AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

VETERINARY PATHOLOGY REPORT

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Management Committee Report

Welcome to the 70th Veterinary Pathology Report.

The Cairns AGM:

The ASVP Annual General Meeting was held on June 25 – 26th in Cairns at the RSL Club on the Esplanade.

Again, the value of holding our AGM as a satellite to a larger associated congress was proven by the good attendance for a relatively remote location, and by the presence of three impressive guest speakers who came to Cairns to attend the Wildlife Disease Association International Conference, which immediately followed our meeting. Bruce Rideout gave us three excellent talks, dealing with broad issues in zoo health as well as showing us that egg pathology can be as intellectually demanding and rewarding as any other

specialty. Ian Barker gave us an up-to-date view of West Nile virus diagnosis, politics and management, which was all the more valuable given the close relationship between this virus and some of our local arboviruses. And Elliott Jacobson told us about some ailments in loggerhead turtles and was again able to take a broad view of the politics that become involved in investigations of this sort. Members are referred to the relevant comprehensive abstracts in the Proceedings book, which has been mailed out to all members who did not attend (please notify your secretary roger.kelly@uq.edu.au if you have not received your copy).

Then our delegates rose to the occasion and presented the usual fascinating smorgasbord of cases, and on Saturday our President ran the business meeting with such efficiency that we were able to head for refreshments in what must have been record time. The annual dinner was associated with minimal morbidity, and we are grateful for Les Sims' efficient local arrangements.

The Australian Animal Pathology Standards Program (AAPSP)
Jim Taylor of the Toowoomba QDPI lab is running the Quality Assurance program with about 20 participating laboratories, using a mixture of real sections and digital images. The limitations of the latter are recognised, but a trial of whole-slide scanning is planned.

The AAPSP management committee (Mike Bond, Tony Ross, Keith Walker, Barry Richards and Roger Kelly) meet by teleconference. The committee is working on the design of an AAPSP page on the Animal Health Australia website which will facilitate access of AAPSP subscribers to quality assurance and continuing professional development exercises. It will also carry digital copies of all issues of the ASVP Veterinary Pathology Report (from the first 1983 issue): these have been scanned and will be set up on the site so that members can search all reports on keywords, authors' names, etc. It is also hoped to include archived copies of the text of all Slide-of-the-Month exercises. Another aim is to allow subscribers to the Program to place diagnostic exercises, interesting cases, etc, on this website using templates that should make it easy to share this valuable information.

Details, including legal issues, for the Second Opinion Service are still being worked out, but it was heartening to receive offers from 28 members to act as second opinion providers for this scheme.

Longer-term projects include re-staining and scanning of the Registry histo collection to produce a resource that will be more accessible to trainees and others.

ASVP Website
It is apparent that the AAPSP webpage on the AHA site will overlap with content already on or proposed for the ASVP website, but the two should be complimentary, and there will be numerous links between the two sites. Having the AAPSP page managed by IT professionals in AHA will lessen the strain on the ASVP webmasters.

A contract is about to be signed with a Brisbane web maintenance company who have provided a detailed quote for upgrading the ASVP pages and (more importantly) making them easier for ASVP webmasters to update.

Membership Payments
For those of you who have not yet paid your annual subs, the committee encourages you to try using internet banking with electronic funds transfer ("Pay anyone" option). About a dozen members have successfully paid their subs using this method. You will need the following information:
BSB Number: 105-068
Account number: 040843440.
and remember to ensure your name is entered with your payment. Let the Treasurer anita.gordon@dpi.qld.gov.au know you have made an EFT payment, and you will be issued with an

electronic receipt once the transaction appears on the ASVP statement. Old-fashioned payments are also welcome, of course.

Management Committee Changeover for 2006
The pointer has been fairly spun, and now points directly at New South Wales as the State to host the next Management Committee. Members (particularly NSW members) are reminded that the present committee MUST stand aside at the next AGM.

AGM 2006
The location of next year's AGM has been decided: it will immediately precede the AVA meeting in Hobart in May, and for the first time will be held over 3 days (Friday 19-Sat.20-Sunday 21). This is because we hope to be able to feature another speaker from the US, the fare to be supported by the CL Davis Foundation. The extra day in our program will allow us to make better use of such a speaker, since we have had pretty full programs at the last two conferences.

A final decision has to be made to choose between Launceston and Hobart: factors include availability of local arrangements organisers, etc.

Roger Kelly (honorary secretary)
for the **Management Committee** Sept. 19, 2005

DRAFT MINUTES OF ASVP ANNUAL GENERAL MEETING, 2005

held Saturday June 25th, 2005, at the RSL Club, The Esplanade, Cairns

The meeting was opened by the President at 1556hrs, who thanked the overseas speakers, then welcomed members and exhorted them to give the committee more feedback in matters of concern to the Society.

ATTENDANCE: 40 members
Angela Begg, Jemma Bergfeld, Shane Besier, Michael Bond, John Boulton, Grant Campbell, Rod Campbell, Mark Carrigan, John Finnie, John Glastonbury, Anita Gordon, Damien Higgins, Celia Hooper, Roger Kelly, Allan Kessell, Philip Ladds, Richmond Loh, Philippa McLaren, Bronwyn Murdoch, Richard Miller, Judith Nimmo, Mandy O'Hara, Peter Phillips, Richard Ploeg, Stephen Pyecroft, Barry Richards, Tony Ross, Janeen Samuel, Jenni Scott, Cathy Shilton, Les Sims, Neill Sullivan, Jim Taylor, Christine Trezise, Shirley Turner, Keith Walker, Pam Whitely, Ian Wilkie, Samantha Wong, Stephen Yeomans

APOLOGIES
Received from Doughty, Sutton, Huxtable, Nunn, Storie, Main, Mackie, Shinwari, Reece, Bastianello, Williamson, Bolton , Phil Summers and Brett Stone

MINUTES OF THE 2004 MEETING
K. Walker pointed out that the words "non-veterinary" in line 2 of Correspondence item (i) were not a true representation of what he has said, and it was agreed to delete these.
The amended minutes were accepted (moved Carrigan, seconded Gordon, carried *nem con*)

BUSINESS ARISING FROM THE MINUTES
(i) Progress toward National Registration (dealt with under Correspondence (i), below)

(ii) Formation of the Australian Association of Veterinary Laboratory Diagnosticians (AAVLD) has progressed and the notice of the inaugural meeting of this group has been circulated to ASVP members *via* the mailing list. The meeting will be held on September 22nd-23rd, 2005 at the PIRVIC facility, Attwood, Victoria, and further details can be got from Peter Kirkland peter.kirkland@agric.nsw.gov.au

Discussion followed on the desirability of holding joint ASVP-AAVLD meetings, with the Friday before the traditional ASVP meeting being used as a common day for activities involving both groups. P. McLaren passed on a message of support from Melbourne University (Jenny Charles) that the ASVP should consider joint meetings with the AAVLD. R. Miller again exhorted members to give feedback on this issue.

CORRESPONDENCE:

(i) **To the AVBC:**

As directed by a motion passed at the 2004 meeting, the secretary sent a letter to the Australian Veterinary Boards Council strongly expressing the Society's dissatisfaction at the anomalous registration status of veterinary specialists including pathologists who make diagnoses on material derived from different States.

(i) **From the AVBC:**

In response to (i) above, an email reply was received from the Council on Feb.9th, 2005, to the effect that an official letter of response to the ASVP complaint had not yet been prepared. This message included a rough transcript of the discussion that was stimulated in the AVBC meeting by this matter, and the message included a request that the transcript be not circulated before the official response from the AVBC was received. R. Kelly then attempted to give members a sense of the AVBC discussion as conveyed in the email message: this was to the effect that even if national registration was accepted as being a good idea (and there were some negative feelings about it among the AVBC), it would remain low on the legislative priorities of the ministers concerned. In other words, the champions of this cause should not hold their breaths. T. Ross commented that the current situation is too expensive for organizations. He also mentioned that the president of the state veterinary councils was sympathetic, and that part-time presidents are more amenable to change than full-time registrars.

(iii) **To Peter Kirkland:**

re the formation of the AAVLD: (see under Business Arising)

(iv) **From Melbourne University Vet School:**

in support of the formation of the AAVLD

REPORTS OF OFFICE-BEARERS

The reports of the **President**, **Treasurer**, **Management Committee**, **Webmaster**, **Australian Animal Pathology Standards Program (AAPSP)** and the **Australian Registry of Wildlife Health (ARWH)** are printed in the Proceedings of this conference.

No additions were made to these reports by the relevant officers, however Damien Higgins spoke to the ARWH report on behalf of Karrie Rose. He gave an overview of the function of the ARWH, and mentioned that Dick Montali has now left, but remains in contact with K. Rose. He advertised the Australian Wildlife Health Investigation Manual, which was to be available at a reduced price of $35 at the WDA conference, and also from the website. He emphasized the features of the soon-to-be-opened ARWH website, which will include a down-loadable guide to submission of pathological specimens from wildlife cases, as well as many other valuable links, and a template to allow veterinarians to submit interesting cases to the website for dissemination (after approval by the moderator). The website of the AWHN was also to be launched.

There being no amendments moved to any of the reports, acceptance of the Treasurer's report was moved by R. Kelly and seconded by R. Ploeg. Carried *nem con*. R. Miller thanked the sponsors of the 2005 AGM (the three private veterinary laboratories, and the CL Davis Foundation).

GENERAL BUSINESS

(i) ASVP Website proposals:
R. Kelly reiterated the difficulties associated with the website; these relate to the lack of visibility of the site to search engines, the lack of user-friendly interface programs to enable the webmaster and assistant webmaster to easily update the various pages, and the resultant generally stale air about the site. He then asked for members' responses to a proposal to spend about $1700 on some professional help to clean up spurious code, raise the visibility to searches, provide a user-friendly interface program, and provide some templates for dynamic page creation.

Les Sims and others spoke in favour of the proposal. Among the additional functions outlined by members were use of the website to outline training opportunities to attract more pathology recruits, and archiving past editions of the VPR and conference proceedings. A. Gordon said that Society finances were healthy enough to stand this sort of expenditure. The meeting agreed to have the management committee proceed with the proposal, after due consultation with the Webmaster.

(ii) Affiliation with the CL Davis Foundation:
Tony Ross moved that:
"The ASVP affiliate with the CL Davis Foundation, and that the ASVP management committee proceed with acceptance of the offer of the CL Davis Foundation to (a) provide a speaker for a third day of the annual ASVP conference, and (b) to provide support for experts to give continuing education workshops in pathology throughout Australia".

The motion was seconded by I. Wilkie

In speaking to the motion, T. Ross agreed that the quality of the CE from CL Davis was variable, but pointed to the success of the speakers whom the Foundation had so far provided: Bruce Williams at the 2004 ASP conference, Fabio del Piero for the AAPSP roadshows this year, and Bruce Rideout at this year's annual conference.

Allan Kessell supported the motion by praising the quality of the CL Davis courses he attended in the US during his preparation for the AVBC exams.

Philip Ladds asked who would make decisions on which speakers were imported, and was told by T. Ross that the ASVP would.
The motion was carried (*nem con*)

(iii) Changeover of management committees:
R. Kelly reminded members of the nasty mess in 2003 caused by failure to obtain nominations for the incoming management committee. He had already approached members in the Northern Territory (which had never provided a management committee) and Tasmania (which last did so in the '80s) asking for expressions of interest. He suggested that, if both these groups declined on grounds of insufficient personnel, the committee should be provided by New South Wales, who in fact should have provided an executive before Queensland was pushed into the knocking box in 2003. T. Ross suggested that NT and Tas could share the job using modern technology.

(iv) OTHER BUSINESS:

(a) Missing histopathology proficiency testing reports: T. Ross mentioned that he has now found the 4th round of histoproficiency testing reports from 2002, which went missing in his office, and is distributing copies.

(b) Life Membership of Professor Ken Jubb: Philippa McLaren asked why Prof. Jubb was not a member of the Society, and asked if it would be appropriate, in view of his contributions to veterinary pathology, for him to be so nominated. R. Kelly undertook to ask him for his response to this proposal.

(c) Committee on veterinary diagnostic pathology laboratories: Rod Campbell, a member of this committee, gave a brief report of its activities. It had met three times, and he foreshadowed a report which would be placed on the SCAHLS website. There were still some confidential aspects, but he felt he could make three points for consideration by the Society: (1) the committee will urge laboratories to develop more collaborative strategies with each other, to form active linkages, hubs, etc): these should have very beneficial effects upon research output, and should provide opportunities for graduate training. (2) The committee was very concerned about the lack of diagnostic veterinary specialists in Australia, as well as their ageing; this deficiency extends to microbiologists as well as to veterinary pathologists. Recommendations will probably include the creation of scholarships for those wanting to specialize. (3) SCAHLS has at present no university representative; this is seen as an important deficiency.

Sims stated that overall national co-ordination is lacking; individual labs can disappear and nothing happens.

ELECTION OF OFFICE-BEARERS

Dick Sutton had indicated his intention to stand down from the management committee because he will be retiring at the end of 2005, and Ian Wilkie had agreed to be nominated in his place. Dick was thanked for his support.
There having been received no other nominations for positions on the management committee, the current committee was re-elected including I. Wilkie.

Stephen Yeomans will be spending a year in the US from August and Peter Phillips has agreed to act in his place as Veterinary Pathology Report correspondent for South Australia (non-elected position).

VENUE FOR THE 2006 MEETING

The Australian Veterinary Association AGM will be held in Hobart in May 2006. In the absence of any suggestions to the contrary, the management committee agreed to work toward arranging the 2006 ASVP annual general meeting in Hobart on the weekend preceding the AVA meeting.

The meeting was closed at 4.52 pm.

Roger Kelly
Hon. Secretary, ASVP

STATE REPORTS

WESTERN AUSTRALIA

State correspondent: David Forshaw

Renal necrosis and urolithiasis in sheep

Cleve Main

Animal Health Laboratory, South Perth

Fifteen or so 6-tooth Merino wethers died in one paddock at Bindoon. Sheep deaths have occurred in this same paddock for the past few years. The property had a history of summer urolithiasis on summer pastures. Swollen kidneys with cortical haemorrhage were evident at necropsy. The mucosa of the urinary bladder was haemorrhagic and contained necrotic foci with numerous small uroliths present in the lumen. It was noted that the urethra was patent. Histological examination of the urinary bladder revealed transmural haemorrhagic cystitis with patchy mucosal loss and necrosis of the mucosal epithelium. Small mineralised clusters were evident in these areas.

The kidney contained changes judged to be unrelated to those in the bladder. These were characterised by severe and universal acute to subacute cortical tubular epithelial necrosis with widespread and numerous proteinaceous casts present in tubular lumens. The nature of the casts varied from amorphous hyaline, granular to globular, the latter possibly due to sloughed necrotic epithelial cells. Additionally there were numerous dilated tubules, some with thin attenuated epithelium, suggesting that the injury was not peracute. Medullary tubules were mostly normal but many contained protein casts and a few contained mineralised casts. Glomeruli were unaffected. An aetiological diagnosis of probable plant-induced injury was accepted and it was also commented that, despite the quite severe damage to the bladder, there was minimal inflammatory cell response. The cause remains undetermined, but it may be a result of the concurrent urolithiasis. The case has similarities to one that was reported in July at Esperance where haemorrhagic cystitis was seen in feedlot weaner sheep with water-belly.

Gourami atypical Mycobacteriosis

Francis Stephens

Animal Health Laboratory, South Perth.

Gourami were imported from a Singapore wholesaler. The fish had red ulcers adjacent to the dorsal fin and reddened areas around the nose. Some fish had exophthalmos and raised scales. Microscopic miliary foci in liver, kidney and choroid body of the eye consisted of interstitial infiltrates of histiocytic cells. These lesions were unusual in that there were no discrete granulomas in some fish. One fish had severe multifocal necrosis of the liver and vacuolated areas that were infiltrated with eosinophilic granule cells and macrophages, which contained intracellular bacteria that were ZN positive. Culture was negative.

Barramundi myopathy

Francis Stephens

Barramundi were airfreighted overnight from Darwin 4 weeks previously. The fish never thrived and lay in a curved shape in lateral recumbency with tremors evident in the tail when stressed. Mortalities increased after a month and the fish became very thin. Microscopically, there was severe, diffuse skeletal muscle necrosis with vacuolation of muscle fibres and mineralisation. There were large numbers of nuclei present, no inflammatory reaction but some fibres had evidence of attempted regeneration. Causes of such muscle lesions include nutritional factors (Vit E/Se deficiency, cachexia), interruption of muscle innervation and exercise-induced myopathy.

Kangaroo choroiditis
David Forshaw
Albany Regional Laboratory

Blindness was noted in adult and juvenile wild western grey kangaroos on two properties at Redmond. Cases have been seen over a number of years but more were seen this year. A head from a chest-shot individual was submitted. No significant gross lesions were noted, but in the retina pigment epithelium was irregularly pigmented and showed markedly irregular morphology with individual and groups of cell protruding into the vitreous chamber. At the ora ciliaris of one eye there was a disorderly mass of fibrino-cellular material overlying the pigment layer and a single nodular protrusion of fibrovascular tissue protruded from the choroid of the other eye. In the optic nerve there was widespread axonal swelling with Wallerian change. At the optic disc there was perivascular accumulation of homogeneous eosinophilic material resembling amyloid. Similar light lymphoplasmacytic infiltrates are present through the rest of the choroid and ciliary body. The inflammatory changes are mild but consistent with Wallal virus infection or "choroid blindness" of kangaroos. We would be interested to learn of a higher than normal prevalence of blind kangaroos elsewhere.

Bovine lead poisoning – residue issues
David Forshaw

10/24 calves died on a property at Porongurup. They were either found dead or blind, depressed and mildly uncoordinated. Necropsies of five animals failed to reveal any gross changes but histopathology of one calf that had survived for a number of days with severe neurological signs revealed unequivocal polioencephalomalacia confined to the tips of gyri and superficial layers of the cortex. Kidney lead levels of dead animals were up to 733mg/kg dry weight (>25mg/kg wet weight in association with a consistent clinical picture are considered diagnostic). The farmer subsequently located an old tractor battery in the paddock, which he suspected had been broken up when some trees were bulldozed in the paddock and then probably exposed after recent heavy rain. The extremely high lead levels in the kidneys of the affected animals and the fact that one affected animal recovered following treatment with thiamine and corticosteroids raised the possibility of violative residue levels in the surviving animals. The mob has been quarantined pending testing of surviving animals. Advice from the Department of Health is that each animal will need to have a negative blood lead test before slaughter. Other references quote <0.1 ppm as background/normal levels.

Copper toxicity in calves
Cleve Main
Animal Health Laboratory, South Perth

Emaciation and chronic scours developed in 5 - 6 week old calves at Busselton, with 4 dead and 10/30 very sick. The calves had been drenched with "Paramax" and Vit B/Se 3 and 2 weeks previously. Mineral deficiency was suspected but (surprise, surprise) liver copper levels of 1302 and 1046 mg/kg were detected. The kidney copper level of one of the calves was estimated at 60 mg/kg. Histological examination of the liver of one of the calves revealed widespread random and periportal groups of hepatocytes with hyper-chromic faintly granular cytoplasm and dark shrunken nuclei. Occasional individual hepatocytes were necrotic and surrounded by small aggregates of neutrophils. The problem with this case is that that no mineral supplements (selenium excepted) were used and the source of the copper has so far eluded investigators.

More copper toxicity in calves
Cleve Main

Sudden death in 8/40, 2 to 4 month-old Angus steers at Pinjarra died suddenly after they were vaccinated, marked, drenched, given B group vitamins, selenium and Coppernate (copper glycinate 60 mg/ml). Five days after the treatments the owner noticed 4 dead steers and 1 sick one that was brought in for hospitalisation. The following day more steers were found dead. A few other young steers looked affected and wobbly but none of the adult cattle showed any signs. Histological examination of tissues from one of the calves revealed universal massive liver necrosis with widespread periacinar haemorrhage. Its liver copper concentration was 893 mg/kg and the kidney concentration 118 mg/kg. The pattern of liver necrosis is unusual and is more commonly seen in blue green algae toxicity and phosphorus poisoning (and calicivirus infections in rabbits). These calves were given 120 mg doses (twice the recommended rate) but treatment with copper has been part of the farm management procedures for several years, apparently without ill effects.

Bovine anaphylaxis & possible copper toxicity
Marc Kabay
Animal Health Laboratories, South Perth

Four of 90 five- to eight month-old Shorthorn/Brahman bulls died after receiving Coppernate (copper glycinate) injection i/m, possibly at 2 -3 times the recommended 60mg dose (Note: for heavier animals the dose is 120 mg). Several animals were diagnosed with anaphylactic shock; one of them died. **Hands up those who know about the potential for anaphylactic shock**.

Another three animals died over a 5-day period. Necropsy of one of these found red urine and jaundice. Unpreserved liver and kidney were submitted and hastily immersed into 10% buffered formal saline solution - but too late. Histologically the tissues were very autolysed, but nevertheless it was possible to discern widespread acute periacinar liver necrosis and possible cellular and granular casts in the renal tubules. The evidence we have so far suggests the animal might have died of chronic copper toxicity. Its liver copper concentration was 816 mg/kg (normal range 20 - 250) but the kidney copper concentration was only 31 (normal range 15 - 25). Injected copper in an animal can cause liver necrosis, destroy red cells resulting in elevated liver enzymes and lead to increased kidney copper concentration and haemoglobinuria. The kidney copper was not all that high in that animal. For toxicity in cattle a concentration of >60 mg/kg would be expected.

The Coppernate label warns of local tissue damage and the chance of anaphylactic shock in young bovines. It also warns that copper deficiency should be confirmed before treatment. One can only speculate on the copper status of the animals prior to injection.

Avian leucocytozoonosis AS-05-1758
Marc Kabay

A red wattlebird from a local wildlife rehabilitation centre was submitted for examination of yellowish 1mm skin nodules below the beak and around the eyes. Histopathological examination provided a few surprises, one of them, the presence of numerous intra-erythrocytic protozoal organisms consistent with *Leucocytozoon sp*. The skin lesions proved to be of two types. Those around the unfeathered peri-orbital areas proved to be classic pox lesions with focal areas of epidermal hyperplasia with numerous ballooning epidermal cells that contained large eosinophilic cytoplasmic inclusion bodies. In contrast the yellowish nodules on the neck were characterised by focal epidermal ulceration and hyperplasia, focal cellulitis and severe necrotising subcutaneous myositis with an exuberant heterophil inflammatory response.

Leucocytozoonosis is a debilitating and often fatal avian disease, especially in young birds. It is transmitted by black flies (*Simulium sp*.), which are fierce biters. Poxvirus is transmitted by contact, mechanically by mosquitoes and possibly flies as well.

Acute Liver Necrosis in Cattle – Boobialla poisoning or Acute Bovine Liver Disease?
Marc Kabay

Photosensitization and neurological signs occurred in 11/900 Holstein cows grazing both dry land and irrigated pastures at Busselton. All affected animals were febrile; one severely affected and down with "altered mental status" and hind limb paresis. That animal died overnight. At necropsy there was a congested mottled liver but no evidence of skin damage. Histological examination of the liver revealed sharply demarcated severe acute midzonal to periportal hepatic necrosis with bridging and severe replacement haemorrhage. Tests on blood samples taken from 9 clinically affected cattle revealed excessively elevated plasma GLDH (180, 870 1080 - 5300 U/L) and GGT (26, 186 -501 U/L) concentrations. Bilirubin values ranged from 5 - 75 μmol/L.

Boobialla (*Myoporum* spp.) was the prime suspect because a subsequent property inspection located a stand of Boobialla plants that had had their foliage stripped by grazing. Rough Dogstail (*Cynosurus echinatus)* infested with the fungus *Drechslera sp* was also found in pastures. The role of both plant and fungus is uncertain. Initial reports of the associated condition (acute bovine liver disease ABLD) came from the Eastern States where investigators described photosensitisation and jaundice in affected cattle. In those cases there was peracute hepatic necrosis with replacement haemorrhage initially centro-acinar (periportal) extending to become midzonal but which may include the entire acinus. In this WA case the problem disappeared when the Boobialla plants were removed.

(*editor's note: it should be borne in mind, however, that pastures in eastern States on which ABLD occurs (usually Boobialla-free) rarely remain toxic for more than a few days)*

Ovine listerial abortion
Cleve Main and Marc Kabay
Animal Health Laboratories, South Perth

Stillbirths and premature births occurred in a flock of 470 Merino ewes at Quairading. The flock was fed silage and oats up until shearing. At the time of the investigation 20 - 30 lambs have been lost. Post mortem examination of 2 lambs revealed little in the way of macroscopic lesions, but did confirm they were stillborn. Histological examination of tissues from the second lamb revealed widespread venous congestion and patchy alveolar congestion and oedema as well as numerous small clusters of gram-positive bacterial rods within alveoli and bronchioles. Similar bacteria were present in the chorioallantois and cotyledons. Bacterial culture yielded *L. ivanovii* from lung, liver and abomasal fluid. A light growth was also obtained from culture of lung of the other more autolytic lamb. This is an unusual isolate in WA, possibly because of the relatively few investigations of abortion outbreaks in sheep. It is not considered to be pathogenic to man except where that person is immunocompromised. *L ivanovii* was isolated from aborted lambs by Stan Dennis when he carried out his perinatal lamb mortality survey (pub AVJ 1975). However AHL records show that many isolates have been recovered from silage.

Encephalitis in a beached whale
John Creeper
Animal Health Laboratories, South Perth

This animal was one of about 150 false killer whales that beached themselves at Busselton. Remarkably, all but one were re-floated and escorted out to sea. Necropsy on the dead whale revealed numerous flukes in the tissue below the blubber and in the colon as well as numerous acanthocephalid worms, *Bolbosoma capitatum*, which have the squid as their intermediate host. The brain of this whale also contained lesions characterised by the presence of wide mantles of plasma lymphocytes, cells that were also present within the vessel walls and occasionally accompanied by endothelial cell necrosis. Many neurons contained a fine green brown granular pigment (ceroid lipofucsin). No eosinophils were detected suggesting the lesions were not associated with the parasites. Distemper due to morbillivirus was considered as a differential diagnosis but was not confirmed by immunohistochemistry on brain. However a viral aetiology is still suspected.

NORTHERN TERRITORY
State Correspondent: Anton Janmaat

(editor's note: The NT lab provided us with a splendid diagnostic case last month. The discussion for this case follows. If any member failed to receive the case and images, it means you have not subscribed to the ASVP mailing list. Visit the ASVP website www.asvp.asn.au to subscribe)

Discussion Of Diagnostic Exercise 10: bovine demodicosis
Cathy Shilton
DBIRD Veterinary laboratories, Berrimah NT

Disease due to *Demodex bovis* infection in cattle in Australia is usually limited to low numbers of small dermal or subcutaneous nodules, important only with respect to damage to hides used for leather. The nodules are formed when mites invade a follicle, the hair falls out and the epidermis subsequently closes over the follicle, which becomes cystic as it distends with proliferating mites (Murray 1976). The cyst may ultimately rupture, setting up a local inflammatory response. Secondary bacterial infection of affected follicles may also occur. It is thought that in most cases infection is acquired at an early age from the dam, with the disease taking months to manifest. It is apparently most common in adult housed cattle in the late winter to summer months, differing from the canine counterpart in which clinical disease is most common in young immunocompromised individuals (Radostits *et al* 2000). Lesions occur predominantly on the neck and anterior thoracic regions, with most animals developing less than 100 nodules that generally disappear after several weeks. It has been hypothesised that the distribution of lesions is related to increased sweat gland activity in the predisposed regions, with the mixture of sweat and sebum favouring mite motility (Matthes 1994).

Severe generalised demodicosis, as in the present case, is considered rare in cattle. In Africa, severity of infection was thought to be related to poor nutritional state and increased environmental temperature (Yager and Scott 1993). In a severe case reported from Kenya, the affected animal was a Friesian heifer that had been treated with glucocorticoids, had severe tick and flea infestations and was found at necropsy to have several concurrent diseases (Mbuthia *et al* 1994). At necropsy the steer in this case was found to have mild unilateral pyelonephritis from which *Arcanobacterium pyogenes* was cultured; no uroliths were noted at field necropsy. There was also generalised peripheral lymphadenopathy due to marked lymphoid hyperplasia, with large numbers of neutrophils within sinuses and a few cortical pyogranulomas with

central bacterial club colonies. Some of the dermal follicular cysts were markedly infiltrated with neutrophils and contained bacterial colonies, therefore it was assumed that the suppurative lymphadenitis was a result of secondary bacterial dermatitis. Conversely, the systemic bacterial infection raises the possibility of partial immunodeficiency. The animal was described by the field vet who performed the necropsy as being in relatively poor condition, but as can be seen in the picture of the live animal, body condition wasn't dreadful. The disease occurred over the dry season when excessive temperature and poor pasture quality could be issues that resulted in stress, but there is no obvious reason why this steer would be more affected by these husbandry factors than the rest of the herd.

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Trypanosomiasis in Sea-Caged Barramundi *Lates calcarifer* in the Northern Territory

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The disease was first diagnosed in the Northern Territory in mid-July 2005 in a single cage of 15 month-old sea-caged barramundi *Lates calcarifer*, and thereafter in other cages and age-groups. Affected fish were lethargic, tended to swim near the water surface, showed incoordination and in some cases appeared blind. Large, single necrotic ulcers of the integument up to about 5 to 6cm diameter, and uni- or bilateral exophthalmos were also described in approximately 10% of cases. Affected fish rapidly became moribund and died. The disease resulted in a mortality of approximately two percent over a period of a week in the cage initially affected. Subsequently, the disease has emerged as a chronic, on-going infection in multiple cages and age groups, with occasional sporadic peaks in mortality.

A proportion of the affected fish showed no external lesions. The large necrotic ulcers described clinically in fish from the initial outbreak extended into and exposed the underlying musculature. The surrounding integument was haemorrhagic. There was no apparent predilection site, with ulcers occurring on abdomen, tail, dorsum and posterior to the operculum. Smaller haemorrhagic erosions and ulcers of the integument up to one cm were also present, in some cases associated with scale loss. The exophthalmos was accompanied by marked intra-ocular and retro-orbital haemorrhage. Splenomegaly was a feature, with spleens up to 7 cm in length with a rubbery consistency. The blood in the majority of cases was watery and anaemic.

Histologically, the gills showed generalised telangiectasis and thrombosis of secondary lamellae with occlusion by cellular debris, together with generalised hyperplasia of primary and secondary lamellar epithelium, oedematous separation of epithelium from underlying stroma, loss of integrity of the secondary lamellar epithelium, haemorrhage and a mucoid exudate. Massive numbers of trypanosomes were present in major blood vessels, in the dilated lamellae and external to the secondary lamellae. The spleen showed congestion, oedema, erythrocyte and reticuloendothelial cell necrosis and focal

reticuloendothelial cell hyperplasia. Multifocal mononuclear cell accumulations were seen in the brain and meninges, on occasions accompanied by vascular hyperaemia and trypanosomes in vessels. In one case, occlusion of meningeal blood vessels by trypanosomes was seen. Massive numbers of trypanosomes were present in the choroid of the eye, accompanied by haemorrhage and oedema (Figure 1). Numerous trypanosomes were also present in the vascular spaces of the myocardium and epicardium, accompanied in some cases by multifocal mononuclear cell infiltrations and an intense epicardial inflammatory cell infiltration. The integumentary ulcers observed grossly were characterised by erosion and necrosis of superficial epithelial tissues, scale loss and loss of dermal tissues extending to the underlying skeletal muscle. An intense generalised inflammatory cell infiltration was present, with hyperaemia, extensive haemorrhage and superficial bacterial colonisation. Some cutaneous blood vessels were occluded by trypanosomes.

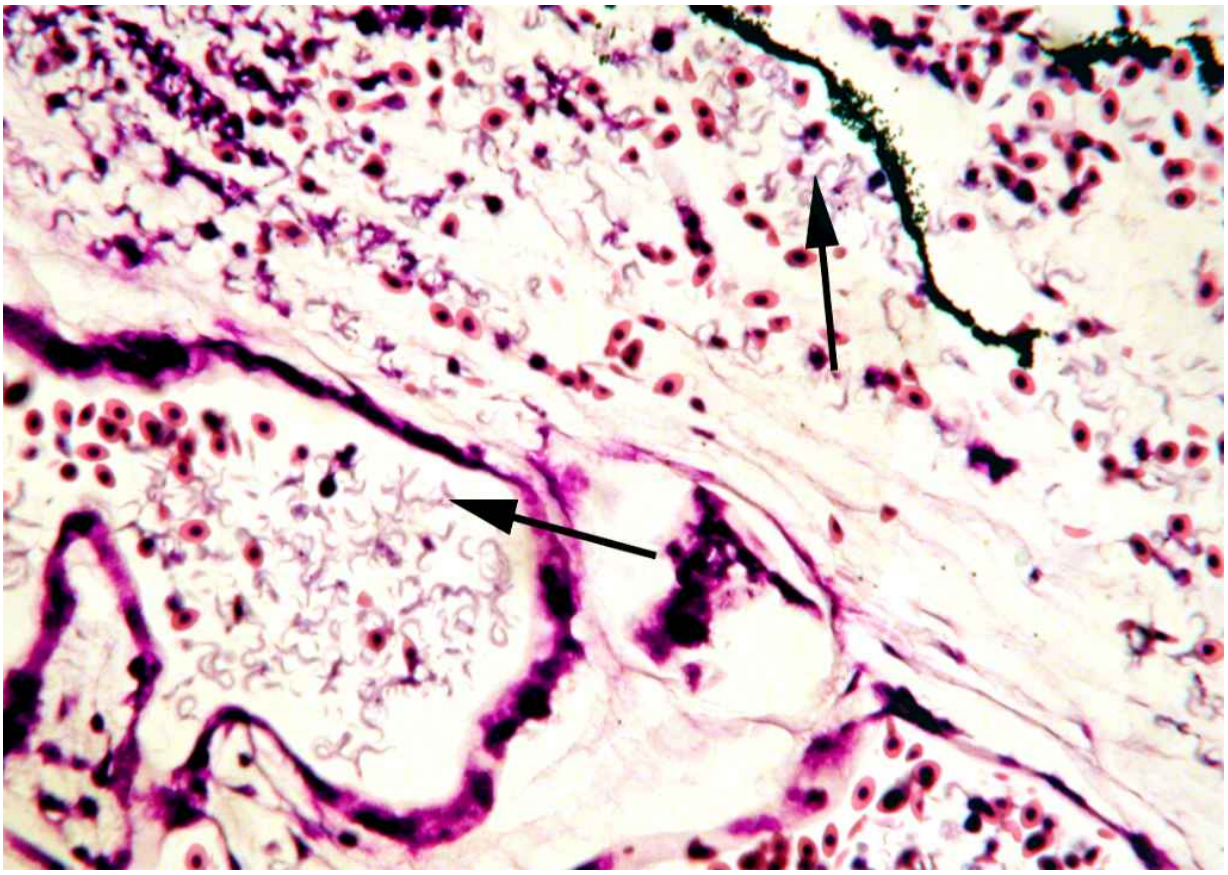

Figure 1. Intra- and extra-vascular trypanosomes (arrows) in choroid of eye. Note relative paucity of erythrocytes and loss of integrity of vascular spaces.

Bacterial culture of kidney, liver and spleen from six fish produced a light growth of *Vibrio harveyi* in two of the fish, with no growth in the remainder. *Streptococcus iniae* was not recovered from enrichment broth inoculated with these tissues. There was heavy, mixed bacterial colonisation of the integumentary ulcers in which *V. harveyi* was the predominant organism isolated. *S. iniae* was recovered on enrichment media from the ulcer of one fish.

Haematozoan parasites typical for the genus *Trypanosoma* were readily observed on microscopic examination of fresh blood smears; on Giemsa-stained blood smears of diseased fish, and on stained (Gram, Giemsa) impression smears of visceral tissues. The parasites measured 13-24 µm in total body length, showed a single flagellum attached to the body as an undulating membrane with its end extending free anteriorly, a nucleus and a single conspicuous kinetoplast. Trypanosomes were also demonstrable in low numbers in stained blood smears of clinically normal fish.

Over 190 species of *Trypanosoma* are reported in fish; however, disease associated with *Trypanosoma* sp. appears unusual. Trypanosomiasis has not been recorded previously in barramundi and has emerged as a disease of importance in their commercial production in the Northern Territory. It is considered that infection was acquired from wild fish in the local area. Although the gills of the barramundi were infected with low to moderate burdens of the haematophagous copepod *Lernanthropus latis*, microscopic examination of wet mounts of the parasite and histological examination of sections of the parasite failed to demonstrate internal trypanosomes. There is as yet no evidence of an intermediate host vector and the possibility of direct transfer cannot be excluded. Clinical signs and lesions, at least in the integument, brain, eye and gill, may in part be attributed to vascular occlusion by trypanosomes with local ischaemia, vascular necrosis and haemorrhage. Further studies are underway to define the epidemiology and pathogenesis of the disease.

QUEENSLAND

State correspondent: Brett Stone

Presumptive septicaemic melioidosis in feedlot cattle

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Haemolytic disease and sudden death involved three 2-3 year old steers on a 2000 head feedlot at Mareeba during the last week of May. The animals originated from two different properties and had been in the feedlot for almost two months. The first two steers died suddenly, whereas the course of illness (aggression, recumbency and seizures) in the third animal was about 24 hours. Field necropsies of all three revealed red urine and watery blood. The liver of the third animal was swollen and orange.

Initial laboratory investigations targeted causes of haemolysis, although no blood samples were received to confirm its presence. Babesiosis, copper poisoning, and gossypol poisoning were ruled out early in the investigation. Consistent histological changes were found in the liver of all three animals; these comprised severe acute to subacute necrosis with a multifocal to coalescing distribution, frequently producing wedge-shaped areas of necrosis involving the periacinar and midzonal parenchyma. There was marked canalicular cholestasis and fatty change of surviving hepatocytes. Other significant histological findings included multifocal necrosis and suppuration in the spleen (Animals 2 and 3), and lung (Animal 3). Occasional haemoglobin casts were present in the kidneys of all animals. No material was available for bacterial culture from Animal 1, and swabs from Animal 2 yielded mixed growth. *Burkholderia pseudomallei* was isolated in pure (or almost pure), heavy growth from kidney, liver, lung and spleen of Animal 3.

From DPI records it appears that melioidosis is diagnosed regularly throughout Qld, primarily in pigs and small ruminants, but cases in cattle are very rare (three since 1987). Forms of melioidosis recorded in Qld cattle include mastitis, pneumonia and placentitis/endometritis (Ketterer *et al*. 1975). Although acute, fulminating infection with septicaemia is recorded in many species, cattle are believed to be resistant to infection, and infections in adult animals tend to be chronic (Sprague and Neubauer 2004). It remains uncertain whether melioidosis was the primary cause of the haemolysis and death in these animals.

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Bovine mycotic nasal granuloma

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A cow from southeast Qld (age and breed unknown) was noted at the meatworks to have dyspnoea, nasal discharge and pedunculated masses on the nasal mucosa. Sections of submitted tissue revealed pronounced nodular and diffuse inflammation of the nasal mucosa and submucosa, accompanied by moderate epithelial hyperplasia. Eosinophils were predominant, with lesser numbers of multinucleate giant cells, macrophages and plasma cells. Fungal chlamydospores were observed within granulomas, singly and in small groups. They were round with refractile walls, which stained strongly with GMS, and faintly with PAS. Occasional budding forms were observed. A pure growth of *Bipolaris* sp., a dematiaceous fungus related to *Drechslera*, was obtained from culture of fresh tissue.

The changes described in this case are similar to those recorded by McKenzie and Connole (1977), except that there was focal destruction of nasal cartilage in the present case.

Reference:

McKenzie RA and Connole MD (1977). Mycotic nasal granuloma in cattle. *Aust. Vet. J.* **53:** 268-270

Candidiasis in a dairy calf

Anita Gordon

Scours and death affected 2-4 week old Friesian calves from a dairy on the Atherton Tablelands in June. Fixed tissues and faeces were received from one calf that had been necropsied after a two-week history of bloody scours, which were unresponsive to empirical therapy, including antibiotics.

Fixed samples of the forestomachs showed pronounced grey-white discolouration and diffuse mucosal thickening. Sections revealed severe, acute, diffuse inflammation of the forestomach mucosa, accompanied by marked epithelial hyperplasia. Fungal elements (branching, septate hyphae, pseudohyphae and budding yeasts) were present within the depths of the epithelium.

Although unable to be confirmed by culture, findings were consistent with candidiasis, probably secondary to inanition and antibiotic therapy. Coccidiosis was suspected to be the cause of the scours because *Eimeria zuernii* oocysts were detected in a scouring herd-mate.

Presumptive organophosphate poisoning in cattle

Anita Gordon

Sixteen of 140 Brahman-cross heifers died on a property in central Qld in August. There was history of access to an old dip and drain pen, and clinically affected animals were observed to be weak, drooling, shaking and slightly ataxic. Laboratory findings included lowered plasma cholinesterase (95 U/l; NR 150-400U/l), and probably significant quantities of the OP compounds bromophos ethyl (14ppm and 6ppm in rumen and small intestinal contents, respectively) and ethion (15ppm and 42ppm in rumen and small intestinal contents, respectively).

The owner was advised not to sell surviving cattle to slaughter for one month, and follow-up muscle biopsies were planned to ensure that there were no violations of residue limits.

Centrifugal metastasis in a feline oral squamous cell carcinoma
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A 15 year-old neutered male domestic shorthair cat developed inflammation and swelling of 3 digits on 3 different feet, at which the animal had been chewing aggressively for the past 2 weeks. The swellings were centered on the nail-bases but extended down into the pad. There was no real response to treatment and a toe was removed surgically for histological diagnosis. At this time (presumably during anaesthetic administration) an ulcer was found in the mouth but was not sampled. The submitted digit was extensively infiltrated by poorly differentiated squamous cell carcinoma. The malignancy did not appear to be arising from the overlying skin, so the client was advised that this might be an example of multiple pedal metastatic SCC such has been described by Pollack *et al* in a cat. The cat was killed and the other digits and the oral ulcer sampled. All the submitted digits were infiltrated by the same sort of squamous cell carcinoma, as was the base of the oral ulcer.

Centrifugal metastasis of carcinoma to distal limbs in such a selective manner is unusual and its mechanism is not understood. There has also been a similar pattern of metastasis observed from mammary malignancy in a cat (J. Nimmo *pers comm*). There is the possibility that primary multicentric nailbed malignancy spreads centripetally to other more central sites, but this seems even more improbable. The condition quite effectively mimics inflammatory nail-bed disease.

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U.S.A.
Correspondent: Paco Uzal

West Nile Virus in California
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The first confirmed case of West Nile Virus (WNV) infection in the US was diagnosed in birds of the State of New York in 1999. Since then, the virus has been spreading westward state by state and California has been awaiting its arrival.

Following the initial detection of WNV in New York State, sentinel chickens and mosquito pools strategically located across California were periodically tested to monitor the appearance of the virus in the state. After a couple of false alarms, the first mosquito pool tested positive in the spring of 2003. The first Californian equine case was diagnosed at the California Animal Health and Food Safety Laboratory in San Bernardino in October 2003. This horse was presented for post-mortem examination after a short period of incoordination, lethargy, ataxia, flaccid paralysis of lips and muscle fasciculation.

Since then, the infection has been diagnosed in multiple avian and mammalian species, including humans, throughout California. During the period October 2003 to January 2005, WNV infection was diagnosed in 540 horses, 3,232 birds and 732 humans (including 27 human fatalities).

At the San Bernardino laboratory, WNV has been incorporated into the list of differential diagnosis for neurological diseases in horses. This list now includes: rabies, equine viral encephalitides, rhinopneumonitis, botulism, equine protozoal myelitis and WNV. The diagnostic workup for WNV includes PCR and virus isolation from pooled CNS (medulla and lumbar spinal cord). Serology (IgM) of CSF and serum (when available) is also run. However, we have found that a strategy that provides a fast

presumptive diagnosis with acceptable accuracy is to process overnight sections of medulla and lumbar cord for histology. Although occasionally we have seen cases of WNV infection without histological CNS changes, this seems to be unusual, and in most cases the histology of these two areas provides a diagnosis of lymphocytic myelitis, which, although not unique to WNV, is highly suggestive of the infection. Immunohistochemistry, although very sensitive for the diagnosis of WNV infection in birds, has a low sensitivity in equine tissues, probably due to the short viremia and the low viral titers in this species.

HONG KONG SAR
correspondent Brad Chadwick

A Hairy Hong Kong case

Brad Chadwick and Roger Kelly
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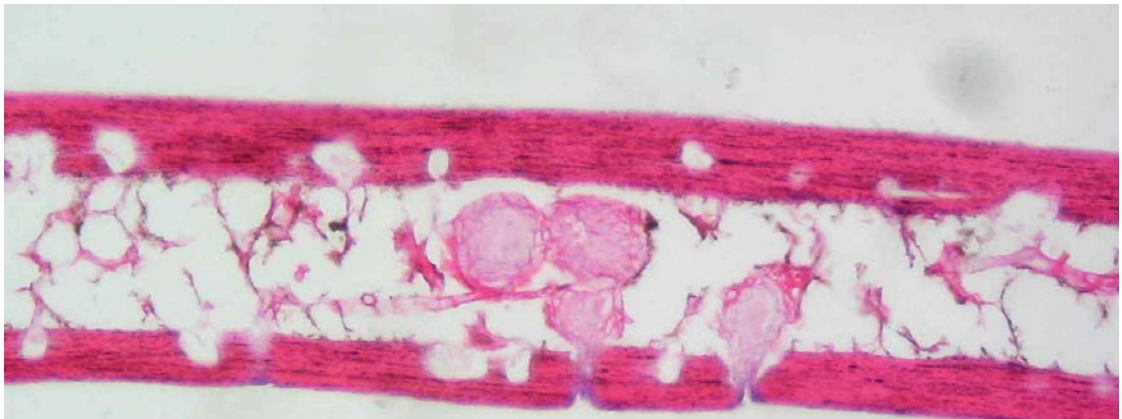
Extensive swimming exercise is an important part of the routine for thoroughbreds in training in Hong Kong, particularly in hot weather. A number of horses lost varying amounts of tail hair without showing other abnormalities. The hair shafts began disintegrating almost up to the skin surface, leaving only a stumpy tail with hairs about 2cm long, while the skin itself seemed not to be affected in any way. Simple microscopic examination of the whole hairs (after KOH digestion) showed some evidence of fungal elements within the hairs, so hairs were processed through paraffin and sectioned longitudinally and stained with H&E in the usual way. Hair shafts were spectacularly riddled with fungal elements, which seemed to be easily able to punch their way through the solid keratin, presumably by using potent keratinolytic enzymes.

The horse swimming pool used in Hong Kong is chlorinated, but it gets heavily used and soiled, so it is assumed that this is the vector for an interesting saprophytic fungus. Attempts are being made to isolate and identify it.



LS of fungus-infected equine tail hair; H&E x 400

(no reports from disease-free Tasmania, South Australia, NSW or Victoria)