "inclusions" (possibly phagocytosed material). The numbers of inclusions and syncytial cells were greater in mice dying at the end of the outbreak. Spleens had multifocal, acute necrosis, plus scattered individual cell necrosis, and abundant inclusions (both intranuclear and intracytoplasmic) as described for the liver. Similar changes were seen in mesenteric lymph nodes and adrenal cortex. Lungs had generalised, moderate to severe, granulocytic infiltration of alveolar walls, and a mild increase in alveolar macrophages. There was multifocal, acute, probably agonal, intra-alveolar haemorrhage, plus focal interstitial aggregates of large, often multinucleate, mononuclear cells with inclusions as above. In some gut sections there were small focal, necrotic areas in the lamina propria, and occasional cells with inclusions as above. There was also widespread individual cell necrosis in the epithelium and occasional crypt abscesses. There were no significant lesions in brain, kidney or heart.

In the surviving mice, there were occasional tiny foci of necrosis, with associated non-suppurative inflammation in the livers, but no inclusions. Lungs had occasional interstitial foci of non-suppurative inflammation, while spleens and adrenal cortex had mild multifocal necrosis, with amphophilic, round to oval, intranuclear inclusions in adjacent cells.

Infections with Mouse Virus Hepatitis (a coronavirus) are well documented in Swiss nude mice. (These mice are athymic and immunocompromised). The virus is almost ubiquitous and often carried asymptomatically by normal mice (both laboratory reared and wild) so strict isolation procedures are necessary to maintain nude mice in the laboratory. In this case, wild mice had gained entry to the "rodent proof" animal house, this being discovered just the same day as the first deaths.

Many of the changes reported here are typical of Mouse Virus Hepatitis infection in nude mice - the widespread necrotizing lesions, syncytia formation, and the eosinophilic intracytoplasmic inclusions. Intranuclear inclusions are, however, not usually a feature, and their occurrence is more suggestive of cytomegalovirus infection. Unfortunately no virologic examination was possible, but electron microscopy will be performed on tissues salvaged from the paraffin blocks.

SCONE DISTRICT VETERINARY LABORATORY

EQUINE INFECTIOUS ANAEMIA IN A THOROUGHBRED FOAL - Angela Begg

A 4 month old thoroughbred foal had a 2 to 3 week history of recurrent pyrexia, deteriorating anaemia and neutropaenia with terminal lymphoma and thrombocytopaenia. A Coombs test was negative. Prior to this, the foal had an episode of diarrhoea at 2 weeks of age and had received a transfusion of blood from its dam because of moderately severe anaemia. In the intervening period the foal was apparently normal although did not grow as well as expected. At postmortem, mild jaundice, splenomegaly, generalised tissue pallor, lymphadenomegaly and a prominent hepatic lobular pattern were found. Histological examination revealed the presence of dense infiltrates of lymphocytes and variable numbers of immature erythroid cells in alveolar walls, liver sinusoids, renal interstitium and glomeruli, splenic pulp and lymph nodes together with prominent haemosiderin accumulation and recent erythrophagocytosis in the lung, liver, spleen and lymph nodes. Equine Infectious Anaemia was confirmed serologically by a strongly positive GDPT. Although E1A infection is prevalent in some horse populations in North Queensland and the Northern Territory, cases of EIA are very uncommon in NSW and this case had serious implications for the export of local horses to certain countries.

EIA infection causes one of 3 clinical syndromes in horses: acute disease characterised by severe pyrexia and anaemia, chronic disease or an inapparent carrier state. Transmission between horses occurs via Tabanid flies (horse flies) or transplacentally to offspring. Iatrogenic transmission via contaminated blood products, contaminated needles and surgical instruments can also occur and colostral transmission is also postulated but not proven. The mare was considered to be the source for this foal but, despite repeated tests, the mare has remained serologically antibody and antigen negative.

RVL ARMIDALE

REPEAT OUTBREAK - EQUID HERPESVIRUS ABORTIONS - Stephen Love and Steven Hum

In 1989 we reported an outbreak of abortions due to EHV1 on a New England thoroughbred stud (Vet Path Report, 1989). During that epidemic, approximately a quarter of the 90 mares at risk lost their foals. There appears to be a recurrence of the disease on the same stud this spring. Two groups of mares have foaled already, apparently normally. A third group of nine mares, 3 to 4 weeks off foaling, began aborting recently, with 6 of the 9 expelling freshly dead foetuses. Gross and histopathological findings in some of the foals at least are consistent with herpesvirus abortion. Virological confirmation is awaited.

OBSTRUCTIVE UROLITHIASIS IN FEEDLOT CATTLE - B A Vanselow

A devastating outbreak of obstructive urolithiasis occurred in a 13,000 head feedlot; predominantly affecting "finisher" cattle. Over a 7 week period approximately 1000 animals were diagnosed with urethral obstruction and immediately sent for salvage slaughter. As a result of early detection, very few animals died from the condition and few carcasses were condemned. Significant financial loss occurred when animals sent for salvage slaughter did not reach the required weight for export to Japan.

The stones were composed predominantly of magnesium ammonium phosphate but calcium oxalate and calcium carbonate were also identified. The urinary pH for 24 affected animals was alkaline (mean pH 8.4). A treatment regime of dietary ammonium chloride (1%), additional sodium chloride (1%), and a reduction in dietary sodium bicarbonate (to 0.25%) resulted in a drop in urinary pH after one week (mean pH 6.4) and after two weeks there was a virtual cessation of clinical cases.

Predisposing factors which may have initiated the outbreak included sorghum as the sole grain fed, alkalizing agents in the diet (sodium bicarbonate and calcium carbonate), and alkaline drinking water high in calcium carbonate and magnesium salts.

Monitoring of urinary pH has now become a routine practice at this feedlot when steers are brought through the race for weighing. Sophisticated equipment is required; a plastic container strapped to the end of a broom handle for collecting the specimen and a hand-held pH meter (from Dick Smith's) for the measurement.

REGIONAL VETERINARY LABORATORY WAGGA WAGGA - John Glastonbury

SHEEP

Following the mild wet summer of 1992/1993, nematodiasis caused major problems over winter and early spring. Submissions from scouring weaners became very monotonous. Severe mortality rates of up to 20.0% occurred in individual flocks.

A 5-year-old Merino ram from Tumbarumba was suspected of having **bluetongue**. It was pyrexic and displayed congestion of the lips and coronary bands. Severe subacute segmental primary contact irritant dermatitis was found in the affected areas histologically. To date extensive virological examinations at AAHL have been negative. The sheep were grazing a pasture containing Medicago spp.

Lupinosis was responsible for a mortality rate of 43.4% amongst 1,450 Merino wethers. The "Gungurra" variety of lupins was harvested in the paddock during the summer of 1991/1992. The next year it was sown to wheat and subsequently undersown with lucerne and subterranean clover. At the time of the deaths there was good 10 to 15 cm lucerne foliage available and lupin plants were estimated at <200 per acre.

Septicaemia colibacillosis was diagnosed in a 2-week-oid Texel lamb. We were unable to serotype the isolate of *Escherichia coli* and histological findings included fibrinopurulent choroido-ependymo, leptomeningo-encephalitis.

Other diagnoses of interest included **hepatogenous photosensitisation** in Merino wethers which had hepatic histological changes suggestive of facial eczema; **rickets** in Dorset x Merino weaners grazing lush oats; **end stage renal failure** due to the ingestion of lesser loosestrife - *Lythrium hyssopifolia*; **nutritional degenerative myocardiopathy** in 4-month-old Merino rams.

CATTLE

Two **Salmonella** isolates we have recovered recently from dairy calves on the same farm have shown *in vitro* resistance to all antimicrobials tested. As they are presently being serotyped we don't know whether they are Salmonella Typhimurium as in Victoria. Severe acute necrotic enteritis associated with salmonellosis in another calf resulted in intestinal rupture and serofibrinous peritonitis.

A 13-month-old Limousin heifer proved a treasure-trove of diagnoses - 170,000 **Trichostrongylus axei** and 36,000 **Ostertagia** sp in the abomasum, acute segmental ulcerative oesophagitis, necrotic enteritis and proctitis, **pestiviral** antigen capture ELISA positive and **Yersinia pseudotuberculosis** as well as **Campylobacter jejuni** recovered from intestinal contents.

Degenerative myocardiopathy associated with possible ingestion of excessive **monensin**, a **granuloma cell tumour** in an 18-month-old Angus heifer and 2 outbreaks of **pyrrolizidine alkaloidosis** in 5 and 8-month-old Herefords were additional highlights.

PIGS

Neonatal pigs had secure **inclusion body rhinitis** associated with acute multifocal necrotic encephalitis. **Multifocal locally extensive encephalomalacia** and acute cerebrospinal angiopathy accounted for a mortality rate of 27.8% amongst 90 8-week-old weaners. Samples for bacteriology were inadequate. Vast numbers of **Clostridium novyi** were demonstrated by fluorescent antibody staining of hepatic smears taken from a 14-week-old gilt which had died suddenly. A 5 month old Duroc x Large White that also died suddenly was found to have lesions consistent with **mulberry heart disease**, mostly in the right atrium, and disseminated intravascular coagulopathy.

DEER

Skeins of **Dictylocaulus filaria** were extracted from the trachea and major bronchi of a 9-month-old Red Deer. Acute haemorrhagic enteritis and typhlitis as well as mild acute multifocal necrotic hepatitis were associated with the recovery of heavy growths of **Campylobacter jejuni** from 4 small and large intestinal sites of a Fallow Deer.

BIRDS

Strong suspicions of an **exotic disease** were roused by the sudden deaths of all 180 pigeons in a local loft. Histological lesions included severe visceral congestion and granulomatous pneumonic aspergillosis. Finches in a neighbouring cage also succumbed but they did not have pneumonia. Extensive virological and toxicological studies were negative.

A 6-month-old "free ranging" pullet caused considerable excitement. It had necrotising myositis of the proventriculus associated with what appeared to the **Sarcocystis** sp. Protozoal forms also were detected in the Bursa of Fabricius, heart and peribronchial muscles.

"**Bumble foot**" caused by *Staphylococcus aureus* accounted for 12.6% of 4000 8-week-old quail. Associated lesions were pyogranulomatous nodular dermatitis, tendovaginits and suppurative synovitis.

Enterotoxic colibacillosis was suspected in 4-week-old broiler chickens. They had acute catarrhal typhitis and many organisms with a morphology consistent with *E coli* adherent to the microvillous brush border of the caeca. Heavy growths of *E coli*, not typeable with our antisera, were obtained from small and large intestinal contents.

The ultimate diagnosis was **hyperplastic goitre** in a Gouldian Finch.

RVL WOLLONGBAR

PYRROLIZIDINE ALKALOIDOSIS IN CATTLE ASSOCIATED WITH *SENECIO LAUTUS* –
Roger Cook, Paul Gill

This year we have diagnosed more cases than usual of pyrrolizidine alkaloidosis in grazing cattle on the mid north coast of NSW (Taree-Kempsey). Most cases had swollen livers with a marked zonular mottling. Many of these cases had a distinctive myxomatous periacinar fibrosis (so-called "veno-occlusive" disease), with or without associated megalocytosis and karyomegaly of hepatocytes. Dr Alan Seawright tested a number of these cases and confirmed the presence of S-bound pyrroles. The myxomatous, periacinar fibrosis follows periacinar oedema with or without associated periacinar hepatic necrosis and has been associated with experimental and natural pyrrolizidine alkaloidosis caused by plants with high pyrrolizidine alkaloid concentrations (e.g. *Senecio jacobea*). Fireweed (*Senecio lautus*) is the most likely source of pyrrolizidine alkaloids in these cases, although fireweed is usually unpalatable to cattle, and is said to contain relatively low concentrations of the hepatoxic pyrrolizidine alkaloid, senecionine. However, natural cases of pyrrolizidine alkaloidosis due to *Senecio lautus* have been reported in NSW (Walker and Kirkland 1981) and south-east Queensland (Seawright et al 1991).

R**EFERENCES**

Seawright AA, Kelly WR, Hrdlicka J, McMahon P, Mattock JR and Jukes R (1991) Vet Rec 129: 198.

Thorpe E and Ford EJH (1968) J Comp Path 78: 195.

Walker KH and Kirkland PD (1981) Aust Vet J 57: 1

QUEENSLAND

DRUNK AND DISORDERLY COW - Ross McKenzie

Ethanol poisoning was diagnosed in an adult Friesian cow at Dayboro in early August 1993. The attending veterinarian reported ataxia in the animal. The cow had access to fermented fruit pulp containing pineapples and apples. (Cider!) An assay of its blood revealed an ethanol concentration of 0.23%. While 'normal values' are not available for cattle, it seems reasonable to interpret this result as abnormally large. Recovery was reported as uneventful. No, the veterinarian did not advise 'Take two aspirin and call me in the morning', nor was a feeding trial with appropriate controls organised! Standard texts on veterinary toxicology provide little information on ethanol poisoning of domestic animals, but cases have been recorded in cattle fed brewer's grain [Hibbs, C.M. et al. (1986) *Proceedings of the 14th World Congress on Diseases of Cattle* 2:733.]

POXVIRUS INFECTION IN AN OSTRICH

Poxvirus infection was diagnosed in a 6-month-old farmed ostrich (*Struthio camelus*) near Beaudesert in early November 1993. The bird had a small raised lesion, 6-7 mm diameter, on the medial side of the right leg, just above the tarsus. Typical poxvirus inclusions were seen histologically in the hyperplastic epidermis.

SUSPECTED NUTRITIONAL CARDIOMYOPATHY IN GOATS - Greg Stone and Laurie Dowling

Sixty of 400 goats died over a 3 month period beginning in early June 1993 on a property near Rosewood in south-eastern Queensland. Animals died acutely with no premonitory signs observed. Histopathological examination of specimens submitted by the attending veterinarian revealed multifocal to coalescing myocardial necrosis with extensive calcification. Analysis of blood sample from 24 of the remaining animals revealed serum Vitamin E levels averaging .08 mg/L (range .02-.17), almost 20 times less than our reference range of 1.5 mg/L. GSH-Px activities were normal. Pasture conditions at the time were very poor and goats were being fed horse pellets and poor quality hay.

DEPARTMENT OF BIOMEDICAL AND ATROPICAL VETERINARY SCIENCES, JAMES COOK UNIVERSITY, TOWNSVILLE - Philip Ladds

Some recent 'cases of interest' include:

DOGS

HISTIOCYTIC ULCERATIVE COLITIS IN A BOXER

This dog had a history of bowel haemorrhage and occasional diarrhoea since 6 months of age. It was euthanased at 18 months of age and necropsy revealed extensive ulceration. Macrophages with PAS-positive cytoplasm were prevalent in the deep lamina propria but extended into the sub-mucosa.

PRESUMED SEVERE LEPTOSPIROSIS IN A BULL TERRIER

This was the second of two dogs from the one Cairns suburb to be presented with identical signs (of hepatitis and renal disease) during one week in April. There was widespread fatty change of the liver with intra-cellular pigment (presumed bile) and some 'individualisation' of hepatocytes. Extensive necrosis of renal tubule epithelium was apparent and associated with attempted regeneration and much intra-luminal debris and pigment.

MYCOTIC NEPHRITIS IN A GERMAN SHEPHERD

This young (2 year old) dog was presented with urinary incontinence, polydypsia and polyuria and low specific gravity of urine. Neurological signs developed and the dog was euthanased. At necropsy the kidneys were enlarged and granulomatous and microscopically fungi (morphology consistent with *Aspergillus*) were prevalent. This case resembled those seen previously (e.g. Day et al *Australian Veterinary Journal* **63**: 55.

BABESIOSIS IN PUPS

Several further cases characterised clinically by listlessness, abdominal pain, vomiting, diarrhoea, haemoglobinuria and jaundice, have been seen. Experience here is that in histological material the intra-erythrocytic organisms are best found in capillaries in the renal medulla.

SYMMETRICAL LEUCODYSTROPHY IN A ROTTWEILER

This 2 ½ year old dog had a history of a proprioceptive defect of several months duration which was progressive leucoencephalomalacia was suspected. Microscopically there was symmetrical vacuolation of white matter of the brain stem and spinal cord associated with proliferation of microglia, astrocytic gliosis and scattered gitter cells. These findings supported the clinical diagnosis leucoencephalomalacia is considered to have autosomal recessive genetic transmission (Reference Chrisman, DL. *J Small Anim Pract* **33**: 500-504).

TRANSMISSIBLE VENEREAL TUMOUR

Pronounced lesions were observed on the penis of a dog examined as part of a dog/human health study in communities in northern Queensland. Histology was typical of TVT. Although seen infrequently, it is evident that this tumour is present in north Queensland as well as the Northern Territory (Reference Locke, KB et al. *Aust Vet* J **5**1:419).

CATTLE

RENAL FIBROSIS AND DYSPLASIA (PROBABLY BVD INFECTION) IN A CALF

A 6 week old calf was presented with abdominal pain and 'CNS spasms' of 23 minutes duration. These were characterised by nystagmus and a 'paddling' movement. Microscopically there was extensive cortical fibrosis of both kidneys with diffuse cystic degeneration of tubules which contained much proteinaceous fluid and some cellular debris.

PROBABLE SPERMATOGENIC ARREST IN A BULL

Clinical examination (on several) occasions of a 2 ½ year old bull imported from overseas revealed consistently poor quality semen with zero-low viability and marked abnormality of spermatozoa. Microscopic examination of testes collected at slaughter showed that although germinal epithelium was of normal height, and earlier germ cell types including primary spermatocytes were well represented, few spermatozoa were seen. No spermatozoa were present in tubules of the mediastinum testis. History, clinical and pathological findings of this case were consistent with the spermatogene arrest/'sticky' chromosomes condition although an earlier periorchitis was a further possibility.

OTHER SPECIES

LYMPHOSARCOMA IN A KOALA

A further case of lymphosarcoma was diagnosed in a female koala, approximately 7 years of age, which was found sick in the wild (on Magnetic Island). Excess fluid was in body cavities and haemorrhages were on serous surface. Microscopically, neoplastic lymphocytes infiltrated most organs (Reference Canfield, PL et al *J comp Palli* **97**: 171-178).

FOCAL NON-SUPPURATIVE (PRESUMED VIRAL) MYOCARDITIS IN A CRIMSON-WINGED PARROT

A parrot found sick and depressed in the wild was submitted. Microscopically there was a multifocal lymphocytic infiltrate of the myocardium and local degeneration and necrosis of epithelium of the oesophagus with intranuclear inclusions in some cells. Second opinion in this case by Neil Sullivan (Veterinary Pathology Services) was that these changes were consistent with circovirus infection.

CRYPTOSTEGIA GRANDIFLORA POISONING - Rob Pierce and Ross McKenzie

Rubber vine (*Cryptostegia grandiflora*) poisoning (cardiac glycoside poisoning) was diagnosed in 2 bulls in a herd in the Rockhampton area in early July 1993 after access to the plant was potentiated by drought conditions. One bull died. Necropsy findings were non-specific. The second bull recovered after therapy with activated charcoal, electrolyte replacement solution and atropine. Clinical signs were diarrhoea, bradycardia and heart block. Clinical chemistry revealed increased concentrations of creatinine, urea and glucose in serum and glucosuria. These abnormalities were eliminated after therapy.

CYLINDROSPERMOPSIS RACEBORSKII (CYANOBACTERIAL POISONING) – Rob Pierce and Ross McKenzie

Poisoning of a group of cattle in a feedlot west of Emerald in early July 1993 was ascribed to the presence of *Cylindrospermopsis raceborskii*, a cyanobacterium (blue-green algae) with known hepatotoxic properties, in the turkey-nest dam supplying the feedlot. Histological and clinical chemistry evidence of hepatic damage was obtained from the cattle after 6 died over a few days. C. *raceborskii*, at a concentration of about 500,000 cells/ml, was the only cyanobacterium detected in a sample of water examined. No pyrrolic metabolites were detected in a liver sample from a dead steer. The liver lesion in one steer from which material was obtained was characterised by hepatocyte foamy vacuolation and irregular size, biliary retention and biliary ductular hyperplasia.

C. *raceborskii* has been associated with hepatic disease of humans on one occasion in North Queensland, toxicity being demonstrated by mouse inoculation with the organism, but has not been implicated in disease of domestic animals previously to our knowledge. Recent work has characterised the toxin as an alkaloid, not a peptide as is the case with the more common hepatotoxic cyanobacterium *Microcystis aerurinosa*.

16.

NORTHERN TERRITORY

BERRIMAH VETERINARY LABORATORIES

LANTANA POISONING - David Pritchard

A mob of two-and-a-half year old Brahman heifers was trucked from near Charters Towers, Queensland. On arrival near Tennant Creek, NT, six were dead and a further eleven sick. Clinical signs were diarrhoea, excessive and rapid weight loss, pale and jaundiced mucosae, depression, and sternal recumbency. Another three of these died over the next day and a sick animal was shot for post mortem examination.

Aspartate aminotransferase, alkaline phosphatase and gamma glutamyltransferase were elevated with all other blood analytes (renal function tests, protein, CK, Ca, Mg and electrolytes) in reference range. Haemoglobin (6.2 g/dl) and haematocrit (.17) were slightly subnormal.

Necropsy findings were pale, icteric membranes and connective tissues swollen, ochre liver, and pale kidneys and skeletal muscles.

Histologically, there appeared to be generalised hepatocellular lipidosis, widespread megalocytosis with binucleated and multinucleated hepatocytes and sporadic abnormal appearing mitoses. Scattered focal aggregations of neutrophils are associated with degenerating, dying and dead hepatocytes. The kidneys had several focal areas of interstitial fibrosis associated with dilated tubules containing hyaline, granular and cellular casts.

On further investigation of the history of the mob it was discovered that the sub-group of 700 in which the illness and deaths occurred had been brought from a property near Rockhampton three weeks before shipping from Charters Towers and had been grazing a paddock heavily infested with *Lantana*. Presumably stress of travel with inanition and consequent fat loading of an already hypofunctional liver produced terminal hepatic failure in more severely affected animals.

EXTRACTS FROM RECENT MONTHLY REPORTS - Anton Janmaat

"Specimens from 17 cattle and one buffalo bull from Kakadu, were received for histological examination under the BTEC scheme. Two cows (one part of a destock and one representing a breakdown) and the old buffalo bull showed lesions consistent with tuberculosis.

Fourteen specimens were pyogranulomas. Of these, eleven were club-forming (with *Actinobacillus* sp. isolated from ten), two were consistent with *Rhodococcus equi* infection (with isolation of the bacterium in both cases) and one was fungal. One submission showed hyperplastic glandular change."

"Specimens from 11 cattle and five buffalo were received for histological examination under the BTEC scheme. Two cows from the VRD region, both representing "breakdowns", and four buffalo from a field test in Kakadu showed lesions consistent with tuberculosis.

Nine specimens were non-tuberculous granulomas (with five club-forming) and one consisted of normal salivary gland and tonsillar tissues."

Just to remind you that some of us are still at it. All but two of the club-forming pyogranulomas in August came from Alice Springs as submissions under the National Granuloma Submission Program.

ARBOVIRUS MONITORING IN THE N.T. IN 1993 - Lorna Melville

Weekly blood sampling for virus isolation was carried out at Coastal Plains Research Station from January to October.

Following good rainfall during the wet season a moderate level of bluetongue virus activity occurred. Sixty three isolates of bluetongue Type 1 were made between March and May, with forty one isolates of EHD Type 5 in January and February; Untyped isolates of EHD made during 1992 were identified by the Australian Animal Health Laboratory as being closely related to EHD-1, a serotype not previously known in Australia.

Another group of viruses new to Australia was isolated this year. Five isolates from the Bunyamwera Serogroup of Bunyaviruses were made in April from three cows, 1 buffalo and 1 bull. One cow, which was about 6 weeks pregnant at the time of infection, subsequently aborted the calf at about 3 month's gestation.

Serology also indicated that bluetongue Type 20 was active again this year. This serotype, which caused the original recognition of bluetongue in Australia, re-appeared in 1992 after an absence of about 15 years.

C A L L F O R P A P E R S

ASVP ANNUAL CONFERENCE 1994
5 - 6 MARCH
CANBERRA

THEME: Neuropathology

KEYNOTE SPEAKER: Dr. Rick LeCouteur
BVSc, PhD, Diplomate ACVIM (Neurology)

Neurology/Neurosurgery Referral Service
Castle Hill Veterinary Hospital

MEMBER PRESENTATIONS: Members are invited to submit presentations for the Sunday session of the conference. Member presentations need not be limited to the theme of the conference and presentations should be 15-20 minutes duration.

Interested members should provide a copy of their paper or abstract typed on A4 paper to the Secretary no later than January 21. No facsimile transmissions please.

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

Incorporated in Victoria.

C/- Toowoomba Veterinary Laboratory, PO Box 102, Toowoomba QLD 4350.
Phone: (076) 314 352 Facsimile: (076) 331 918

Honorary President: John Gibson
Honorary Secretary: Jim Taylor
Honorary Treasurer: Russell Graydon

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28 July 1993

Mr. Peter Fletcher
Civil Aviation Authority (Biological/Medical Transport)
Allan Woods Building
25 Constitution Avenue
CANBERRA ACT 2601

REGULATIONS GOVERNING AIR TRANSPORTATION OF DANGEROUS GOODS

I am writing to you on behalf of the executive of the Australian Society for Veterinary Pathology. Our membership comprises veterinary pathologists employed in laboratories, universities and research institutions throughout Australia. At our recent AGM, concerns were raised about the current regulations governing the air transportation of dangerous goods. It is apparent that the regulations will have a major economic impact on the veterinary and human health care professions with increasing costs for both airfreight and packaging. Like the CAA we are concerned about the safe transport of dangerous goods, however, we believe that costs can be contained within acceptable levels without compromising safety aspects. The regulations which most concern our members are Instruction 650 and 602. We offer the following comments for your consideration.

(1) The majority of samples handled by our members come under Regulation 650. The requirement that the external packaging have a minimal external dimension of 100mm ignores the fact that existing approved cylindrical labmailers are 80mm diameter. This 100mm requirement does not increase the safety of the packaging but does increase total freight costs. Estimates by our members indicate that freight costs will rise by as much as 18%.

(2) Our members also believe that the current restrictions on weight and volume for packing Instruction 650 will severely jeopardise the animal health programmes in Australia. Surveillance programmes and export testing protocols require that large numbers of serum samples from clinically normal animals be transported with minimum delay to interstate laboratories. Clearly in these cases weight and volume restrictions mean the cost and labour component of packaging become unreasonable. We believe that weight and volume restrictions should not be applied to such low risk goods provided they otherwise conform to IATA Instruction 650.

(3) While we basically support the 602 packaging instructions, the society believes that some rational risk assessment needs to be applied to the definition of infectious substances under these instructions. A classification system for veterinary pathogens identifying specifically those agents requiring 602 packaging could be developed. We understand that this has been implemented in Canada after successful negotiations between air transport authorities and veterinary and human health care professionals. Many veterinary pathogens do not infect humans and once in the courier system represent a negligible threat to animals. Much of the animal health research effort in this country is underpinned by the interchange of laboratory isolates. This

would be compromised if a classification system based on risk assessment is not implemented. We totally support the 602 packaging of those agents that are known to pose a real risk to our animal industries and the public (i.e. zoonotic and exotic disease agents etc.)

(4) Our society also seeks clarification on what constitutes "approved" packaging under Instruction 650. It has been brought to our attention that some sections of the dangerous goods packaging industry advocate that only they can supply "approved" packages, while Instruction 650 does not mention any necessary approval for packaging. Many of our members already have available packaging that conforms to Instruction 650.

We believe that through open communication and co-operation that dangerous goods regulations can be successfully implemented with no compromise in safety but at a cost acceptable to those industries involved.

Yours sincerely

John Gibson
HONORARY PRESIDENT
AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

Civil Aviation Authority
AUSTRALIA

GPO Box 347 Canberra ACT 2601
Australia
Telephone (06) 268 4111
Telex 6221
Fax (06) 268 5683

F93/0444

Dr John Gibson
Honorary President
Australian Society for Veterinary Pathology
C/- Toowoomba Veterinary Laboratory
PO Box 102
Toowoomba Qld 4350

Dear Mr. Gibson

REGULATIONS GOVERNING THE AIR TRANSPORTATION OF DANGEROUS GOODS

I refer to your letter dated 28 July 1993 on the above subject. Please note that the letter was not received in the Authority until 23 August 1993.

It is important to explain the position of the Authority in relation to the IATA requirements and their relation to the requirements of the ICAO (International Civil Aviation Organisation) Technical Instructions.

The Authority strongly recommends adherence to the sensible practice of consigning any cargo in good quality packaging for air transport. The IATA requirements relating to Packing Instruction 650 when consigning non-infectious Diagnostic Specimens and Biological Products are obviously designed to provide protection not only for these sensitive substances but also for those required to handle or carry the package. However, as these substances are not included in the dangerous goods list in the ICAO Technical Instructions, they are therefore not dangerous goods and are not covered by the existing legislation. The standards relating to Packing Instruction 650 may only be insisted upon by the IATA member airlines/freight forwarders etc.

It is agreed that the minimum dimension requirement of PI 650 is not related to aviation safety. The Authority has no objection to the use of 80mm minimum. dimension containers (or smaller) for these non-infectious substances. Also there is no legislative restriction on quantities of non-infectious Diagnostic Specimens or Biological Products which may be consigned or carried in a package. However, you should be aware that any consignments which do not comply with the IATA requirements may be rejected by the airline operators, especially the IATA members, and this is a matter that would need to be taken up with them.

In relation to Packing Instruction 602 for Infectious Substances, the standards prescribed in the ICAO Technical Instructions (and also included in the IATA Dangerous Goods Regulations) relating to classification, packaging and quantities must be adhered to in full. The only advice I can offer in relation to the possible easing of what you may consider to be a very restrictive definition of Infectious Substances is that the ICAO Dangerous Goods Panel will, in October this year, consider the introduction of the World Health Organisation's four risk groups for Infectious Substances. It would be speculation on my part to suggest what, if any, changes may result from the Panel's considerations.

It is understood that the Panel may also consider classifying Diagnostic Specimens and Biological Products as dangerous goods. Any changes to the legislation as a result of these considerations are unlikely to have any affect before 1st January 1995, the date of issue of the next Edition of the ICAO Technical Instructions.

There exist three methods by which packaging manufacturers may obtain approvals for their dangerous goods packaging. If the packaging manufacturer is approved (certified) by the National Association of Testing Authorities (NATA), he may test and approve his own product. Otherwise, he can have a NATA approved laboratory do it on his behalf.

Another method is for a non-NATA approved manufacturer to test the packaging himself, provided the test equipment is available, and have the tests witnessed by a Competent Authority.

It is suggested that if in doubt about the "approved" nature of any packaging offered or purchased, you should seek assurance from the supplier that the packaging approval process is in accordance with one of the above requirements.

Please do not hesitate to contact me again if any matter requires further clarification.

Yours sincerely

Peter Fletcher
for Manager
Airworthiness and Operations

September 1993

Head Office
ORANGE

NSW Agriculture

161 Kite Street,
Locked Bag 21
ORANGE NSW 2800

Telephone (063)91 3100

Dr John Gibson
President
Australian Society for Veterinary Pathology
Toowoomba Veterinary Laboratory
203 Tor Street
TOOWOOMBA QUEENSLAND 4350

Dear John

At the recent July meeting of the S.C.A.'s sub-committee on Animal Health Laboratory Standards, state representatives discussed the possible involvement of a representative of privately employed veterinary pathologists at future meetings. Although there are many matters discussed at S.C.A.H.L.S. at which the attendance of a private pathologist would not be appropriate, there are specific matters on which their input would be particularly beneficial for all concerned. Specific examples include the involvement of private laboratories in exotic disease cases - both from the initial diagnosis to involvement in subsequent monitoring.

It is important that the nominated private pathologist is seen and accepted as being representative of the whole group rather than from one private company. With this in mind, I write in the hope that your Society, being representative of both government and privately employed pathologists, would be in a position to nominate a suitable person.

Meetings of SCAHLS are held once a year, usually in July. I look forward to hearing from you in the near future.

Yours faithfully,

RAY WEBB
on behalf of KEITH MURRAY
Chairman, SCAHLS

7 October 1993

AGRICULTURAL RESEARCH COUNCIL
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Announcing:

NOTES ON OSTRICH DISEASES

F W Huchzermeyer

In recent years the ostrich industry has experienced a revival and has again spread rapidly not only beyond the borders of the Little Karoo but beyond the borders of South Africa itself and across other continents. This worldwide growth of ostrich production has also fanned a new interest in the veterinary aspects of ostrich farming. For many years our knowledge of ostrich diseases was somewhat akin to alchemy, based on a few ancient publications, some whispered information and a lot of guess work. Even now it is in a state of rapid flux. New facts continue to come to light, so this book will require frequent updating. Its title is meant to indicate this state of incompleteness. It is based on personal experience in ostrich pathology, a general poultry pathology background and a literature survey, in some cases data from other ratite species, particularly rhea and emu, have also been included.

The book will have 120 pages with 24 full colour illustrations in addition to black and white illustrations, 20 tables, a complete bibliography and will be in soft cover. It is expected to appear towards the end of 1993.

The projected price is R60.00 + R3.00 handling + VAT = R71.82

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ONDERSTEPOORT VETERINARY INSTITUTE
PRIVATE BAG X5
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REPUBLIC OF SOUTH AFRICA

Name: ……..
Postal address: ……..
……..
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