AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

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APPOINTMENTS

Chairman - Registry of Domestic Animal Pathology	Tony Ross
Newsletter Editor	Cleve Main
Continuing Education - NRDAP	Phil Ladds

CONVENOR - SLIDE OF MONTH

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PRESIDENTS REPORT

The major event for the Executive since the AGM has been interaction with the National Review of Laboratory services launched by the AAHC. No doubt many of you will have also interacted with it in some way. Barry Richards is of course one of the three members of the Review team, and I represented the Society when the team visited Murdoch University during the Western Australian leg of its national tour. In addition the society has made a written submission to the Review, which was initially drafted by me and refined by the Executive. This submission is included in the mailing for the information of members. The impact of recommendations made when the Review submits its report towards the end of this year will be of great significance for the Society. We should be hopeful that there will be some positive outcomes for all of us. In the meantime I would appreciate receiving information and critical comment from you. Several members have already obliged in this way, so join them and feed us your opinions and knowledge.

Another task we are getting started on is the venue and programme for the 1999 meeting. The plan is to schedule it for Melbourne on the weekend before the AVA meets in Hobart. This year's meeting set a high standard all round and gives the next organizing committee something to aim for. One thing I would like to see - as many presentations as possible by the younger members, particularly those in training.

That's it for now - best wishes to you all

A/Prof. Clive R. Huxtable
HONORARY PRESIDENT

EDITORIAL

Well, here it is the September issue of the Report and I can't believe it; we've got contributions from nearly all States. Well done!

I have responses from several members regarding delivery of the Report to their email addresses. By the time you read this, those members will have received their electronic copy a week ago. So if you want to join a growing trend, drop me a line or send an email (cmain@agric.wa.gov.au) advising me of your address and I will add you to the list.

Some of you may be getting a little anxious, as you haven't received your copy of the last ASVP Conference Proceedings. If it makes you feel any better, I haven't received mine either. Tony Ross tells me they're on their way and you should have your copy in the near future.

CONTINUING EDUCATION COURSES, 1998 - PROGRAMME NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY. Phil Ladds has come and gone. We in the West have been reminded how much reproductive system pathology we either never knew or had forgotten. The reports that are filtering in to me via the grapevine give me the strong impression that, Australia wide, everyone that attended his sessions enjoyed them tremendously. - Thanks Phil

On a more serious note and in conjunction with this year's "Registry - Continuing Education" courses in each state, Phil has held discussions with various pathologists about aspects of Quality Assurance in veterinary anatomical pathology. He is to report (with recommendations) to the ASVP on the outcome of these discussions by 30 September. At the end of discussions at each centre, pathologists were asked to complete and return a questionnaire. So that the report can be truly representative, Phil would greatly appreciate receiving completed questionnaires from pathologists who did not attend the above discussions or have not yet returned their questionnaire. The questionnaire accompanies this Report

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Phil also wanted to alert everyone to the ILSI (Int. Life Sciences Inst.) meeting on **"IMMUNOCYTOCHEMISTRY"** to be held in St. Louis, Missouri on Nov. 16 in conjunction with the ACVP meeting (Nov. 17-20).). Further information can be obtained by email: rsi@ilsi.org or at http://www.ilsi.org

List of Members - It was intended a current list of financial members be published in each edition of the Report. However, there has been a change of staff at the Secretariat's office in South Australia. Associated with the change are a few other problems that have lead to a backlog of ASVP work. A number of cheques for this year's subscriptions have yet to be banked. So anything I publish in this issue would be incorrect and cause confusion. RESULT - There's no list in this issue.

STATE REPORTS

Western Australia - David Forshaw

Layer Mortalities - John Creeper, Agriculture WA, South Perth

Mortalities in a commercial layer flock commenced at one end of a row of cages and proceeded over a 2 week period towards the other end. The birds (from a different supplier) in adjacent rows were unaffected throughout the outbreak.

On necropsy, affected birds showed enlarged livers and spleens. Histologically, changes of periellipsoid amyloidoisis within the spleen and lymphocytic portal hepatitis were seen in several birds. Big Liver and Spleen antigen was detected from birds and Pasteurella multocida was recovered. The significance of the BLS antigen in these birds is not known.

Fish myopathies - John Creeper, Agriculture WA, South Perth

During periods of very cold weather, there have been several cases of skeletal muscle myopathies in both tropical fish and commercially raised Silver Perch. There are extensive histopathological lesions for which no causal infectious agent could be identified. Has anyone else encountered this problem?

Bird myopathies - John Creeper, Agriculture WA, South Perth

A breeder of fancy poultry experienced 75% mortalities in birds between 15 and 26 weeks of age. Affected birds show a general decline prior to death. Grossly, the lesions were restricted to musculature of the breast and legs. At both sites the muscle contained a mixture of brown liquid, pale muscle and areas of fibrous tissue. About 60 %of the muscle mass was affected. Histopathologically there was coagulative necrosis and masses of gram positive cocci in chains. Cl.perfringens was cultured from the liquid which oozed from the muscle. The birds are on a commercial ration, there is NO coccidiostat used.

Comments from anyone who has seen similar changes would be appreciated.

Erysipelas Septicemia in Unmarked Lambs - Marc Kabay, Agriculture WA, South Perth

An uncommon form of Erysipelas - septicemia with high mortality - was seen recently in young unmarked lambs at Koorda. Erysipelas usually presents in sheep as polyarthritis in lambs following marking or as post dipping lameness in any age group. Morbidity may be high but mortality rates are usually low.

In late May a mixed age flock of 450 Merino ewes was introduced to a paddock containing a lush growth of capeweed, radish and regrowth wheat. When lambing started in mid June, many lambs died at 3 to 5 days after birth, without prior signs of illness. Lambs died on their sides, without struggling, and usually with the head drawn back. About 50 percent of the lamb flock was lost.

Necropsy findings of three lambs were unremarkable. All had large milk clots in the abomasum and one had a mild interstitial pneumonia. *Erysipelothrix rhusiopathiae* was isolated from lung and heart blood. The cause of this outbreak is unknown, but it is known that the bacteria are common in soil and faeces and can enter the circulation via the umbilicus or the feet.

Multiple problems in a multiple species bird flock - Mandy O'Hara, Shane Raidal, Murdoch University

10 x Japanese quail, 4 x silky bantams, 2 x pigeons and 1 x chukar partridge were submitted from a commercial producer to the Division of Veterinary and Biomedical Sciences, Murdoch University for post mortem examination. The birds were submitted to investigate the problems of:

- multiple skin tumours in the bantams,
- low hatchability and facial and per-ocular swelling in the quail
- weight loss in the pigeons, and
- ocular swelling, mucoid nasal discharge and respiratory noise in the partridge

The skin lesions in the bantams were attributable to Mareks disease and fowl pox with a secondary bacterial pyoderma.

The facial and ocular swelling in the quail was associated with cystic follicular hyperkeratosis and diffuse epidermal orthokeratosis. These lesions were non-inflammatory and suggestive of a nutritional or metabolic origin, most likely Vitamin B deficiency.

The weight loss in the pigeons was associated with a protozoal ingluvitis due to *Trichomoniasis sp*. In addition, there was a marked diffuse hyperplastic enteritis with basophilic intranuclear inclusions within enterocytes suggestive of adenovirus enteritis. The pigeons, quails and bantams all had a hyperplastic lymphoplasmacytic colitis with intra-glandular protozoa consistent with *Spironucleus* spp (formerly known as *Hexamita* spp).

The ocular swelling and nasal discharge in the partridge was associated with a chronic hyperkeratotic sinusitis. Bacterial culture was negative. This bird also had diffuse facial epidermal orthokeratosis and follicular hyperkeratosis similar to the quail suggesting a common nutritional or metabolic origin. There was a mild diffuse subacute tracheitis and multifocal mild granulomatous bronchitis consistent with a bacterial aetiology, although culture was negative.

The multitude of problems identified within the different bird species suggests significant failings in the management and husbandry of the operation. A farm visit is scheduled to further address husbandry and management practices in association with implementation of preventative and treatment strategies for the diseases identified.

New South Wales - Paul Gill

NSW Veterinary Histopathology Group Meeting.

The final meeting for this year will be on Saturday 14 November at the Sydney vet school. Anyone interested in veterinary pathology is welcome to attend. Please contact Malcolm France for more details (02 9351 2023; france@vetp.usyd.edu.au). The usual mailing will go out to those on the group's mailing list a few weeks before the meeting.

Exocrine pancreatic insufficiency in laboratory rats - Malcolm France, University of Sydney

Wistar rats in a biochemical study began losing weight. The researchers also noticed that their food consumption had increased and that their faeces were sometimes pale. On gross examination the pancreata were reduced in size (sometimes markedly) and had a granular appearance. Histologically there was interstitial fibrosis, loss of acini and a generally mild lymphocytic infiltrate. Mitotic figures were relatively numerous in surviving acini. The study was an investigation into diabetes and although the islets appeared normal histologically, the researchers decided to euthanase all 70 rats.

Lesions of this type (referred to as 'pancreatic atrophy') are recognised as an age-related change in Wistar rats and have been induced by a variety of experimental regimes including copper deficiency and administration of various compounds. The animals in this case, however, were of mixed ages and clinical signs appeared in different groups more or less simultaneously. The rats were fed one of several freshly prepared 'synthetic' diets and an error in formulation seems a likely explanation for the problem.

Investigations are continuing and I would be interested to hear from anyone with suggestions as to which dietary or other factors might be worth investigating.

Adult onset hind limb ataxia due to ceroid lipofuscinosis in ferrets - Malcolm France, University of Sydney

A 3rd year veterinary student (Francette Geraghty) brought in a pet ferret for post mortem examination. The animal was adult and had developed hind limb ataxia and incontinence over the last 3 or 4 months. On gross examination the brain appeared small and there was moderate hydrocephalus. To my surprise, histological examination revealed eosinophilic, hyaline inclusions in neurones in virtually all central and peripheral locations. These stained with PAS and luxol fast blue and were auto fluorescent leading to a diagnosis of neuronal ceroid lipofuscinosis (NCL). Subsequently, Francette has encountered another ferret with similar clinical and pathological findings. The inclusions were examined under EM by Rosanne Taylor who found them to be of the 'fingerprint' type often associated with subunit c accumulation.

NCL does not appear to have been recognised previously in ferrets although in addition to these 2 confirmed cases, we know of 2 more Sydney ferrets exhibiting similar clinical signs. The adult onset and predominance of thoraco-lumbar signs make an unusual clinical presentation for a storage disease. This makes me wonder if NCL is being seen but perhaps dismissed as intervertebral disk disease or some other form of acquired spinal lesion. Unfortunately no family history is available for any of the affected animals.

Kikuyu grass (*Pennisetum clandestinum*) poisoning of cattle - Steven Hum, Elizabeth Macarthur Agricultural institute

On 27th of February, 9 bulls were removed from a 6 acre paddock consisting of kikuyu grass on a property next to the Nepean river, near Windsor and replaced by 80 heavily pregnant Friesian cows for calving. The next morning 2 cows were found dead and large proportion of cattle appeared depressed, dehydrated, had staggering gait, salivation, rumen stasis and inability to swallow. The group were immediately removed

from the pasture except for 16 very sick cows which could not be moved, all of which died by the next morning. Two cattle were submitted to the laboratory for post mortem examination.

Both animals were dehydrated and in good body condition. Autolysis was advanced in the first carcass, which weighed 550kg; she had calved a few hours before death and had severe multifocal haemorrhages throughout the liver and on the serous surfaces of visceral organs. The second carcass weighed 570kg, had a full term foetus in the uterus and there were petechial haemorrhages over serous surfaces of visceral organs. The mucosa of the rumen and omasum was sloughing off in both animals and the sub mucosa was congested.

The significant histologic lesions were similar in both cattle and confined to the omasum and rumen. There was a marked neutrophil infiltration and under running of the stratum corneum separating it from the basal layers and leading to ulcerations in some places. Similar damage was seen in the rumen but lesions were more superficial and less widespread. There was a diffuse submucosal vascular congestion in both forestomachs. In addition in cow one, there were acute multifocal haemorrhages and septic thrombosis in the liver, kidney and lung consistent with terminal septicaemia. There was no evidence of grain feeding; the rumen contained only kikuyu grass. The aqueous humour Ca, Mg, and BOHB level were normal and negative for Nitrite/Nitrate in both animals. There were no active protozoa detected in the rumen liquor of the second cow.

These findings of toxic/chemical omasitis and rumenitis are consistent with previously reported cases of kikuyu grass poisoning.

A further 12 cattle died from the same group in the next two days exhibiting similar clinical signs but no post mortem examinations were conducted.

Cattle deaths associated with the grazing of rapidly growing kikuyu grass have occurred in New Zealand. South Africa and Australia. In New Zealand and in South Africa previous feeding on the pasture by the armyworm caterpillar (*Spodoptera exempta*) is considered to be of epizootiological significance although kikuyu pastures so invaded do not invariable become toxic. This hypothesis assumes that toxic substances may be produced by the plant against the invading caterpillar which may also be toxic to livestock.

In Western Australia, however, two outbreaks were reported on kikuyu grass which apparently had not been invaded by insect pests, and the outbreaks took place in an area where the armyworm caterpillar apparently does not occur. The possibility of mycotoxin involvement has also been investigated. Evidence from the isolations of pasture fungi, so far has not suggested a mycotic cause. In some studies the epidemiological evidence of lush growth following drought-breaking rains suggest that a plant toxin may be released under certain meteorologic or climatic conditions.

The circumstances leading to kikuyu grass poisoning on this property has not been ascertained. Large areas of pasture were infested by armyworm caterpillars in all paddocks in the previous month. Also, the 27th of February was a hot day and temperature reached 40°C with cool change late on the afternoon and some rain the next day. The 9 bulls grazing on the paddock for three months before their removal on 27th of February and other cattle grazing on similar pastures on the property had no problems.

The unpredictable nature of the outbreaks of kikuyu grass poisoning, the sudden onset and heavy losses which may follow and the inability to predict, advise or prevent outbreaks occurring can be very frustrating both for the manager and the veterinarian.

Ref: Wong PTW, Roth IJ and Jackson ARB (1987) *Aust Vet J*, 64 No 8, 229 - 232.

Northern Territory - Anton Janmaat

Tuberculosis - the National Granuloma Submission Program works - Anton Janmaat, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

Continuing on from the last newsletter.....the second diagnosis of tuberculosis this year concerned lymph node lesions from an aged cow from a station in the Victoria River District. The gross diagnosis by the Katherine abattoir inspector, who submitted the nodes under the national granuloma submission program, was a confident tuberculosis. Two bronchial and a mediastinal lymph node were submitted and all showed lesions histopathologically consistent with tuberculosis including the presence of acid-fast bacilli.

The third diagnosis of tuberculosis this year was in a de-stock from the property on which the first case (reported in the June 1998 ASVP newsletter) was diagnosed this year. This latest diagnosis was greeted with grunts of relief by the regulatory people rather than the usual sighs of despondency and the inevitable question.... are you sure?

Septicaemia in a Shar Pei pup - Helen Parkes, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

We recently did a post-mortem examination on a 10 week old. Chinese Shar Pei puppy. The pup had died suddenly, and showed signs consistent with acute septicaemia. Pasteurella multocida was isolated from various lymph nodes, lung and other tissues. The interesting thing about the case was the book (produced by the Chinese Shar Pei Club of America) that arrived with the pup, described the many congenital, degenerative and infectious disease problems associated with this breed, as well as their behavioural problems. Read the book first if you're thinking about getting a Shar Pei.

"Sudden Death" in a cat associated with Dirofilaria immitis infection - Helen Parkes, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

Post-mortem examination of a 7 year old, apparently healthy, male Abyssinian cat with a history of "sudden death" (? snakebite, by the submitting vet) revealed firm lungs that did not deflate fully and a knot of heartworms in the venacava. Histological examination confirmed pulmonary changes related to the presence of heartworm, and a heartworm antigen test was strongly positive. Unfortunately, heartworm disease in cats isn't always so blatantly obvious on PM!

Victoria - Malcolm Lancaster

Immunohistochemistry for Transmissible Encephalopathies - Peter Hooper, CSIRO -AAHL, Victoria

AAHL has obtained a very good monoclonal antibody, effective in indirect immunohistochemical tests for transmissible encephalopathies. This is important as we are all now launching into the TSE's surveillance program.

The monoclonal is that reported by O'Rourke et al. It gives very strong clear cut reactions in tests on the collection of positive blocks of scrapie and BSE retained by AAHL (and which most of you have slides). The reactivity appeared to be confined to target areas where there are most lesions, e.g. the solitary and trigeminal spinal nuclei in BSE, and in some grey matter of the medulla and perivascular "amyloid" deposits in scrapie. This was a good indication that the antibody was specifying the abnormal isoforms of the prion protein.

The development of this test is very well timed.

O'Rourke et al (1998) Monoclonal antibody F89/169.1.5 defines a conserved epitope on the ruminant prion protein. J Clin Microbiol 36 1750-1755.

Basal cell tumour in a green tree frog - LeeBerger, CSIRO, AAHL, Victoria.

A large (ll0gm) free ranging green tree frog (Litora caerulea) was collected from Lismore High School as it was lethargic and had multiple ulcerated 3-20mm diameter nodules on the dorsal skin of the body and limbs.

The frog died in transit. Histologically, the nodules were found to be composed of epithelial cells arranged in varying stages of glandular differentiation as well as areas undergoing squamous metaplasia with keratin production. The mitotic rate was low and there was little variation in nuclear and cell size. The appearance was consistent with that of a basal cell tumour.

Cutaneous granulomas containing acid-fast organisms in long footed potoroos - Dr. Judith S. Nimmo Wilkie Victorian Veterinary Pathology Services, Dr Rosie Booth, Healesville Sanctuary.

A colony of long-footed potoroos had dwindled from 15 to three individuals. Many of the deaths had been due to pulmonary or systemic mycobacteriosis with M. avium and M. terrae having been isolated from lesions in the past.

Of the three remaining animals two had multiple cutaneous lesions. These were red, raised, alopecic but non-painful nodules about 1cm in diameter. Histologically the lesions consisted of a solid sheet of foamy macrophages interspersed with mast cells, located in the superficial and deep dermis extending up to the dermo-epidermal junction. No organisms were seen in the lesions with Gram, PAS or ZN stains. However, a modified acid-fast stain (Fites) revealed small numbers of acid-fast organisms within the macrophages. The organisms varied from rod shaped to beaded. To date the organism has failed to grow in culture. It is likely to be an atypical mycobacterium or possibly a Nocardia sp which will also stain with modified acid-fast stains.

Lead Poisoning in a Wedge Tailed Eagle - Dr. Judith S. Nimmo Wilkie, Victorian Veterinary Pathology Services, Dr. Rosie Booth, Healesville Sanctuary.

An adult wild wedge-tailed eagle was found in a paddock. It was moribund, unable to fly or right itself and exhibiting neurological signs. The bird had a normal haemogram which included a PCV of 0.47. Red cell morphology was normal except for some mild polychromasia. Blood lead levels on this bird were 13.71 umol/L. (normal levels for mammals are < 1.71umol/L). The source of the lead is unknown but is likely to be ingestion of shot rabbits or lead poisoned waterfowl. Small metallic objects were seen in the gizzard on Xrays of the bird.

Possible lysosomal storage disease in neonatal Southdown lambs - Malcolm Lancaster VIAS Attwood, Judith Nimmo Wilkie WPS South Yarra

A Southdown stud owner reported that lambs had been born in the last 3 seasons with neurological signs and a thickened leathery skin. The Iambs exhibited continuous shaking of the head and limbs and couldn't stand and suckle. Histological lesions were seen in the nervous system, skin and mononuclear phagocyte system. Many neurons in the brain and spine were degenerate or necrotic, with some enlarged and pale. Occasional blobs of degenerate myelin were evident in both CNS and peripheral nerves. Macrophages with large cytoplasmic volume were present in thymus, lymph nodes, spleen and liver. The skin was orthohyperkeratotic and the hair follicles were irregularly arranged. Copper levels were within the expected range.

South Australia - Ruth Reuter

Adenovirus pneumonia in guinea pigs - John Finnie, Veterinary Division, IMVS, Adelaide

Four young adult guinea pigs from a Melbourne laboratory animal facility were found dead with pathological features of adenovirus pneumonia.

Microscopic examination of lung revealed a severe, acute, necrotising, exfoliative bronchitis and bronchiolitis. There was extensive necrosis and desquamation of respiratory epithelium in affected airways, with scant remaining intact, viable, lining epithelial cells. Lumina were frequently occluded by cellular and streaming nuclear debris, admixed with degenerate leucocytes and fibrin. Recognisable sloughed epithelium was deeply eosinophilic and sometimes contained large (5-15 um diameter) basophilic, round to ovoid, intranuclear inclusion bodies with margination of nuclear chromatin. Fewer inclusion bodies were found in intact lining epithelium. The lungs were markedly congested with a light, diffuse infiltration of the alveolar interstitium by inflammatory cells, mainly lymphocytes with fewer plasma cells, macrophages and neutrophils. There was also a mild, non-suppurative tracheitis with lymphoplasmacytic infiltration of congested and oedematous lamina propria and mildly hyperplastic lining epithelium, but inclusion bodies were not detected in the trachea. No pathological changes were found in other tissues.

Respiratory infections in guinea pigs are predominantly bacterial, with few viral pathogens described. However, pneumonia associated with an adenovirus has been reported in Europe, North America and, occasionally in Australia. Cavian adenovirus pneumonia appears to be a disease of low morbidity but often high mortality, with affected animals often found dead or sometimes showing premonitory respiratory distress. Young guinea pigs are most susceptible, although the virus appears not to be highly contagious, and clinical disease is often precipitated by stress. The virus has been poorly characterised since in vitro cultivation methods are lacking and present diagnostic serological techniques are not specific for guinea pig adenovirus, but polymerase chain reaction analysis suggests that this guinea pig agent is a distinct adenovirus.

Pelican Mortalities in Country SA - Ruth Reuter, VPS/VETLAB, Primary Industries South Australia, Adelaide

An inspector working with the Department of Environment, Heritage and Aboriginal Affairs (previously known under a variety of names including National Parks and Wildlife) received a report of excessive mortality in pelicans along the Murray River. 50 birds were found dead, with numerous others showing signs of weakness and apparent ataxia. The dead birds all appeared to have succumbed around the same time. Local residents were concerned that the deaths were due to malicious or toxic causes.

Most of the dead birds were decomposed and unsuitable for post mortem examination. However, a freshly dead bird was obtained and submitted to the laboratory. On external examination, the feathers were rough and dirty. There were teeth marks on the right leg, with extensive haemorrhage in the subcutis in this region. The other main findings were emaciation, an empty digestive tract and atrophic liver and kidneys.

The week before the birds were found dead, the temperatures in the area dropped to -6°C overnight, with frost. The Murray River at this location is turbid, with few fish present. The post mortem examination, in conjunction with these observations, suggested that hypothermia associated with starvation was responsible for the "sudden" deaths. The rough feathers indicated that the bird had not been preening itself. The bite marks on the leg could have been inflicted by a dog attempting to catch the moribund bird.

Queensland - Bruce Hill

Myxosporidian infection in a dolphin fish (Coryphaena hippurus) - Wendy Townsend, QDPI, Yeerongpilly Veterinary Laboratory

A local fisherman contacted the laboratory about the abnormal appearance of a dolphin fish. The fish looked perfectly normal when it was caught, and a friend who gutted the fish noted no abnormalities. Upon filleting the fish however, the fisherman found that its muscle had "turned to mush". The remains of the partially filleted fish were brought to the laboratory for examination. No skin lesions were evident, but all the muscle remaining on the carcase showed extensive liquefactive necrosis. Surprisingly little odour was noted, considering the extent of liquefaction.

Histological examination revealed extensive muscle degeneration and liquefaction, with minimal inflammation or haemorrhage. Numerous myxozoan spores resembling Kudoa, were seen throughout the affected muscle.

Several genera of Myxozoans, including Kudoa, have a spore stage within the white muscle of wild fish. In live fish, numerous parasitic foci in the muscle give it a mottled appearance. After the fish dies the flesh rapidly softens and can turn into either a thin jelly-like substance or thick viscous material, which has minimal odour. It is thought that in live hosts, proteolytic enzymes are released by the parasites and continually removed by the bloodstream, or that the enzymes are localised strictly within parasitic pseudocysts (muscle fibres replete with one large, or many small plasmodia). After death it is thought that these enzymes accumulate and/or diffuse outwards causing autolysis of host tissues.

Reference: Lom J, Dykova I: (1992) Protozoan Parasites of Fishes. In Developments in Aquaculture and Fisheries Science (26), Elsevier, Amsterdam, pp 174-175

Pituitary-dependant hyperadenocorticism in a cat - John Mackie, VPS, Brisbane

A 16-year-old spayed female domestic shorthaired cat had a history of polydipsia, polyphagia, abdominal enlargement, and ventral alopecia, thinning of the skin, a tendency to bruise easily and a non-healing abscess on the pelvic limb. Ultrasonography revealed bilateral adrenal enlargement. At necropsy the referring veterinarian noted apparent enlargement of the pituitary gland and both adrenals. Histologic examination revealed an adenoma of the pars intermedia, bilateral adrenal cortical hyperplasia, and an atrophic dermatopathy and hepatocellular vacuolation.

Hyperadrenocorticism is relatively rare in cats but resembles the canine disorder in many other respects. It has also been associated with a skin fragility syndrome in cats.

Mulga Fern (Cheilanthes sieberi) poisoning in cattle - Jim Taylor, QDPI, Toowoomba Veterinary Laboratory

Fourteen of a group of 40 cross bred dairy heifers died after exhibiting localised swelling of the throat, epistaxis, laboured respiration, ptyalism and an occasional bloody scour. At post mortem there were extensive petechial and ecchymotic haemorrhages throughout the subcutaneous tissues and on the serosa of abdominal and thoracic viscera. Multifocal haemorrhages were present throughout the intestinal lumen and urinary bladder contents were haematuric. There was marked perilaryngeal oedema and necrosis in the local lymph nodes. Stem material of mulga fern (Cheilanthes sieberi) was present throughout the rumen contents. Haematology showed a severe pancytopaenia and sternal bone marrow sections were markedly depleted of haematopoietic cells. Sections of tissue around the larynx revealed a marked necrotising cellulitis and lymphadenitis with numerous bacteria present in sections.

Localised swelling of the throat and occasional necrotic cheilitis and stomatitis are reported in cattle with mulga and bracken fern poisoning and are assumed to be localised infections secondary to the marked pancytopaenia and possible bacteraemia.

(Jubb. Kennedy and Palmer, Pathology of Domestic Animals. 4th edition, Chapter 3 page 165).

Lymphocytic vasculitis in a dog

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A group of at least 10 puppies owned by a pet shop developed vague respiratory signs of wheezing and mild pyrexia. The group was treated with antibiotics but one pup died. A second 8 week old West Highland White developed acute laryngeal oedema and also died. A post mortem performed by the attending vet found only laryngeal oedema.

Histologic abnormalities were detected in the larynx, liver and heart. Sections taken from the larynx and laryngeal muscles showed oedema of the lamina propria and marked interstitial oedema. The most outstanding changes were in medium sized veins in the laryngeal muscles. Some of these vessels were completely blocked by lymphocytes and thrombi. Lymphocytes nested in the walls of the vessels, some had undergone karyorrhexis and there was a scattering of nuclear dust and cellular debris. Lymphocytes and a small number of plasma cells, neutrophils and histiocytic cells were present in cuffs surrounding the vessels. The liver showed mild focal acute periacinar hepatocyte necrosis and haemorrhage. Heart muscle showed small foci of mononuclear inflammatory cells within the myocardium.

Lymphocytic vasculitis is a very uncommon lesion and may be associated either with resolving stages of immune complex vasculitis or may represent a primary cell mediated immune response directed against the vessel wall. The source of antigen in most cases of vasculitis is never determined. Several of the puppies in the same group went on to develop blue eye. An exhaustive search for adenoviral inclusions proved negative and it was felt that the liver and heart lesions could be ascribed to hypoxia.

The pathogenesis of canine adenovirus-1 infection is described in Jubb and Kennedy. Virus multiplication occurs first in the tonsils, where it produces a tonsillitis. This can sometimes be severe and may be fatal, with oedema of the throat and larynx. No description of a vasculitis is given. Tonsilar tissue in this case showed a reactive hyperplasia but no evidence of necrosis or inflammation.

Pups arriving at this pet shop had only been vaccinated for parvovirus and leptospirosis. Canine adenovirus-1 vaccination was not given until 12 weeks of age. On contacting the attending vet, two months later, vaccination of the canine adenovirus-1 is now given on arrival and there have been no more cases either of laryngeal oedema or blue eye. Perhaps the development of blue eye in the other pups was in fact a red herring as far as this WHW was concerned. These pups are all coming from farms into a group of young stressed individuals and it is likely that a number of pathogens are present.

Perhaps a respiratory pathogen was responsible both for the vague respiratory signs and also provided the antigenic stimulus for the vasculitis. Bacterial culture results of trachea! swabs were negative. I would be very interested to hear from anyone who has seen a similar case to this.

13.

Feline cowpox virus infection

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United Kingdom

A 6 year old female DSH developed acute ulcerative lesions on the face and on one paw. Biopsies from these areas showed severe ulceration. Intact epithelium was hyperplastic, spongiotic and showed ballooning of the cells of the stratum spinosum. Some of these cells contained large eosinophilic intracytoplasmic inclusions. Follicular epithelium was necrotic with hair free in the dermis surrounded by cellular debris and a very marked infiltrate of mixed inflammatory cells. Eosinophils were prominent in this infiltrate. Some fragments of follicular epithelium and adnexal epithelium were present and cells also showed inclusions.

Feline cowpox virus is fairly common in the UK from spring to autumn with most cases seen in autumn. The diagnosis is usually straightforward, although older severely inflamed lesions can be a challenge. Often there is little intact epithelium left and the inclusions are not present in older lesions. The severe necrosis of the follicular epithelium and the severe inflammation will usually suggest Pox virus infection. This virus is an Orthopoxvirus virus. It is presumed that small wild mammals act as the reservoir of infection and that cats become infected after hunting. The lesions resolve after 4-6 weeks in immuno competent individuals. More severe disease is associated with immuno suppression, for example, if the cat is treated with corticosteroids. In these cases infection in the lungs produces a pneumonia which carries a poor prognosis. Older lesions with few or no inclusions and lots of eosinophils can be a diagnostic challenge as they need to be differentiated from lesions of the eosinophilic granuloma complex. The virus is potentially zoonotic but infection of humans appears to be extremely rare. The virus is present in Europe but we see cases only rarely from European clients. Apparently cow pox is not present in Ireland.

STATE REPRENTATIVES

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AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY,
NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY -

QUALITY ASSURANCE AND ACCREDITATION IN ANATOMIC PATHOLOGY

- MEMBERS QUESTIONNAIRE

1. Respondent Details

a. Years since graduation _____

b. Post graduate (pathology) qualifications held:- Masters Degree _____ PhD _____

MACVSc _____ FACVSc _____ Dipl. ACVP _____ MRC Path _____ FRC Path _____

Other (please specify) _____

c. Number of years worked as a veterinary pathologist _____

d. Approximate percentage of time spent during this period involved in:-

Diagnostic pathology _____ % Research in Pathology _____%
Teaching Pathology _____%

2. Minimum Qualification/Experience

Please indicate what you, as a practicing veterinary pathologist, regard as the **minimal** acceptable qualification/experience for a veterinarian conducting:-

a. gross and histopathological examinations under indirect specialist (eg FACVSc) supervision in a diagnostic laboratory and writing preliminary reports. (Tick one)

- BVSc (new graduate) _____

- BVSc + 1-3 years supervised experience _____

- MACVSc _____

b. gross and histopathological examinations **independently,** writing, signing and dispatching final reports to clients (Tick one)

- BVSc (new graduate) _____

- BVSc + appropriate supervised experience _____

- MACVSc _____

- FACVSc (or equivalent) _____

3. Continuing Education and Proficiency Testing

So that standards deemed acceptable as above will be maintained by a practicing veterinary pathologist with time, please indicate your attitudes to the following:-

a. "on the job" experience by a conscientious pathologist automatically maintains competence so that any form of structured/formal continuing education and record of it is unnecessary.
Agree _____ Disagree _____

b. Some structured continuing education (SCE) for all practicing veterinary pathologists is,
Desirable _____ Essential _____

c. Demonstration of regular participation of a pathologist in such SCE and formal recording of this should be Voluntary _____ Compulsory _____

d. The ACVSc has accepted the role of examining pathologists and awarding the appropriate qualification. Which organisation, in your opinion, should accept the role of providing and or documenting SCE in pathology as outlined above? The ACVSc _____ the ASVP _____the Veterinary Schools _____ the Postgraduate Foundations _____ Others (please specify) _____

e. All formal CE initiatives and documentation of participation in these will need to be self-funding. Bearing in mind such factors as the professional satisfaction of keeping abreast with developments in ones specialty, and taxation concessions for CE programmes, please indicate what you would regard as an appropriate **total** annual expenditure (i.e. inclusive of airfares, accommodation etc.) per pathologist on SCE and proficiency testing. $100-200 ____ $200-400 ____ $400-1000 ____ $1000-$1500 ____ >$1500 ____

f. Assuming that formal accreditation for a pathologist's participation in CE and Quality Assessment initiatives is given and advertised (as is, for example, NATA accreditation in **anatomical pathology** would justify the expense and effort involved? Yes _____ No _____ Maybe _____

g. In view of the trend towards species-oriented, specialty **clinical** practice in veterinary medicine, do you feel there may be merit in ongoing CE and accreditation in veterinary pathology that is species related (e.g. avian pathology, commercial livestock, companion animals)? Yes _____ No _____

h. In regard to possible **CE programs** that could be established by one or more of the above organisations, please indicate your likely preference for the following (Indicate A - most support, E - least support)
- Preparation/Circulation of Training Modules
- Organisation and conduct of an annual 2 day [species, system or technique (e.g. immunocytochemistry) oriented] workshop utilizing invited national or international leaders in that field. Such a workshop being coordinated with the ASVP AGM.
- Continuation of the ASVP "Slide-of-the-month" program, perhaps with improved coordination and monitoring funded by a direct monetary contribution by all receiving microslides
- Expanded "pathmail" participation, with rostered contributions (interesting/unusual cases; literature updates; mini-reviews etc) by participants in their areas of expertise.
- Expansion of the referral service as offered by the Registry, perhaps with arrangements in place for rapid "second referrals" through the Registry to recognise specialists.

i. In regard to **Proficiency Testing/Quality Assurance** in anatomical pathology and possibly cytology, please indicate (bearing in mind the likely cost involved) if you would be prepared to subscribe to, and actively participate in:-
- Maintenance and submission of a detailed Continuing Professional Development Diary of work related and CE activities. Yes _____ No _____
- Examination and reporting back on peer-reviewed "mystery slides" distributed regularly by e.g. the Registry. Yes _____ No _____

PLEASE ATTACH ANY FURTHER COMMENTS OR SUGGESTIONS ON CONTINUING EDUCTION AND QUALITY ASSURANCE ➔