VETERINARY PATHOLOGY REPORT

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President's Report

Although it has only been two months since our Annual Conference, which was held here in Adelaide, there have been several developments involving the ASVP. The website now contains items such as the conference minutes and proceedings, in addition to other information previously located there. Hopefully this will expand more in the future, as members begin to take advantage of this rapid means of communication.

Round 4 of the Histopathology Quality Assurance program was circulated by Tony Ross in June, and those results will be forthcoming. This program was strongly supported at the annual meeting, and all labs are encouraged to participate in this essential program.

A submission was forwarded to the Government Inquiry into Veterinary Pathology Services in Victoria. This article is also present on the website, in the members only section. Change is constantly occurring, particularly in the area of technology, and is a feature of many aspects of our lives. I have also felt the "winds of change" recently, having been with IDEXX/VPS for more than 11 years. As a result, I have tendered my resignation to IDEXX laboratories, and will be leaving Australia in October. I have accepted a position as diagnostic pathologist in Herefordshire, UK, where I will be staying for the next 2 - 3 years. Although I hope to retain my membership in the ASVP, an interim President must be appointed to fill the gap until the change of executive in Sydney.

As with any move, there are always mixed feelings. I have enjoyed my association with the ASVP and my colleagues here in Australia, and hope to keep in touch during my absence via the 'Net'. I will follow developments here with interest.

Ruth Reuter
Honorary President

Editorial/Secretary's Report

As you will be aware, this Executive established the ASVP website in order to promote a rapid, cost-effective means of communication between members and feedback suggests that the consultant employed to construct the site has performed admirably. However, I have believed for some time that we needed to have an ASVP member maintain the site "in house" to meet the needs of pathologists more effectively. Cleve Main has kindly agreed to assume this role, and I thank him for this valuable contribution to the Society. Any suggestions regarding the site operation should be directed to Cleve at cmain@agric.wa.gov.au

We have compiled an ASVP response to the Victorian government enquiry into the provision of veterinary pathology services (see website) and many of the issues raised therein also apply more generally to other States.

We recently received an invitation from the AVA to send an ASVP observer to the AVA Policy Council meeting in October to "seek to strengthen the relationship with, and involvement of, veterinary pathologists in the Association". Stephen Yeomans has agreed to fill this role, with Keith Walker standing in for Steve at the first meeting. The notion of affiliation of the ASVP with the AVA has a long history, with the majority of pathologists continuing to favour a separate existence, but we might as well know what they are prepared to offer! The right of ASVP members not to join the AVA would probably be a non-negotiable condition of any such affiliation, together with our choice of meeting venue.

Tony Ross has agreed to coordinate the 2003 ASVP Conference in Sydney and has proposed the weekend of April 12 and 13 at EMAI, immediately after the Sydney Postgraduate Foundation Gross Pathology course.

We will soon, unfortunately,be losing the services of Ruth Reuter and Julia Lucas, who are departing for the UK and USA, respectively. I would like to thank them both for their substantial contribution to the Society and wish them well in their new pathology endeavours. Peter Phillips has agreed to wear two hats until next year's hand-over to Queensland - President and Treasurer - and Stephen Yeomans (Gribbles, Adelaide) will be the SA Rep.

John Finnie
Honorary Secretary

STATE REPORTS

NSW – Paul Gill

No report

NT – Anton Janmaat

Possible Coffee Senna toxicity in a herd of gemsbok

Cathy Shilton, Berrimah Veterinary Laboratories (BVL), NT Department of Business, Industry and Resource Development

All animals in a herd of three aged adult gemsbok (Oryx gazella) died within a four-week period in March and April. The gemsbok were part of a collection of African ungulates kept in large paddocks containing native plants. The first two gemsbok to die were not observed to be sick. The third was observed to be lethargic and weak in the hind legs for two weeks prior to being found dead.

Carcasses of the first two gemsboks to die were too autolysed for meaningful post-mortems. Relevant post-mortem findings in the third gemsbok were limited to a fine red and tan zonal pattern in the liver. Histologically, there was severe diffuse bridging periacinar hepatic necrosis with associated haemorrhage. Hepatocytes at the periphery of affected regions were variably shrunken or vacuolated. There was moderate anisokaryosis of remaining hepatocytes, and mild cholangiolar proliferation.

Sections of muscle that had been randomly selected from the hind limbs, fore limbs and neck all showed similar histological changes. There was marked diffuse subacute myodegeneration characterised by hypercontracted or fragmented fibres with either vacuolated, hypereosinophilic or granular cytoplasm. Myocyte nuclei were hypertrophic and occasionally situated in the centre of myocytes. A few degenerate myofibres were infiltrated by macrophages or contained peripherally aligned satellite cell nuclei. Representative sections of various other tissues, including brain and spinal cord, did not reveal significant microscopic findings.

The manager of the facility found grazed mature coffee senna plants (*Cassia occidentalis* - identification confirmed by Andrew Mitchell, NAQS botanist) in the paddock where the gemsbok were kept. Coffee senna is a toxic plant that typically causes skeletal muscle necrosis with or without concurrent hepatic necrosis. Several potential toxins are found in the plant. The particular toxin that causes the muscle and hepatic lesions is unknown. The plant is usually unpalatable to hoofstock, reducing the chances of toxic exposure. However, the pastures where the gemsbok were kept had been sprayed by a herbicide containing 2,4-dichlorophenoxyacetic acid (2,4-D) approximately six weeks prior to the first death. Weeds exposed to this herbicide may become sweet tasting and fruit prior to dying. Both factors may have increased the likelihood of intoxication, since a sweeter taste may have rendered the plant more palatable, and seedpods are reportedly the most toxic part of the plant.

Fatal hookworm infection in an adult dog

Cathy Shilton, Berrimah Veterinary Laboratories (BVL), NT Department of Business, Industry and Resource Development

A 5 year-old intact male Akita breed dog died following several days of severe haemorrhagic diarrhoea. Faecal flotation revealed large numbers of hookworm eggs. A faecal sample submitted to the laboratory a few days prior to death did not reveal obvious pathogens on general aerobic or anaerobic bacterial culture, and neither Salmonella nor Campylobacter grew in enrichment culture.

The referring veterinarian performed the necropsy and submitted formalin-fixed tissues to the laboratory. The major gross findings were that most of the small intestine and all of the large intestine were filled with blood. Hundreds of nematodes were partially embedded in the mucosa of the submitted section of formalin-fixed small intestine.

Histopathological examination of the small intestine revealed severe regional ulcerative enteritis. There was haemorrhage within the intestinal lumen and multiple deep ulcers extending to the level of the muscularis mucosae. The ulcers either contained nematodes or were the approximate width of nematodes, and filled with necrotic tissue, neutrophils and haemorrhage. The mucosal epithelium adjacent to the ulcers was variably hyperplastic or attenuated. Intestinal villi were generally short and blunt, and there was marked diffuse infiltration of the lamina propria with eosinophils and lymphocytes.

Parasitological examination identified the nematodes as *Ancylostoma spp*. Histopathology of the lung revealed moderate acute neutrophilic and eosinophilic bronchointerstitial verminous pneumonia. There were numerous approx. 25x50 µm deeply basophilic stippled structures within the pulmonary interstitium, small capillaries and alveoli, consistent with morphology of migrating nematode larvae.

The severity of the intestinal ulceration and haemorrhage in conjunction with pneumonia and likely anaemia was deemed sufficient to have caused the death of this dog. Typically, hookworm infection is only fatal in puppies. Therefore in this case there may have been decreased host resistance, due either to immunosuppression or lack of previous exposure to the parasite. Alternatively, there may have been overwhelming infection. This animal functioned as a guard dog, and was likely tethered and constantly occupied the same small section of ground. This, and the warm, moist, tropical conditions present at the time may have allowed large numbers of infective larvae to develop in the environment, with subsequent massive percutaneous infection.

Localised vacuolation of the straight proximal tubules in cattle

Anton Janmaat, Berrimah Veterinary Laboratories (BVL), NT Department of Business, Industry and Resource Development

The kidneys of two Brahman cows four and five years old from a property near Daly Waters, submitted under the NTSESP, showed severe vacuolation of the straight proximal tubule cells confined to the outer stripe of the outer zone of the medulla. The vacuolation was visible with the naked eye as a pale band in the sections. There was considerable mineralisation lower in the medulla especially at the junction of the outer and inner zones.

The liver showed marked vacuolation of hepatocytes, more pronounced centrilobularly, variation in nuclear size of the hepatocytes and prominent Kupffer cells, some with yellow-green pigment. Neutrophils were prominent in the sinusoids and there was cholangiolar cell hyperplasia in and around the portal triads with mild to moderate mononuclear cell infiltration.

These changes suggest a toxin. The cattle had access to surface water and an algal toxin is a possibility. The localisation of the tubule cell vacuolation is possibly a dose-related response. Another cow submitted two days previously showed the same changes in liver and kidney except that there was severe degeneration and some necrosis of proximal tubule cells with proteinaceous material in the lumen. The marked vacuolation of the tubule cells extended along the entire length of the straight proximal tubules.

QLD – Bruce Hill

Myelocytomas in broiler chickens

Anita Gordon
Yeerongpilly Veterinary Laboratory

During March 2002, a broiler enterprise in SE Qld recorded mildly increased mortalities in 28-day-old birds. Affected birds were depressed, lame, had ruffled feathers and wet, whitish-yellow droppings. Colibacillosis was suspected, and four birds submitted for necropsy.

Necropsy findings were not uniform. No birds showed evidence of enteritis or septic arthritis. Bird 1 had fibrinopurulent airsacculitis and pneumonia, whereas bird 3 had fibrinous pericarditis, perihepatitis and peritonitis. Birds 2 and 4 had friable, cheesy masses within the cranium and Meckel's diverticulum, respectively. Histological examination revealed these to be neoplasms, rather than abscesses, and similar tumours were evident in the lungs of both birds. Neoplasms consisted of closely packed round cells with eccentric nuclei and strongly eosinophilic cytoplasmic granules. Mitoses were frequent, and there was minimal stroma. The neoplasms were consistent with myelocytomas. The 'J' strain of Avian Leukosis virus is said to be the most common cause of this disease (Reece R pers. com.).

In this case, mortalities were caused by at least two diseases: myelocytomatosis, and septicaemic colibacillosis (E. coli was isolated from liver and lung of Bird 1).

Anthrax in Queensland

Toowoomba Veterinary Laboratory

In January 2002, anthrax was diagnosed as the cause of death in cattle on a Wandoan property shortly after returning from agistment on a property at Dirranbandi. Most animals were found dead. One animal was observed staggering with respiratory difficulties and muscle tremors immediately prior to death. Fourteen died from a group of 150.

Post mortem findings were splenomegaly with raspberry jam appearance on cut surface; scattered ecchymoses on a number of organs; swollen and oedematous mesenteric lymph nodes and serosanguinous pleural and peritoneal effusions. Histology showed little change in tissues apart from large numbers of Gram positive bacilli in vessels in tissues with little post mortem change. A non-haemolytic *Bacillus sp.* was isolated in pure growth from a swab of the spleen.

The culture was sent to Queensland Health Scientific Services (QHSS) PC3 lab in Brisbane and Bacillus anthracis was confirmed by PCR. Isolates were also referred to the Department of Agriculture, Western Australia's Animal Health Laboratory at South Perth where the isolate was confirmed by gamma phage typing.

On trace back to the agistment property, environmental samples were collected from sites of deaths of Wandoan cattle prior to return. These were cultured by QHSS and found to contain anthrax spores. Another agistment animal from a different property subsequently died on the Dirranbandi property and cultured positive for *Bacillus anthracis*. Control measures and vaccination programs were implemented on both properties.

SA – Stephen Yeomans

***Isospora suis* infection in piglets.**

Peter Phillips, IDEXX Laboratories, 33 Flemington Street

Tissues from 2 piglets were received with the history that a whole litter of 10 2-days-old piglets had died overnight. The submitting practitioner that the intestines were red/purple and that there was no fat present.

Histopathological examination revealed no pathology in the livers (x2), lung, kidney, spleen or heart (x1). In the small intestines of both pigs there was a huge invasion by coccidian parasites into enterocytes. At this stage there appeared to be no significant rupture of cells or inflammatory response. There were large numbers of coliform bacteria on the surface of the enteric villi, particularly in one piglet.

Bacterial culture produced a mixture of haemolytic and non-haemolytic *E.coli*. No serotyping was carried out.

A diagnosis of neonatal coccidiosis (*Isospora suis*) with possible colibacillosis was proffered.

TAS – Philip Ladds

Mount Pleasant Laboratory report

Philip Ladds

Les Gabor, who worked at this laboratory for several years, recently left us to take up a contract position at the Atlantic Veterinary College on Prince Edward Is., Canada.

New staff to join us in the pathology area are Stephen Pyecroft (from Queensland), and Richmond Loh and Brad Chadwick - both from Western Australia. Also to help maintain diagnostic strength in terrestrial animal pathology, Roy Mason (who retired in 1999) has commenced "helping out" on a part-time basis.

Congratulations to Judith Handlinger, Stephen Pyecroft and Kevin Ellard (DPIWE field aquaculture veterinarian) who all recently obtained Membership of the ACVSc by examination in the discipline of Aquatic Animal Health. Judith has on previous occasions examined in this area!

Some interesting cases were as follows:

Bovine: Further cases of polioencephalomalacia occurred in 5-6 month old calves. Pestivirus infection was confirmed in 8 month old calves that had pyrexia, diarrhoea and oral lesions. Lesions characteristic of atypical interstitial pneumonia

were seen in animals from two separate herds; a dietary cause seemed likely. Salmonellosis was diagnosed as the cause of illness and death of seven two-week-old calves in a group of 50. Mycotic placentitis was confirmed in one conceptus from a property on which five cows in a group of 150, had recently aborted.

Two severe outbreaks of hepatopathy, diagnosed as Acute Bovine Liver Disease occurred in June. In one outbreak 13 Angus cattle died suddenly, while in the other outbreak in a dairy herd two cows died; a further 50 were ill and milk yield dropped by about 11%. In both outbreaks there was severe hepatic necrosis and markedly elevated "hepatic leakage" enzymes. In view of this it seemed surprising that most affected dairy cows recovered.

Ovine: Inappropriate drenching technique was the cause of illness and death of lambs on two properties. In lambs from one property there was marked facial oedema, pharyngeal ulceration and associated myositis. On the second property, slow-release drench capsules were lodged in the mid-oesophagus causing obstruction, ulceration and associated inflammation.

Urolithiasis was the cause of death of 20 of 500 wether lambs (See ASVP Adelaide Conference Proceedings 2002, p. 22); dietary factors including access to subterranean clover seemed to be causally involved.

Listeric encephalitis was observed in two flocks.

Campylobacter jejuni was diagnosed as the cause of abortion in at least 12 ewes in a flock of 4000. Microscopically, lesions were most conspicuous in foetal lung and brain.

Other domestic species: Examination of an aborted cria from an alpaca herd ruled out an infectious cause but revealed histological congenital goitre. The thyroid/bodyweight ratio (0.75 g/kg) was high compared to that in related species (0.2 - 0.4).

Abortion of a foal was associated with a sub-acute diffuse placentitis from which the bacterium *Oerskovia xanthineolytica* was isolated. There is a report in the literature of this organism causing abortion in a mare in SE Queensland in 1982 (See Thomas RJ & Gibson JA (1982), *Aust. vet. J.* 58, 166-167).

In a severe outbreak of Fowl Cholera in partridges, a total of 90 (of 2000) plump birds died suddenly. *Pasteurella multocida* was isolated from a number of birds which all had remarkably similar multiple haemorrhages.

Wildlife: Examination of several Tasmanian Devils (*Sarcophilus harrisii*) with extensive multiple tumors, especially of the face around the teeth and gums, revealed multicentric sarcoma. Immunohistochemical and ultrastructural characterisation of this tumour is being done. The condition appears to be an important cause of illness and death in this species. Booth (Proc.233, Postgrad. Fdn, Univ. of Syd, 1994, p.426) states that dasyurids, especially Tasmanian Devils "... appear to be susceptible to a wide variety of hyperplasias and neoplasias..." (See also Griner LA (1979) Neoplasms in Tasmanian Devils. J. Natl Cancer Inst. 62, 589-595).

Microscopic examination of tissues from a Little Penguin (*Eudyptula minor*) that died after 11 weeks with a wildlife carer and was about to be released, revealed

marked squamous metaplasia of peri-ocular glands; avitaminosis A, was suspected.

Necropsy and follow-up examination of a raven (*Corvus sp.*) suspected of having been poisoned revealed massive numbers of hepatic inclusions consistent with circovirus infection.

The laboratory is collaborating with Dr Rupert Woods, the Australian Registry of Wildlife Health, and the Tasmanian Nature Conservation Branch in a study of mortality of chicks of the threatened Shy Albatross (*Thalassarche cauta*) on Albatross Is. in Bass Strait. Preliminary investigations have revealed a fibrino-haemorrhagic enteritis, probably with subsequent septicaemia, as a major problem; a range of bacteria, but especially *Staphylococcus aureus* was isolated. In addition most chicks had multiple focal pustules or ulcers on non-feathered skin. Histological changes consistent with poxvirus infection were seen in one chick only but it was felt that had examinations been conducted earlier more proliferative pox lesions would have been seen.

An interesting finding in one chick was extensive infiltration of skeletal muscle of the neck with lymphosarcoma-like cells in which mitoses were common.

The study confirmed the presence of a range of ecto- and endo-parasites which are currently being identified, and virological/EM studies at AAHL on (ground) ectoparasites from (both sick and apparently healthy) chicks have revealed the presence of what appears to be a Bunyavirus. Serological studies at AAHL revealed that all chicks were negative for Avian Influenza and Infectious Bursal Disease. One, of 37 chicks, however, gave a titre of >80 in the Haemagglutination Inhibition test for NDV; all other birds were negative.

VICTORIA – Malcolm Lancaster

Abalone mortalities .
Mark Williamson Victorian Institute of Animal Science Attwood, Victoria

Mortalities in abalone were reported from a farm in south west Victoria in January, which were attributed to an unclassified *Vibrio spp*. Mortalities were found in 4 of 90 land-based marine tanks, three tanks contained black-lipped and the other, green-lipped abalone. Mortalities in each tank varied from 2 to 5% in tanks containing approximately 15,000 abalone. Tanks on the farm contained 1, 2 or 3 year old abalone, however, affected tanks contained only 2-year-old abalone. Histologically there was necrosis and suppurative inflammatory lesions in the foot of each abalone and a *Vibrio sp.* was cultured from these lesions in both of the abalone species. The Vibrio species isolated was most closely related to *Vibrio vulnificus* biotype 2. Samples have been submitted to the Department of Fisheries, Tasmania for further classification. This *Vibrio sp.* differs from other species that have caused mortalities in abalone in Victoria, namely *V. harveyi, V. parahaemolyticus* and *V. alginolyticus*. Antibiotic sensitivity testing showed the bacteria to be sensitive to tetracycline and mortalities decreased dramatically within 4 days following antibiotic administration.

WA – David Forshaw

Western Australian Department of Agriculture
Animal Health Laboratories; South Perth and Albany.

Bovine - Allergic dermatitis?(Marc Kabay).

In a repeat episode of a vesicular disease alert at Busselton at the same time as last year, multiple lesions (dozens of evenly spaced 1-2 cm diameter papules) developed on the udder skin of 3/400 dairy cows. Histologically there was multiple subcorneal pustule formation containing neutrophils and eosinophils and occasionally complete necrosis of the epithelium. There was also a moderate to heavy infiltrate of neutrophils and eosinophils in the underlying dermis. Epithelial lesions extended into hair follicles. Dermal blood vessels appeared normal. Distribution and pathology suggested a contact allergen but none has been identified so far.

Bovine - Microphthalmia associated with Mucosal Disease Virus infection(Marc Kabay).

Congenital eye deformities were found in a single Charolais calf. Histopathological examination revealed poorly myelinated optic nerve with retinal dysplasia and failure to form posterior chamber. No significant lesions were found in the the brain.

Embryonic losses, coincident with an outbreak of mucosal disease were recorded in this 100 cow herd in 2001. In this year's case, calving had just started. The calf was found to be antibody +ve and antigen -ve. Although this is reported as a common sequel to *in utero* infection with pestivirus we rarely see it and cerebellar hypoplasia is far more common.

Canine - Neospora dermatitis(Cleve Main)

The subject was a two year old female dog with a skin nodule. Histologically there was a large circumscribed sub-epidermal granuloma with masses of 4-6 x 1.0 micron basophilic organisms within macrophages. Most were contained within small cysts approx 50 microns long. The cyst wall was difficult to discern. *Neospora sp.* was cultured from the lesion.

Bovine - Perennial Ryegrass Staggers(David Forshaw)

During the 2001-2002 summer season, there were numerous reports of staggers in the southern region, far more than usually encountered. In one case, there was 100% morbidity with 9/80 calves dead. After moving to a bare paddock and hand feeding, the animals were still affected 3 weeks later. Usually signs of ryegrass staggers abate much sooner. Ryegrass samples from one property sent to Department of Natural Resources and Environment, Hamilton, Victoria, contained high levels of lolitrem B. The syndrome is associated with seasonal conditions - early summer rain allowing regrowth of ryegrass followed by a prolonged drought. There have been reports of large numbers of animals affected in Victoria and NSW this year as well.

Bovine - "Sprouted barley deaths" (Jeremy Allen, John Creeper, Russell Graydon)

Weight loss and 50 deaths in late pregnant and recently calved heifers were reported at Merredin. The animals were fed sprouted barley grown intensively in an hydroponic arrangement. The cause of death not established. Blood chemistry on a recumbent animal was unhelpful, with no elevation of liver enzymes evident. Clinical chemistry on about 30 animals covering (all classes of stock) revealed that at least a third showed evidence of severe liver damage. Unfortunately no animal necropsied showed any histopathological evidence of liver pathology. Clinically there is a short illness manifest as apparent sore hind feet, to hindquarter stagger, to collapse and death. Farmers believe that there was a slight difference in the clinical signs between the Murray Grey and the Santa Gertrudis cows. The Murray Greys showed a period a significant weight loss before actually dying. They think that the different clinical signs may be related to how much of the fodder was being eaten. Alpacas fed fodder at lower rate started to lose condition like the Murray Grey cows so the farmer stopped feeding the fodder. They all improved.

Aspergillus spp were cultured from the sprouted grain samples submitted. On the basis of this, and with the clinical chemistry, the private practitioner advised the owner that the problem was a mycotoxicosis.

Both farmers claimed to have seen a bluey fungus with a patchy uneven distribution on the sprouted grain for the first time this year. They say they also saw a white material on the green sprouts which they assumed was spider web material (fungal hyphae??).

The clinical chemistry indicated active liver damage progressing (very high GLDH) 3-4 weeks after the sprouted barley factory material had been removed. This would not support the diagnosis of mycotoxicosis resulting from the sprouted barley as it was removed from the ration immediately the illness was seen. However the farmers believe that deaths were due due to the sprouted barley as the animals started to improve when it was removed from the ration. The animals that were eventually put back on all the same feed except for the sprouted barley, all thrived.

There may have been some contaminant introduced to the systems this year, and we have missed it. There was a lot of *Pyrenophora semeniperda* on barley grain in WA this year. If this fungus got into a liquid culture, it could certainly have produced mycotoxins capable of producing the disease as reported.

Information was received that a similar problem was occurring in South Africa but this could not be confirmed by contacts in S. Africa. Many farmers in S. Africa have a similar system where they feed sprouted maize. They have had cases where the maize becomes infected with *Aspergillus clavatus* and causes high mortalities with a tremorgenic syndrome with cardiac,skeletal muscle, brain and spinal cord lesions. The fungus is quite obvious growing over the grain and sprouts. *Aspergillus clavatus* has also been reported elsewhere to infect sprouted wheat and sprouted barley and to cause the same problem. Although we cultured two *Aspergillus spp.* from the supsect material the clinical signs were not the same as those reported in S.Africa.

Ovine - Chlamydial abortions? (Mark Kabay)

Abortions in (360/600) two year old merino ewes were reported after the farmer noticed several lambs in placenta (some fresh and some mummified) on the ground and 60% of flock with a bloody vaginal discharge. Later, the number of ewes reported with a discharge reduced to 16/90. One lamb was born alive, stood, but died during the day. Placenta taken from that ewe and lamb were submitted for necropsy together with bloods and vaginal swabs from several ewes. 9/11 ewe blood samples positive for chlamydia. Histopathological examination of lamb tissue was not very revealing - some focal necrosis with calcification in the brain but no intralesional organisms seen.

Nothing cultured and all other tests (leptospira, brucella, listeria, salmonella etc.) negative.

Ovine vasculitis - Aetiology unknown(David Forshaw)

A veterinary practioner reported 50/1000 weaner sheep dead with history of lethargy and ill thrift. Only one animal examined initially. From his necropsy, the practitioner described consolidation of 75% of lung volume and a large area of haemorrhage in the meninges over the cerebral cortex. Histologically the lung had severe diffuse interstitial pneumonia with smooth muscle hyperplasia, some alveolar macrophage formation and increased cellularity of the alveolar wall. There was also severe non-suppurative vasculitis of the renal arcuate vessels. The cellular response was mostly lymphocytes and plasma cells and mostly perivascular. There was a similar but milder vasculitis in mesenteric vessels and a very mild lymphoid vasculitis in the meninges associated with massive subarachnoid haemorrhage. CAE ELISA (Maedi-Visna check) negative and Pestivirus PACE negative. The lesions are similar to Malignant Catarrhal Fever lesions in cattle. Brain and lung samples were PCR positive for MCF virus but the significance of this is not clear. Subsequent tests on blood samples collected from other animals in the flock as well as samples taken from flocks in other areas inidicate that in company with sheep producing countries in other parts of the world, Ovine Herpes Virus-2 is ubiquitous in Western Australia.

Ovine abortions and encephalitis - *Listeria ivanovii*(David Forshaw)

Pregnant cross bred ewes due to lamb in 4-6 weeks. Fed silage two weeks prior. Approx 12 off colour with putrid vulval discharge. Heavy growth of *Listeria ivanovii* from swab of one. Then lost 50/350 ewes with neurological signs - blindness, running into fences, head tilt, recumbency, torticollis, death. Similar lesions in three necropsied. Severe meningoencephalitis centred on the cranial medulla with marked mononuclear perivascular cuffing, microabscess formation and gliosis. *L.ivanovii* isolated from brain swabs of all three. *L.ivanovii* is not a common cause of encephalitis (has this been reported in sheep?). It is also unusual to see abortions and encephalitis in the same listeriosis outbreak. Culture of silage bales initially revealed *L.ivanovii* in 5/7 bales sampled. Later sampling revealed *L.ivanovii* in only 2/10 bales.

Scrub Wallaby - Calcinosis Circumscripta(Russell Graydon)

Chronic proliferative lesions on plantar aspect of feet between toes. Have been surgically removed but recurred. Fibrous capsule surrounding zone of calcinosis within which there are occasional bony spicules.

Ovine - Mycotic meningoencephalitis(John Creeper)

Three to four month old lambs with neurological signs of opisthotonus, paddling and nystagmus over 2-3 days. One dead. Histologically there was a severe cavitating meningoencephalitis with intra-lesional fungal hypahe affecting the cerebellum. Vasculitis was also present in other parts of the brain. Fungal stains demonstrated large numbers of fungal hyphae morphologically resembling Aspergillus sp. In our experience this is quite a rare lesion in sheep in WA. Lesions in cattle are usually associated with Mucor sp or Rhizopus sp and secondary to rumenitis or abomasitis from any cause eg. grain overload.

Bovine - Photosensitisation associated with "Rough Dogs Tail" (Jeremy Allen, Russell Graydon)

A syndrome of photosensitization caused thirteen deaths and affected 30 of 130 Friesian cows grazing in a particular paddock. Two months earlier cows in this paddock had also had signs of photosensitization. The suspect paddock was grazed for the second time for one week and on the first evening, the whole herd was lethargic and depressed and there was a drop in milk production. The next day, 8 cows died overnight and several were sick. There were 5 more deaths the following day and 17 others had signs of severe photosensitization. One cow was euthanased. Skin lesions consisted of full thickness necrosis consistent with photosensitisation. Liver sections showed acute necrosis of scattered individual and small groups of hepatocytes, with no distinct zonal pattern. Some liver enzymes were raised [GGT 128 U/L (ref 0-35), GLDH 18.6 U/L (ref 0-40), bilirubin 79 umol/l (ref 0-10) with direct 20]. Rough Dogstail (*Cynosurus echinatus*) was found to be a prominent pasture species in the affected paddock. This may represent the first WA case of "Acute Bovine Liver Disease" as reported in Victoria, South Australia and Tasmania, although the liver lesion is not as severe, nor is the clinical chemistry completely consistent with descriptions of cases in the Eastern States. In the current case the skin and liver lesions seemed to be out of synchrony for a secondary photosensitisation. The skin lesions were of greater age than the liver lesions. However, some Eastern States descriptions seem to indicate this occurs. One case has been reported in sheep in Tasmania. A *Drechslera sp.* was cultured from a sample of the Rough Dogstail collected from the affected pasture, but nothing was cultured from pasture in the adjacent paddock (predominantly ryegrass). A *Drechslera sp.* has been isolated from Rough Dogstail in paddocks associated with Acute Bovine Liver Disease in Victoria, and at least one *Drechslera sp.* is reporded to produce hepatotoxins.

Barramundi - Septicaemia / Neoplasia (Brian Jones)

Barramundi juveniles out of quarantine for 1 month, 10-12 mortalities per day. Fish are feeding well but many are pale with swollen bellies and haemorrhage in front of the vent. Several bacteria were isolated, including a heavy growth of *Aeromonas hydrophila*. Sherwanella algae and *Proteus vulgaris* were also isolated from the ascitic fluid of the 2 fish that were tested. An incidental (probably) finding was tumour masses in the kidney and pancreas with tumour cells also visible in subendothelium in other organs. Morphology suggests an endocrine tumour (possible pancreatic islet cell).

Comments: Septicaemia was diagnosed but the significance of the neoplasia is unknown.

Barred Grunter - Hepatic protozoa?(Brian Jones)

Barred grunter (carp) ex Perth Zoo with severe hepatitis associated with an unidentified microsporidian or rickettsial parasite. The organism was present in large numbers in hepatocytes, however it has not been seen previously by us and has not been identified. This large organism (to 50 microns) looks to be budding in some places and to contain zoites in others. Thought to be likely a secondary pathogen taking opportunistic advantage of a fish weakened by age.

Murdoch University

Presumptive Cytomegalovirus Encephalitis in a Pig
Mandy O'Hara Senior Pathology Registrar
Division of Veterinary and Biomedical Science Murdoch University
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An 8-week-old male weaner/grower pig in poor body condition (5.28kg) was submitted for necropsy with a history of head tilt, wasting, poor growth, lethargy and death. Post mortem examination of the pig found a moderate, subacute, suppurative left otitis externa and media and left sided suppurative rhinitis. There was also a mild, acute, bilateral bronchopneumonia and severe, subacute pustular dermatitis affecting the ventral abdomen. The cause of death was not definitively determined at necropsy.

Bacteriology cultured a heavy growth of *Actinobacillus pleuropneumoniae* from the middle ear and heavy growth of coagulase positive Staphylococcus from the skin. Histological examination of lungs, skin and middle ear confirmed the gross findings. Sections through the nasal turbinates identified a severe, subacute, diffuse, suppurative rhinitis with numerous, large, basophilic intranuclear inclusions within mucosal tubuloalveolar glands (presumptive Cytomegalovirus). Similar inclusions were also identified within nuclei of scattered renal tubular epithelial cells.

Examination of sequential transverse sections through the brain identified a mild to moderate, subacute, necrotising inflammatory reaction of greatest severity around the cerebellar peduncles. At the junction of the cerebellar peduncles, medulla and cerebellum there was a mild to moderate, multifocal, perivascular and interstitial infiltrate of lymphocytes and plasma cells accompanied by rare neutrophils. Associated with the infiltrate was a unilateral, focally extensive area of malacia and early gliosis in one cerebellar peduncle. There was mild gliosis within the cerebellar white matter and scattered acutely necrotic neurons and swollen axons within the cerebellar molecular and granular layer. The pons demonstrated mild to moderate, diffuse, acute axonal necrosis and malacia of the white matter tracts. Throughout the leptomeninges was a mild, multifocal infiltrate of lymphocytes and plasma cells, which extended into the underlying cerebral cortex associated with mild gliosis. Throughout the cerebral cortical white matter were multifocal areas of degeneration, necrosis and mild gliosis.

The pigs death was attributed to the meningo-encephalitis, the cause of which has not been definitively determined, although a systemic infection with Cytomegalovirus (CMV) is suspected. Cytomegalovirus usually causes an asymptomatic/low morbidity infection in sucker pigs, however death may occur, usually during the systemic phase of infection. Reports of histological changes in the nervous system of pigs infected with Cytomegalovirus are rare, although there is mention of focal gliosis and inclusions in glia within the central nervous system of systemically infected pigs in Dungworth's section on inclusion body

rhinitis in Volume 2 of Pathology of Domestic Animals 4th Edition. We are currently trying to develop an immunoperoxidase stain to determine if the lesions in the nervous system are associated with demonstrable CMV infection. Most causes of non-suppurative encephalitis in pigs are exotic, however the nature and distribution of lesions within the CNS and other organs makes diseases such as Aujesky's disease, Rabies, enterovirus, hog cholera, coronavirus, and the various paramyxoviruses unlikely.