THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Regional Veterinary Laboratory,
Private Mail Bag, Wagga Wagga. N.S.W. 265O
069 230920
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EDITOR: Ian LINKS

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OFFICE BEARERS 1985.

ELECTED EXECUTIVE.

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SECRETARY:	Dr. Ian Links, BVSc..DIP. BACT. *	(069 230934)
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COMMITTEE:	Dr. John Searson, BVSc.. MVSc. *	(069 230928)
	Dr. Roger Cook. BVSc., PhD., DIPLOMA ACVP. +	
ADDRESSES:	* As per letterhead.	
	+ N.S.W. Department of Agriculture, Regional Veterinary Laboratory, WOLLONGBAR. 2480 (Phone: 066 297511)	

NON-ELECTED STATE REPRESENTATIVES.

Dr. Mark Carrigan, BVSc..
N.S.W. Department of Agriculture,
Regional Veterinary Laboratory,
ORANGE. N.S.W. 2800 (063 636700)

Dr. Roger Kelly. BVSc.. PhD.
Department of Veterinary Pathology and Public Health,
University of Queensland.
BRISBANE. QLD. 4067 (07 3772565)

Dr. David Obendorf,
Department of Agriculture,
Mt. Pleasant Laboratories, P.O. Box 46,
SOUTH LAUNCESTON. TAS. 7250 (003 415215)

Dr. Peter Phillips, BVSc.,
Institute of Medical and Veterinary Science,
Veterinary Science Division,
Box 14 Rundle Street, P.O.,
ADELAIDE. S.A. 5OOO (08 2287537)

Dr. David Pass. BVSc.. Msc., PhD..
School of Veterinary Studies,
Murdoch University,
MURDOCH. W.A. 6150 (09 3322211)

Dr. Owen Williams,,
Arid Zone Research Institute,
P. 0. Box 2134,
ALICE SPRINGS. N.T. 5750 (089 522344)

Dr. Sue Friend,
Department of Veterinary Paraclinica1 Science,
School of Veterinary Science,
University of Melbourne,
WERRIBEE. VIC. 3030 (03 7413500)

MINUTES A.S.V.P. ANNUAL GENERAL MEETING MELBOURNE, 19 MAY, 1985.

Minutes of the 2nd Annual General Meeting held in the Veterinary Research Institute, Parkville, Sunday 19 May, 1985, at 8.30 a.m.

(1) Present

I. McCausland {President}, L. Badcoe, C. Bourke
C. Button, R. Ca11inan, B. Car, B. Carrigan, W. de Saram,
S. Friend, M. Forsyth, D. Gardener, R. Giesecke, J. Glastonbury,
L. Gleeson, W. Hartley, K. Harrigan, I. Jerrett. R. Jones
P. Ladds, M. Lancaster, C. Lenghaus, I. Links, S. McOrist,
L. Melville, G. Mitchell, M. Nunn, P. Phillips, R. Rahaley
R. Reece, J Seaman, H. Smith, E . Straube, R. Sutton, V, Tham
R. Whittington.

(2) Apologies

R. Badman, R. Campbell, M. Hindmarsh, J. Humphrey, K. Johnston,
T. Nicholls, G. Riffkin, J. Searson, L. Sims, R. Webb, J. Webber.

(3) Minutes of the 1st A.G.M. held In Sydney. Sunday 20th May 1984, accepted as a true record (Harrigan - Giesecke).

(4) Business arising from Minutes - none.

(5) Treasurer's Report
Moved that the report be accepted (Rahaley - Kerrigan). Carried.

(6) Membership
Current membership is 135.

(7) Elections

President: J. Glastonbury.
Secretary: I. Links.
Treasurer: R. Whittington.
Committee: J. Searson.
Committee: R. Cook.
(Gleeson - Friend). Carried.

(8) General Correspondence

(a) Financial members of AAVP were re-imbursed.

(b) Association with A.C.V.Sc. R. Rahaley president of Pathobiology Chapter. Predicted exciting period, objective to expand membership. Past President of Chapter attending Council meeting in NZ where the principle of introducing fellows to the Chapter without examination, as proposed in the Jubb letter, would be discussed. Moves in all States except W.A. towards registration of Veterinary

specialists (in hands of Veterinary Boards). Phil Ladds stressed that members of the Chapter and ASVP should be aware that "unqualified" pathologists do work in Australian Veterinary Laboratories.
General correspondence was accepted (Phillips - Carrigan).

(9) Industrial Registration
Bill Dobson an Industrial Consultant addressed the meeting. He talked about the industrial laws and systems and he indicated that they could be used as a vehicle to establish and promote professionalism. He discussed the advantages of being recognised by a statutory organisation. Establishes exclusivity.

A motion was passed "That the ASVP investigate the issue of industrial recognition and registration." (Kerrigan - Gleeson). Carried.

(10) Membership Fees
Moved that the fee remain $10.00 (Rahaley). Carried.

(11) Slide of the Month
C. Huxtable offered to continue to co-ordinate service. Some slides were of poor quality and staining. Some were broken in transit (recommended that tops of containers be taped and cotton wool used as padding). State representatives could include comments from members about the monthly slides in the newsletter . Any controversies could be discussed at Annual Meeting.

(12) Vet. Path. Report
Excellent in current format. Four per year.

State Representatives:

NSW	M. Carrigan
QLD	R. Kelly
S.A.	P. Phillips
W.A.	D. Pass
N.T.	O. Williams
VIC.	S. Friend
TAS.	D. Obendorf

(13) General Business

(a) New Zealand Pathology and Slide Registry. Hartley Leaving NZ in September. Service taken over by Keith Thompson. Hartley establishing a registry of marsupials/ native birds/zoo animals at Taronga Park.

(b) Lymph Node Pathology in Cattle. Phil Ladds made a request For interesting and unusual pathological lesions in bovine lymph nodes for the AMRC atlas of lymph node Pathology.

(14) Next Meeting

Resolved that the next meeting should be in Brisbane, back to-back with another conference. The theme will be immunopathology.

R.T. JONES
Public Officer.

INCOMING PRESIDENTS REPORT.

The new executive of the Australian Society for Veterinary Pathology is gradually assuming responsibility for maintaining the Society in its present healthy state. On behalf of all members we would like to thank Ian McCausland and the inaugural executive for placing the Society on such a solid foundation. Two areas that have already exercised the minds of the executive have been the planning for the conference in 1986 and the possibility of establishing an Australian Registry of Veterinary Pathology.

In this issue of The Veterinary Pathology Report we will explore these two item· in greater detail. Also included are reminders about the Australian College of Veterinary Scientist's examinations Phil Ladd's request for examples of pathological lesions in lymph nodes, and your subscriptions to the Society. Finally we have the "answers" for the cases presented at the 1985 Conference and the very valuable contributions from the State Correspondents.

ANNUAL CONFERENCE 1986.

The Annual Conference of the A.S.V.F. will be held on 17th and 18th May, 1986 in Brisbane. This is the weekend immediately following the A.G.M. of the Australian Veterinary Association. Please note these dates in your diaries and make a firm commitment to attend.

Immunopathology will be the theme for the first day of the Conference. We would appreciate receiving your ideas on the format for this day and possible guest speakers. With regard to the format two alternatives present themselves:

1. The present format of having one or two guest speakers accompanied by cases contributed by members.

2. Because many of us are relatively inexperienced in the field of immunopathology we would attempt to attract more guest speakers, perhaps aided by suitable demonstrations.

It is hoped to provide the answers for the immunopathology cases prior to the conference in 1986 so that members can read about the subject and take a more active part in the discussion.

The second day of the Conference will follow a similar format to previous years. If you have an interesting case or startling research findings start getting your ideas together now.

Remember the 1986 Conference of the Australian Society for Veterinary Pathology will be in Brisbane on 17th and I8th May, 1986.

AUSTRALIAN REGISTRY OF VETERINARY PATHOLOGY.

At the workshop conducted by Bill Hartley in Sydney in May, 1985, the idea of establishing an Australian Branch of the Australasian Registry of Veterinary Pathology was floated. Participants from all States agreed that Sydney would be a suitable location for such a registry. Accordingly the executive of A.S.V.P. has taken steps to bring the idea to fruition.

Generous donations, each of $1,000:00 have been obtained from the New South Wales Department of Agriculture and the Post-Graduate Foundation in Veterinary Science, Sydney University. This money will be used by Bill Hartley to copy "Kodachromes" relevant to Australia, presently held in the Registry in New Zealand. Hopefully this most valuable work will be completed by Bill before he returns to Australia during September 1985.

Mr. George Knowles, Director General of Agriculture in New South Wales, has kindly agreed to the Registry being temporarily housed at the Regional Veterinary Laboratory, Glenfield. It will be relocated with the RVL to the John Macarthur Agricultural Institute currently under construction at Camden.

The executive feels that the establishment of this Registry is an exciting prospect for Veterinary Pathology in Australia. It will allow the accurate documentation of the wealth of pathological material generated in this country over the years. It will also open an ideal avenue for training of veterinarians in pathology. Because of its attachment to a veterinary diagnostic laboratory, close proximity to the University of Sydney and the relatively central location of Sydney for pathologists from throughout the country, we are sure that the John Macarthur Institute will provide an ideal home for the Registry.

Obviously there are many details yet to be finalised. It is hoped to resolve these when Bill returns to Australia. However, in the end it will be your Registry, so If you have any comments or suggestions please let us know the before September.

AUSTRALIAN COLLEGE OF VETERINARY SCIENTISTS.

Applications to take the Preliminary or Membership Examinations in 1986 close with the:

Chief Examiner (Dr. E.W. Moodie),
Australian College of Veterinary Scientists,
P. 0. Box 34,
INDOOROOPILLY. QLD. 4068

on 1st September. 1885.

Final details of the Membership Examinations are not available but they will be held in January/February, 1986.
If you have any queries about the format of the examinations, or require application forms please contact:

John Glastonbury,
Regional Veterinary Laboratory,
Private Mail Bag,
WAGGA WAGGA. N.S.W. 2650. (069 230929)

Applications for Fellowship Examinations in 1986 have now closed but the Chief Examiner will be able to answer any queries.

Within the Pathology Chapter you can nominate the particular speciality in which you wish to be examined. These are not strictly defined but are assessed by the Chief Examiner for their suitability. Possible areas would include pathology, bacteriology, virology, parasitology, clinical pathology etc. The examinations consist of a written component (essay type, probably 3 hours·, with some choice, held at a venue local to the candidate, tentatively on 28th January, 1986) and an oral/practical examination (tentatively 20/21st February, 1986 in Sydney for Australian candidates and Palmerston North for New Zealand candidates).

The award of Membership of the College indicates that the person has demonstrated competence superior to that of his/her colleagues in some area of veterinary activity. Candidates must produce evidence of a satisfactory background in their chosen field, and in their examinations, must demonstrate that they have a sound theoretical understanding of the subject and that they would be competent to handle practical problems. Criteria suggested include:

a. The candidate should demonstrate knowledge and skill at a level which would make him or her a person suitable to give their immediate professional colleagues advice on problems in the areas of work concerned.

b. In terms of under graduate examination·, the candidate should perform at the 7O percent, or credit, or third class honours, or five (on a scale of seven) level.

c. The candidate should give evidence of having read in the general area of his work.

One further criterion for membership is that by the time of examination you must have been engaged in a full-time veterinary occupation for a period of at least four years. The cost of the Membership Examination is $200 while the Preliminary Examination is free.

The ball is firmly in your court. Help make the Pathobiology Chapter a strong vital organisation and College Membership/Fellowship worthy recognitions of expertise in Veterinary Pathology. Apply now.

Post-Graduate Refresher Course on Gross Pathologv.

Tom Hungerford has recently contacted us about a proposed refresher course in Gross Pathology to be organised by the Post-Graduate Foundation with the assistance of Bill Hartley and our association. As his letter contained the statement, "as pathology is the very basis of veterinary science", how could we refuse to help? The course has been tentatively set down for 18-20 May. 1987, in Sydney.

At this stage the programme is being drawn up and possible lecturers are being selected. If you have any ideas about topics which should be covered, suitable speakers, or the overall format could you please let Bill Hartley or the Executive know as soon as possible please.

If this course is in fact held in Sydney in May, 1987, it will be possible to organise our Annual Conference for one of the adjoining weekends. This should ensure an excellent turn-out for both events.

SLIDE OF THE MONTH.

In consultation with Clive Huxtable it was decided that a more detailed history and description should be included by the submitter for circulation with the slide of the month. We feel that the areas to be covered should include history, description of the significant histopathological findings, morphological and aetiological diagnosis, comments and references. The paper could be folded in the normal manner so that only the contributor and history is visible.

We have two main reasons for proposing this change. It will assist our junior members in thoroughly working up their cases and assessing the merits of those from other institutions. Consequently, it should help people in their preparation for the membership examination of the Australian College of Veterinary Scientists. Secondly, it was proposed at our recent annual meeting that there be more discussion on the "Slides of the Month" via our Vet. Path. Reports. A more concise manner of setting out the contributor's findings and the reasons behind their diagnoses should promote more logical discussion.

Any pertinent comments on the slide of the month should be referred to the secretary for publication in the next Vet. Path. Report.

BOVINE LYMPH NODE PATHOLOGY - (See Vet. Path. Report October, 1984).

Phil Ladds would still like to receive submissions for his colour atlas of bovine lymph node pathology to be published with AMRC funding. Slides, blocks, wet tissues, and/or colour photographs are required of the range of bovine lymph node lesions seen in Australia. Phil can be contacted C/- Graduate School of Tropical Veterinary Science, James Cook University of North Queensland, Townsville, Qld. 4811 - phone (077) 814428.

DIARY OF COMING EVENTS.

We would like to include a diary of coming events, visits of overseas experts etc. in future editions of Vet. Path. Report. Please advise the secretary of any items likely to be of value on a State, Federal or International basis. We also intend to send copies of the Vet. Path. Report to veterinary pathologists from Australia and New Zealand currently resident In North America. Please advise if you would like to be included.

VET. PATH. REPORT PUBLICATION DATES.

JULY, OCTOBER, JANUARY, APRIL.

TYPING OF VET. PATH. REPORT SUBMISSIONS.

It is easier to edit the report if submissions are in a standard format. We would prefer letter Gothic 12 type (Prestige 12 or similar if Gothic unavailable) with single spacing.

MEMBERSHIP FEES.

The membership fee is still $10.00 and was due on 30th June, 1985. Please fill in the invoice attached to the bottom of the application for membership sheet. Please Pay PromptIy. Members who do not pay will be removed from the mailing list.

NEW MEMBERS.

Under our constitution, new members must be nominated and seconded by current members. An Application Form and membership criteria are attached. Please make new members of staff at your institution aware of the ASVP and encourage them to join.

FOWL PLAGUE OUTBREAK - Bendigo, May, June 1985 (prepared by Bob Jones).

The recent outbreak of fowl plague involved a single poultry complex of layer, broiler breeder, broiler grower and processing works. A total of 116,000 birds comprising 39,000 layers, 16,000 broiler breeders end 61,000 broilers were present on the property at the start of the outbreak.

Upper respiratory tract disease, primarily sinusitis, had been present since 17th May in broiler breeders in four sheds (Sheds 7, 8, 9 & 10) and in broilers (Sheds 13 & 14). Increasing morbidity due to upper respiratory tract disease caused the owner to instigate parenteral medication with injectable Tylosin and Streptomycin to 5,000 30-week-old broiler breeders (Shed 7) on 23rd May. The following day the birds were sprayed with malathion for control of lice and given levamisole in the drinking water. Over the following weekend a very high mortality rate was recorded in this shed with deaths approaching 75% of total.

In the three remaining broiler breeder sheds (Sheds 8-10) morbidity and mortality rates had increased over the same period but were well below those in Shed 7.

The clinical signs varied depending on age and breed of bird affected. Broiler breeder hens and cocks all showed signs referable to upper respiratory tract involvement with swollen wattles, swollen infraorbital sinuses and eyelids, crackling respiratory sounds and head shaking. Mucus was often present exuding from the nares. Cyanosis of the combs and wattles occurred terminally and was more marked in cocks. Torticollis was present in a small number of cocks. Heavy mortality was seen in caged laying hens on June

1st. Many live birds were very depressed, were hot to touch, and had prominent oedema of combs and wattles. Wattle and comb cyanosis was also seen.

Onset of signs and death appeared to occur more rapidly than in broiler breeders.

Two broiler breeder hens from each of Sheds 7 & 8 were submitted for autopsy by the owner on May 27th. The changes seen were marked fibrino-necrotic cellulitis beneath the skin over the head and neck. Eyelids, wattles and combs were oedematous and fibrino-purulent exudate was present in infraorbital sinuses. Oedema was present in the neck. Hyperaemia of the trachea was variable. Visceral lesions were not present in all birds but when present consisted of marked fibrinous airsacculitis, pericarditis and peritonitis, congestion and consolidation of the lungs and splenic enlargement. Dead broiler breeders had similar changes but in addition showed marked muscular congestion and greater incidence of airsacculitis and peritonitis. No haemorrhages were found in broiler breeder birds.

Caged layer hens destroyed for pathological examination had marked oedema of wattles and combs, petechial haemorrhages on the serosal and mucosal surfaces of the proventriculus, petechial haemorrhages of the vent and thoracic pleura, and fragile ovules, many of which had burst into the abdominal cavity. Dead birds from the same cages had marked fibrinous peritonitis, markedly hyperaemic tracheas with haemorrhage into the tracheal lumina as well as fragile ovules and oedema of the neck, wattle and combs. Not all the changes described occurred in every bird.

Bacteriology was carried out on the early submissions (May 27 and 29) but failed to indicate a bacterial cause for the peritonitis and airsacculitis. Haemophilus paragallinarum was isolated from nasal sinuses and eyes of some birds.

Histopathological changes were commonly present in the wattles, eyelids and skin consisting of a subacute vasculitis and cellulitis with a marked perivascular mononuclear cuffing reaction. In the trachea there was marked submucosal mononuclear cell accumulation and degeneration and necrosis of tracheal epithelium. Changes in visceral organs were non specific and consisted of increased mononuclear cell aggregations in the liver and acute fibrinous perihepatitis, airsacculitis and pneumonia.

The gross changes found in these birds have been attributed to Avian Influenza virus (A.I.) which was present in both layers and broiler breeders. We cannot be sure that all the changes described are due to A.I. as both Mycoplasma gallisepticum and Haemophilus paragallinanum are known to be present on the property and their contribution to the pathology must be taken into account. What is interesting is the variation in clinical signs and lesions amongst different ages and breeds of birds and the lack of proventricular and tracheal haemorrhages fitting the written description of fowl plague amongst broiler breeder birds.

The primary clinical sign and lesion of fowl plague in the outbreak was wattle, comb and head oedema.

FISH PATHOLOGY WORKSHOP, RVL BENALLA. MAY 1985.

The RVL Benalla, under the auspices of the Standing Committee on Agriculture, hosted a fish pathology workshop from 27-30 May, 1985. Dr John Humphrey, who is in charge of the Australian Fish Health Reference Laboratory organised the workshop. The AFHRL is funded by the Commonwealth Department of Health (Quarantine) and the Victorian Department of Agriculture and Rural Affairs and is incorporated into the fabric of the RVL Benalla.

The course aimed to familiarise practicing diagnostic pathologists with fish pathology techniques and to give these people the confidence to perform routine fish pathology. The course also aimed to demonstrate the complexities and difficulties of fish pathology and so make these diagnostic pathologists aware of their limitations and when they should seek assistance from the AFHRL.

The 3 ½ day program was very intensive and involved lectures and workshops from 8.30am until l0.00pm.

An overview of aquaculture established that aquaculture will be an intensive area of activity and growth in the next five years. Following the overview, the problems and mechanisms of export of eyed ova to the Northern Hemisphere were covered. The next section involved the anatomy, histology and histopathology of fish and this was followed by workshops on bacteriological and parasitic techniques used in the diagnosis of diseases of aquatic animals.

Lectures and workshops on the second day involved diseases of native and ornamental fish and salmonids.

The third day involved an excursion to a trout hatchery to investigate the practical problems related to the discovery of an exotic disease in a commercial enterprise. The practical and bureaucratic problem raised by this exercise was brought home to a11 who attended.

The final session on Thursday consisted of lectures on the diseases of molluscs and crustaceans and, most importantly, a review and discussion of achievements of the workshop by those attending.

As the workshop was held under the auspices of the Standing Committee on Agriculture, a report and recommendations will have to be forwarded to the SCA. The meeting agreed on nine recommendations and these will be submitted to the SCA at a future date.

The notes from the workshop will be made available as soon as possible and will be available through Dr John Humphrey at the RVL Benalla (057} 62 2933.

RENAL PATHOLOGY, CASE REPORTS, ANNUAL CONFERENCE ASVP MELBOURNE MAY 18TH, 1985 - Histopathological findings, morphological and aetiological diagnosis and comments:

Submitter.
W.R. Kelly
Dept. Veterinary Pathology Public Health, University of Qld., ST LUCIA, 4067

Histopathology.

Glomeruli - generalised and diffuse thickening of glomeruli basement membranes and parietal layer of Bowman's capsule microfilaria were present in many glomeruli.

Scattered small interstitial foci of mononuclear leucocytes, particularly plasma cells, mild interstitial oedema.

Haemosiderosis proximal and distal
Haemoglobinuric nephrosis.
Small foci mineralisation medulla.

Diagnosis.

1. Dirofilaria immitis infection.
2. Membraneous glomerulopathy.
3. Haemoglobinuric nephrosis.
4. Chronic multifocal non-suppurative interstitial nephritis.
5. Microangiopathic haemolytic anaemia.

Comment.

Electron micrographs - swelling of endothelium, thickening of glomerular basement membrane.

Two components - pre-renal
Renal - haemoglobin casts, directly toxic and causes obstruction, ischaemia.

Microangiopathic haemolytic anaemia due to presence of worms.

Submitter.
L. H. Badcoe
University of Melbourne, Veterinary Clinical Centre,
Princes Highway, Werribee, Vic. 3030

Histopathology.

Proteinaceous to hyaline casts. Nephrosis causing cellular casts in medulla.
Multifocal interstitial granulomas - lymphocytes plasma cells, eosinophils, multinucleate giant cells, cortex and medulla.
Cortex interstitial fibrosis and granulomas caused pressure atrophy and loss of tubules, distal convoluted tubules dilated with hyaline casts.
Eosinophils.
Medulla - interstitial pale homogeneous material - amyloid?
Ischaemic glomeruli, infarction.
Hyperplasia of collecting tubular epithelium.

Diagnosis.

1. "Hairy vetch", Vicia villosa. poisoning.
2. Multifocal chronic granulomatous interstitial nephritis.
3. Hyperplasia of collecting tubules.

Comment.
Tubular rupture/herniation due to ischaemia rather than toxic, may also occur in immunological conditions. Immunological basis for lesions most likely according to the submitter.

Submitter.
P.W. Ladds,
Graduate School of Tropical Veterinary Science,
James Cook University, Townsville, Queensland, 4811.

Histopathology.

Mass consisted of variable sized follicles surrounded by simple low cuboidal to cuboidal epithelium containing eosinophilic colloid. Resorption vacuoles present at periphery of many follicles. Follicles were present in lymphatics at periphery. No mitotic figures were found. Hyperplasia with pseudostratification in a small number of follicles.

Other sections, large areas of coagulative pressure necrosis. Some foci with increased amounts of interstitial tissue.

Diagnosis.

Metastatic thyroid adenocarcinoma?

Comment.

Immunoperoxidase stain for thyroglobulin was equivocal.
Teratoma?

Submitter.

J. Boulton
Regional Veterinary Laboratory
Wollongbar. N.S.W.

Histopathology.

Severe congestion. Subcapsular metanephric mesenchyme. Slight ballooning mucoid degeneration of renal pelvic epithelium. Many glomeruli contained fibrin thrombi in capillaries. Slight nephrosis loops of Henle and hyaline droplet change straight segments of proximal tubules.

Small numbers of interstitial vessels contain fibrin thrombi, medial oedema and endothelial necrosis.

Diagnosis.

1. Erysipelas.
2 Intravascular coagulopathy.
3. Schwartzman reaction.
4. Renal hyaline droplet change.

Comment.

Erysipelothrix rhusiopathiae virulent strains produce neuraminidase which split membranes →vascular damage. Intact bacteria activate Hageman factor. Clotting cascade thus stimulated.

Tom Horsfall protein→ hyaline casts → lower nephron.

Submitter.

R.J. Whittington
Regional Veterinary Laboratory,
Wagga Wagga. N.S.W. 2650

Histopathology.

Foci of mineralisation medulla. Fatty change proximal tubules. Small multifocal interstitial foci of mononuclear leucocytes. Slight segmented nephrosis straight segments of proximal tubules. Many nuclei of proximal tubular epithelial cells and to a lesser extent distal convoluted tubular epithelial cells contained large eosinophilic inclusion bodies.

Granular cast formation.

Diagnosis.

1. Lead nephrosis
2. Renal fatty change.
3. Chronic multifocal non-suppurative interstitial nephritis.

Comment.

Rarely find the inclusions with Z.N. if not visible with H & E.

Osteoclasts best site to search for lead inclusions.

Submitter.

W.R. Kelly,
Dept. Veterinary Pathology & Public Health,
University of Queensland,
ST LUCIA. QLD. 4067

Histopathology.

Atrophy of medulla with much interstitial hyalinized atrophy. Suppurative pyelitis. Wedge shaped areas of interstitial fibrosis, extending to cortex where base of wedge formed. In these areas, small numbers of mononuclear leucocytes, atrophied tubules, obsolescent glomeruli, bile pigment. Focal periglomerular fibrosis with hyperplasia of parietal Bowman's capsule.

Mineralisation medullary tubules. Tom Horsfull protein casts.

Metastatic mineralisation gastric mucosa.

Diagnosis.
1. End stage kidney.
2. Acute suppurative pyelitis.
3. Renal mineralisation.
4. Renal bile pigment staining.

Comment.
No specific morphological or aetiological diagnosis. Renal dysplasia, chronic tubular disease and infarction were presented as possible primary aetiologies.

Submitter.
P.T. Hooper,
Veterinary Research Institute, Park Drive,
Parkville, Vic. 3052

Histopathology.

A. Karyomegaly of tubular epithelial cells and some glomerular cells. Tubular regeneration. Proteinaceous casts.
B. Kidney, generalised proliferative glomerulonephritis, karyomegaly. Metaplasia of parietal layer of Bowman's capsule to cuboidal cells.

Liver, diffuse fibrosis, karyomegaly, fatty change.

Diagnosis.

1. Pyrrolizidine alkaloidosis (Crotolaria sp.).
2. Generalised proliferative glomerulonephritis (B).
3. Chronic toxic hepatopathy (A).

Comment.

Metaplasia of Bowman's capsule not published previously.

Submitter.

J. R.W. Glastonbury,
Regional Veterinary Laboratory,
Wagga Wagga. N.S.W. 2650.

Diagnosis.

1. Phytogenous chronic copper poisoning.
2. Haemoglobinuric nephrosis.
3. Renal hyaline droplet change.

Submitter.

I. J. Links,
Regional Veterinary Laboratory,
Wagga Wagga. N.S.W. 2650

Histopathology.

Marked tubular dilation and atrophy. Oxalate crystals subepithelially and in lumens. Proteinuria interstitial oedema. Neutrophils in collection tubular lumens. Cellular casts, small number.

Diagnosis.

1. Oxalate nephrosis.

Comment.

Not birefringent but optically active crystals.
Dilation very important.
Acute deaths first 1-2 days. Die from hypocalcaemia. More chronic deaths, renal failure.

Submitter.
M. Carrigan, K. H. Walker and I.R. Littlejohns,
N.S.W. Department of Agriculture,
Agricultural Research & Veterinary Centre,
Forest Road,
ORANGE. N.S.W. 2800.

Histopathology.

Foci of mineralisation medulla. Multifocal non-suppurative chronic interstitial nephritis. Glomeruli were hypercellular in mesangial areas, lymphocytes and proliferation. Equivocal thickening of basement membranes.

Diagnosis.

1. Mucosal Disease.
2. Mesangioproliferative glomerulonephritis.
3. Multifocal chronic non-suppurative interstitial nephritis.
4. Renal mineralisation.

Comment.

Mucosal disease virus could not be confirmed as the inciting agent of this immunological condition because fluorescent antibody stains were not performed, electron micrographs not examined and viral antigen not eluted from the kidney.

Submitter.
R. T. Badman,
Regional Veterinary Laboratory,
Bendigo. Vic. 3550

Histopathology.

Acute necrosis of most of cortex, "tongues" of normal tissue remaining at corticomedullary junction. Severe interstitial congestion and haemorrhage. Possible fibrin thrombi in ghosts of glomeruli. Thrombosed vessels.

Diagnosis.

1. Renal cortical necrosis.
2. Haemophilus suis septicaemia.
3. Disseminated intravascular coagulopathy.

Comment.

Pathogenesis - multiple small infarcts that coalesce. Generalised Schwartzman reaction requires a primary or priming dose followed by a second dose. Not immunological. Can’t be transferred by hyperimmune serum.

Submitter.
P. T. Hooper,
Veterinary Research Institute,
Park Drive,
PARKVILLE. VIC. 3052.

Histopathology.

A. Tubular lumens widely dilated. Several extensive segmental necrosis of proximal tubular epithelium.

Numerous proteinaceous casts. Slight mononuclear leucocyte response interstitially. Slight tubular regeneration.

B. Slight evidence of regeneration, segment piling up of cells. Epithelial cells of tubules were generally plumper with vesicular nuclei which had prominent nucleoli. Slight interstitial mononuclear leucocytes, response. Exceedingly eosinophilic proximal tubular epithelium - hyaline droplets? Isolated hyaline casts. Dilation of tubules. Cytomegaly.

Diagnosis.

1. Nephrosis (amino acid induced).

Comment.

Restricted to the rat. Rat kidneys - remove some nephrons surgically, remaining nephrons will become necrotic.

Submitter.
C. Mitchell.
Regional Veterinary Laboratory, Benalla.

History.

A 3 year old female quarter-horse presented with weight loss and was subsequently necropsied.

Gross Pathology.

Roughened irregular surface. Cortices reduced in width. Cut surface of cortex had a granular appearance.

Histopathology.

2. Thickening of the glomerular capillary basement membranes.
3. Thickening of basement membrane of parietal layer of Bowman's capsule. Crescent formation.
4. Segmental granular positive staining for IgM, capillary basement membranes and mesangium.
5. Electron dense subendothelial deposits, splitting of the basement membrane, deposits in mesangium, together with fibrillar material. Fusion of epithelial foot processes.

Diagnosis.

1. Chronic membranoproliferative glomerulonephritis.
2. Equine infectious anaemia.

Comment.

Acute exacerbation of a chronic disease process. Chain specific IgM immunofluorescent.

RENAL PATHOLOGY: ANNUAL CONFERENCE ASVP MELBOURNE, MAY, 1985.

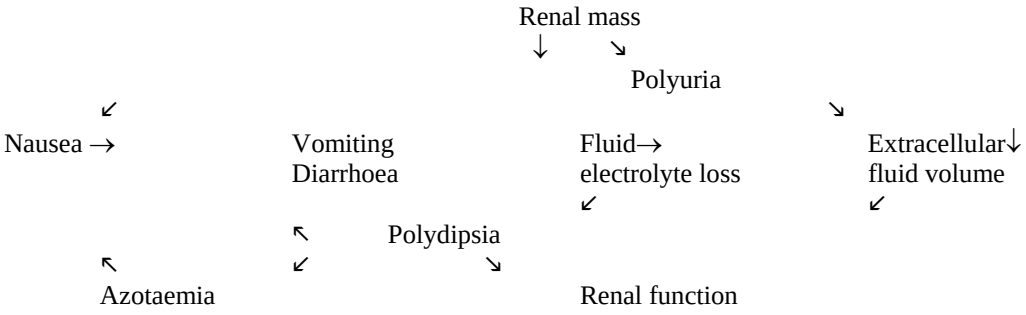
"EXTRA-RENAL EVIDENCE OF RENAL FAILURE."

Louie Filippich - University of Queensland.

Must look at other organs to diagnose renal failure at post-mortem.

Extra-renal Lesions of Renal Failure.

1. Dehydration.



2. Emaciation.
   Reduction calorific intake. ↓
   Alimentary tract changes → vomiting and diarrhoea.
   Reduction in basal metabolic rate due to decrease in glucose utilisation and/or Na. transport.

   Abnormal metabolism
   - carbohydrate - insulin resistance
     metabolic acidosis
   - nitrogen - insulin resistance
     hyperglucogonaemia
     growth hormone
   - fat

3. Alimentary Tract Changes

   a. Halitosis - alaphatic amines by bacteria metabolising creatinine.
   b. Stomatitis - necrotising lateral medial border of tongue, vascular problem.
   c. "Gastroenteritis" - ulceration, oedema, congestion, bleeding.

4. Anaemia.

Not correlated well with a degree of renal failure - normochromic, normocytic, non-regenerative.

Mechanisms - I
   Renal mass ↓ → erythropoetin ↓ → erythropoiesus ↓

   Chemical factors modified → reduced survival of erythrocytes.

Insidious toss of blood from alimentary trace, primarily due to platelet dysfunction, the platelets themselves are abnormal.

5. Cardiovascular.
   Hypertrophy of myocardium.
   Not well studied.

6. Osteodystrophia.
   a. Renal osteodystrophy.
   b. Metastatic calcification.
   c. Dystrophic calcification.

Ca, PO_4,. PTH interaction.
PO_4, excretion $\downarrow$ plasma PO_4 $\uparrow$ $\rightarrow$ plasma Ca^{2}+ $\downarrow\rightarrow$ PTH $\uparrow$
$P0_4\uparrow$ excretion, Ca from bone, reduces Ca excretion in urine, calcium absorption $\uparrow$

At 25% GFR, PTH can no longer control plasma PO_4

a. Renal Osteodystrophy.

Rare at p.m., die of renal failure before it develops.

Due to PO_4, $\downarrow$ excretion, decrease in 1:25 $(OH)_2$
D_3 $\rightarrow$ Ca absorption and resorption $\rightarrow$PTH $\uparrow$

1:25 $(OH)_2$ D_3 controls Ca absorption at duodenum/jejunum level.

b. Metastatic Calcification.

Abnormal chemical balance$\rightarrow$ mineral deposition in normal tissue. Occurs in stomach, lungs and kidney.

Calcium/phosphate product > 5.65 $\rightarrow$ soft tissue calcification. Still need a predisposing factor which is pH. Above 3 tissues have an alkaline phase because they excrete acid.

c. Dystrophic Calcification.

Pulmonary aorta, pleura.
Retention of N_2 containing metabolites plus local cellular structural changes. These are not pH dependant sites.

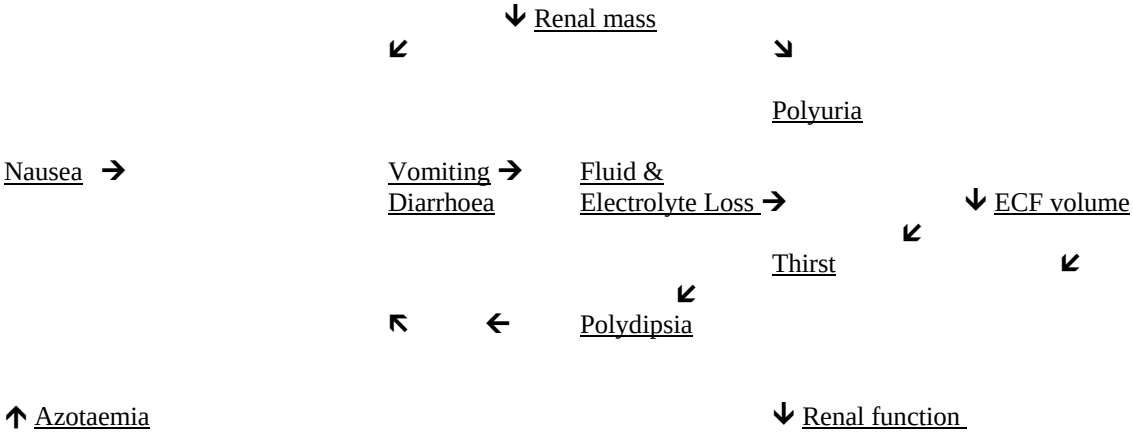
7. Uric Pneumonias / Pulmonary Oedema.

Coagulative defect? Pulmonary oedema relatively early manifestation, dilation of lymphatics.

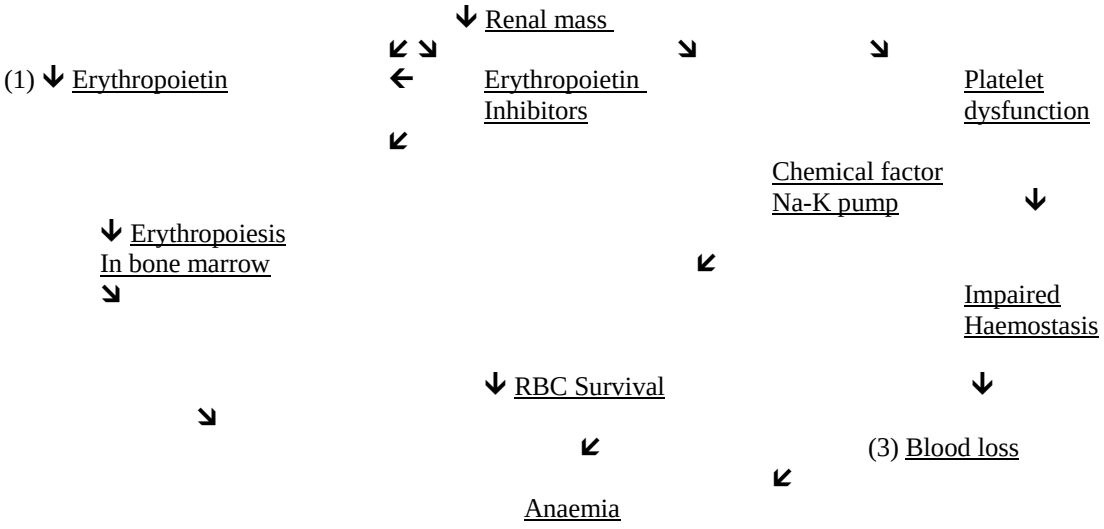
8. Perirenal Oedema.

Ruminants.

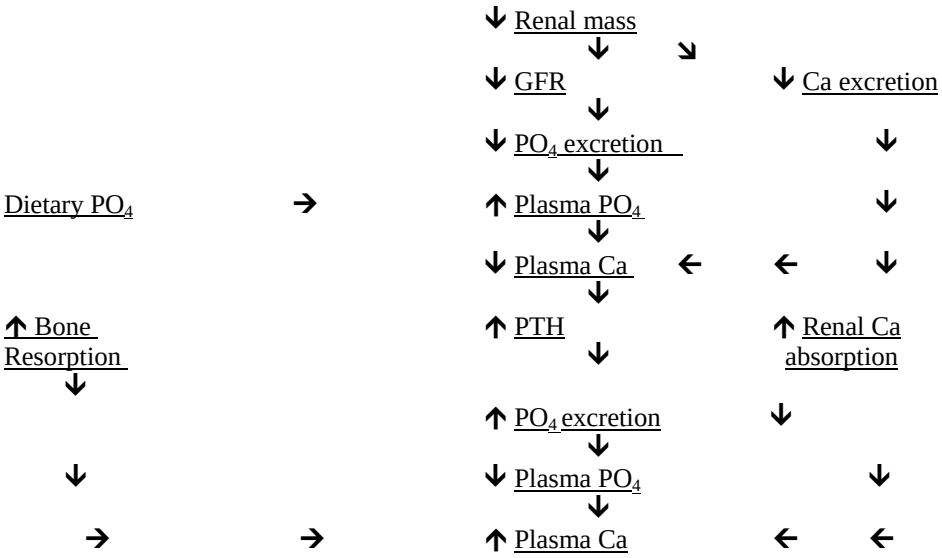
(A) DEHYDRATION



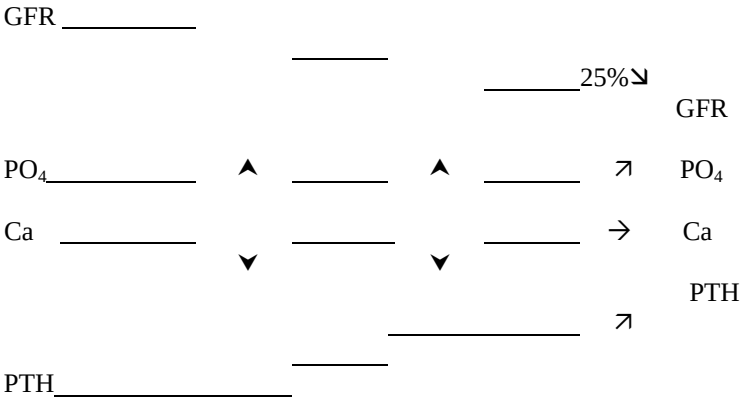
Anaemia in Renal Disease



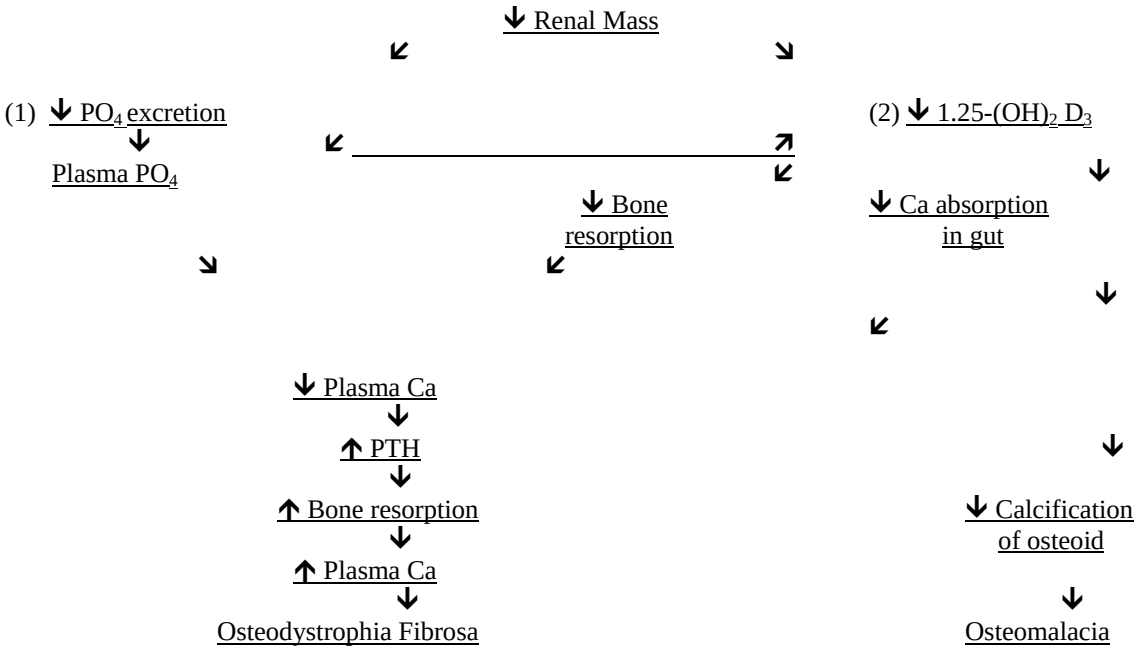
Phosphorus - Calcium - PTH axis in Renal Disease



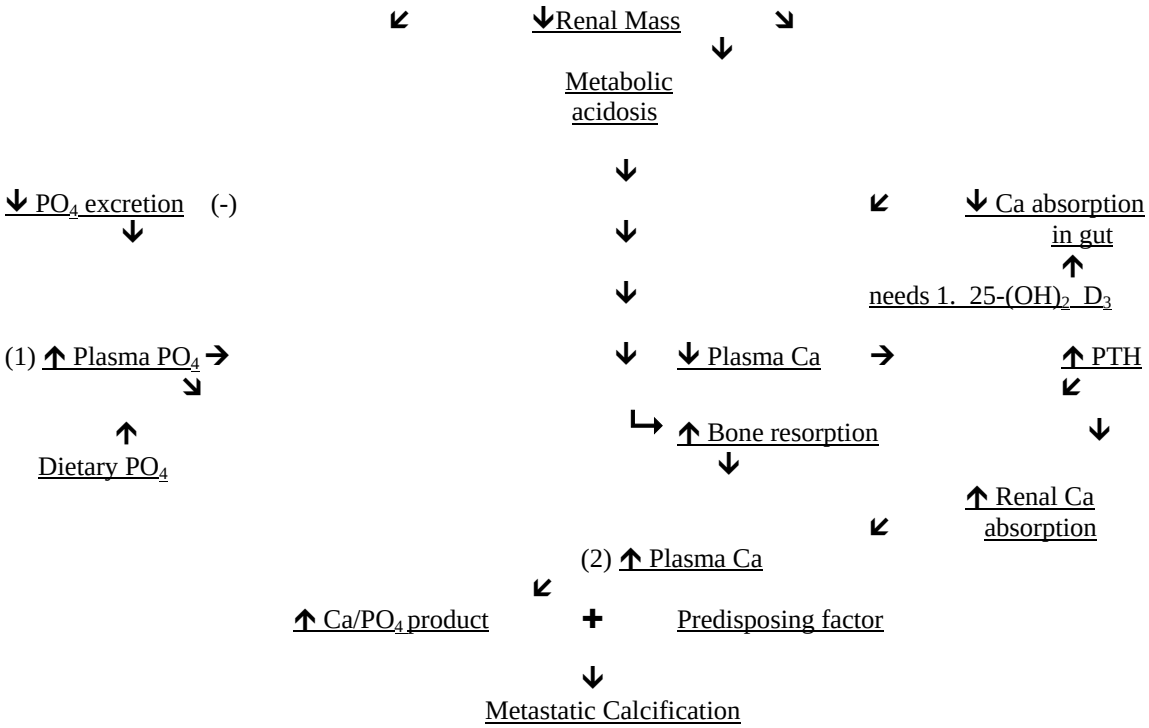
Pathogenesis of Secondary Hyperparathyroidism in Renal Disease.



Pathogenesis of Renal Osteodystrophy



Pathogenesis of Metastatic Calcification



HISTOLOGICAL TECHNIQUES

While on the subject of renal pathology Dr John Dowling from The Royal Melbourne Hospital suggested the use of Masson's trichrome or combined sliver methenamine/Masson's trichrome or similar stains to demonstrate fibrin and basement membrane/fibrin respectively in thin sections of kidneys. It appears that his techniques have not yet been published. Does anyone have a routine method for these stains? Also is there a foolproof method of ensuring adhesion of thin sections to the slide during repeated washing steps? Do you have experience with the use of microwaves to speed up fixation of tissues and performance of special stains (e.g. ZN, silver stains). If so please advise the Secretary for inclusion in the next Vet. Path Report.

STATE REPORTS.

QUEENSLAND

ARI. Yeerongpilly (Fraser Trueman): Three outbreaks of suspected toxic algal poisoning in cattle have been investigated. In one of these, 30 animals died after showing jaundice and photosensitization. Histopathology revealed severe liver injury with periportal fibrosis, bile duct hyperplasia, bile retention and vacuolation of remaining hepatocytes. In all three outbreaks cattle were drinking at dams with distinctly green-coloured water. Ancystis sp. and Anabaena sp. were identified in water samples on occasions.

Sporadic outbreaks of encephalomyocarditis virus infection have been recorded in S.E. Queensland piggeries. In one instance, 90 growers died, and the virus was isolated from rat faeces collected at the piggery.

Vet. School, Q'ld. Uni.: While on the EMC theme, some grower pigs submitted here with EMC showed remarkably similar gross pathology to that of mulberry-heart disease.

NEW SOUTH WALES - Prepared by Mark Carrigan.

STAFF

Peter Mylrea, formerly Director, Animal Health Research has been appointed Deputy Chief, Division of Animal Health and commences duty on July 1. Steven Hum, a Hungarian graduate, has taken up a 2 year temporary appointment as Veterinary Research Officer at the RVL Orange.

DISEASE HIGHLIGHTS

Vitamin A Deficiency in Pigs

In the last 6 months two cases of Vitamin A deficiency in pigs have been investigated in the central area of the State. The problem occurred on two intensively run piggeries both of which were feeding home prepared feed.

In the first piggery, the diet consisted of wheat, 4% meat meal and a food additive of minerals and vitamins. Over a 3 to 4 month period 5 gilts had developed a posterior paralysis which progressed to lateral recumbency within 1-2 weeks. Host pigs exhibited a slight head tilt, to the right or left, in the terminal stages. Necropsy findings on the gilt presented for post mortem included evidence of increased C.S.F. pressure with coning of the cerebellum and flattening of the ventral aspect of the brain stem. In the bladder there were numerous yellowish nodules 1-2mm in diameter adherent to the epithelium. Histopathologically the most significant CNS changes were located in the cervical spinal cord - there was dilation of the central canal and leakage of CSF with resultant cavitation of the dorsal and dorso-lateral grey and white matter. Large focal areas of malacia with moderate glitter cell response were seen in the grey matter lateral to the cavitation. Moderate to severe Wallerian degeneration in the cervical cord, with mild Wallerian degeneration in the thoracic and lumbar cords. Bladder - thickening of the transitional epithelium due to epithelial hypoplasia and foci of keratinisation of the surface epithelium. Biochemical analysis of liver detected no Vitamin A.

In the second piggery the food was a home mix of 900kg wheat, 50kg meat meal and 50kg of blood meal. The problem was seen in 5½ month old pigs which had been on the above diet since weaning. Over a month period the pigs (40-50) had developed clinical signs - initially these included coughing in the majority of pigs, this did not respond to antibiotics. At five months of age the majority of pigs developed a severe watery diarrhoea and 3 pigs showed hind limb weakness which progressed to paralysis. Two pigs were autopsied - one had severe diarrhoea and was coughing, the other had posterior paresis. Both pigs had mild coning of the cerebellum and the pig with diarrhoea had intestines distended by very watery contents. There was no evidence of pulmonary pathology. A pathogenic Escherichia coli was cultured from the G.I.T. of both pigs. Histopathology in the pig with paralysis showed severe Wallerian degeneration in all white matter tracts in the thoracic cord, with mild Wallerian degeneration in the cervical and thoracic cord. In the scouring pig there was mild Wallerian degeneration at all levels of the spinal cord. Liver vit. A levels were 0.25 m moles/kg in the paralysed pig and 4.08 m moles/kg in the scouring animal.

Suspect 2, 4-D Poisoning in Dogs

The death of 6 adult kelpies and 7 pups was investigated. These animals presented with severe diarrhoea, conjunctivitis and mild keratitis. Necropsy findings included a mild gastritis and enteritis in all dogs, with an ochre coloured liver and pale kidneys in pups, while the adult dog had congested liver and kidneys. The main histopathological changes were in the liver. In the liver of a pup there was a severe necrotising hepatitis with the normal sinusoidal architecture obliterated due to a marked swelling of hepatocytes, many of which (>50%) were undergoing coagulative necrosis. Bile duct epithelial cells were moderately hypertrophied and there appeared to be slight proliferation of the bile ducts. No inclusion bodies were detected. Further investigation by the submitting practitioner revealed that the dried dog food had probably been contaminated with 2,4-D (Estercide 80 - approximately 1 litre). Dogs are the most susceptible animals to 2,4-D poisoning and the LD50 is l00mg/kg for a single dose but 20mg/kg repeated daily quickly causes clinical signs.

Congenital Blindness - White Shorthorns

Ray Webb, at the Orange laboratory (063 - 636764) is currently collecting information on and cases of a condition of congenital blindness in shorthorn cattle. Affected calves are full term and appear normal except they are totally blind. The condition is one of retinal dysplasia. All affected calves have white coat colour; they are not albinos. Investigations to date indicate that it is possibly of genetic origin, probably autosomal recessive. It has a number of features different to a condition described by Leipold et al

(Am. J. Vet. Res. (1971) *32* : 1019
Cornell Vet (1974) *64* : 367
Can. Vet. J. (I974) *15* : 2:34)

He currently has 4 bull and 2 heifer animals and would be grateful to hear of any similar cases that may be encountered by others. Any notes on "Favourite Techniques" for processing eyes would also be appreciated.

TASMANIA

Prepared by David Obendorf

1. Ross River Virus Infection is suspected in causing fever, leg stiffness, inco-ordination and temporary algactia in a mare on Tasmania's East Coast. The mare was one of a sentinel herd to monitor RRV infection. Prior to February 1985, RRV titres were negative or <1:10. On 20th February her titre was 1:320. Mosquitoes collected from the paddock where the horses were sentinelled yielded RRV, the virus-carrying species was Aedes flavifrons.

2. Neonatal Canine Herpes Virus infection vas diagnosed in a 2 week old puppy. Blotchy eccyhmotic haemorrhages occurred throughout the renal cortex end serosa of the small intestine. The liver was congested and the lungs moist. Diffuse hepatic, splenic, myocardial and pulmonary foci of necrosis were observed. Eosinophilic intranuclear inclusions were associated with foci of necrosis particularly in the liver.

3. Mortalities in caged birds (budgerigars and finches) due to intestinal coccidiosis have been investigated this autumn. Severe haemorrhagic enteritis accompanies the development and shedding of the coccidia. Other deaths in both parrots and budgerigars have been attributed to heavy burdens of Ascaridia platycerci. Burdens up to 1800 adults and immature nematodes have been removed from individual birds. Mucosal damage, intestinal impact ion, perforation of the bowel and biliary migration may occur in these circumstances.

4. Yersinia pseudotuberculosis, Y. enterocolitica, Haemolytic E. coli, Pseudomonas and Salmone1la (Group B) are regularly isolated from weaner sheep and young cattle with primary parasitic gastro-enteritis.

5. Acute fluke in lambs. Good seasonal conditions in southern and south eastern Tasmania have allowed fluke transmission to begin again. A recent consignment of 6 to 8 month old lambs from the Bridgewater sale was heavily infested with fluke. Many carcases were condemned outright due to poor body condition, internal adhesions and peritonitis. In some lambs immature fluke had burrowed across the diaphragm and commenced tracking in the lungs!

Possible Toxoplasma abortion in a cow.

Two cows in a dairy herd aborted in May 1985, one on 5/3/85 and one on 15/3/85. The foetus from the latter had Listeria monocytogenes in its abomasum, but no bacteria were cultured from the first foetus. However, this foetus did have a non-suppurative encephalitis and myocarditis suggestive of a protozoan abortion. Relevant serological results were:-

	25/3/85	30/4/85
Cow 1 Sarco CFT	-	-
Toxo IFAT	1/32	1/8
Cow 2 Sarco CFT	-	-
Toxo IFAT	1/16	1/8

These suggest Toxoplasma infection in the recent past.

Cattle are regarded as being relatively resistant to Toxoplasma infection and rapidly eliminate the organism from their tissues. Hence, they do usually have persistent serological titres for Toxoplasma.

Most evidence to date suggests that protozoal abortions in cattle are due to a Sarcocystis sp. which has not yet been completely identified. In this instance there is a suggestion that Toxoplasma gondii could have been involved.

(AmyI) oodinium infestation in sea-caged rainbow trout.

During late summery/early autumn, rainbow trout held in sea-cages in south-eastern Tasmania suffered substantial mortalities.

The outstanding lesions were gill inflammation, hyperplasia and fusion. These changes were associated with vast numbers of protozoan parasites identified as (amyl) oodinium.

At the time of the outbreaks weather conditions had been consistently warm with clear sunny skies. However, the water temperatures were not excessive for trout.

However, a consistent finding was that the nets used on the cages were heavily fouled with algae and seaweed; much more than would have been expected for the time they had been in the sea. This led to reduced water exchange and a build up of waste food and faecal material. Obviously, the combination of poor environmental conditions and parasite load led to the outbreaks.

It has been suggested that the fouling of the nets probably was due to a combination of:

(a) suitable substrate (i.e. the structure of the net cords)
(b) long hours of sunlight
(c) possible phosphate build-up from run-off from the surrounding farmland

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VICTORIA Prepared by S. Friend

BAIRNSDALE REGIONAL VETERINARY LABORATORY (Rob Seller)

AHTHRAX OUTBREAK:- In a11, eleven cattle died suddenly in area spanned by four neighbouring properties in South Gippsland. Anthrax was confirmed in four cattle (including three bulls) on two properties. Necropsy showed peritoneal haemorrhages with haemorrhage into the intestines, and an intensely congested spleen. Histologic changes were congestion and haemorrhage with large numbers of bacilli in the spleen. Blood smears from affected stock showed large numbers of capsulated sporulated rods. 1

B. anthracis, which was typically rough on blood agar and mucoid on bicarbonate agar, was isolated. Infective blood from carcasses killed guinea pigs in 24 hours with terminal septicaemia. The bacteria isolated are being used to produce a fluorescent antibody suitable for anthrax, which should make diagnosis easier in future, particularly differentiation of B. anthracis from B. cereus. One of the properties was connected with a swamp which has been used as a carcass dumping site for some years - probably since settlement began in the area. The swamp was implicated previously in a 1941 anthrax outbreak in the area, and in a 1976 outbreak on an East Gippsland property, where agisted cattle involved also came from this area. All cattle on the quarantined, and on some neighbouring properties, were vaccinated and there have been no further suspicious deaths.

B0VINE ABORTIONS:- Most of the diagnostic material seen at this time of year is from abortions. Fungi (Aspergillus spp and Mortierella wolfii and Campylobacter foetus are the most common infectious agents isolated. M wolfii has a greater predilection for systemic dissemination to the viscera and brain, and fatal pneumonia due to M. wolfii is occasionally seen in the aborting cow. A serologic test developed at this laboratory, is proving to be a sensitive indicator of M. wolfii infection and abortion. Protozoal abortions have also been quite common and histologically there are characteristic foci of necrosis and gliosis scattered in the foetal brain. In only 10% of cases can protozoal organisms be found in the. lesions. Experimental studies to date have suggested that these are probably Sarcocystis cruzi.

POISONING OF WILD BIRDS:- Poisonings continue to be a significant cause of mortality in wild birds. Separate incidents recently in eastern Victoria have included rainbow lorikeets, magpies, rosellas and mountain ducks. Necropsy findings usually consist of moderate congestion of the lungs, and evidence of recent ingestion of grains, corn or other toxin coated materials in the crop. Normal ingesta are often present in the gizzard. Analysis of crop contents, or suspect bait, is usually the most rewarding. Organophosphates or metaldehyde are the usual toxins detected, and all these incidents were possibly due to landowners or orchard owners poisoning to reduce bird damage to their crops. Prosecutions are rare due to the frequent lack of admissible
evidence.

HAMILTON REGIONAL VETERINARY LABORATORY (Cor Lenghaus)

PARVOVIRUS INFECTIONS

The essential differences between parvovirus infection of kittens and pups were well illustrated recently. Three 4-week old kittens from one litter were submitted alive with varying degrees of ataxia, tremor and inco-ordination. These kittens had all been apparently healthy 6 days previously. All of five 3-week old silky terrier pups in one litter died suddenly after a short period of crying out. Two pups were submitted for examination.

At post mortem the cerebellar folia of the kittens were less well developed than expected. Histologically there was total (premature) absence of the external granular cell layer of the cerebellum. The molecular layer, Purkinje cell layer and internal granular cell layer were partially degenerate or dysplastic. Focal mineralization was present in the cerebellar folia. One kitten had typical parvovirus intranuclear inclusion bodies in cardiac myocytes, without any associated inflammatory response. This kitten also had severe thymic atrophy, not present in the others.

Gross post mortem changes in the pups included wet lungs and areas of pallor in the left ventricle, extending from the coronary groove to the apex of the heart. Histologically there was degeneration of myofibres, oedema and some diffuse inflammatory cell infiltration in the heart. Parvovirus intranuclear inclusions were present in a small number of myocytes. Lesions in other tissues were attributable to acute heart failure.

Both cerebellar hypoplasia and myocarditis are now comparatively rare because of immunity after natural exposure or vaccination of queens and bitches. The reasons for overt parvovirus disease referable to such distinct, specific, target tissues is not well understood, but probably relates to the host-specific ontogeny of both these target tissues and the immune response.

SCHOOL OF VETERINARY SCI. MELBOURNE {Bruce Car. S. Friend)

Caprine Galactostasis

A clinicopathological study of "hard-udder" (in goats is in progress at the VCC. Twelve goats with “hard-udder” have been donated to the Veterinary School and material has been taken for virological, serological and light and electron microscopic examination. Eight of the goats had clinical and histological findings consistent with “big-knee“. Three goats were positive, on the agar gel immunodiffusion test (AGID) for Contagious Arthritis-Encephalitis (CAE). Previously, positive goats and goats with seropositive relatives have been negative on the AGID test.

Mammary pathology was similar in a11 cases. There were small amounts of normal secretory tissue juxtaposed to secretory tissue in various stages of involution. There was marked thickening of interlobular septa, which contained many lymphoid follicles and were most prominent around lactiferous sinuses. Large corpora amylacea were numerous in affected glands. Pending further results we feel that “hard-udder” may be another manifestation of the CAE complex.

Canine Toxoplasmosis

A 12 week-old boxer pup was presented to the VCC with profound muscle weakness, atrophy but no pain. The dog was leukopenic, neutropenic, lymphopenic and hypoproteinaemic. Serum electrophoresis results revealed low albumin and Y globulin but increased a globulin levels. Cerebrospinal fluid examination revealed a pleocytosis and increased protein and creatine phosphokinase (CPK) levels. Serum CPK values were also elevated. The dog died and was necropsied. Mottling of the skeletal muscle was the only change observed at post mortem, however, microscopic examination of the tissues revealed a severe necrotizing and granulomatous meningoencephalitis, pneumonitis, myocarditis and polymyositis with focal but extensive hepatic necrosis. Although many cysts and bradyzoites were seen in most tissues, the large numbers of cysts and the severity of the myositis were spectacular. Later it was discovered that the dog had been treated with steroids. The toxotitre was 1/128. In contrast, another puppy with toxoplasmosis was submitted shortly after the first case, with severe jaundice. It had a severe necrotizing myocarditis, with large numbers of toxoplasma cysts. There was extensive hepatic necrosis, but cysts were difficult to find.

BENALLA REGIONAL VETERINARY LABORATORY.

Porcine Angiopathy (Malcolm Lancaster):

A group of seven-week-old piglets showed nervous symptoms ranging from head tilt to recumbency with mild convulsions . Some were also scouring. Two neurologically abnormal animals were necropsied. Moderate mesenteric oedema and enlarged oedematous mesenteric lymph nodes were present in one.

Chronic oedema disease was diagnosed on the basis of the histological lesions. Arterioles in the brain, intestine, lymph nodes and liver showed medial degeneration or hypertrophy, sometimes with adventitial mononuclear cell accumulation.

Mixed faecal flora was cultured from the intestinal tracts. While a heavy growth of beta-haemolytic E. coli is associated with acute oedema disease, this may not be the case in the chronic form. The occurrence of oedema disease in Victoria in the last decade has apparently been rare.

Geeldikkop in sheep (Christopher Button):

An outbreak of hepatogenous photosensitivity in sheep occurred in the Cobram area in February, 1985. Clinical and biochemical findings and post-mortem liver lesions were typical of a condition known as "geeldikkop" (yellow, swollen head). The condition is reputed to be the result of simultaneous ingestion of Tribulus terrestris ("caltrop" or "bindii") and the sporidesmin-containing spores of the fungus Pithomyces chartarum. Approximately 50 first-cross weaner lambs were affected and 28 died.

T. terrestris is an annual herb which grows after summer rains. In the Cobram outbreak, it grew on the margin of a stubble field irrigated from an adjacent orchard. P. chartarum is an ubiquitous fungus which flourishes under conditions of high temperature and humidity.

WESTERN AUSTRALIA, (prepared by David Pass)

Murdoch. WA

Oedema disease in Pigs (7). An unusual form in pre-weaned pigs.

In recent months, a large intensive piggery has experienced a dramatic rise in pre-weaning mortality from 14% to 30%. May of the dead pigs have been 2-3 weeks old and have been in excellent condition. Most of the pigs were found dead but others were found recumbent and exhibiting chronic convulsions with periods of rigidity and opisthotonous. There were no gross· lesions present, but in a number of animals, focal areas of acute necrosis were present in the brain stem, predominantly in the medulla. Distinct vascular lesions were not seen in the majority of sections. One pig, however, had lesions consistent with the cerebral angiopathy of oedema disease. Pure growth of E. coli or mixed growths, predominantly E. coli was obtained from gut, brain, lung and kidney of numerous pigs. Several of these isolates of E. coli were typed. All the isolates were 0141. This serotype has been associated with oedema disease elsewhere.

Suspect Vitamin A deficiency in ducks.

A commercial duck producer had 90 young adult Pekin-cross ducks with bilateral "conjunctivitis". The birds were anorexic and lethargic. The diet consisted of bran and pollard and there was no access to any green feed. Only one bird was necropsied. Thick yellowish caseous plaques were present over the base of the tongue and pharyngeal mucosa. The lacrimal ducts were distended with dry friable material and small yellow plaques were present on abdominal air-sacs. Histologically, there was extreme squamous metaplasia of epithelial glands typical of vitamin A deficiency.

Dept. of Agriculture. W.A.

(1) CHRONIC CALCIUM DEFICIENCY OF SHEEP

Experiments at Wongan Hills Research Station by field veterinary officers have duplicated the reported laboratory appetite simulating effects of adding 2% finely ground limestone as a supplement to sheep fed ad lib oats only rations.

(Peet et al 1984 A.V.J.61 : 195, and Peet et al, 1985 A.V.J. 62 : 138).

The previous field experiments over the Summer/Autumn of 1983/84 failed to show any significant difference in growth rate or feed intake between groups, but unseasonable intermittent rain over that period provided green pick in the stubble pastures and a probable source of calcium.

This Summer/Autumn (1986/85) has been more typical with drought-like conditions applying from December onwards. The supplemented group of 50 weaner sheep gained an average of 3 kilos per head over the oats only group after 70 days - mimicking the laboratory findings.

(2) PORCINE ATROPHIC RHINITIS IN WESTERN AUSTRALIA (R.B. Richards)

Atrophic rhinitis was diagnosed in two W.A. pig herds. Clinical signs included depressed growth rates, epistaxis and shortening and lateral deviation of the snout. Grossly there was virtually complete absence of both dorsal and ventral nasal turbinates with remnants visible as small 1 x 2 cm leaflets in the posterior nasal cavity. The histologic lesion was consistent with an atrophic process and although the nasal mucosa had mild multifocal inflammation, inclusion bodies were not present. Pasteurella multocida isolated from the nasal cavity of a number of pigs and several isolates were toxigenic for the mouse test; one was also dermonecrotic for guinea pigs and cytotoxic for Vero cells. It is believed to be the first diagnosis of severe atrophic rhinitis in Australia associated with typical clinical signs and the presence of toxigenic P. multocida.

(3) TORTICOLLIS IN GOATS (Prepared by D.P. Rasali, Nepalese Veterinarian)

A syndrome where goats develop torticollis has been seen in Western Australia in a commercial milking herd of 80-90 milkers and about 200 young replacement animals. It was claimed by the owner that cases were recognised before visual signs were apparent and were treated ineffectually with antibiotics but successfully with cortisone.

Usually young goats were affected and some matured with permanently twisted necks. The ligamentum nuchae of these animals was taut and the cervical spine curved to the side. It could be held straight without the animal showing any discomfort.

Out of 13 dead goats examined, six were adult and the others 4 months to 1 year old. Intestinal contents from two of the adult goats gave positive entero-toxaemia tests (type D). Salmonella orion was isolated from the intestinal contents of the same two goats and two other young goats.

One two year old maiden doe which recovered from torticollis the previous year redeveloped the condition about two weeks prior to death. It was found to have bony thickening of the right arch of the fourth cervical vertebra. Some adjacent muscles showed mild individual fibre necrosis. The vertebrae of an 8 month old animal which had a twisted neck for 2 days were boiled. They were found to be soft enough to crumble on squeezing with fingers. The cortical bone of these vertebrae was very thin. Blood calcium levels of 4 affected goats were slightly below the normal range of 9.5 to 10.5 mg%. Examination of brains, segments of spinal cords and middle ears from the affected animals showed no indications of any change.

Although there were concurrent problems of enterotoxaemia and Salmonella infection in the flock, it was recognisable that bone abnormality was present in vertebrae of two affected animals. An imbalance of dietary calcium and phosphorus is likely to be the aetiology of the condition. Similar isolated cases have been seen in Victoria and in New Zealand but the cause has not been determined (Sandra Baxendell, Personal Communications). We would be interested to know any other information on this condition.

(Replies to Mrs D. L. Hopkins)

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY
Incorporated in Victoria

Telephone:
(069) 230920
Telex:
69714 (Disvet)

Regional Veterinary Laboratory,
Private Mail Bag,
Wagga Wagga,
NEW SOUTH WALES, 2650.
AUSTRALIA.

EXTRACTS FROM THE RULES OF THE ASVP
APPLICATION FOR MEMBERSHIP

3. (1) a. A person who -
i. holds a Degree of Veterinary Science registerable within Australia, and
ii. is actively engaged in, or has had a minimum of 2 years experience in gross, microscopic or clinical veterinary pathology, and
iii. is nominated and approved for membership as provided in these rules is eligible to be a Full Member of the Society on payment of the annual subscription.

b. A person who -
i. holds a Degree of Veterinary Science which is not registerable in Australia, and
ii. is actively engaged in, or has had a minimum of 2 years experience in gross, microscopic or veterinary clinical pathology, and
iii. is nominated and approved for membership as provided in these rules is eligible to be an Associate Member of the Society on payment of the annual subscription. Associate Members have all rights and obligations of Full Members except that they may not vote at any meeting and may not be elected to the Committee of Management.

c. Distinguished scientists who do not hold a veterinary degree may become Associate Members after a motion recommending them as such, is passed at a general meeting, and on payment of the annual subscription payable under these rules.

(2) A nomination of a person for membership of the Society:-
a. shall be made in writing in the form set out in Appendix 1, and
b. shall be lodged with the secretary of the Society
c. shall be accompanied by the sum payable under these rules as the first year's annual subscription.

ENTRANCE FEE AND ANNUAL SUBSCRIPTION

4. (1) The annual subscription will be determined by majority vote at the Annual General Meeting and is payable in advance before the first day of July each year.

(2) A list of non-financial members shall be issued on or about 31st December each year. Any member who has not paid the annual subscription within 28 days of this notice will be removed from the Register (see Rule 7 (1) (a).

(3) Any member removed under subclause (2) may be re-instated by -
(a) payment in full of all annual subscriptions owing, or
(b) re-nomination as described in Rule 3.

(4) A new member who joins the Society on or after 1st April shall not be required to renew the annual subscription until July the following year.

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY
Incorporated in Victoria

Telephone: (069 230920
Telex: 69714 (Disvet)

Regional Veterinary Laboratory
Private Mail Bag, Wagga Wagga,
N.S.W., AUSTRALIA.

I, …….....
(Full name)
of …….....
…….....
(Address)

desire to become a Full Member/Associate Member of the Australian Society for Veterinary Pathology. In the event of my admission as a member, I agree to be bound by the rules of the Society for the time being in force.

Place of employment …….....

Details of experience in Veterinary Pathology …….....
…….....
…….....
…….....

Academic qualifications (State granting institution after each qualification).
…….....
…….....

I enclose $10 as advance payment of the annual subscription.

…….....
(Signature of applicant)
…….....
(Date)

I …….....
Being a Full Member of the ASVP nominate the applicant who is personally known to me, for membership of the ASVP.

…….....
(Signature of Proposer)

…….....
(Date)

I …….....
Being a Full Member of the ASVP second the nomination of the applicant, who is personally known to me, for membership of the ASVP.

…….....
(Signature of Seconder)

…….....
(Date)

MEMBERSHIP RENEWAL.

The Treasurer,

Enclosed please find $10 for annual membership of the Australian Society of Veterinary Pathology to 30th June, 1986.
NAME …….....
ADDRESS …….....
PHONE NO: ……

Signature …….
Date …….