AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

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VETERINARY PATHOLOGY REPORT
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INCOMING PRESIDENT'S REPORT

The year 2000 has started off with a number of changes for Australia. We have seen the predictions of disaster due to the Y2K bug proved wrong. The Sydney Olympics has received a lot of publicity, both good and bad. The GST has also arrived with much less pain than anticipated in most quarters. Another change this time affecting the ASVP directly has been the move of the Executive from Western Australia to South Australia. Your new Executive is now getting organised, and looking forward to working on your behalf for the next three years.

Some of the issues which arose during the recent conference in Perth, and which we will need to consider during the next year, include the future of the Slide of the Month and the National Registry, which will need continued support. Tony Ross has kindly agreed to be involved in the slide circulation and the Registry, and there has been discussion in committees such as SCAHLS regarding funding for this type of work. Proficiency testing of pathologists has also been an issue for some time and continues to be debated. At this stage there has been general agreement that these are worthwhile endeavours, but as yet the prospect of funding from government sources is uncertain.

Ruth Reuter

Proposed future conference dates

2001	**Melbourne**	**May 12-13**
2002	**Adelaide**	**May 4-5**

OUTGOING PRESIDENT' REPORT

At the AGM last year I was voted in as President to enable the Western Australian members to continue their term as the Executive Committee following Clive Huxtable's departure to the USA. Having not been an active member of the Society for several years I was apprehensive in taking on the role, I am however, pleased that I did because I have enjoyed becoming more involved again and it has allowed the more important members of the Executive to get on with running the Society. My thanks to Cleve Main (Secretary and News Letter Editor) Jeremy Allen (Treasurer) Barry Richards (Committee Member and Convenor of the Working Group on Professional Standards), John Jardine Dave Forshaw (Committee Members) Rod Reece (Slide of the Month Coordinator) and Tony Ross (Chairman of the Registry of Domestic Animal Pathology).

Phil Ladds completed his Continuing Education course on the Lymphoreticular System during the year. We thank you Phil for your unflagging enthusiasm for Veterinary Pathology. We are very fortunate to have such people. This year we have another enthusiast, Roger Kelly to carry on this relatively recent initiative in presenting a course on the liver.

Compared to the previous two years under Clive's Presidency this year has been very quiet. At the last AGM a decision was made to form a Working Party to progress the work that had already been done on a business plan entitled Animal Pathology Standards in Australia. The report on progress in this area will be presented at this meeting. I wish to thank Barry Richards, Roger Kelly, John Mackie and Judith Nimmo Wilkie for giving their time to work on this project.

It has been very gratifying to see how the Veterinary Pathology industry willingly sponsors conferences such as this. On behalf of the members of the ASVP I sincerely thank Agriculture WA, CSIRO AAHL, Veterinary Pathology Services, Victorian Veterinary Pathology Services, Vetpath Laboratory Services, Alpha Scientific and Idexx CVDL for their generous support of this conference.

On behalf of this Executive we congratulate the incoming Executive.

David Pass

Australian Society for Veterinary Pathology Annual General Meeting, June 2000

TREASURER'S REPORT

Financial Members
As at 23 June 2000 the Society had 119 financial members. There are also 22 members who are one year in arrears and 10 members who will be 2 years in arrears on 1 July 2000

Income and Expenditure
Two sets of Income and Expenditure Statements are presented. The first is for the 1998/99 financial year and the second for the current financial year to 31 May 2000

Statement of Income and Expenditure of ASVP for 1998/99

Opening Balance
$8,656.50

Income
Subscriptions
$4,665.00
Conference (1998) Deposit Repaid
$500.00
Conference (1998) Surplus
$680.39
Conference (1999) Registrations
$3,290.00
Conference (1999) Sponsorship
$500.00
WA Pathology Seminar Surplus
$60.00
Interest
$15.68

$9,711.07 $18,367.57

Expenditure
Veterinary Pathology Report
$1,124.95
Secretarial Services
$543.39
Conference (1998), Printing Proceedings
$521.00
Conference (1999), Deposit on Facilities
$1,200.00
Conference (1999), Guest Speaker Honorariums/Expenses
$1,043.25
Audit
$445.00
Ministry of Fair Trading (Incorporation Annual Fee)
$32.00
Bank Fees and Charges
$245.61

$5,155.20

Closing Balance
$13,212.37

Statement of Income and Expenditure of ASVP from 1 July 1999 to 31 May 2000

Opening Balance
$13,212.37
Income
Subscriptions
$1,930.00
Conference (1999), Balance of Cash Advance
$193.44
Conference (1999), Sponsorship
$1,200.00
Conference (2000), Sponsorship
$2,100.00
Interest
$52.20
$5,475 64 $18,688.01
Expenditure
Veterinary Pathology Report
$713.90
Secretarial Services
$277.65
Conference (1999), Balance for Facilities
$760.00
Conference (1999), Catering
$2,680.00
Conference (1999), Printing Proceedings
$453.18
Conference (2000), Deposit for Venue
$500.00
Annual Subscription to FASTS
$288.00
Bank Fees and Charges
$165.54
Closing Balance
$5,838.27 **$12,849.74**

Statement of Income and Expenditure for 1999 Annual Conference

Income
Registrations
$3,290.00
Sponsorship
$1,700.00
Balance of Cash Advance
$193.44
$5,183.44 $5,183.44
EXPENDITURE
Venue
$1,960.00
Catering
$2,680.00
Guest Speaker Honorariums/Expenses
$1,043.25

$5,683.25 **($499.81)**

NOTES

We have applied for an ABN but have yet to be advised what it is. Details of the Office Bearers of the Society will need to be changed with the ATO in due course.

Accounts for subscriptions for 2000/2001 will go out shortly. We are not required to charge the GST because we have a turnover of less than $50,000.

Mark Williamson, our Property Officer for several years has moved to the USA. Malcolm Lancaster has agreed to take over the role of the Property Officer.

During the year we have considered moving banks because of the high fees charged by the ANZ. At the same time, Barbara Gill who runs the Secretariat has advised that the AVA (SA) office is remote from the nearest ANZ bank, and this results in banking taking longer than she would prefer. We decided to leave the task of moving banks to the new SA Executive. They could choose one closer to the AVA (SA) office.

I would recommend that the new Executive move at least $6,000 into a higher earning form of deposit.

Jeremy Allen
Honorary Treasurer

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY
MINUTES OF ANNUAL GENERAL MEETING 2000

Held at Fremantle Western Australia
Saturday June 24 4.00 PM.

Attendees: Allen J, Carrigan M, Charles J, Collett M, Creeper J, Finnie J, Forshaw D, France M, Jardine J, Johnstone A, Jones B, Kabay M, Kelly R, Kessell A, Lancaster M, Lucas J, Main C, Mitchell G, Nicholls T, Pass D, Phillips P, Pritchard D, Rahaley R, Richards B, Ross A, Rozmanec M, Slocombe R, Smits B, Taylor D, Walker K, Wilkie J.

Apologies: Barton M, Campbell Prof R, Carlilse M, Copland M, Glastonbury J, Hull S, Huxtable C, Mackie J, McOrist S, Miller R, Nunn M, Philip Ladds, Reece R, Reuter R, Rothwell T, Samuel J, Sutton R, Yoeman S.

Minutes of AGM held Melbourne 1999- Ross/Richards - carried - subject to the following amendments
(i) Correct spelling of Janeen Samuel's name
(ii) Amend comments attributed to Janeen Samuel to read "fewer gross post mortems were being done and that this could lead to standards falling in time",
(iii) Note that ASVP is an affiliate member, not an associate member.

Business Arising

- Hamilton Histopathological Slide Collection - The CVO Victoria advised that the glass slide collection has been retained by the Department, but the Administrator/Liquidator had taken control of the paraffin blocks.

- Training CD - President Dave Pass spoke to Clive Huxtable who explained that his reference to CDs was limited to a wish to have things such as Roger Cook's collection of pig pathology transparencies put onto a CD. There was no inference that the matter of training modules be resurrected.

- SOM - circulation list has decreased to 28 Australian plus 10 overseas contributors. There have been no contributions of cash in lieu.

- FASTS - President David Pass read out the contents of a letter that explained that affiliate membership was being phased out leaving full membership the only option. This would entail a substantial increase in subscriptions. After a brief discussion it was moved that the Society does not renew its membership.

Ross/Pritchard - carried.

Correspondence	Inwards:	Dealt with under Business arising from minutes.
	Outwards:	A letter of congratulations sent to John Howell on his Order of Australia award.

Reports

President's Report	attached	**Mitchell/Rahaley**
carried		
Secretary's Report	attached	**Mitchell/Phillips**
carried		
Treasurer's Report	attached	**Kelly/Kabay**
carried		
Bulletin Editor's Report	attached	**R Slocombe/Finnie**
carried		
SOTM Convenor's Report	attached	**Rahaley/Kabay**
carried		
Registry Management C'tee Report	attached	

After presenting his report, Chairman Tony Ross recommended that the meeting consider two proposals, the second to lie on the table until the Report of the Working Party on Professional Standards had been presented.

(i) The glass slide collection needs repair.

(ii) The database is getting old and is not capable of integrating with other databases.

A budget item is needed to allow an update so that it can communicate with the Taronga Park database. If full funding is achieved, an expression of interest in the position of Registrar and a hosting institution will be requested.

Keith Walker asked about the financial position of the Registry. Tony Ross explained that there is enough money to do this year’s courses and probably next years as well. The Registry has funds of about $57,000 remaining.

Moved Rahaley/Phillips that the report be accepted - carried.

NOTE: The Secretary left the meeting at 5.10 PM. Committee member David Forshaw took over recording of the minutes.

Report of the Working Party on Professional Standards.

Chairman Barry Richards tabled his report and distributed copies to members. He pointed out that SCAHLS was due to meet in Canberra in 2 weeks time and he would argue the case for Tony Ross's suggestions. He was optimistic about the response, but suggested that if the response were negative, he would submit that the current arrangements be continued.

Moved that the report be accepted and distributed to the wider membership. **Jones/Pritchard - Carried**.

An extensive discussion followed and is reported in some detail;

Barry Richards recommended that the Working Party continue to report to members before the next AGM and implement the new system.

Tony Ross proposed that a trial Mystery Slide scheme (x 4/year) either in the form of a CD or sections

- describe morphology
- DDx
- anonymous grading by facilitator
- score and helpful comments provided by facilitator.
- the scheme to be funded by ASVP.

Bronwyn Smits advised that a proficiency program in operation at Prince Edward Island in Canada used such a scheme.

Terry Nichols asked about NATA's attitude to such a scheme. Barry Richards replied that if such a scheme existed then it will be used. Terry Nichols continued the discussion by pointing out that an NATA approved scheme would raise standards and bring the discipline together.

Malcolm Lancaster asked if a proficiency testing scheme would include a histopathological description. Barry Richards replied that difficulties with length and subjectivity of marks made it unlikely, but maybe it should be included in the trial.

Judith Wilkie added her support for such a description to be included.

Marc Kabay's opinion was that the trial should include everything.

Geoff Mitchell agreed with B Richards's opinion of the difficulties of including a morphological description.

Rob Rahaley stated that as the SCAHLS meeting is only to approve funding, the meeting should support the scheme now rather than trial it.

T. Ross added that if funding became available, we should go ahead.

Roger Kelly agreed that it should be done now, preferably with a CD using his proposal (at an earlier meeting).

Keith Walker argued that the Report should be accepted and a trial implemented. A final report should be submitted to the next AGM.

Moved Tony Ross/Jeremy Allen that:

- The ASVP strongly endorses Proficiency Testing and Continuing Education for veterinary pathologists in Australia.
- The ASVP propose an ongoing and evolving scheme for the above.
- If the decision from SCAHLS is not forthcoming in 3 months, a trial program is instituted with funding from the previously endorsed SCAHLS National Business Plan.

Ron Slocombe voiced his dissent regarding proficiency testing and urged greater emphasis on continuing education. He had added concern over SCAHLS being responsible for proficiency testing.

Barry Richards explained that the scheme would be jointly run with an ASVP nominee in control SCAHLS are only interested in the scheme as a way of maintaining professional standards. He also added that in his opinion, examination of computer images is no substitute for examination of sections.

J Wilkie stated that there is a need to get overseas material for the Registry if it is to be used for proficiency testing.

Ron Slocombe described a software package - internet clubroom with free software offered for Continuing Education televideo. Contact Ron for details.

Ron said that his US experience is that all referral imaging is done electronically without film.

The motion was put and carried.

Election of Office Bearers

President R Reuter

Secretary J Finnie

Report Editor J Finnie

Treasurer R Rahaley

Committee Members
M Copland, Julia Lucas, Steve McOrist

Moved J Allen/G Mitchell - Carried.

There being no further business, the meeting ended at approx 6pm.

Cleve Main
Hon Secretary 1999/2000.

New South Wales

Coccidiosis in chickens - John Glastonbury, Regional Veterinary Laboratory Camden, Elizabeth Macarthur Agricultural Institute, NSW Agriculture.

As part of surveillance for Newcastle Disease, mortality amongst 4-week-old chickens on the south coast was investigated. The owner was described as being "a grower of organic plants but does not know much about chickens". In 1 day, 20 out of 100 chickens died after appearing dopey and having drooping wings and fluffed-up feathers for 12 hours prior to death.

Chickens submitted to the laboratory were in very poor body condition, dehydrated and anaemic. Their caeca were distended with blood and necrotic debris and the tunica mucosae were diffusely congested and haemorrhagic. Severe, necrotic, erosive and haemorrhagic typhlitis was found histologically. In all birds there was massive accumulation of various coccidial stages with a morphology consistent *Eimeria tenella* in the tunica mucosa. Multifocally, infiltrates of mononuclear leucocytes were evident in the tunica submucosa and tunica muscularis, and in some birds, this was accompanied by early fibrosis.

Interestingly, in the Bursa of Fabricius of one bird, the lamina epithelial mucosae was heavily parasitised by gametogenous stages of *E. tenella*.

E. tenella is one of the most pathogenic coccidial species. The second generation of schizogony, which occurs deep in the lamina propria, is the most pathogenic stage and is at its peak 4 days following infection. The exact cause of death is not known but toxic factors as well as anaemia are thought to be involved. Parasitism of the Bursa of Fabricius has been recorded in the literature but this is the first case we have observed.

Despite finding one perivascular cuff and a focus of gliosis in the mid brain, all examinations for Newcastle Disease virus were negative.

Infectious laryngotracheitis - John Glastonbury, Regional Veterinary Laboratory Camden, Elizabeth Macarthur Agricultural Institute, NSW Agriculture.

Fifty week-old layers developed gasping respiration prior to death. Over 4 days, 30 out of 8000 birds died each day.

Consistent post mortem findings were haemorrhagic and necrotic casts in the larynx and proximal trachea, mild mucoid pharyngitis and mild diffuse congestion of the remainder of the tracheal tunica mucosa.

Microscopic examination of the tracheas revealed copious necrotic and haemorrhagic debris in the lumens, squamous metaplasia of the lamina epithelial mucosae obliterating mucous glands, segmental erosion of the tunica mucosa and severe infiltration of the propria/submucosa by mononuclear leucocytes. Syncytia associated with the luminal necrotic debris were observed in three of six tracheas examined; in three, eosinophilic, intranuclear, inclusion bodies were identified with confidence.

Infectious laryngotracheitis virus was detected in one of six tracheas by ELISA.

Detection of the characteristic inclusion bodies is generally thought to confirm a diagnosis of infectious laryngotracheitis. However, it must be remembered that inclusion bodies only can be demonstrated consistently during the early stages, first 1 to 5 days, of infection. Therefore, it is always prudent to examine a number of birds in varying stages of the disease process.

Bracken fern. *Pteridium esculentum*, toxicosis - John Glastonbury, Regional Veterinary Laboratory Camden, Elizabeth Macarthur Agricultural Institute NSW Agriculture.

In addition to demonstrating some interesting pathological changes, this case highlights problems associated with the introduction of full cost recovery charging.

On the south coast, a practitioner was called to investigate the "sudden death" of six out of 60 15-month-old Holstein heifers. He conducted a necropsy of the most recent death; which had occurred 2 hours previously. His post mortem findings and conclusion were "It had signs of septicaemia with serosanguinous fluid in the body cavities with degeneration of liver and perirenal tissue. Found one area of darkened muscle on the diaphragm-muscle was swollen and shiny. Suspect blackleg (animals of uncertain history)". Specimens submitted to the laboratory included fresh skeletal muscle (presumably the diaphragm), air dried smear of skeletal muscle and skeletal muscle, liver, spleen, kidney, and small intestine fixed in 10% neutral buffered formalin. There was a plea to save costs by eliminating the possibility of blackleg first!

A section of muscle was examined the day after receipt at the laboratory and it revealed a haematoma with no evidence of inflammation or bacterial infection. A Gram stained smear of the diaphragm contained no bacteria.

Further discussion with the practitioner ascertained that the owner's wallet would extend to a further section, this time of liver and heart. The former was decided upon because the practitioner then recalled that grossly the liver contained miliary yellow lesions.

Microscopically, the liver was observed to contain focal/locally extensive areas of acute coagulative necrosis, in which there were spectacular septic emboli in branches of the portal vein, terminal hepatic venules and sinusoids, and mild acute segmental fibrinous inflammation of the overlying capsule. Not a single leucocyte could be found. Bracken fern poisoning was offered as the most likely aetiological diagnosis. This result was reported 5 days (including a weekend) following receipt.

The wallet was extended more as the owner desired definite proof. From the next animal to die with similar findings, a piece of sternum was fixed in formalin. Multiple petechial haemorrhages, small pockets of metarubricytes, isolated degenerating megakaryocytes and no evidence of myelopoiesis were the microscopic findings. This result was available 2.5 weeks after the original investigation.

The atrophy of the bone marrow is consistent with the aplastic pancytopaenia described in bracken fern poisoning. Death results from thrombocytopaenic haemorrhage and terminal septicaemia consequent to neutropaenia. Ischaemia/infarction associated with the septic emboli would account for the foci of hepatic necrosis. The offending bacteria in this instance could not be defined because of the costs associated with bacterial culture and Gram staining of histological sections.

There are a number of problems related to charging brought to light by this investigation. The total cost of laboratory testing to the owner for four submissions was $371.40. A definitive answer was not provided until 2.5 weeks after the initial visit by the practitioner. If the laboratory had been contacted at the time of the first visit and a broader range of samples examined without fear of costs, the diagnosis could have been available 1 day after receipt. There were at least four telephone conversations, which are costly in terms of time and money, with the practitioner. Scientifically, the case was not "completed" as the offending bacteria were not identified by either culture or special staining. Hullo real world!

Hepatitis and encephalitis in a kangaroo - Paul Gill, Regional Veterinary Laboratory, Wollongbar

An adult (7-8 year old) female Eastern Grey kangaroo died during a sudden onset of generalised convulsions. The animal had been living in captivity for about 6 years and had progressive weight loss and a refractory non-regenerative anaemia for about 1 year.

The animal was thin with no detectable subcutaneous or visceral fat. The liver appeared to be small and nodular with multiple, 2-10mm diameter, closely spaced nodules through the organ. Microscopically the liver consisted of variably sized nodules of parenchyma separated by bands of condensed stroma and fibrous tissue heavily infiltrated by lymphocytes, plasma cells and the occasional giant cell. This kangaroo also had mild to moderate, non-suppurative meningoencephalitis.

Chronic liver failure would explain this animal's weight loss and mild, chronic non-regenerative anaemia. Aetiological diagnoses for either the chronic hepatitis or the non-suppurative encephalitis are elusive. I'd be pleased to receive any opinions or comments about this case

12.

Northern Territory

Case study: visceral nematodiasis and haemorrhagic peritonitis in mature barramundi cod *cromileptes altivelis* - John Humphrey, Lois Small & Kevin Formiatti, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

History and clinical signs

Multiple deaths in barramundi cod *Cromileptes altivelis* held for broodstock purposes have occurred over the past 12 months. Fish were originally collected from Barrier Reef waters and transported to Darwin Aquaculture Centre. Fish are held in large tanks supplied with filtered, continuous flow seawater. Diet consists of fish and squid held below -20°C before thawing for feeding. Signs include failure to grow, swollen abdomen, lethargy, loss of stability in the water column, unilateral or bilateral exophthalmos, inappetence, integumentary ulceration and haemorrhages.

Gross pathology

The abdominal cavities of affected fish showed moderate to severe fibrinous adhesions and excessive serous or sero-sanguineous exudate. The visceral organs and mesentery were frequently immobilised by extensive bands of fibrous tissue, in many cases extending to the peritoneal surface. Moderate to large numbers of iridescent, green-black, linear or irregular firm masses were present embedded in the fibrous tissue of the visceral organs and mesentery. In some cases, distension of the bile duct was present.

Sub-gross examination of the mesentery and fibrous tissue of the visceral mass frequently showed low to moderate numbers of coiled nematodes. In some cases, the nematodes were partially encapsulated by green-black sheaths.

Histopathology

Extensive fibrosis was present in the mesentery of the visceral tissues. Numerous irregular, effete, melanotic parasitic bodies or tracts were present in the fibrous tissues of the visceral mass and mesentery, with occasional similar bodies in the mucosa or wall of the stomach, pyloric caeca and intestine. Atrophy of exocrine pancreatic tissue entrapped in the fibrous tissue was evident. Marked congestion of blood vessels in the mesentery and fibrous tissue was present.

In most cases, multiple sections of the visceral mass showed single or multiple, viable nematodes in the mesentery or in the mucosa of the stomach, pyloric caeca or intestine.

Parasitology

The coiled nematodes were morphologically consistent with *Terranova* sp type 11 larvae.

Bacteriology

Photobacterium damsela subspecies damsela was recovered in pure culture from the peritoneal fluid in one case. Bacteriological examination of peritoneal fluid and visceral organs in other cases was unrewarding due to post-mortem change and/or earlier antimicrobial therapy.

Diagnosis

Chronic visceral nematodiasis, fibrosis and pancreatic atrophy. Acute peritonitis.

Comments

It is believed that the fish become infected with the nematode larvae before capture with migrations of nematodes resulting in fibrosis of the visceral mass and subsequent loss of pancreatic tissue. It is thought that physical constriction of the alimentary tract, as well as compromised pancreatic function may lead to poor growth rates and digestive disturbances leading to peritonitis. Alternatively, migrating larvae may facilitate peritoneal invasion by pathogenic gut organisms.

The melanotic iridescent bodies appear to represent degenerate parasites. The presence of these bodies as well as viable nematodes suggests successive episodes of parasitism. While unlikely, the possibility exists that recurrent infection occurs from the feeding of frozen/thawed fish or squid. Freezing for prolonged periods at less than -20°C is thought, however, to inactivate any larvae present.

The definitive host for *Terranova* is thought to be species of shark, in which the adult parasite embeds itself into the gastric mucosa.

Treatment is problematic. The fibrosis appears progressive and related to the presence of viable nematodes as well as degenerate or effete parasites and pancreatic acinar degeneration. As such, treatment of viable parasites may not change the course of the syndrome. Preliminary experimental studies in the use of Ivermectin to control the parasites indicate the drug is not successful in eliminating the visceral parasites.

It is noteworthy that the syndrome of severe peritoneal fibrosis and nematode infection has been recorded in recently captured *Cromileptes altivelis* in Australia and a similar or identical syndrome is described in Indonesia. The species is now endangered in the South-East Asian region. In addition to habitat destruction, the possibility exists that visceral nematodiasis is a contributing factor to the decline of the species.

Queensland

Parasite diversity in an ill-thrifty calf - (Anita Gordon, Yeerongpilly Veterinary Laboratory, Queensland Department of Primary Industries)

A group of 8-10 month old Hereford calves being held at the research farm of a pharmaceutical company were considered "slow". One died prior to experimental infection with 3000 larvae of each of *Haemonchus* sp., *Cooperia* sp. and **Oesophagostomum** sp., and one animal became moribund two weeks after. The moribund animal was killed and submitted for necropsy.

Necropsy revealed an extremely stunted and emaciated carcase with an impressive diversity of parasites: *Dictyocaulus viviparus, Haemonchus placei, Trichostrongylus axei, Cooperia pectinata, Cooperia punctata, Trichostrongylus colubriformis, Bunostomum phlebotomum, Nematodirus* sp., *Moniezia benedeni, Trichuris* sp., and *Oesophagostomum radiatum.* Histology also revealed infections by *Eimeria* sp. and *Sarcocystis* sp.

Infection with *D. viviparus* was associated with moderately severe verminous pneumonia. Significant burdens of many of the gastrointestinal helminths were also present.

Equine abortion possibly caused by *Oerskovia xanthineolytica* - (Anita Gordon, Yeerongpilly Veterinary Laboratory, Queensland Department of Primary Industries)

Numerous abortions occurred during 1999 on a property in central Queensland running miniature ponies. These were not investigated to any extent through DPI laboratories. In June 2000 the only remaining mare on the property also aborted, and numerous samples were submitted to YVL. Although foetal and placental histology was unrewarding, *Oerskovia xanthineolytica* was isolated in pure, culture from swabs of foetal brain, lung and stomach. Subsequent Gram-stained sections of lung revealed numerous Gram-positive coccobacilli, consistent with this organism, within bronchioles and alveoli. The bacteria were mostly extracellular, although one or two clumps were noted within alveolar macrophages. This is suggestive of terminal invasion of the foetus, which may have died from the effects of a placentitis. Since no histological lesions were apparent in the portion of placenta submitted, it was concluded that the mare might have had a focal placentitis. Thomas and Gibson (1982) describe a more convincing case of equine abortion due to this organism.

Reference: Thomas, R. J. and Gibson, J. A. (1982) *Aust. Vet. J.* **58**: 166-7.

Yersinia pseudotuberculosis enteritis and cholangiohepatitis in cattle - (Ross McKenzie and Wendy Townsend, Yeerongpilly Veterinary Laboratory, Queensland Department of Primary Industries)

Yersinia pseudotuberculosis was isolated from the liver and small intestine of a Brahman cow necropsied after sudden onset of diarrhoea. This was one of 20 sick animals in a group of 200 of which four had died. Diarrhoea was first noticed amongst weaners with high morbidity but no mortalities. Subsequently adult animals were affected. Histological examination of tissues revealed a cholangiohepatitis and microabscesses in the Peyer's patches of the intestine, suggestive of Yersiniosis. *Yersinia pseudotuberculosis* was also isolated from the faeces of a diarrhoeic cohort. *Yersinia spp* are recognised as human pathogens causing gastroenteritis and septicaemia with renal and arthritic sequelae.

***Mycoplasma agalactiae* mastitis in dairy goats** - (Jim Taylor, Toowoomba Veterinary Laboratory, Queensland Department of Primary Industries)

A Saanen dairy herd had up to 80% of its 80 milkers severely affected with mastitis. Udders were firm and hot with watery milk containing caseous clots. Affected does occasionally developed diarrhoea and depression prior to or during the onset of mastitis but were afebrile. Kids, including those suckling affected

does remained unaffected, but two does died. An affected doe and milk samples from a number of affected cohorts were submitted for examination. Grossly the affected gland had caseous plugs in the intra and interlobular ducts. Histologically there was a chronic interstitial mastitis characterised by a moderate infiltrate of lymphocytes and plasma cells throughout the interstitium and multifocal exudates of neutrophils and caseous material into the ducts. Mammary tissue and milk samples yielded *Mycoplasma agalactiae* on culture, with no growth of aerobic bacteria. In Mediterranean countries, *M. agalactiae* produces contagious agalactia, an acute, high mortality septicaemia in does and kids with chronic sequelae of arthritis, mastitis and keratoconjunctivitis.

M. agalactiae has been isolated from normal, mastitic and pneumonic goats in Australia on previous occasions, but strains isolated are believed to be different to those causing contagious agalactia overseas.

Low virulence Infectious Avian Laryngotracheitis in Layers -(John Gibson, Toowoomba Veterinary Laboratory, Queensland Department of Primary Industries)

Infectious laryngotracheitis was diagnosed in a large layer flock on the western Darling Downs. The property housed 205,000 birds and 10% of a flock of 24,000 birds had mild respiratory signs. Affected birds had a slight cough and snicker with no effect on production and no associated mortalities. The birds were 33 weeks of age and had been vaccinated at 4.5 and 12 weeks with live attenuated ILT virus vaccines. Microscopic changes included a mild to moderate rhintotracheitis in four of the eight birds submitted for necropsy. An infiltrate of lymphocytes and a lesser number of heterophils thickened the tracheal and nasal turbinate mucosa. In some areas sloughed epithelium formed syncytia containing intranuclear eosinophilic inclusions. ILT virus was isolated from pooled trachea cultures.

Infectious Pustular Vulvovaginitis and Balanoposthitis in a dairy herd - (John Gibson, Toowoomba Veterinary Laboratory, Queensland Department of Primary Industries)

Numerous cows in a group of 45 mixed parity Friesians developed a severe erosive and purulent vulvovaginitis, five days after the introduction of a 2-year-old bull. The bull also developed a balanoposthitis at this stage. Bovine Herpesvirus I was isolated from the semen of the bull and vaginal discharges of five of eight cows sampled.

Botulism in a case of Newcastle disease virus exclusion - (Jim Taylor, Toowoomba Veterinary Laboratory, Queensland department of Primary Industries)

Six birds were submitted from a flock of 70 mixed poultry for Newcastle disease exclusion. The owner reported the death of 11 birds in the previous week with another 10 birds sick, mainly chickens. Most birds had died suddenly but some clinical signs observed included depression, lethargy, ocular and oral mucoid discharge, green diarrhoea and terminal convulsions. The owner contacted QDPI concerned about Newcastle disease. All birds examined were severely depressed and reluctant to move, sitting with their wings drooped. The only lesion observed at necropsy was mild haemorrhage over the caecal tonsils. No histological lesions were detected in a range of tissues examined including brains. Samples referred to AAHL were negative for ND virus. Crop contents collected from three birds each gave a strong positive reaction in an ELISA for botulinum toxin.

16.

South Australia

Superficial dermatitis due to *Aspergillus* in a hand reared koala - Julia Lucas Idexx - Veterinary Pathology Services

A 3 month old, hand raised koala developed generalised severe seborrhoea. Several sections of skin were examined and there was marked parakeratotic hyperkeratosis. Within the keratin there were large numbers of fungal organisms both yeasts and hyphae. The hyphae were septate and branching. There was minimal inflammatory response in the dermis or epidermis. There were also large numbers of small cocci bacteria. The fungus was identified on culture as *Aspergillus spp*. and is histologically consistent with *Aspergillus spp*.

The koala was euthanased and there was no evidence of systemic infection.

Superficial skin infection by *Aspergillus* and other fungi is reported in humans with immunosuppressive such HIV - AIDS, transplant patients and myeloid or lymphoid neoplasia[1,2,3,7,8]. It is also reported in premature neonates [5]. Aspergillus has been identified as a common isolate from the nails of humans and this could be a source of infection in this case. It is also reported in the skin fungal flora of cats with FIV infection [6]. One study of the microflora of the Koala pouch did not identify Aspergillus specifically[4].

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17.

Murine intestinal amyloidosis - John Finnie, Veterinary Division, IMVS

Several apparently healthy, Swiss mice submitted for routine health evaluation had lesions of amyloidosis.

While deposits of homogeneous, eosinophilic amyloid material, also Congo red-positive and birefringent in polarised light, were found in renal glomeruli and interstitium, liver and lung, the most striking deposits were in the intestine, The amyloid was so abundant that there was almost total effacement of the lamina propria.

Amyloid deposition in the small intestine is occasionally encountered in domestic animals with systemic amyloidosis and rarely is responsible for the principal clinical presentation (malabsorption and protein-losing enteropathy).

Spontaneous amyloidosis is a common disorder in certain strains of mice, occurring with a high prevalence at either a relatively young age or of mature onset. It may be primary, as is likely in the present case, or secondary to chronic inflammatory diseases or intestinal helminthiasis.

Tasmania

Department of Primary Industries Water and Environment Mount Pleasant Laboratory Report - Phillip Ladds DPIW & E

Cases of general interest

Bovine - These include *Neospora* sp. abortion, *Rhodococcus equi* lymphadenitis and metastatic adenocarcinoma in abattoir lymph node specimens submitted as part of the national granuloma programme, and enteritis and sudden deaths in adult dairy cattle due to bacterial infection (see separate report).

Ovine - Abortions due to campylobacteriosis and listeriosis were confirmed. Several severe outbreaks of yersiniosis, one involving ~20% of yearling sheep, with many deaths, were diagnosed. Abattoir surveys of older sheep entailing submission and histopathology of enlarged mesenteric lymph nodes plus segments of ileum have proved to be a cost effective method of detecting flocks infected with ovine Johne's disease.

Avian - Tissues sent to AAHL from a domestic chicken with a non-suppurative meningoencephalitis, were found negative for Newcastle Disease virus infection. A severe granulomatous typhlitis in a Golden-necked pheasant which died suddenly in a sanctuary was associated with heavy *Heterakis* sp. infection and a concurrent fibroma-like change in the affected tissue - suggesting the nematode may have been causally involved.

Wildlife - Non-suppurative meningitis in a platypus which appeared to have died of severe traumatic haemorrhage was associated with frequent pseudocysts, presumed to be those of *Toxoplasma gondii*. Vitamin A deficiency, with typical squamous metaplasia, was diagnosed in a native wood duck. Death of a mature White breasted eagle (*Haliaeetus leucogaster*) was confirmed as being due to (probable malicious) poisoning, analysis of stomach contents revealed 14mg/kg of Mevinphos, As an incidental finding in the kidney of this bird, many eggs, considered to be those of the fluke *Renicola* sp., were present - largely unassociated with any pathological change.

Bacterial septicaemia in dairy cattle - Les Gabor and Philip Ladds

A dairy herd on King Island had experienced sudden death of three, 18 month old heifers and a dry cow on the one day. Necropsy by the practitioner revealed a reddened abomasal mucosa, sand in the abomasum and loose watery bowel contents. There were ecchymotic haemorrhages on the epicardium. Formalin fixed sections of liver, abomasum, heart and diaphragm were submitted along with faeces from ten cattle, and rumenal contents of the autopsied animal.

One day later, one more heifer died, and 15 had developed watery scours.

Histologically there was a severe mononuclear inflammatory infiltrate of the myocardium. The abomasum was markedly autolytic and non-diagnostic. Apart from slight oedema of the liver, and marked oedema of the diaphragm, little histological change was apparent. Examination of tissues from a further animal revealed a sub-acute but focal myocarditis with frequent polymorphonuclear neutrophils, focal hepatic necrosis and scattered microabscesses, and an acute haemorrhagic abomasitis. Probable bacterial septicaemia was diagnosed.

Faecal culture revealed growth of group B *Salmonella* spp., *Yersinia pseudotuberculosis* and haemolytic *Escherichia coli*. Apparently the heifers had been kept in a crowded rain-soaked pen with no feed for over 24 hours, which may have precipitated the outbreak.

Flounder with systemic *Uronema* sp and enteric microsporean infection - David Taylor and Judith Handlinger, Fish Health Unit, DPIWE, Mt Pleasant

A small commercial Greenback flounder (*Rhombosolea tapirina*) operation was reporting low level mortalities from 2 tanks containing 50g fish. Point mortalities peaked at 5%. The rearing system consisted of flow through seawater of ambient salinity and temperature with a total of 3000 fish. The diet of the 2 affected tanks had been recently changed to one of high oil content, supplied by a commercial fish meal manufacturer. Affected fish exhibited swelling of the abdomen and general listlessness (not surprising for flounder?). Gross internal examination revealed multiple small white nodules present over the serosa of the gut. Microscopically, the gut was distended and the wall massively thickened. The nodules consisted of xenomas within the lamina propria packed with a microsporean parasite, morphologically similar to *Glugea stephani*. The intestinal epithelium was attenuated. In many cases the xenomas were so numerous and large as to nearly completely obstruct the lumen of the gut. Occasionally, rupture of the luminal epithelium with release of mature spores was seen. A majority of the fish presented for examination also had a ciliated protozoan, consistent with *Uronema* sp, within the gut lumen, between xenomas, and widely distributed throughout the body. The degree of inflammatory reaction to the parasites was seen to vary from occasional and scattered eosinophilic granule cells (EGC's), to intense reaction with tissue necrosis, oedema, macrophages, neutrophils and EGC's. Other tissues infected included the choroid of the eye, swim bladder, kidney, myocardium and connective tissues. Thrombi and intense inflammatory reaction were present extending into the heart lumen.

Uronema sp parasites were present within blood vessels of the epicardium. The epidermis and gill tissue did not appear to be altered.

We have observed the presence of the gut xenomas in wild caught flounder previously. *Glugea stephani* is common in the intestine of 11 species of marine flatfish in the northern hemisphere[1]. Prevalences of up to 50% of the fish stock are reported. Xenomas develop from infected neutrophils and progress to replace the normal structures of the intestinal wall. Heavily infected intestine may also become completely occluded resulting in intestinal failure and death. *Uronema* parasites have been implicated in mortalities of cultured and native fish species[2,3,4]. Although the entry point for the *Uronema* is not proven, it is likely that there was extension through the damaged intestinal epithelia.

[1]Lom J and Dykova I. Microsporea. *In*: Protozoan Parasites of Fishes. Elsevier, Amsterdam, 1992

[2]Veterinary Pathology Report, ASVP, April 1999

[3]Fatal encephalitis due to the scuticociliate *Uronema nigricans* in sea-caged, southern bluefin tuna *Thunnus maccoyii*. Munday BL, O'Donoghue PJ, Watts M, Rough K, Hawkesford T. Diseases of Aquatic Organisms. 1997, 30: 1, 17-25

[4]Ulcerative dermatitis associated with *Uronema* sp infection of farmed sand whiting *Sillago ciliata*. Gill PA and Callinan RB. Australian Veterinary Journal. 1997, 75: 5, 357

Victoria

Ichthyosis in a Jack Russell Terrier - Judith S. Nimmo Wilkie, Idexx/CVDL, Mount Waverley, Victoria

An 18 month-old Jack Russell Terrier was presented with a non-pruritic skin condition affecting the ventral abdomen, chest and axilla. There were large keratin flakes adherent to the skin in the affected areas. Skin scrapings were negative for ectoparasites.

Skin biopsies showed a thick layer of compact orthokeratotic hyperkeratosis that was affecting the follicular infundibulae. Hair shafts were normal and some were emerging ensheathed in keratin. Inflammation was minimal. A histological diagnosis of ichthyosis was made and die dog is being successfully treated with vitamin A orally and papaw ointments (which are rich in vit A.) topically.

Ichthyosis has been reported in several breeds of dogs including Jack Russells as well as occasionally in cross-breed dogs. It is usually present from birth, in this case no lesions were noted until after one-year of age and were fairly localized, but it is possible that mild lesions had gone unnoticed earlier.

Chronic Sebaceous Adenitis in a German Shepherd - Judith S. Nimmo Wilkie,Idexx/CVDL, Mount Waverley, Victoria

A female German Shepherd, 8 years of age, had an alopecia of three months duration that was characterised by broken hairs and adherent white scales, commencing on the neck and ears and spreading to the dorsum. The condition was non-pruritic but the dog was malodorous

Skin sections showed hyperkeratosis and keratin plugging of hair follicle orifices. The most striking lesion was an almost complete absence of sebaceous glands on any of the sections examined. An occasional hair follicle had a unilateral focus of granulomatous inflammation at the level of the isthmus. These were composed of macrophages, lymphocytes, plasma cells and occasional neutrophils. Apocrine sweat glands were normal.

Sebaceous adenitis is a condition of unknown aetiology and pathogenesis but is believed to be a developmental and inherited defect in some breeds such as standard poodles. Vizslas, Akitas and Samoyeds also show a strong breed predilection for this condition. It is less often diagnosed in other breeds but Old English Sheepdogs, Toy Poodles, Belgian Sheepdogs, Chows, Collies, Boxers, Lhasa Apso Springer Spaniel and German Shepherds had been diagnosed as well as occasional mixed-breed dogs.

The condition is usually progressive and immune-mediated destruction of the sebaceous gland is thought to be a possible pathogenesis. Alternative hypotheses include a defect in keratinisation with subsequent obstruction of the sebaceous ducts resulting in inflammation of the gland or abnormalities in lipid metabolisms resulting in the keratinisation defects and sebaceous adenitis.

Mild cases may respond to topical treatment to reduce scale or omega-6 and omega-3 fatty acid supplements but more severe cases may need vitamin A or even cyclosporin treatment Once there has been complete destruction of the sebaceous glands as in this case, the prognosis is not favourable.

Western Australia

Animal Health Laboratory, Agriculture Western Australia, South Perth

Sea lion death, cause unknown

Adult male Australian sea lion, about 5 years old, found stranded, in poor body condition and suffering from respiratory distress on a metropolitan beach. It was collected by CALM wildlife officers for observation and treatment. Laboratory tests on a blood sample revealed an elevated white cell count and blood urea. Although treatment with antibiotics gave a transitory response, the condition worsened and it was euthanased (i/v and i/c barbiturate) as it was thought that it might have TB. A subsequent necropsy revealed plum coloured, almost fully collapsed lungs. The hepatic venous sinuses were distended with clotted blood The stomach was empty, apart from several squid beaks lodged in the pyloric area The intestines were essentially empty. Both kidneys seemed to be markedly enlarged.

Histopathological examination revealed focal acute to subacute renal cortical tubular necrosis, with most tubules containing proteinaceous material. Pulmonary alveoli were collapsed and some contained proteinaceous material. There was also severe acute/subacute periacinar necrosis in the liver, presumably due to hypoxia.

We still have much to learn about diseases of pinnipeds. The lung collapse is intriguing, but not thought to be associated with stress. The hepatic venous filling can be associated with a dive response and, therefore, with an animal in acute terminal stress. (Nick Gales pers comm). The aetiology of the kidney and lung changes was not determined.

Quokka cryptococcal pneumonia and protozoal gastritis.

Adult female quokka from the Perth Zoo was found dead in its enclosure. Black circular lesions were found in the lungs, the stomach contained multifocal necrotic foci along one border, the kidneys were mottled with haemorrhagic foci, and the heart was pale with some mottling.

Microscopically, there was a severe erosive and haemorrhagic gastritis that penetrated deep into the submucosal area. Associated with the damage were numerous circular organisms closely resembling an *Entamoeba* sp (thought to be *E. histolytica*). These were present in the deeper layers of tissue and in some of the capillaries adjacent to the laminar muscularis. Lung lesions seen at necropsy proved to be aggregates of macrophages which contained pigmented material (probably dust etc). Of interest was the isolation of *Cryptococcus neoformans var Gatti* from lung culture. There was, however, no evidence of Cryptococcus in any areas of the lung section. In the heart, there was a chronic progressive multifocal cardiomyopathy.

Bovine pyogranulomatous pneumonia.

As always, we are alert for cases of notifiable diseases and hopefully quick to acknowledge the assistance of colleagues in private veterinary laboratories.

In this instance, Vetpath Laboratory Services notified AHL that a smear of a lung abscess taken at the necropsy of a l0 year old bull contained numerous acid fast bacteria. By that stage, the body had been taken to the local knackery and the viscera including lungs had, in turn, been collected by a local rendering service. To cut a very long story short, the lungs were recovered (somewhat worse for wear) and samples taken for histopathological examination and mycobacterial culture.

Grossly, there were multiple, millet sized abscesses throughout the lung parenchyma. Surprisingly, the microscopic picture was one of multiple myogranulomatous necrotising pneumonia with no giant cells, no

fibrosis and no acid-fast organisms. Within the granulomas, there were focal colonies of gram positive cocci and rods as well as some gram negative rods. Despite an intensive search of smears of lesions in the unfixed tissues, only one acid fast organism could be located. PCR techniques allowed it to be identified as a mycobacterium, but not *M. bovis, tuberculosis, intracellulare* or *avium*.

A very big effort, for very little result - but we tried. The good news, of course, was that it was not a case of bovine tuberculosis. The assistance of Vetpath Laboratory Services is gratefully acknowledged.

Ovine hepatic necrobacillosis

Twelve feedlot lambs being fed on oats, lupins and *ad lib*. hay for 5 weeks died. A lamb in thin to average body condition was presented for examination. Its abdomen was filled with proteinaceous fluid, fibrin tags and clotted fibrin. There were large areas of necrosis in liver and some areas of lung. Fibrinous visceral adhesions were present in both thorax and abdomen.

Histopathologically, the hepatic and pulmonary lesions were consistent with the sawdust liver complex seen so often in grain fed animals and, less frequently, in animals suffering from acidosis/rumenitis. There was additional, and more chronic, widespread periportal fibrosis with cholangiolar cell hyperplasia and also acute, and in some places severe, periacinar necrosis.

The cause of additional lesions is unclear. The periportal fibrosis may have been caused by facial eczema and the periacinar lesions may be a terminal phenomenon or the results of a more acute toxic insult. Contamination of the ration with wild radish seed has been suggested as a possible aetiological factor as this seed is able to induce both liver and kidney lesions.

Avian fibrous osteodystrophy.

50/200, 9-week-old broilers in a small rural poultry farm died and most of the rest of the flock were unable to stand (hock sitting). Their diet was comprised of 10% meatmeal, 16% lupins and 75% wheat (no additives).

Examination of 2 affected birds revealed soft vertebral and cranial bones that were easily cut with a scalpel. Metatarsal bones could be broken by hand, but tended to flatten as they did so. Long bone epiphyses were easily sliced through.

Histopathogically, there were severe dysplastic lesions. The cartilage at the physis appeared to be of normal thickness, but the zone of vascular penetration was more disorderly. Of far greater significance were abnormalities present in the zone of ossification. At that site, there was marked osteoclastic and osteoblastic activity, but negligible ossification. There were scant osteocytes within trabeculae and, in the medullary spaces, there was an excess of young fibrous tissue. In many areas, cortical bone was replaced by dense fibrous tissue. Similar changes were evident in the mid-shaft areas where the cortical bone adjacent to the periosteum had been replaced by fibrous tissue, which was also present on the inner perimeter of the cortex.

Avian cardiomyopathy

5 deaths/day occurred in a flock of 100, 6-month-old ostriches. Necropsy of 2 birds revealed haemorrhage in the mesentery and congestion of the small intestine. Histopathological examination of tissues from both birds showed similar heart lesions that were characterised by focally extensive areas of necrosis, degeneration and mineralisation of cardiac myocytes. The causes of myopathy in ostriches include ionophore toxicity from in-feed coccidiostats and vitamin E/ selenium deficiency. Liver vitamin E concentration was estimated to be 1.5 mg/kg., which is considered deficient for avians and certainly lower than accepted AHL values for normal emus and ostriches. Further investigation revealed that the farmer had reduced the amount of premix by 66% as a cost-saving exercise. This would have seriously reduced available vitamin E as well as other vitamins and minerals. The problem disappeared when the birds were treated with indictable selenium/vitamin E,

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