THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
S.A. Department o£ Agriculture
Central Veterinary Laboratories,
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Registered by Australia Post Publication No. VBG 6333

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Number 24 April, 1989

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INSERTS : TREASURER'S NOTICE

: CONFERENCE REGISTRATION - REQUIRED BY 1 MAY

DEADLINE FOR VPR COPY 2 JUNE

PRESIDENT'S PAGE

Apart from my annual report which will appear in the Conference Proceedings this is my last opportunity to have a say as President of the ASVP. I have appreciated the honour of being elected to the office and only hope I have filled it adequately. If I have done the latter it is very largely due to the tremendous support received from my Committee. I publicly acknowledge and thank them.

I am greatly concerned at present with award restructuring. I believe that the professions as a whole are being placed under attack and must carefully guard their positions. It seems only yesterday that Veterinary Pathologists won a heady victory in establishing an award £or themselves in South Australia. This award has since been rescinded but Veterinary Pathologists have retained their separate classification and classification criteria. We are now asked to attend a meeting with Veterinary Officers and Scientific Officers (Veterinary) at the Public Service Association with a view to award restructuring. Perhaps some of you in other States are hearing similar calls.

As I understand it the concept of award restructuring is to reduce the number of employment classifications and allow greater overlap of duties, which all sounds logical when one hears the causes of some demarcation disputes. There does seem to be a paradoxical situation, however, when the Government is looking to reduce classifications and our profession is moving more to specialization and registration of veterinary specialists.

To use an analogy I believe we should prepare our defenses against this tide, which will surely ebb again, lest it breaches our levees and permanently erodes our position.

Peter Phillips.

1989 CONFERENCE UPDATE

Papers on exotic diseases and subjects related to exotic diseases diagnosis will be contributed by AAHL scientific staff. There will also be a session on the role of State Veterinary Laboratories during exotic disease outbreaks. Some papers on exotic diseases by non-AAHL staff have also been received.

Some papers on non exotic diseases have been received and every effort will be made to include in the programme a session on these diseases.

Should you be attending the Conference please return the registration form to the Hon. Secretary as soon as possible at least by 1st May, 1989, so that Noreen Nicholson can arrange breakfast, etc. at Deakin University, and AAHL can prepare name tags for participants' I.D.

A bus will be hired to provide transport between Deakin University and AAHL for those staying at Deakin.

TRAINING COURSE UPDATE

TRAINING COURSE IN RECOGNITION OF EXOTIC DISEASES 14-19 MAY - AAHL

Commencing on the Sunday evening, after the ASVP Conference, a training course will be held for pathologists and laboratory-based Veterinarians. The number wil1 be limited to 16 Veterinarians nominated by the State CVOs .

The course will be organised by Peter Hooper as a practical 'hands on' course, with morning sessions working with animals, afternoons in the lab on histopathology and research into new tests, and evenings spent on reviews and exercises. Participants will be expected to have studied Exotic Diseases - Volume 9 of Animal Health in Australia, by W.A. Geering and A.J. Forman.

Participants will be unable to contact animals for a period of 7 days following the course.

TREASURER'S REPORT

Despite Australia Post increasing its charges, the subscriptions are still covering the day-to-day running costs of the ASVP, and the Hon. Treasurer recommends that annual subscriptions remain the same as in 1988/89.

However, it has become apparent that the current financial year used i.e. 1/5 to 30/4 presents some difficulty to the membership and the Hon. Treasurer. Many members pay their subs at the beginning of the calendar year, and some have become confused when they receive another notice in June telling them that further payment of subs is required. The conclusion of the financial year at the end of April also results in a short time being available for production of a Treasurer's report for inclusion in the proceedings of the AGM/conference in mid May. I therefore move a motion (seconded by V. L. Tham) that the financial year be changed to coincide with the calendar year. Many other societies have their AGMs at different times to the end of their financial years without difficulties.

Finally, review of the membership records shows that the following people have been unfinancial for 1986/87, 87/88, and 88/89; C.A. Bourke, A. Ghawi, P. Harper, R.J. Seiler, M.J. Shiel, and C.L. Ungku. Can members please contact one of the Executive if they know why these subs ere outstanding e.g. if the person has moved to a new address, has extenuating circumstances etc. If we don't hear from either these members, or someone else who knows why they haven't paid, their names will be removed from the ASVP mailing list after the A.G.M.

Tammy Utteridge
Hon. Treasurer

EDITOR'S REPORT

The Veterinary Pathology Report is a very healthy publication, with contributions being received from 28 Australian laboratories on a regular basis and occasional news from overseas members (more would be welcomed). The current circulation is 172, of which 30 are overseas. The increasing use of the VPR for advertising job opportunities and conference details is most rewarding.

The value of the Report as a quick referral and awareness resource is evident and has made the Editor's task of limiting the number of pages difficult indeed. While an attempt has been made to publish all articles received as soon as possible, the increasing length of contributions has led to some carry-overs to the following issue. In general the standard of contributions has been high.

Currently articles printed are being indexed, by species, state and issue for the previous five year period in the belief that this will further increase the Report's value.

The cost of production of the Report is considerably reduced by the provision of facilities for typing and copying, and time given freely by members of the Committee. Succeeding Committees may consider if any advantages would be gained by having the Report professionally printed.

In conclusion I would like to thank the State representatives who, by various means, have usually met the deadlines. Regrettably the use of Fax has been limited by the necessity to retype the articles for reproduction, but has allowed some editing prior to arrival of the hard copy.

Robin Giesecke
Hon. Editor

STATE REPORTS

WESTERN AUSTRALIA (Dave Pass)

PERTH ANIMAL HEALTH LABORATORIES (J. Dixon)

Renal Failure in Koalas

A mature female died at Perth Zoo at the end of December 1988. The cause of death was renal failure, due to extensive renal fibrosis. There was some oxalate-type crystals present in the tubules which stained positive with Pizzoiato stain. The question is where did these crystals come from and are they common in koala kidneys?

One month later at the end of January 1989 her ten month old youngster died after a few hours illness. It had been doing well up till then.

General bacteriological cultures were negative but kidney problems were suspected because of the suspected subcapsular cysts noted at post mortem. There was evidence of focal pyeolonephritis and fibrosis and a few crystals were present in kidney tubules but these were negative to Pizzolato stain.

Perhaps this information, such as it is may be helpful to some colleague and any relevant observations would be welcome.

Lymphocytic leukaemia in a Northern Quoll (Native Cat)

A mature female died on 28/12/88 at the Zoo. The main post mortem finding was a spleen which was larger than the liver. The spleen and other major organs showed evidence of severe lymphocytic leukaemia. Bacteriology and virology cultures were negative. Immunology for F.E. virus was negative.

A similar case occurred last year in the same pen which houses several animals. Specimens have been lodged with the Registry at Taronga Park,

Renal Lesions in a Chapman's Zebra

A 23 year old stallion died in December 1988. It had a seminoma in one testicle. Both kidneys showed excessive polypoid hyperplasia of the renal pelvic epithelium. Thin strands 10 centimetres or more long and 2 to 3 millimetres thick were embedded in thick tacky mucus. These filled the pelvis of the kidney and proximal ureter and I think caused partial obstruction to the urinary flow.

They appeared to be unattached to the renal pelvic epithelium but this may be an illusion due to the very fragile attachment which would rupture at the slightest pressure.

I do not know if this is a common finding in aged Zebras or not, but I do not recall seeing anything like it in any horse kidney that I have examined. Any comments?

PERTH ANIMAL HEALTH LABORATORIES (Ron Peet)

Suspect Ergotism in Sheep

Thirty of 1200 three year old merino wethers wore found dead in a paddock which consisted almost entirely of annual rye grass (Lolium rigidum) feed. They had been on the paddock six weeks and all had appeared well when last seen 12 days previously by the non-resident owner.

Annual Ryegrass Toxicity was suspected but had not been previously diagnosed in that area, and the sheep had no abnormal "rocking horse" gait when driven. No nematode galls were found on pasture or in soil samples submitted to plant pathology, but many sclerotia of Claviceps spp were found infecting the seed heads. Two live sheep were submitted for post mortem examination and they were recumbent, not paddling, apparently conscious and showing marked muscular rigidity, but with some flexion of the forelimbs.

Differential diagnosis suggested included ARGT, polioencephalomalacia, enterotoxaemia (animals had not been vaccinated) tetanus and convulsive ergotism.

There were no remarkable gross post mortem findings, but histopathology of the brain of one animal revealed spheroids and degenerating neurones in the posterior brainstem and medulla oblongata of one sheep (Sheep 1) but not the other. However, there was marked fibrinoid necrosis of vessels in the myocardium of Sheep 2. Fifteen sections of both sheep brains were examined, then re-cut and re-examined with the same findings. The histopathology was considered consistent with an acute vascular toxicosis with the most likely aetiology being vascular toxins from ergotism.

To my knowledge this is the first time convulsive ergotism has been diagnosed in Western Australia.

References

Seawright, A.A. (1982) Animal Health in Australia, Vol. 2, Chemical and Plant Poisons, Australian Government Publishing Service, Canberra, p. 124.

Testicular Calcinosis in Goats

Testicles from adult ma1e goats are currently being collected in two Western Australian Abattoirs and exported to Taiwan for human consumption. Protocol at one abattoir requires removal of the tunica albuginea, but not at the other.

Occasional white "spots" were seen under the visceral serosal surface at the abattoir after the tunicae were removed. Many testicles were condemned (10-15%) of older (6 tooth) bucks. Some of these were forwarded to the Animal Health Laboratory for microbiology and histopathology. Microbiology yielded non-significant organisms, but histopathology revealed the numerous white spots to be testicular calcinosis lesions.

These stained strongly positive for calcium salts using von Kossa's stain, and probably would taste very gritty if masticated. I have not yet performed that test.

References

Ladds, P.W. (1985) Pathology of Domestic Animals, Jubb, K.V.F., Kennedy, P.C. and Palmer N., 3rd Edition, Academic Press, New York Vol. 3, P432.

ALBANY REGIONAL LABORATORY (Ruth Reuter)

Pox Infection in a Magpie

Several magpies on a property east of Albany had large wart-like lumps on the head, particularly around the eyes. One severely affected juvenile was submitted for examination. Large, necrotic fly blown masses were found at the mucocutaneous junctions of the eyes and beak. The animal was depressed and emaciated. On necropsy several small grey raised areas were found in the mouth on the soft palate. The spleen was enlarged and there were caseous yellow foci scattered through the liver. Microscopically the epidermis of the masses was acanthotic with ballooning degeneration of epithelial cells and large eosinophilic intracytoplasmic inclusion bodies characteristic of pox virus present. In the dermis there were large structures resembling schizonts of apicomplexans. With the kind assistance of Dr Bill Hartley these were identified as probably asexual stages of Leucocytozoon. The granulomas in the liver appeared bacterial in nature; however, overgrowth of the culture plates with Proteus prevented isolation of the aetiologic agent.

Avian Ascariasis

A young Scarlet parrot approximately four weeks out of the nest appeared depressed one evening and died the next day. The bird was one of eight in a cage. The other seven, plus 30 other psittacines in the aviary were clinically normal. The flock had been treated with tetracyclines six months previously following a positive diagnosis of Psittacosis. On necropsy the intestinal tract was grossly distended. The intestinal lumen was obstructed by very large numbers of ascarids. Impression smears of liver taken to rule out concurrent Chlamydial infection were negative on F.A.T. It appears that heavy contamination, probably by the parents, occurred while the bird was still in the nest.

Workload at the Laboratory

Following the introduction of fees for laboratory services in October 1988, submission of samples has dropped significantly. At present our accession rate is 51 percent compared to the same period last year. The most striking decline is in necropsy and histology material. It certainly gives one the opportunity to clear the paper backlog which tends to develop in a laboratory!

NORTHERN TERRITORY (Lorna Melville)

BERRIMAH AGRICULTURAL LABORATORY.

Acute bluetongue in sheep (L. Melville)

The 1989 virus sentinel program commenced in late January and by early February we had recorded Australia's first field case of acute bluetongue disease in a sheep.

The sentinel program has been running in various forms for a number of years now. Sheep were first introduced as a sentinel flock in 1976 before the first recovery of bluetongue virus in Australia. In recent years the sentinel herd has consisted mainly of cattle with sheep, goats, horses and pigs being added at various times. Sheep were last used in 1984 and while bluetongue viruses (BLU) Types 1 and 21 were isolated from these animals there was no clinical disease associated with the infection.

The sheep sentinel herd this year consists of 10 animals. These animals were transported by road from Queensland, held at Adelaide River for one week, then transported to Coastal Plains Research Station east of Darwin on 1st February. On 2nd February all sheep were bled and were in good health. Baseline clinical chemistry showed all values normal.

On 9th February the sheep were again bled and number 74 was found to be acutely lame and unable to walk to the yards. Following examination it was decided to move the sheep back to Berrimah Agricultural Research Centre for close observation due to the severity of clinical signs seen.

Clinical signs included severe congestion and ulceration of the nasal mucosa, facial oedema, congested buccal mucosa, excessive salivation with wetting of the skin around the muzzle and severe coronitis on all feet. The sheep was depressed and anorexic with a temperature of 41.4°C. There were petechial haemorrhages on the skin. The sheep was kept under close observation over the next few days and become recumbent and progressively depressed. It finally died on 12th February.

The necropsy snowed severely congested and oedematous lungs which oozed fluid and froth when incised. There was extensive froth in the major airways. There were several large haemorrhages at the base of the pulmonary artery and sub endocardial haemorrhages associated with the papillary muscle of the left ventricle. Area of pallor were seen in both cardiac and skeletal muscle particularly the shoulder area. There was mild congestion in the abomasum and small intestine.

Significant laboratory findings have included a neutrophilia and very elevated CPK and AST. Routine bacteriology on lung and heart blood yielded no growth. Salmonella was isolated from the small intestine. Histopathology confirmed the acute pulmonary oedema and congestion. There was extensive cardiac and skeletal muscle necrosis.

Serology showed a blue-tongue AGID conversion from 0 to 3 and initial serum neutralization suggests that ECU Type 23 is involved. Bluetongue type 23 virus has been isolated from the blood sample collected on 2nd February.

Bovine TB in Bali Cattle (M Bell)

Two groups of Banteng/Bali cattle exist in the Northern Territory. One group on the Coburg Peninsula and one in the adjacent Murganella area. The Coburg bantengs have had an initial negative survey for the presence of bovine TB which will be confirmed by a second sampling in early 1989. It has been assumed that the isolation of the herd and the low stocking density has kept them free of bovine TB. The bantengs at Murganella are surrounded by buffaloes which have a high incidence of bovine TB. The question arose, "is there a special situation with bantengs such that they might not be as susceptible to bovine TB as cattle and buffalo?".

Three cull Bali cattle were infected with M. bovis to ascertain their susceptibility to this disease. Two were given 1×10^6 intravenously and the other 1×10^5 subcutaneously in front of the point of the shoulder. The latter animal died 5½ weeks later. M. bovis was isolated from mediastinal bronchial, retropharyngeal and prescapular lymph nodes. The remaining two animals were tested at 6 weeks post infection using the gamma-interferon test (CSIRO) and intradermal caudal fold test. Both animals responded very strongly to these tests.

At post mortem (8 weeks) both showed miliary pulmonary TB and invasion of the mediastinal and bronchial lymph nodes. From this limited trial, it would appear there is little difference in susceptibility between Bali cattle and normal bovines to M. bovis.

QUEENSLAND (Fraser Trueman)

ANIMAL RESEARCH INSTITUTE

SUSPECT BOTULISM (Fraser Trueman)

Three large cattle mortalities were investigated in SE Queensland during late 1988. The first case was in a large feed lot where only two pens of cattle were involved. Over a 10 day period 237 head of 770 adult cattle died.

The second case involved a dairy farm where a total of 109 animals of different ages died from the total herd of 271.

In the third case 30 milking cows died out of a total of 380 animals on the property.

Clinical symptoms were similar in all cases and consisted of a stiff stilted gait, muscle weakness progressing to sternal then lateral recumbency and death. Other symptoms included depression, partial anorexia, stasis of rumen and weak jaw muscle tone. Early in the outbreaks deaths were acute, but later it took up to 72 hours for animals to die. Some recoveries were recorded.

Necropsies were generally uneventful, and extensive laboratory testing failed to detect any infectious or toxic agent. The feed lot ration included 4% chicken litter, which has been associated with outbreaks of botulism overseas. The second case was associated with supplementary feed prepared on the property, as only animals receiving this feed developed symptoms. The symptoms were reproduced when the feed was fed to a sheep.

The third case was also probably feed related as these cows received a feed lot diet including brewers grain, vegetable waste and molasses. In the first two cases the ration was quickly changed, but new cases and deaths continued for another 10 days. All animals received botulism vaccine Type C and D.

Laboratory tests failed to detect botulism toxin from any case, but culture of rumens did detect Clostridium botulinum spores.

Chicken Anaemia Agent Syndrome – (CAA) was suspected in 2 broiler flocks in Brisbane. One involved chickens aged between 10 and 14 days which had excessive numbers of runts. Haematological examination revealed 23 out of 37 birds to be anaemic. The anaemia was predominantly a normocytic, normochronic anaemia. This, together with the increased fatty area present in the bone marrow is suggestive of infection with Chicken Anaemia Agent. Organ lesions were generally mild with the main lesions being vacuolated hepatocytes in the liver, increased numbers of reticulo-endothelial cells in the spleen and mild heterophil infiltrations in the thymus and spleen. There appeared to be no major secondary pathogen involved with only seven birds having a moderately elevated white blood cell count.

The other possible CAA case was in 32-day-old broilers showing poor feed conversion. Four sick and 4 healthy birds were presented. Two of the 4 sick birds had a normocytic, normochronic anaemia and one had red cell parameters at the lower end of normal. At 32 days of age, this is consistent with previous suspect CAA properties which showed a prevalence of anaemia of approximately 50% at this age. None of the 4 healthy birds were anaemic. The most severely affected bird had severe lymphocytic depletion of the thymus and moderate depletion of the Bursa of Fabricius with some fibrosis. This bird also had severe proventriculitis, myocarditis, purulent inflammation of the lungs and Inclusion Body Hepatitis. Both sick and healthy birds had a mild thymitis, mild to moderate liver degeneration and mild purulent inflammation of the lungs. The mild liver lesions may reflect early infection with Inclusion Body Hepatitis (IBH). It is possible that the IBH represents a secondary infection due to possible immunosuppresion resulting from earlier CAA infection.

An unusual case of mass hysteria throughout 100% of a flock of 1,400 commenced soon after beak trimming and was still continuing at 13 weeks. Another younger flock in the same shed on the same feed and water showed no signs. Birds were dying with secondary bacterial infection due to fight wounds. Examination revealed the presence of Marek's Disease but this did not adequately explain the hysteria present. The birds were in fair condition, very nervous and had numerous skin lacerations. No significant findings resulted from haematological, histological, biochemical and virological examinations. It is possible that the birds had a genetic predisposition to hysteria.

VETERINARY PATHOLOGY SERVICES - BRISBANE

HEPATITIS IN A DOBERMAN BITCH (Geoff Mitchell)

A 10 year old Doberman was presented with progressive weight loss over several weeks and on clinical examination had ascites. (Temperature was normal).

Clinical Pathology

* Chemistry		Ref. Range	*	* Haematology		Ref. Range	*
* T. bilirubin	38.0	0-8 u mol/L	*	* Hb	143.0	120.0 – 180.0 g/L	*
* ALP	1034.0	20-70 U/L	*	* PVC	43.0	.37 - .55 L/L	*
* ALT	961.0	15.70 U/L	*	* WBC	32.3	6.0 - 17.0	*
*				*	* mature neutr., normal platelets		*

These results suggested ongoing obstructive liver disease. "Chronic active" hepatitis of female Dobermans was considered likely although neoplasia, ascending cholangiohepatitis, etc. could not be excluded.

On the basis of the clinical condition and the above findings the owner decided to euthanase the dog. Grossly the liver had a multinodular appearance. Histologically there was chronic active hepatitis characterised by extensive fibrosis and necrosis with diffuse neutrophilic and plasmacytic infiltration and biliary hyperplasia.

Chronic hepatitis of Dobermans is an interesting disease about which not a lot is known. It has some similarities to copper associated hepatitis of Bedlington Terriers although it does not result in the Heinz body anaemia as seen in Bedlingtons. Although copper accumulates in the liver of affected Dobermans, it may be secondary to liver disease and not a primary copper storage disease. In Dobermans there is a marked predominance of females affected (39 of 41 in one study). Clinically they present with weight loss, ascites, jaundice, polyuria and polydipsia. Biochemically ALP, ALT and bilirubin are increased while albumin is often decreased. The histology described in this case is typical.

Most dogs are in advanced liver failure by the time they are examined and survival is usually poor i.e. weeks to a few months. Low copper diets, d-penicillamine, nutrition and prednisone have all been tried with little success. Early diagnosis may help improve the effectiveness of therapy. It is assumed that this is a genetic disorder but the inheritance pattern is not known as yet.

UNIVERSITY OF QUEENSLAND (Roger Kelly)

An outbreak of cardiomyopathy in unrelated dairy cows:

There has been a low but fairly continuous incidence (about 9 cases) of fatal cardiomyopathy in unrelated dairy cows on a prison farm near Brisbane. Animals have been affected from 5 years of age, and show a remorseless progression of heart failure. At necropsy, there are the changes expected of right and left-sided heart failure, such as passive venous congestion of the liver, ascites, and variably severe pulmonary oedema. The heart itself, however, shows only a mild degree of dilatation of both ventricles.

The myocardial histology is characterised by asymmetry of fibre diameters, and central vacuolation: frank fibre necrosis is rare. These changes are not specific, and overlap those seen in an apparently heritable cardiomyopathy which has been described in young Friesian cattle, and which has also been seen in the Brisbane area.

The cause of this disease of older cows is unknown: investigation has not revealed exposure to toxins such as the ionophore growth promotants.

Smoke inhalation and cerebrocortical necrosis in a dog:

A German Shepherd dog made the TV news after Christmas by being trapped in a burning garage and getting rescued by the fire brigade. It was temporarily revived, but died about 4 days later without having fully regained normal neurological function.

At necropsy, besides having particles of carbon mixed with proteinaceous exudate in its smaller airways, the brain showed distinct lamina and focal polioencephalomalacia. The clinicians who admitted the dog stated that the mucous membranes were dull red, which suggests the probability of CO poisoning.

Nitrate-nitrite intoxication in goats:

During a spell of rainy and overcast weather recently, a band of about six goats died suddenly on a stand of pearl millet Pennisetum americanum. Ironically, this strain had been specially bred for low cyanogenic glycoside levels. The tissues were brownish and the aqueous humor was positive for nitrite with the diphenylamine test.

TOOWOOMBA VETERINARY LABORATORY

SUSPECT BOTULISM (Jim Taylor)

Botulism was suspected as the cause of a large mortality in a 25,000 head feedlot near Toowoomba. Seven hundred steers in two adjacent pens were affected. A total of 283 head died over a 10-day period. Clinical signs were muscle weakness progressing to sternal recumbency, lateral recumbency and death. Early in the outbreak the mortality rate was 100% with only 20 or so animals showing muscle weakness and sternal recumbency surviving in the last few days. No gross or histological abnormalities were detected in the 20 to 30 animals necropsied. Biochemical profiles on a number of animals showed no abnormalities. Extensive testing for myotoxins, pesticides, heavy metals and monensin was negative. Botulinum toxin was not detected using mouse inoculation. The suspected source of the toxin was chicken litter containing an occasional chicken carcass. The litter was hammermilled and added to the ration at a rate of 4%. Feed was mixed by the truckload with one load feeding two pens. In this case the two affected pens received the same mix of feed. No other animals in the complex including those in adjacent pens were affected.

IXIOLAENA BREVICOMPTA POISONING (John Gibson)

Ixiolaena brevicompta was diagnosed as the cause of skeletal muscle necrosis in sheep and goats on several properties in the St George/Dirranbandi area. Mainly young animals were affected with clinical signs of muscle weakness, staggering, knuckling, collapse and death. Serum GOT levels in affected cases ranged from 3000 to 4000 I.U./L with CPK levels from 800 to 25,000 I.U./L. Histological sections of skeletal muscles showed varying degrees of myonecrosis. No other lesions were detected. In all cases animals were grazing pasture containing Ixiolaena brevicompta. The plant was identified in rumen contents of affected animals.

CHRONIC FURAZOLIDONE POISONING (Jim Taylor)

A number of vealer calves from a property near Crows Nest suffered severe haemorrhagic diathesis. The calves were continuously medicated with furazolidone in their milk over a period of four weeks. Clinically affected animals had pale mucus membranes with petechial haemorrhages, epistaxis and passed melenic faeces and frank blood. Haematology showed severe anaemia, granulocytopaenia and thrombocytopenia. Two calves were submitted for necropsy. There were extensive haemorrhages throughout the carcases. Histological sections of sternal bone marrow showed severe hypocellularity and a total absence of megakaryocytes.

CONGENITAL CARDIOMYOPATHY IN HEREFORD CALVES (John Gibson)

Congenital fatal cardiomyopathy in curly-coated Hereford calves was diagnosed in two herds on the Darling Downs. Affected calves, although appearing normal at birth, except for the tight curly hair coat, soon showed poor exercise tolerance. Calves collapsed and died usually after a short period of exertion. All calves died before six weeks of age. Selenium status of the herds and affected calves were normal. Losses only occurred in one herd when a purebred Poll Hereford bull was mated to his daughters. The other herd involved horned purebred Herefords.

CITRULLINAEMIA (John Gibson)

Citrullinaemia was diagnosed as the cause of progressive nervous signs in Friesian calves on a dairy farm at Bell. Levels of citrulline in CSF, plasma and urine were 640, 1760 and >5000 um/L, respectively.

NEW SOUTH WALES (Jim Rothwell)

NATIONAL REGISTRV - GLENFIELD

ERYTHROCYTIC PROTOZOANS IN BIRDS (Dr. W. J. Hartley)

Asymptomatic infection by blood protozoans is quite commonly seen in some species of birds in coastal Eastern Australia and is probably seen in other areas. Occasionally sporadic deaths can be attributable to these infections.

LEUCOCYTOZOON SPP. This protozoan is seen all the year round in the Sydney area as an unapparent infection in Pied Currawongs, Magpies and several other species of birds. Occasionally it is associated with failure to thrive and death in the young Pied Currawong. In the latter case blood smears showed up to 20% of erythrocytes containing large circular non-pigmented micro and macro gamonts. Schizonts were present, particularly in cardiac muscle.
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HAEMOPROTEUS SPP. This organism is usually seen as an asymptomatic infection sporadically in water birds and occasionally other species. The protozoan is seen as large sausage-shaped pigmented gamonts in erythrocytes. A suspected Haemoproteus spp is involved with a lethal disease of Pied Currawongs in the Sydney area in the summer months. The disease is associated with the presence of rows of megaloschizonts in skeletal, cardiac and gizzard musculature and massive inflammatory myonecrosis. Zoites released from the schizonts enter erythrocytes to develop first into ring forms and later into large pigmented sausage-shaped gamonts which may occupy most of the erythrocyte cytoplasm.

A further lethal suspected haemoproteus infection is seen in psittacines in North-east coastal Queensland. This is associated with enormous numbers of free intracytoplasmic schizonts in blood vessels, particularly in the lung and spleen, and the presence of pigmented Haemoproteus-like gamonts in erythrocytes.

PLASMODIUM SPP. Asymptomatic malaria may occur in wild and aviary birds in susceptible environments. Occasionally deaths, perhaps stress induced, do occur in association with very heavy plasmodium infestation of erythrocytes and profound anaemia.

The life cycle in malaria infections is quite complicated, but briefly erythrocytic schizogony is preceded by one or more stages of schizogony in cells of the R.E.S. Erythrocytic schizonts may contain up to 20 zoites, these are liberated and invade other erythrocytes to form ring and then round to oval pigmented gamonts.

IDENTIFICATION. The Diff Quik staining method applied to freshly prepared blood smears is a very effective and quick method of demonstrating blood protozoans. Leucocytozoons are easy to identify because of size and are non-pigmented. Haemoproteus and Plasmodiun spp are difficult because both are pigmented. Also, dual infections may often occur.

In tissue sections Leucocytozoons are easily seen. The others are more difficult to see in an H & E, but when mature gamonts are present pigment granules are readily discernible.

REGIONAL VETERINARY LABORATORY WAGGA WAGGA (John Glastonbury)

SHEEP

Encephalitis/ Histophilus ovis

Eight 2-year-old Merino ewes died over a period of 2 weeks. They had been running on a phalaris pasture after returning from agistiment in the western division of New South Wales. The animals were in good body condition and had been vaccinated against Enterotoxaemia twice as lambs and had received a booster vaccination in May 1988. Pathological examinations were dominated by the presence of acute multifocal embolic suppurative meningoencephalitis, nephritis and myocarditis and in one case subacute suppurative polysynovitis. Heavy pure growths of Histophilus ovis were recovered from the brain, kidney and synovial fluid of the last animal presented for examination.

Oxalate Nephrosis

During the summer months mortality rates varying from 0.7 to 6.6% were attributable to this disease on 7 farms. In most outbreaks the sheep had been grazing stubble paddocks but in only a few was there a history of access to sorrel (Rumex spp). The diagnosis was confirmed histologically by the detection of subacute chronic nephrosis, dilated nephrons and the presence of birefringent crystals with a morphology consistent with oxalates.

Eperythrozoonosis

A 6 tooth Merino ewe was presented to the laboratory. Although the animal was extremely anaemic with a PCV of 6% it was in good body condition. Numerous organisms with a morphology consistent with Eperythrozoon ovis were detected both on the cell surface and extracellularly. Histologically there was a moderate generalised periacinar necrosis and slight to moderate degrees of haemosiderosis in the liver, kidney and spleen. Follicular hyperplasia and neutrophilia also were observed in the spleen. It is most unusual to diagnose infection with Eperythrozoon ovis in animals of this age.

FISH

Coccidiosis

Following the diagnosis of coccidiosis in Murray Cod at the Narrandera Inland Fisheries Research Station 53 Murray Cod and Trout Cod fingerlings were submitted to the laboratory. Infection with a coccidosis parasite Goussia lomi was confirmed in many of the fingerlings examined.

RABBITS

Myxomatosis

A recently established rabbitry was decimated when virtually all of its stock of 50 Angora rabbits died. Affected animals were either chronically ill for a period of 2 to 3 weeks showing severe conjunctivitis or died relatively suddenly after appearing depressed for less than 48 hours.

In the acute cases histological evidence of viral infection was not dramatic. A very mild vesicular palpebral dermatitis was evident and only occasional eosinophilic intracytoplasmic inclusion bodies could be detected. However in the chronic cases these lesions were much more obvious and necrotising.

Animals which died acutely had extremely severe pulmonary congestion and oedema. In all cases there was evidence of marked compromise of the immunological system. Chronic cases had secondary mycotic pneumonia, possibly due to Aspergillus fumigatus while acute deaths displayed terminal bacteraemias. Material has been submitted to Dr Steve Robbins of the CSIRO Division of Wildlife and Ecology for virology.

REGIONAL VETERINARY LABORATORIES - ARMIDALE

SALMONELIA ANATUM IN PHEASANTS (S. Hum, B. Walker)

Several one week old pheasants have been received from a hatchery. About 50% of chickens appeared sick and 35% died out of 1600. Deaths usually peaked at around one week of age. Affected birds appeared depressed, huddled around lamps, laid on their sides and kicked convulsively. Many seemed to recover and walk away but a few minutes later clinical signs recommenced; birds became progressively worse and died.

The most consistent gross pathologic findings were: caecal distension with yellow foamy ingesta and enlarged pale liver. Histologically exudative and granulomatous epicarditis, acute focal peritonitis and a necrotising typhlitis were detected with frequent typhoid nodules in the liver.

A group E Salmonella sp was isolated from a variety of sites of several birds and was subsequently identified as S. anatum. Twenty four embryonated eggs (some containing dead embryos) and 13 yolk sacs were cultured for salmonellae with negative results suggesting a horizontal transmission.

Changes in management have been made and included improved hygiene by staff collecting eggs and handling chickens, using double the concentration of formaldehyde to fumigate eggs also, chickens hatching late were no longer kept. Following this the death rate halved and progressively improved.

REGIONAL VETERINARY LABORATORY - WOLLONGBAR (Paul Gill)

CID IN AN ARAB FOAL

A 3-month-old Arab foal with a history of generalised dermatophilosis had a severe bilateral catarrhal nasal exudate which persisted despite treatment. Its haemagram showed anaemia, lymphopaenia (I%) and hvpoproteinaemia with an A/G ratio of 0.57. Necropsy findings included multiple abscesses in liver and lungs and miniscule lymph nodes and thymus. Nocardia spp. was grown from liver and lung. Basophilic intranuclear inclusions consistent with adenovirus were present in the epithelial cells of the pancreatic ducts and bronchi.

SUSPECT CEREBELLAR ABIOTROPHY IN PIGS

Three piglets were presented from a 1000 sow piggery. Two piglets were by the same boar, the other was sired by an unrelated boar. All piglets had a history of ataxia progressing to inability to stand despite good muscle tone and intact spinal reflexes. There were no gross lesions in brain, spinal cord, vertebral canal or middle or inner ears. Microscopic examination revealed segmental amphophilic swellings throughout the cerebellar granular layer and, less extensive, in white matter extending into the cerebellar peduncles. There was segmental demyelination of small numbers of axons in the affected white matter with some extension of the demyelination into the medulla and spinal cord. No identifiable pesticides were found in brain or fat.

ADENOVIRUS IN A PARROT FINCH

8/200 parrot finches died after a 12 hour illness characterised by rapid respiration. There were no gross lesions. Basophilic intranuclear inclusions were present in a few hypertrophied Kupffer cells and in a large number of reticuloendothelial cells in the spleen. The liver sinusoids were extensively infiltrated by lymphocytes.

VICTORIA (Susan Friend)

REGIONAL VETERINARY LABORATORY - HAMILTON

BALDY FRIESIAN CALVES (Cor Lenghaus)

In 1987 two calves became illthrifty, exhibited joint pain, drooling salivation and developed granulating skin lesions of the flanks, axilla and over the limb joints. They were killed at several months of age when it appeared they would not recover. No abnormal ties apart from skin lesions were detected at autopsy.

In 1988 three Friesian calves from the same sire and the same property as above, developed similar disease from six weeks of age. They were unresponsive to zinc therapy and were referred to the RVL when about three months old. The calves were in poor body condition and appeared depressed. The skin was very dry and scurfy, with severe pediculosis. There was hair, loss and crusty skin thickening of the dorsal midline, dorsum of the ears, axilla, flank, inguinal region and over the stifle, hock, knee and fetlock joints. Some areas were raw and bleeding, as following trauma. There were no other significant findings at autopsy. The thymus was considered of normal size for the age of the calves.

Histologically all tissues except for the skin were unremarkable. Skin pathology consisted essentially of a localised hyperkeratosis, both ortho and parakeratotic, restricted to the grossly abnormal areas of alopecia and encrustation.

This condition of "Baldy Calves" can be differentiated from the clinically similar "Lethal Trait A-46" syndrome also seen in Friesian-type cattle. The latter do respond to regular zinc supplementation, and have thymic hypoplasia due to a severe depletion of cortical lymphocytes, which is also expressed in other lymphoid tissues.

REGIONAL VETERINARY LABORATORY - BAIRNSDALE

WHITE MUSCLE DISEASE IN YOUNG RUMINANTS (K Button, K. Thomas)

Necrotizing myocarditis (3 kids, one calf) and necrotizing myositis (one lamb, one kid) were seen in several unweaned ruminants recently. When given, history included an inability to keep up with the flock (lamb), sudden death (2 kids) and apparent nervous signs followed by collapse (calf). Gross necropsy findings were suggestive of left and more particularly right sided congestive cardiac failure (ascites, hydrothorax, lung oedema) with pale streaky subendocardial lesions in the ventricles or skeletal muscles. Histologically, lesions were acute to subacute myonecrosis. These cases were consistent with white muscle

disease due to selenium/Vitamin E deficiency and blood samples from cohorts or, one property have been submitted to confirm this possibility.

CLOSTRIDIAL ABOMASITIS IN A KID (K. Button, N. Skilbeck, K. Slee)

A 7 day-old Angora kid died unexpectedly. At necropsy, the kid was in good condition with adequate fat reserves. The abomasum was grossly dilated with clotted milk. The abomasal mucosa showed dark streaky haemorrhages and the wall was thickened, oedematous and extremely friable. Histologically there was necrosis of all layers with severe oedema and apparent microscopic gas bubbles. Clostridium perfringens (untyped) was isolated from the abomasal content. Epsilon toxin was identified in abomasal content by counter current immunoelectrophoresis.

EPERYTHROZOON OVIS IN WEANED LAMBS (K. Button, K. Slee, N. Barton)

Ten of 500 recently weaned Merino lambs died on a Bengworden farm. One submitted for necropsy was anaemic (Ho 5.3 g/dl) and slightly jaundiced. Numerous E.ovis parasites were noted on the blood film. In addition the lamb had a moderate parasite burden (5000 Nematodirus sp, 5000 Ostertagia sp and close to 1000 Trichostrongylus sp) and had a marginal liver copper concentrate of 0.26 mmol/kg (dry weight). This combination of factors adequately explained the deaths. On a second farm near Bairnsdale about one third of 1000 Merino weaners showed weight loss and weakness despite adequate feed and low worm burdens. Approximately 15 died. Blood samples showed regenerative anaemia (Ho 3-4g/dl). but no E.ovis parasites could be found on blood smears of clinically affected lambs. Many clinically normal lambs from the same mob showed numerous E.ovis parasites.

VERTEBRAL OSTEOMYELITIS DUE TQ SALMONELLA DUBLIN IN CALVES (R. Seiler, J. Browning, K. Slee)

Spinal abscesses in calves are seen reasonably frequently during spring. The presenting signs are usually progressive posterior paresis because the location of the abscess is usually in the thoracic (T5-7) or lumbar vertebrae (L2-5). The expected bacterial isolate is C.pyogenes. Two recent cases were presented with forelimb weakness and knuckling. One calf was also obviously distressed when its neck was manipulated. Both had grossly evident osteomyelitis with sub-epiphyseal fractures in the vertebral body of C7. S. dublin was isolated from the lesions, end from other organs, and there was histological evidence of systemic Salmonellosis.

REGIONAL VETERINARY LABORATORY, BENDIGO

KHANCOBAN X DISEASE - AN UPDATE (R. T. Jones)

In the January 1989 issue of the Veterinary Pathology Report (Number 23), I reported on an outbreak of a fatal nervous disorder in pigs. I suspected a ration toxicity of unknown cause and invited comments and suggestions from ASVP members.

I am very pleased to report that 3 members contacted me. David Pritchard from Hamilton suggested that the nature of the outbreak and the clinical signs exhibited by the pigs were consistent with those he had seen in arsenilic acid poisoning. Arsenilic acid poisoning was my initial preferred diagnosis but we proved conclusively that arsenilic acid was not involved in this outbreak. Tony Ross from Glenfield suggested Vitamin A toxicity and thiaminase activity from ingestion of a coccidiostat as two possible causes. Chris Bourke from Orange drew my attention to his publication on tunicamycin-like intoxication in pigs eating water damaged wheat.

I am not able to confirm or disprove any of the latter suggestions, as very little tissue remains and we haven't got any of the original feed sample.

I am very grateful that 3 members contacted me, shared their experiences and knowledge and offered assistance with the investigation.

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2, Frantora, E. Ofukany, L. Mraz, A. Koncek, M. and Blanar. M. (1986) - Monesin poisoning in swine. Veterinarstri 36 (6):270.

3. Miller, D.J, O'Connor, J.J and Roberts, N.L (1986) - Tiamulin/salinomycin interactions in pigs. Vet. Rec. 118 (3):73.

REGIONAL VETERINARY LABORATORY, BENALLA

MULTIPLE CONGENITAL CARDIAC ANOMALIES IN A THOROUGHBRED FOAL (J Mackie)

A four month old Thoroughbred foal was found dead. The foal had a history of becoming distressed and cyanotic whenever it was handled. On post-mortem, the heart was greatly enlarged and the following cardiac anomalies were present: (1) absence of a ventricular septum (2) a dextroposed aorta (3) right ventricular- hypertrophy (4) the cardiac end of the pulmonary artery terminated blindly under the epicardium adjacent to the left longitudinal groove (5) a patent ductus arteriosus.

The liver was enlarged and dark with an accentuated, black, lobular pattern, characterized histologically as periacinar necrosis tending to massive necrosis in some areas. The lungs were oedematous.

The cardiac abnormalities appear to be due to: (1) anomalous development of the membranous septum which divides the truncus arteriosus into the pulmonary artery and aorta, and (2) failure of the upgrowth of the muscular portion of the interventricular septum. This is in contrast to tetralogy of Fallot, where the abnormalities are due to anomalous development of the membranous septum only.

RED SPOTS IN SLAUGHTERED PIGS (Malcolm Lancaster)

Large numbers of pig carcases at two abattoirs had to be skinned due to very obvious reef spots on the scalded skin surface. The dermal histological reaction was very similar in both groups – a predominantly eosinophilic perivascular dermatitis. The epithelium of pigs from the first group had large eosinophilic intracytoplasmic inclusions characteristic of pig pox, but the second group had only scattered degenerate keratinocytes. Many blood engorged Culex annulirostris were collected at the second abattoir. It was concluded that these mosquitoes were probably responsible for the skin reaction but controlled biting experiments using native versus previously mosquito exposed pigs were not carried out.

Coccidiosis in Budgerigars (Malcolm Lancaster)

A budgerigar breeder noted diarrhoea, depression and deaths in several birds including juveniles and adults. Histologically, an ulcerative granulomatous enteritis was associated with oocysts in the lamina propria. Other stages of the life cycle were seen in one bird; with little reaction. The coccidium has not been speciated.

AUSTRALIAN ANIMAL HEALTH LABORATORY, GEELONG.

SWINE VESICULAR DISEASE (P Hooper, A Forman)

In the January issue of the Veterinary Pathology Report we reported on experimental vesicular exanthema and phototoxic vesicular-bullous dermatitis. Since then we have been working with swine vesicular disease.

A series of 3 groups of pigs were inoculated. In the first group, 4 pigs were inoculated with tissue culture material into the skin immediately above the heels of the hind feet. In the second, 2 pigs were inoculated with a suspension containing vesicular material from the first, and in the third, 2 pigs with s suspension of a materiel from the second. There was distinct increase in virulence. In the first, only one pig had a rise in temperature and there were vesicles in the feet of all 4 pigs but only after 4 to 7 days. In the second, both pigs had vesicles after 2 and 3 days and both had temperatures of the order of 40.5 C for 2 days. In the third group, vesicles appeared after 2-3 days but temperatures rose to 40.9 C for 3 days and the spread of vesicles from foot to foot was more rapid.

Only one head lesion was seen, a small snout vesicle in the second group. In the third group, vesicles also developed on the knees, hocks and accessory digits.

From a pathologist's view point, the neuropathology was, extraordinary considering the minimal clinical signs of nervous disease. Both pigs in the second group had clear-cut nonsuppurative meningoencephalitis when they were euthanased at 5 and 7 days post-inoculation. Predominant lesions were perivascular cuffing and glial nodules. All parts of the brain were affected with a slight predilection for the olfactory lobe and the midbrain. One of the 2 had lesions in the spinal cord as well.

Pigs in the first group which were euthanased after 4 weeks did not show significant neuropathology. This could reflect either the lower virulence of the tissue culture material, or of recovery. Pigs in the third group are still alive at the time of writing (13/2/89).

VETERINARY RESEARCH INSTITUTE

APPARENT MALICIOUS INJURIES TO DOGS (Robin Condron)

Two recent cases of apparent malicious injury occurred to dogs in Melbourne recently.

Each dog was found dead with several steel skewers stuck into the body or injuries consistent with stabbing. One issue was whether the skewers were placed before or after death, an issue familiar enough to forensic pathologists but rarely encountered by veterinary pathologists.

In one dog, massive bruising was evident around the neck, and there was little evidence of haemorrhage around the sites of skewer infection.

These types of malicious attacks on dogs and also on caged animals have been thought to be mainly committed by young juveniles.

In both dog cases, there was a lack of continuity in the process of transferring the affected dogs from discovery to our autopsy, making our evidence non-admissable.

Practitioners reed to be aware that for autopsy evidence to be used in court, bodies need to be delivered person to person.

INFECTIOUS BURSAL DISEASE (Don Barr, Steve McOrist)

The avian section at the V.R.I. in conjunction with poultry veterinarians is investigating a sustained epidemic of infectious bursal disease (IBD) in commercial chicken flocks in Victoria.

Clinical signs have been lameness and high mortality in meat chickens 4 to 7 weeks of age. Pathological lesions consisted of necrosis of the epiphyseal plates of the proximal femoral and tibial shafts in the lame birds. Other lesions of synovitis and serositis due to E. Coli. and mycoplasmas were common. The consistent finding was of bursal shrinkage and necrosis from 3 weeks of age. Histologically, necrosis of bursal follicle lymphocytes and stromal fibrosis were evident Serology indicated that IBD maternal antibody titres were only persisting for 1 to 2 weeks, leaving the chickens susceptible. The antibody vaccinal status of the breeder of parent flocks and the time of natural IBD challenge in different flocks is being investigated.

GALAH ENTERITIS (Steve McOrist)

Wild-caught galahs (Cacatua roseicapillus) have been noticed over many years to suffer from a diarrhoea and wasting syndrome.

Galahs do not breed in captivity so all birds for the pet trade are caught at a young age, tamed, then sold. During the taming process, up to 50% of the birds can die with signs of diarrhoea, anorexia and wasting. Gross lesions consist of a dilated fluid filled duodenum, and poor body condition. Histological lesions consist of an enteritis with fused, stunted villi, proliferation of crypts and a moderate mucosal mononuclear cell infiltration. Electron microscopy of faeces has shown high numbers of piconavirus (enterovirus) particles.

These viruses are difficult to culture and standard avian viral culture techniques were unrewarding. However, it is likely that this condition is a viral enteritis. Attempted treatment of the birds has been unrewarding. In some instances, there has been evidence of co-infection with the psittacine beak and feather disease virus

CENTRAL VETERINARY DIAGNOSTIC LABORATORY

In January, 1989, CVDL began testing for antibody to feline immunodeficiency virus (FIV). The test being used is an enzyme immunoassay system (ELISA) to detect antibody to FIV in fresh serum or plasma. (Cite (8), Agritech Systems Inc.)

To date, 43 cats have been tested and FIV antibody was detected in the sera of 14. The majority of these FIV positive cats were castrated males with an age range of 3-10 years. Most were febrile, lethargic, depressed and other clinical signs included weight loss, stomatitis, gingivitis, dermatitis, conjunctivitis, rhinitis, recurrent bronchopneumonia and diarrhoea. Ten of the 14 were tested for feline luekaemia virus FeLV) antigen (Leukassay F II, Pitman-Moore Inc.) and were found to be negative.

FIV has been isolated from the lymphocytes of cats in the USA (1) and the U.K. (2). FIV antibody has been detected in the blood of cats in Japan (3). Europe (4) and Australia (5). A survey on feline sera collected during 1970-1988 suggests the FIV infection in cats in Australia has occurred as early as 1972 (5).

In conjunction with Michael Studdert, Veterinary Science Faculty, University of Melbourne and Chris Birch, Department of Virology, Fairfield Hospital, we are attempting to isolate FIV from antibody positive cats. A retrospective study is underway to examine sera collected during 1985 from sick cats. One hundred and eighty-four sera have been tested using an ELISA test and 41 were positive for FIV antibody. Six of these sera were FeLV positive and the other 35 were FeLV negative.

References:

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(3) Ishida.T, Washizu.T, Toriyake.K, Motoyshi.S. Jpn J Vet Sci 50: 39, 1988.

(4) Feline Advisory Board, Report on a Presentation by Dr.Hans Lutz, University of Zurich, British Small Animal Association Meeting /April 7, 1988.

(5) Sabine.M, Michelsen, J, Thomas,F. Zheng, M. Aust Vet Practit 18: 105, 1988.

UNIVERSITY OF MELBOURNE

LYMPHOSARCOMA IN A CAT - An Unusual Manifestation (Jenny Charles)

An eight year-old Siamese cat was presented with apparent bilateral otitis media, right unilateral vestibular and facial nerve paralysis. Horner's syndrome and left unilateral lysis of nasal turbinates.

At necropsy. both tympanic bullae contained copious gelatinous white-cream material, with similar material present in the caudal left nasal cavity. The only other abnormality was enlargement of mesenteric lymph nodes.

Histologically there was effacement of nasal mucosa and soft tissues of both tympanic bullae by small neoplastic lymphocytes arranged in a follicular pattern, with similar neoplastic involvement of mesenteric and colonic lymph nodes.

BOVINE ADENOVIRUS INFECTION (Jenny Charles]

A group of thirty 8-12 month old dairy calves exhibited chronic ill-thrift and recent severe scouring, with apparent passage of necrotic mucosal casts and blood. The only calf necropsied was in very poor nutritional condition, with evidence of moderate abomasal (Ostertagia sp.) and distal ileal and caecocolonic Oesophagostmum sp.) endoparasitism. There was mild femoral osteopaenia. Peyer's patches and mesenteric lymph nodes were hyperplastic and intestinal contents were watery but without blood or mucosal injury.

Diffuse intestinal lamina proprial plasma lymphocytic and eosinophilic infiltrates were thought to reflect helminthosis. Very large numbers of amphophilic intranuclear inclusions were evident within endothelial cells of abomasal and intestinal mucosal submucosa and in postcapi1lary venules of mesenteric lymph nodes. Minimal necrosis was as yet associated with the presence of adenoviral inclusions in the calf examined, but infection was considered responsible for severe diarrhoea/dysentery in more seriously affected animals.

TASMANIA (Judith Handlinger)

MOUNT PLEASANT LABORATORIES - LAUNCESTON

TASMANIAN TRICHINELLA - Opening a can of worms (David Obendorf)

Cast your minds back to the January 1988 Report (p20). Remember the item from Tasmania on tiger quolls with spinal degenerative lesions and the array of incidental parasitic infestations? To some of you, I suspect, this item might have been quickly passed over (or even gone unread) and dismissed as merely a quaint wildlife investigation with little further consequences. Far from it, the incidental collection of parasites contained at least one nematode which sparked an exotic disease alert in Tasmania. In that article we reported several muscle parasites whose occurrence in these carnivorous marsupials was completely unexpected and new. At the time, we concluded that the intensity of the metacestode (Anoplotaenia dasyuri) and the Sarcocystis infections in the skeletal muscles was highly suggestive of coprophagic feeding by these quails. (Tasmanian devil being the source o£ these faecal delicacies). In addition, we

found several nematode larvae within skeletal muscle cells in one of three tiger quolls. At the time, these nematodes were too quickly dismissed by me as an “ascaridoial” larvae (perhaps of Baylisascaris tasmaniensis). A classic case of "justifying my preconceived prejudices", I fear! I had concluded that if Tasmanian devil faeces were the source of the metacestodes and sarcocysts in the muscle of the quolls then the nematodes might be as well. It all seemed logical (some might say teleological) after all, since Baylisascaris is another intestinal parasite of Tasmanian devils. Baylisascaris produces eggs which, when infective, undergo visceral larval migrans and encystment in body tissues of intermediate hosts.

Perhaps this was not a colossal calamity, but things got interesting when in May 1987, we decided to write up the quoll cases for publication. With the help of several Australian wildlife parasitologists (Paul Presidente, David Spratt, Jack Arundel and Ian Beveridge) we discovered a new parasite of Australian wildlife and opened up a can of worms! Perhaps ‘dabbling’ parasitologists like me should just leave well enough alone. For heaven's sake - Trichinella sp. in Australian wildlife! What next, Rabies?

The nematodes in the quoll muscles produced no capsule as is typical of Trichinella spiralis in pigs and rodents. After dissecting some larvae out of formalin-fixed muscles and examining them, it all became clearer. Diagnostic features were the spirally coiled larvae (approximately 2½ turns), 0.7-1mm in length, the presence of flattened stichocytes, a slowly tapering anterior end and a blunt rounded posterior end.

After an extensive pig survey (1768 pigs from 149 properties) no Trichinella infection could be detected by digests of diaphragms. Nevertheless, a state-wide survey demonstrated a 30% prevalence of infection in Tasmanian devils. AAHL submitted muscle containing Trichinella to Northern American laboratories for species identification. The nematode was identified as T. pseudospiralis, a species which is considered to have low infectivity for domestic animals (e.g. pigs) or synanthropic rodents (black and brown rats and house mice). Unlike T. spiralis, T. pseudospiralis is maintained and transmitted by carrion feeding birds (such as corvids and raptors) in the northern hemisphere, to date no birds with Trichinella larvae have been found in Tasmania.

Currently we are attempting to determine the major cycling of Trichinella in Tasmania. The evidence suggests that devils are the major hosts with quolls being minor hosts in some regions. Cannibalism amongst devils is the most plausible route of infection, with carrion-feeding of devil carcases being the source of infection for quolls. We are currently checking preserved Tasmanian devil and quoll material in museum collections for Trichinella. This may help in determining if the nematode is an 'endemic' parasite of Tasmanian carnivores. So far we have not found Trichinella in thylacine muscle. No kidding.

Post script: The early misdiagnosis of Trichinella does not change the original suggestion of coprophagic transmission for the other parasitic infections in the quoll muscles.

SPOROZOAN ABORTIONS IN DAIRY CATTLE (David Obendorf)

We are currently investigating a property on which sporozoan type abortions have been diagnosed for the past three years. The situation is made more interesting in that several individual cows have aborted in two consecutive years and one cow in three consecutive years. This phenomenon is somewhat contrary to Sarcocystis abortions in that previous abortion is thought to confer active protection against subsequent abortion (as for Toxoplasma abortions in sheep and goats). Cows are aborting at five to seven months of gestation with focal granulomatous encephalitis lesions and foci of necrosis and periportal infiltrates in the foetal liver.

Has anyone investigated similar abortions in cattle?

MEMBERSHIP NEWS

Rodney Oliver, a new member, has joined the Mount Pleasant Laboratories in Launceston, as Chief. Previously he was head of the Virology Section, Central Animal Health Laboratories at Wallaceville, New Zealand. He gained his PH.D from Washington State University in 1979, after working at Ruakura Animal Health Laboratory, New Zealand, from 1972-1975.

SOUTH AUSTRALIA (Robin Giesecke)

WATER BUFFALO DEATHS - CHAPTER 2 (Peter Phillips)

In the last VPR (No. 12, p6) I reported on deaths of buffalo in S.A. due to travel stress and Pasteurella/Pseudomonas/Streptococcus infection, with successful treatment with Terramycin.

Alas, I claimed success too early, as the deaths were halted temporarily only with Terramycin and the Pasteurella etc. were, to a large degree, red herrings!

Deaths continued and were again halted temporarily with 1/M Terramycin on one more occasion until at last after the death of some 60 of the 164 cows and 1 of the 8 bulls. The pathology being basically a polyserositis with a non-suppurative meningo-encephalitis suggested sporadic (sic!) bovine encephalitis (SBE) however, we had difficulty in proving this diagnosis although we steadfastly put our faith in tissue pathology and stuck to it.

Fortunately, thanks to Quarantine's Lou Schinckel, the tenacity of the attending private practitioner, Ron Baker, and the persistent efforts of Sonia Neville, VETLAB's Virology Technical Officer, Chlamydial antigen was detected by ELISA and culture/IFAT techniques in the serum of several sick animals much to this particular pathologist's relief. A further Terramycin treatment and removal of the remaining buffalo to better, sheltered, wallow-free paddocks saw the deaths cease and some calves hit the ground and survived.....touch wood.

SBE has a largely unknown epizootiology but is known to occur in South Australia in the hot dry months almost exclusively. We can only postulate that these stressed, immunologically naive animals encountered the Chlamydia in conditions that favoured the organism's survival and transmission .

Tribulus terrestris TOXICITY IN SHEEP (Peter Phillips, Bill de Saram)

Severely jaundiced lambs in poor condition from separate outbreaks in the Port Pirie and Loxton areas were investigated.

Two live lambs were brought to VETLAB from the Loxton case and displayed the following plasma biochemistry.

	Lamb 1	Lamb 2	Norma1 Range
Total Bilirubin (µMol/L)	252	258	0-7
GGT (U/L)	167	125	30-80
AST {U/L)	2,377	1,808	40-200
GLDH (U/L)	25	40	0-20

The lambs had thickened dark ears, severe conjunctivitis with corneal ulceration and marked encrustation of noses and mouths.

On post-mortem examination, carcases were extremely jaundiced with the livers and renal cortex being particularly so.

Whilst the livers of these two lambs did not show significant changes, the renal cortices had obstructed nephrons with granulomatous foci which included acicular clefts.

Heavy rains had fallen at Christmas time followed by two months of hot, very dry conditions and the wheat stubble on which the ewes with their lambs were grazing had been invaded by prolific growths of calthrop (Tribulus terrestris) .

Ref: Glastonbury, J. et al, A.V.J. (1984), 61: 314-316

SOUTH EAST REGIONAL VETERINARY LABORATORIES - STRUAN

Sheep Diseases Associated with Grazing Haifa White Clover (Michael Hindmarsh)

A mortality rate > 4% was indicated by 20% of producers of certified Haifa seed responding to a survey conducted in the south-ease of the State recently. Deaths had occurred in Merinos and prime lambs in good condition and vaccinated for enterotoxaemia. "Red gut" was also seen but was recorded also on pure stands of lucerne, Trikkala subclover, Paradana clover and Paraggio medic in the region.

Other conditions reported in sheep grazing Haifa clover included tender wool, unthriftiness, scouring and increased mortality rate in sheep which had lupinosis.

The main problems occurred in irrigated stands of Haifa clover (which could be of low palatability), or after rain in mild to warm sunny weather. Similar problems were reported in the western districts of N.S.W. by Dr. Greg Curran. Haifa clover stands giving rise to these conditions were shown to have low fibre (18% ADF), high energy (11.5-12.5 HJ/kg DW) and very high protein (> 30% CPE) content. Provision of wheaten or sorghum straw, in conjunction with the Haifa clover, had reduced mortalities.

Campylobacter Abortion Storm in Merino Ewes (Michael Hindmarsh)

A flock of 886 Merino ewes run in one 17 hectare paddock and trail-fed oats after joining in March began aborting in early June. Abortions continued into July. Campylobacter jejuni was isolated from placental and foetal material.

Scouring galahs feeding on the oat trails were considered to be the source of infection in this case. No Campylobacter were recovered from crows, usually suspected as the source of infection.

The feeding of oat grain which attracted the galahs, and the high stocking rate for the ewes which allowed for rapid spread of infection, were the major predisposing factors.

VETERINARY PATHOLOGY SERVICES – ADELAIDE (Rob Rahaley)

MYCOBACTERIUM INFECTION IN CATS:

Several cases of cutaneous mycobacterial infection have been diagnosed histologically in Adelaide suburban cats. Lesions are typically focal granulomas containing myriads of acid fast, slender, rod-shaped, bacteria within macrophages. The bacteria are morphologically consistent with M. lepraemurium, the presumed cause of "cat leprosy".

Cutaneous infection of cats with mycobacteria is via skin abrasions or, possibly, from rat bites. Treatment involves surgical excision of the lesions, which do not usually recur. Dapsone treatment has also been recommended but there may be some problems with supply of this drug at present. Streptomycin and isoniazid are toxic for cats. The disease is apparently far more prevalent in Adelaide than in the eastern states.

THIAMINE DEFICIENCY IN A CAT:

An adult cat was recently submitted for necropsy after it developed behavioral changes and ataxia. Histological changes in the brain included laminar cerebrocortical necrosis, and perivascular haemorrhage in the inferior colliculi and lateral geniculate bodies. These changes were highly suggestive of thiamine deficiency.

The cat had been in a boarding kennel for one month and fed exclusively on a fish-based commercial canned feed ("Snappy Tom") but the thiamine levels in the feed have not been assayed and a causal relationship not established.

Cats are uniquely susceptible to thiamine deficiency and development of this disease is frequently associated with fish-based diets containing thiaminases. The disease has also been reported as a sequel to anorexia or snail intestine disease.

JOBLINE

EXCHANGE OPPORTUNITY

Dr. Jack Rhyan, Pathology Supervisor in the Department of Livestock Diagnostic Laboratory Division at Bozeman, Montana, U.S.A., is anxious to explore the possibility of a job-swap with a diagnostic pathologist in Australia or New Zealand sometime in the next 3-4 years.

Anyone interested in exploring the opportunity such an exchange would provide could contact Dr. Rhyan D.V.M.. M.S., at the Department of Livestock Diagnostic Laboratory, P.O. Box 997, Bozeman, Montana 59771, U.S.A.

PROVISION OF MATERIAL

APPRECIATION OF MATERIAL PROVIDED FOR ACVSc PATHOLOGV EXAMS

Phil Ladds wishes to thank all those who provided material for the 1989 ACVSc Pathology Membership examinations. Contributions received from Clive Huxtable, Neil Sullivan, Bruce Parry, Steven McOrist, Terry Rothwell, Rick Speare, Richard Sutton, Roger Kelly, Rick Parker and from the Registry, through Bill Hartley, broadened the scope of the examination and enhanced its value. Material provided for these examinations is placed in the Registry for use by candidates preparing for future examinations

REGISTRY

REQUESTS FOR MATERIAL:

These are some disease entities not, or poorly represented in the National Pathology Register. If you have good examples of the diseases below please send them to the Registry.

CATTLE
Mucosal Disease
Leptospirosis (abortion & hacmoglobinuria)
Yersiniosis

PIGS
Swine Erysipelas (heart,joint,septicemia)
Septicemic Salmonellosis
Mulberry heart disease

DOGS
Dirofilaria lungs
Bordetella Pneumonia
Skin conditions

DEER
Acute M.C.F.
Yersiniosis
Lungworms

SHEEP
Chlamydia abortion
Yersiniosis
Pneumonias

POULTRY
Coccidiosis
Tuberculosis
Lymphoid tumours, not Marek's

HORSES
Cyathostomiasis
Sarcoids
s/c fungal infections

Also protozoan diseases including coccidia in any type of animal (domestic & native).

BILL HARTLEY
National Registry of Domestic Animal Pathology
Regional Veterinary Laboratory
Roy Walts Rd.Glenfield 2167

DEPARTMENT OF AGRICULTURE, N.S.W. - NEW ENGLAND, HUNTER AND METROPOLITAN REGION.

REQUEST FOR MATERIAL

The Regional Veterinary Laboratory, Armidale requires ovine and bovine isolates of Campylobacter foetus.

If anyone can help please contact Steven Hum on 067 734805.

Thank you.

Steven Hum
Veterinary Research Officer
ARMIDALE

27th February 1989

WORLD ASSOCIATION OF VETERINARY LABORATORY DIAGNOSTICIANS

Vth International Symposium
Guelph, Ontario, Canada
June 25-30, 1989

Registration - full Conference $300 Canadian
- Mycoplasma Workshop (June 28) - no charge

Completed registration forms and payment should be returned before May 1 to :

WAVLD Symposium
Conference Office, Maritime Hall
University of Guelph
Guelph, Ontario, Canada NIG 2W1

($50 late fee added after May 1. No phone registrations)

Satellite meeting: on the Haemophilus/Actinobacillus/Pasteurella group
:Regional meeting on Paratuberculosis (separate registration)

Further information: Robin Giesecke (08) 126 0571.

ASVP CONFERENCE & AGM 13-14 MAY

DON'T FORGET TO SEND YOUR REGISTRATION FORM TO:

DR. V.L. THAM
VETLAB
GPO BOX 1671
ADELAIDE