THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
the Department of Primary Industry, Tasmania,
Mt. Pleasant Laboratories,
P.O.Box 46, Kings Meadows, TAS. 7249.

Registered by Australia Post Publication No. VBG 6333

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Number 29 December, 1990

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DEADLINE FOR NEXT VET. PATH. REPORT IS MARCH 1, 1991

PRESIDENTS PAGE

The economy may be in recession but there are some bright spots on the veterinary pathology scene.

The new facilities at the Elizabeth Macarthur Agricultural Institute at Camden are first class. Post mortem, animal handling and disposal facilities are top class.

Central Veterinary Diagnostics in Brisbane have moved into new facilities in East Brisbane. Tragically their Adelaide laboratory was gutted by fire, but is now back in operation with the help of staff, clients and suppliers.

On the local scene, the Mt Pleasant Animal Health Laboratory has been commended for being highly respected by primary industry in Tasmania. The commendation is due in no small way to the efforts of our veterinary pathologists, Roy Mason, David Obendorf and Judith Handlinger.

While Roy Mason is in New Zealand in late November, he will represent the ASVP at the Annual Meeting of the New Zealand Society for Veterinary and Comparative Pathology at Palmerston North.

In this journal, you will find an article on specialist registration. This was present at the recent meeting of the sub committee on animal health laboratory standards (SCAALS). The article advances the views of the major employers of veterinary pathologists in Australia.

Merry Christmas
Best Wishes for 1991.

Rod Oliver
PRESIDENT

EDITORS REPORT

This VPR issue is the last for the year. I hope that 1990 has been kind to you. I would like to thank all our contributors around Australia and overseas who have taken the time and effort to send in case reports, news topics or letters to the editor. From the feedback we do receive from readers, the VPR serves a useful role, but please tell us if you want anything added, deleted or changed.

Did you notice that your last VPR issue was printed on recycled paper? Blame your "greenie" editor for that. At the moment it actually costs a little more than chlorine-bleached paper but as more environmentally-conscious organizations like us switch over to unbleached, recycled paper, the price will come down. Only a small step I know, but we've got to make a start!

Judging on the "Letters to the Editor" section in recent issues, it appears that opinions on specialist registration of veterinary pathologists and accreditation of VP's and Vet Labs are many and varied. I will not enter the fray, but I feel that the VPR has been a useful forum to air this topic.

I don't know about anyone else, but I think that Peter Phillip's Logo for the Society is pretty good, even Tassie gets included as an apostrophied P! If there are any challengers, send them to me or bring them· along to the next Conference.

Incidentally, preparations for the 1991 ASVP Conference at Elizabeth Macarthur Agricultural Institute in May are well in hand. For details, see page 5 (a registration form is also enclosed).

Thanks again and all the best for 1991.

David Obendorf
Honorary Editor

1991 ASVP SCIENTIFIC MEETING

THE AUSTRALIAN SOCIETY OF VETERINARY PATHOLOGY INC.
Scientific programme 11 & 12 May 1991

VENUE

ELIZABETH MACARTHUR AGRICULTURAL INSTITUTE - CAMDEN - NSW

Saturday morning 11 May 1991

ONCOLOGY

* UV radiation induced carcinogenesis
Joint presentation by Vivienne Reeve, Paul Canfield (Dept. Veterinary Pathology Sydney and Gavin Greenoak (Dept. Animal Science Sydney)
* Diagnosis of common canine cutaneous neoplasms, Terry Rothwell (Dept. Veterinary Pathology Sydney)
* Cytotoxic drug treatment of canine lymphosarcoma, Jill Madden (Dept. Pharmacology Sydney) Radiation therapy of small animal neoplasms, Graeme Allan (Dept. Veterinary Clinical Studies Sydney)
* Neoplasms of wildlife, Paul Canfield and Bill Hartley (Taronga Zoo)

Afternoon ONCOLOGY CONTINUED

* Tumours of large animals, Tony Ross (Elizabeth Macarthur Agricultural Institute Camden)
* Case Reports (Reports on endangered species welcome) Australian Society of Veterinary Pathology AGM

Evening - Social Function

Sunday morning 12 May 1991

* Principals of liver pathology. (General reactions of liver to injury, circulatory factors in liver disease plus examples of more recently recognized liver disease). Roger Kelly (Department of Veterinary Pathology Brisbane)

* Advances in hepatotoxicity. (Recent developments in diagnosis of pyrrolizidine alkaloid intoxication plus other results of other research in hepatotoxicity). Alan Seawright (Department of Veterinary Pathology Brisbane)

* Case Reports (Reports on endangered species welcome)

Please submit a resume of your case report to the Secretary ASVP by January 1991 for inclusion in the ASVP Proceedings publication.

SPECIALIST REGISTRATION

The following paper was presented to the inaugural meeting of the Sub-Committee of Animal Health Laboratory Standards by Russel Rogers of the Animal Research Institute, Queensland.

It raises a number of issues relating to the appropriateness of specialist registration for veterinary laboratory diagnosticians. The Sub-Committee on Animal Health Laboratory Standards as representatives of the major employer of veterinary pathologists in Australia has an interest in specialist registration (SCAHLS) as representatives of the major employers of veterinary pathologists in Australia has an interest in specialist registration.

Feedback to Russel Rogers (ARI, Yerongpilley) or Rod Oliver (DPI, Tasmania) would be welcome.

SPECIALIST REGISTRATION

The response to my discussion paper of 12th March was mixed in that some respondents took the view that the existing system established academic standards for competence and that evidence of ability to apply that knowledge was not an adequate substitute for academic rigour. However, there was a substantial response indicating that SCAHLS should discuss the issue in detail and reach agreement on the need for specialist registration and the criteria for this.

As the major employers of veterinary laboratory diagnosticians, I believe we have a moral responsibility to ensure our professional standards are not only high put also relevant to our needs. Some specific concerns with the ACVS route to specialist registration follow:

1. The existing system, with its emphasis on examination at fellowship level (2x3-hour papers, a practical and an oral examination) is appropriate for training institutions but not for the front line activities engaged in by diagnostic laboratories.

2. Ability to pass such examinations is not necessarily associated with ability to cope with Industry problems, or to be a productive scientist in terms of adding to knowledge.

3. The Pathobiology Chapter of the College has a reputation for imposing unreasonable standards in its examinations for membership. The ability to memorise detailed information, which is readily available in the literature, is of little value compared with the ability to elucidate complex or new disease problems or to perform productive research.

4. The largest body of laboratory diagnosticians is employed in State diagnostic laboratories whose activities differ substantially from those of universities and of private pathology laboratories. These differences include the broad range of disciplines and associated equipment, the substantial research activities and networks of regional laboratories in each State.

5. It is accepted that there should be some formal component for specialist registration to demonstrate sound knowledge of the discipline. My personal preference for this is American course work type post-graduate degrees. However, membership of the Australian College, in the appropriate chapter, or research degree, would be acceptable.

6. There should be a substantial requirement based on proof of relevant expertise through performance with the emphasis on commercial livestock. Methods of evaluating performance need considerable discussion.

7. Evaluation should be done by proven performers in the field of diagnosis and research into diseases of commercial livestock. Such people would be drawn from State Departments, CSIRO, Principal Laboratory Offices and the Australian Society for Veterinary Pathology. I do not believe university staff, practitioners or private pathologists are sufficiently familiar with the above activities to evaluate applicants.

8. State diagnostic services are undervalued in this country. My branch has approximately $700,000 per year in industry research funds. These are competitive and are allocated to proven performers. It is unreasonable to be judged by other than one's peers, or to be denigrated through ignorance.

9. Limited training resources should be directed to those areas most relevant to the employer's needs. We can no longer afford to do otherwise

The following statements are provided for discussion by SCAHLS:

1. The route to specialist registration via membership/fellowship of the existing subdivisions of the Pathobiology chapter is not the most appropriate for laboratory diagnosticians.

2. SCAHLS has a responsibility to dearly indicate the standards and skills it considers necessary for the efficient functioning of government diagnostic laboratories.

3. SCAHLS should consider developing a proposal for recognition by the ACVS of a new membership subject, entitled 'Diagnosis and Control of Livestock Diseases'.

NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY

TRAINING COURSES IN NSW

A highly successful training course was run by the Registry in early November. Twelve full-time and three part time participants, led by Bill Hartley reviewed the pathology of the central and peripheral nervous systems, skeletal and cardiac muscle, the foetus and placenta.

The course was held over five days at the Elizabeth Macarthur Agricultural Institute. There was a Kodachrome slide presentation by Bill Hartley, video microscope discussions and individual and group study on single and multiple head microscopes.

Pathologists from organisations (such as the universities) who are not currently contributing to the funding of the Registry paid a participation fee.

TRAINING COURSE IN VICTORIA

At the tine of writing a series of training courses were scheduled to be held in Victoria. Presentations and discussions at Bendigo, Benalla, AAHL and Attwood were followed by a more formal training course at Attwood.

SKIN CONDITIONS AND TUMORS

The Registry collection is deficient in most skin conditions and tumours. If you have common or uncommon cases of skin problems and neoplastic processes in companion animals, fast-gaited animals and farm animals; please send them to:-

Dr. W. J. Hartley
National Registry of Domestic Animal Pathology
Private Mail Bag 10
CAMDEN NSW 2570

Tony Ross.

PIG PATHOLOGY

A REQUEST FOR ASSISTANCE FROM LES SIMS

Many of you would have received a questionnaire regarding the production of a book on porcine pathology. The response to the survey was excellent, with many supportive comments and constructive suggestions received. Most respondents felt there was a need for the work. We now have to see whether the members of the Pig Research and Development Corporation (to whom we have applied for funding) feel the same way. To those who replied, I thank you sincerely, your contributions were invaluable in putting together the proposal.

For those who were not surveyed, the ultimate aim of this project is to bring together relevant practical information on the diagnosis of pig diseases so that laboratory and field workers have a one volume guide to assist them in their day to day work. At present, information on the pathology of pigs is fragmented, incomplete and variable in quality. This work intends to "fill in the gaps" and present the material in a clear and consistent manner.

The book would include chapters on the pathology of each of the body systems (following a standard format - e.g. normal structure, development and special features, effects of autolysis, responses to injury, significance of lesions, including differential diagnosis of specific changes etc.) and separate information on the specific diseases of pigs, including information on the best available tests for confirmation of a diagnosis.

It is anticipated that considerable use of the resources of the Australian Registry of Veterinary Pathology would be made and that the material used in the production of this book would be added to the collection of the registry. Members of the ASVP will play a key role as contributing authors.

If you feel that you have something to offer in the area of diagnostic pig pathology (e.g. good cases or expertise in a specific area) please contact Les Sims at the Bendigo Regional Laboratory (PO Box 125 Bendigo, Fax 054 484950). As yet, no contact with overseas members of the society has been made on this project and any information from across the Tasman or from North America would be greatly appreciated.

LETTERS TO THE EDITOR

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Dear Sir

The problem of evaluation of competence and the designation of specialist status on veterinary pathologists, so well aired in The Veterinary Pathology Report No. 28 raises many important questions. As a veterinary pathologist educated in Australia (BVSc, MVSc) and the second Australian to become a Diplomate of the ACVP (David Dodd was the first - although I always thought he was a New Zealander) I would like to point out some of the experiences in North America over the last 40 years. I have some perspective on this as I have been an ACVP Diplomate since 1963, served on the Examination Board for 4 years (1 year as Chairman) and have been the Editor of Veterinary Pathology now for 2 years.

The licensing "of specialists" always causes disruptions in a society because some people will inevitably be excluded and perhaps some who are included will later be judged to have been an unsuitable choice. Although the initial process is painful, no matter how it is done, there are always lessons to be learned from history. The early history of the American College of Veterinary Pathologists has recently been published in Veterinary Pathology 27(6). The article points out that the original members were "grandfathered" into the organization in 1949-1950, but this procedure was soon replaced by an examination. A totally different approach was used to identify the first clinical pathologists who were to become Diplomates of the ACVP. There was no grandfather clause. An examination was set by several prominent American clinical pathologists, whose competence in specific areas was above reproach. Those people who passed that examination, and some famous and well-known ones did not, then became the Examination Committee for the next year. Also, one or two of the original examiners never took the examination and never became full members of the ACVP. Despite that, the procedure was regarded as fair. In other words there was no grandfather clause for the clinical pathologists and only for a very short period for the anatomical pathologists after that college was started in 1949.

COLLEGE OF VETERINARY MEDICINE

The University of Tennessee Institute of Agriculture

The reason for classification as a specialist is really very important. One major stimulus here in the U.S. was the need to have competent people read and evaluate studies on drugs to be submitted to the Food and Drug Administration. This also provided an economic stimulus for people to make the sacrifice in time and money to prepare to take the examination. Whether or not this stimulus exists in the Australian scene, you can judge best, but it is very difficult to demand such a heavy sacrifice of individuals without there being some economic response to reward them later or even to recompense them for the time spent. Significantly, most Australian veterinary pathologists are employed in diagnostic laboratories. In the U.S., relatively few board pathologists work in these laboratories and there can be no doubt that the lower salaries play a significant role.

In an egalitarian society such as the one in Australia, the policy for selection becomes more difficult and this dilemma is reflected in the statements in the letters in The Veterinary Pathology Report No. 28,1990. For example, on Page 6 we read "we must not lose sight of the spectrum in considering the registration of veterinary specialists" and "insistence on rigid criteria for registration as a specialist is unworkable" and "the only standards worth achieving are those we set ourselves as individuals". On Page 7 there is the statement that "we search our souls for a workable, equitable and consistent registration system - which does not exclude the bulk of practicing and competent pathologists". If the position is to be taken, that the standard should be set to include the vast majority of the people working in pathology, rather than to set a high standard and then allow applicants to demonstrate proficiency, the veracity of the examination will undoubtedly come into question.

Standard of Examination. The setting of standards is always difficult but Australians and New Zealanders are used to harsh, anonymous examinations and the ACVP examination is of this type. The ACVP examination was not proposed to license competent pathologists but to identify the top quality pathologists. The ACVP has been most careful not to imply that "competency" was indicated by passing the examination as this would also imply that failure to pass indicated incompetence. This is certainly not so.

Despite all of this, I would earnestly commend a stiff examination as a method of evaluation. MS and Ph.D degrees are never sufficiently "broadbased" to evaluate this type of competence and frankly, in the case of the Ph.D., which should be a research specialist degree, usually in a well-defined area, it should not be able to perform this task. If the trainee had excellent experience in the Ph.D. program, this should mean that he/she should be able to handle a stiff examination, completely separate from that offered for the Ph.D. One area of real concern in any examination is the breadth of the examination as well as its depth. It is very difficult for pathologists working in industry and expertly trained in rodent pathology to become really familiar with the diseases of large animals. Now with the advent of marine animal pathology, the breadth and depths of the examination becomes even more of a problem. This matter has been discussed widely here but the final decision has been to leave the ACVP examination as it is - both extremely wide and requiring an extreme depth of knowledge.

Training. One memorable aspect about the ACVP and unusual for a North American organization, is that it has not been concerned with the mode of postdoctoral training. The only criterion is the examination. Approximately 15 years ago some people did raise the question of “authorizing” post graduate training and evaluating the “quality” of the training and facilities. However, there is a distinct penalty to pay for this approach because the authorization of training sites raises the idea of “implied warranty”. Thus, applicants could, and have in the past, thought that because they have attended a program which has been highly recommended, have paid the money and have worked hard, that they are, therefore, guaranteed a pass. In North America, most of the training faculties are supplied by agencies other than the American College of Veterinary Pathologists. For example, the C.L. Davis Foundation, the Armed Forces Institute of Pathology, and Cornell University all offer short courses and are involved in training in veterinary pathologists. It is a real advantage to completely separate the “licensing organization” from those promoting or authorizing training.

My personal view is that the Australian Society for Veterinary Pathology performs a remarkable service. It provides a home for all veterinary pathologists in Australasia, its publication Veterinary Pathology Report is one that I really look forward to receiving and the slide-of-the-month club is excellent and has no doubt been a real influence of elevating interest in proficiency in histopathology. Perhaps it should be the "working" organization for persons involved in veterinary pathology and specialist status be evaluated by the ACVSc. When I returned to Australia in 1964, I had visions of an Australian veterinary pathology organization -1 even thought it might be called "College of Australian (or Australasian) Veterinary Pathologists" but there were few potential members in those days. The situation is very different now. I would agree that the Australian College of Veterinary Scientists has done itself a disservice in requiring a two tier examination, specifically because the first examination does not confer a specialist status and thus is difficult to discern its use. Also, without the economic recompense to justify the time and the money spent on obtaining the training to pass the FACVSc examination, it may be difficult to entice potential applicants and perhaps this is what is happening now. In fact, the FACVSc (Veterinary Pathology) examination is of an extremely high standard and should be supported and continued.

However, the answer to the Australian dilemma is not to declare all persons working in veterinary pathology to be specialist pathologists and then expect to receive recognition. One possible approach is to establish, as has been done in North America, a group classified as “veterinary laboratory diagnosticians” who better recognizes the wide expertise of many of the veterinarians working in diagnostic laboratories, rather than to try to include them under the category of "veterinary pathologists". To misquote a previous Australian prime minister - Robert Menzies, "we will not become specialists merely by declaring ourselves to be specialists". This is another example where justice must be done and justice must be seen to be done. In other words, the "specialists" have to demonstrate that they really are specialists in the eyes of the public, the veterinary community and especially in the eyes of related health professionals.

Sincerely yours,

M.D. McGavin, M.V.Sc.. Ph.D M.A.C.V.Sc.
Professor and Director of
Necropsy Laboratories

MDMcG:jg

STATE REPORTS

WESTERN AUSTRALIA - Ruth Reuter

ANIMAL HEALTH LABORATORIES, SOUTH PERTH

Coccidiosis in camels (Ron Peet)

Two of 15 camels died suddenly at Geraldton, Western Australia. These animals were being used for tourist camel rides and showed no signs of ill health until found dead. Both animals had a bloody discharge from the anus. Postmortem examinations revealed blood clots and apparent haemorrhage in both small and large intestines. There were no other visible lesions and salmonellosis or an acute toxicosis was suspected. Fresh liver and a small intestine swab were submitted to AHL, but no Salmonella sp. was cultured and the liver was negative for arsenic.

Histopathology of small intestine from the same animal revealed a markedly haemorrhagic mucosa with numerous eosinophils present. Large Coccidia-type organisms (probably schizonts) were visible in these sections and there was an interstitial pneumonia in the lung with marked perivascular and peribronchial lymphoid/plasma cell aggregation. There were no other lesions in sections of brain, heart, large intestine, kidney, adrenal, rumen or spleen from this animal.

Severe haemorrhage with shock due to coccidiosis was considered to be the cause of death in these animals and faeces were collected from the other animals for parasitological examination. However, no oocysts were found and no further deaths occurred. The schizonts seen in the small intestine section were much larger than those commonly seen in cattle or sheep and are probably specifically camel coccidia, a number of which have been described (Boid et al 1985).

Reference

Boid R., Jones T.W. and Luckins A.G. (1985) Br. Vet. J. 141:87.

White Rhino Disease - a saga (Ron Peet)

A 22 month old white rhino female recently imported into the Perth Zoo began losing weight coinciding with loss of appetite over a 7 week period. She had apparent bilirubinuria and isosthenuria (Murdoch University Veterinary Hospital) and was given antibiotics for five days then oral "trimidine" for 14 days. During this period she was stung by two bees which caused a skin reaction. At about three weeks from the onset of inappetance, proteinuria but no bilirubinuria and an apparent "normal blood liver panel" were recorded. She was given cephalosporin for four days which produced diarrhoea – treatment stopped. At five weeks severe weight loss was noted and the animal refused to eat. However, she did drink electrolytes and was given more "trimidine, tagamet and mucotuss". The animal was then anaesthetised at, Murdoch University veterinary hospital and a suspected inflammation of the pharyngeal area diagnosed. Elevated BUN and creatinine levels but normal liver function tests were noted at this time. Given mycostatin with I/V fluids, oxytetracycline for four days, penicillin for 1½ days and other supportive pain killing and corticosteroid drugs. Faecal cultures on 4-5 occasions over this period were negative for Salmonella spp.

The animal unfortunately died on a wet Saturday morning. Postmortem examination revealed copious quantities of blood-stained fluid in the abdominal cavity with a markedly reddened gastrointestinal tract with splash haemorrhages on the serosal surface and distended haemorrhagic lymphatics. The kidneys were swollen with apparent subcapsular focal haemorrhages. The urinary bladder had a markedly oedematous thickened wall and apparent proliferative haemorrhagic mucosa. Opening the gastro-intestinal tract revealed an apparent haemorrhagic gastroenteritis with markedly thickened haemorrhagic mucosa in the caecum and colon.

These were the main gross changes apart from a circular apparent abscess approximately 5cm diameter in the prescapular muscular area. Histopathology of this lesion revealed myonecrosis with marked PMN/mononuclear infiltration and thrombosis of surrounding vessels. Histological examination of the gastrointestinal system showed a relatively focal haemorrhagic gastritis, but a severe diffuse haemorrhagic enteritis especially in the caecum and colon with marked fibrinoid necrosis of some mucosal and submucosal vessels with occasional thrombosis. There was a marked glomerulitis in the kidneys characterised by haemorrhage and necrosis of glomeruli and thrombosis of some vessels. Special stains (Von Kossa) also proved positive on gut sections revealing marked mineralisation of the muscular tunics. A severe haemorrhagic cystitis was present in the urinary bladder with fibrinoid necrosis and microthrombi in some vessels.

There was marked depletion of lymphoid follicles in the spleen with no evidence of regeneration and marked hypertrophy of the adrenal cortex of the adrenal glands with some haemorrhages. There were no other significant changes in other organs examined which included brain, liver, pancreas, heart, skeletal muscle and lung. Microbiology was relatively unrewarding:-
Klebsiella pneumoniae grew from an aspirate of urine from the urinary bladder, Strep group D from the caecum. Pseudomonas aeruginosa from the colon, n/h E. coli from mesenteric lymph node. No growth from liver and lung and various samples were also specifically cultured for Salmonella sp, Mycoplasma, Pseudomonas pseudomallei, Haemophilus Yersinia, Mycobacteria and anaerobes with negative results.

Fresh samples were also sent to AAHL where spleen and mesenteric lymph node samples were used to prepare 10% tissue suspensions in PBS and inoculated onto BTY, BHK, Vero, PK 15, MDBK and BK cell cultures. No cytopathogenic effects were observed in any of the cell cultures after two serial passages.

Fungi were cultured from the lung, but the animal had aspirated feed in extremis and in the light of negative histopathology, this isolate was not considered significant.

Diagnosis

None made, but gross and histological changes seen were considered to be possibly consistent with endotoxaemia, amyloidosis, uraemia, unknown toxaemia and an unusual form of malignant catarrhal fever - or iatrogenic factors.

Any other suggestions would be appreciated. Also I am intending to be "out of town" for the next rhino death and I still do not know who ended up with its horn - but it wasn't me!

Enterotoxaemia in ewes (David Forshaw)

A mob of 300 ewes with lambs at foot were moved from a paddock of very light feed to a lush clover pasture within which there were also large numbers of double gees. Over five days six ewes died exhibiting nervous signs of mild convulsions and frothing at the mouth before dying. The ewes had not been vaccinated. No lambs were affected.

Postmortem examinations of the ewes revealed swollen haemorrhagic kidneys, increased volume of pericardia! fluid and petechial haemorrhages through the mesentery and pericardium. Small intestinal contents were forwarded to the lab as well as a range of fixed tissues for histopathology.

Histologically, lesions of widespread vascular leakage were observed in the brain. Examination of smears of gut content failed to reveal any clostridial organisms but a mouse protection test on filtrates of gut content was positive for epsilon toxin. A diagnosis of enterotoxaemia was made.

In the meantime the farmer had drafted the lambs off and put the ewes back into the paddock with the light feed after vaccinating them with pulpy kidney vaccine. He left the lambs on the lush paddock and did nor. vaccinate them (despite advice to the contrary). 30 more ewes died over the next two weeks but no lambs died.

Presuming that all of the ewes died from the same cause why didn't the lambs also die from enterotoxaemia? Differences in diet?

Enterotoxaemia in marron? (Jeremy Langdon)

A low but noticeably elevated mortality occurred in marron (freshwater crayfish) over two weeks following feeding with split field peas. Moribund animals displayed ataxia and aimless limb movements. Necropsy revealed yellow pea oil in an otherwise empty proventriculus, and a nodular, bright yellow digestive gland ("hepatopancreas") surrounded by congealed haemolymph. Smears and sections contained masses of Gram negative rods and lipid droplets filling the digestive tubule lumena and replacing their necrotic lining epithelium. Pathologic change was generalized in most glands, but in a few cases some tubules were unaffected and certain animals recovered from the disease. Such animals contained sclerotic, mummified tubular casts amongst the normal digestive tubules when examined 12 months later.

The predominant bacterial species was Aeromonas hydrophilaa, which produces enterotoxin, haemolysin and protease. Bacteraemia was not a consistent or frequent finding. The likely pathogenesis, given the clinical signs, thus appears to be one of enterotoxaemia. The accumulation of pea oil in the proventriculus and digestive gland suggests incomplete digestion of the peas, which may have triggered the overgrowth of bacteria in the mid-gut digestive gland.

REGIONAL VETERINARY LABORATORY, ALBANY

Footrot eradication (Ruth Renter

Since the State Reference Laboratory for footrot is located at Albany, the recent decision of the WA Department of Agriculture to embark on a 10 year eradication programme for ovine footrot has had a dramatic effect on the Albany Laboratory. Eight new enthusiastic stock inspectors have joined the field services in the South Coast region and the results of their labours are starting to flow in to the laboratory. This has filled the void left by the decline in pathology specimens which, in my view, has been primarily associated with institution of laboratory charges coupled with the "rural crisis". In the short term the laboratory will be concentrating on footrot and, thanks to the excellent research work of our parasitologist Brown Besier, worms.

REGIONAL VETERINARY LABORATORY, BUNBURY

Mycotic rumenitis of red deer (Cleve Main)

Mortalities occurred in a group of 76 young red deer kept on a tourist-based farm sited on a flat low lying area with very little shelter. At the time the weather was cold and rainy. Violent downpours and strong cold winds frequently occurred. Prior to the deaths, the deer were given supplementary feed in the form of pellets and apples. The pellets were available in 3 X 4 gal. buckets (= 30kg), and the apples in a 4 gal. bucket.

Following the diagnosis of death by exposure in one of the animals, the deer were penned in sheltered yards and the rations increased to 7 buckets of pellets and 7 buckets of apples daily. The deer were fed directly from buckets rather than from a trough. Over the following 8 days several animals sickened and died until a total of 25 were depressed, reluctant to move and usually were found dead the next morning.

Postmortem examinations of early cases revealed large paintbrush haemorrhage over the serosal surfaces of the forestomachs and congestion of other visceral organs. More chronic lesions were characterised by severe peritonitis with marked adhesions of the omentum to the abdominal organs. In two of the deer hepatic necrobacillosis was also evident. The mucosa of the forestomachs was in many places stiff, matted and firmly adherent to the underlying tissue.

Histopathological examination of the rumen and reticulum revealed marked interstitial haemorrhage, vacuolation, vesiculation and necrosis of the epithelium as well as infiltration of polymorph neutrophils. Many of the mucosal blood vessels were thrombosed. The presence of branching non-septate fungal hyphae in these thrombi and in other areas was demonstrated with a Grocott's stain. In one deer fungal hyphae were demonstrated in a Peyer's patch associated with an area of intestinal haemorrhage and also within a parabronchial thrombus in the lung.

The lesions in deer with more severe chronic lesions were characterised by severe fibrino-haemorrhagic inflammation of the serosal and muscle layers. Thrombi and fungal hyphae were more numerous. Necrosis and infiltration of the full depth of the wall with polymorph neutrophils was widespread. In the liver there was focal coagulative necrosis. In these areas vasculitis, thrombosis arid fungal hyphae were evident.

SOUTH AUSTRALIA - Vin Ling Tham

Pinnal Vascular Disease (P. Phillips)

As a follow up to my previous article on this entity (VPR (1990) No. 27, p. 27) one aspect missing from my report was that a second practitioner had been consulted and treated the dog. This practitioner, once a dachshund breeder himself, phoned me and said he had seen this condition before and it always responded to treatment with thyroid hormones despite the dogs often having apparently normal T_3 and T_4 levels. His treatment of "Maverick" returned the dog's ears to normal, albeit with scalloped edges!

Mystery Slide January, 1990 (P. Phillips and V.L. Tham)

A couple of comments have been received following distribution of our January Slide-of-the-Month with a questionable arsenic aetiology. These comments coupled with a review of pathology of material from elsewhere leads us to suspect that the plant Lesser Loose-strife (Lythrium hysopifolia) was likely to be responsible for the renal pathology at least.

Thank you to those recipients who responded with their thoughts.

Equine Herpesvirus (EHV1) Abortions (Peter Phillips)

In mid-October, two mares from the same property aborted on the same day. One mare was due to foal on the 28th October and the other on 5th November. The aborted almost full-term foetuses were submitted as Mare Abortion Packages.

On necropsy, one of the foals had excess blood-tinged peritoneal fluid, a white filmy liver capsule and superficial petechiation of the spleen. The other was unremarkable.

On histopathological examination, one foal had widespread necrotising bronchiolitis with oedema and mixed inflammatory exudate in the lung, oedema and mononuclear inflammation in the perivascular areas of the liver and patchy necrosis in the thymus associated with eosinophilic intra-nuclear inclusions. The other foal's pathology differed in that the lung only had oedema and some inflammation while the liver had foci of hepatocellular necrosis associated with intra-nuclear inclusions. The thymic histopathology was as for the first foal.

The histological diagnosis of EHV1 was confirmed by virus culture and electron microscopy and serology demonstrated an anti-EHV 1 antibody titre of > 1/256 in both mares.

While EHV 1's presence in SA has been suspected, this is the first confirmation of it for nearly a decade.

VICTORIA.

REGIONAL VETERINARY LABORATORY BENDIGO

INTRODUCTION OF CHARGING FOR ALL LABORATORY TESTING (Copied from RVL Bendigo Newsletter).

On 1st October 1990, RVL Bendigo introduced charging for all laboratory testing. A dual price list was introduced to provide for a 'public good' component of laboratory testing of farm animals (i.e. the whole industry and not just the farmer benefits from the information obtained). Companion animal fees apply to dogs, cats, horses, cage birds and wildlife. Production animals include cattle, sheep, deer, pigs, goats and poultry.

Several new packages have been introduced to provide improved services for production animal clients. Two haematology and biochemical profiles are now available for ruminants (sick ruminant and downer cow). Several other packages are also available including examinations for scouring calves and cows (which include tests for the main bacterial, parasitic and viral pathogens), mastitis testing (10 samples from a herd) and herd testing for specific minerals or enzymes.

The only tests which are offered free of charge are those which form part of the Department sponsored disease control programs. The two main diseases which fall into this category are Johne's Disease and Footrot. Tests for Brucelta abortus as part of a disease investigation and testing performed as part of an animal welfare investigation are also undertaken free of charge.

There is a minimum charge for any submission of $10. The charge for post mortem examination ($50 companion animal and $25 production animal) will include all necessary testing to obtain a diagnosis with the exception of virus isolation and toxicology testing.

Our first 4 weeks of charging has seen a 5% reduction in accessions compared with the same period last year. Submissions from live animals (including testing for haematology, biochemistry, serology, bacteriology and parasitology) has been unaffected by charging but the number of autopsies has declined.

Pathologists need to look at alternative ways of doing things and become more involved in clinical pathology.

The challenge for laboratory staff is to sell the value of the services offered.

CONVULSIONS IN AN ADULT HORSE ASSOCIATED WITH BIRTH INJURY (Les Sims)

A 3 year old thoroughbred gelding was presented for necropsy with a history of epileptic fits for approximately one year. The fits had been observed first when the horse was being broken in and were felt to be exercise induced. Over the next 12 months the fits became more frequent and were not related to any specific stimulus. The birth of this horse had been difficult (prolonged and manually assisted) and he did not stand for the first week of life.

Post mortem examination revealed evidence of skin trauma consistent with previous convulsive episodes. There was an area of loss of tissue in the cerebrum approximately 3 to 4 cm in diameter just caudal to the lateral fissure in the left temporal lobe . The tissue was dark and on the cut surface there was some cavitation. The left cerebral hemisphere appeared markedly smaller than the right. In the body of the cerebellum there was discolouration of some folia.

On histological examination there was extensive chronic malaria in the affected cerebrum with almost total loss of cerebral grey matter. There were some haemosiderin laden macrophages present. In the cerebellum there was also loss of tissue with some folia reduced to stumps while in others there was partial preservation of architecture in the outer cerebellum but loss of the granular and purkinje layer in the inner region resulting in the formation of mushroom-like folia - consistent with the change described as ulegyria.

The lesions seen were considered to be consistent with a birth injury. Similar lesions are reported to occur in humans with birth trauma. It was likely that the cerebral lesion was due to damage to a branch of the middle cerebral artery, although no specific vascular lesion was found. Lesions in the temporal lobe are commonly associated with epiletiform seizures in animals. The interesting feature of this case was the time lag between the birth injury and the appearance of clinical signs.

QUEENSLAND - FraserTrueman

YEERONGPILLY VETERINARY LABORATORY

Vaginitis due to Haemophilus somnus (G. Storie)

Purulent discharge from the vulva occurred in 5 beef cows 48 hours after mating with an introduced stray bull. Depressed appetite, pain and anxiety were also present in the affected animals. *Haemophilus somnus* was isolated from 3 of 4 vaginal swabs submitted. Two swabs gave a mixed culture of micrococci and H. *somnus* and one swab gave a pure culture of H. *somnus*. Haematological examination showed a left shift (increased band neutrophils) in 3 of 4 blood samples submitted.

Fluorosis (G. Stone)

Shifting lameness with swollen phalangeal area and subsequent loss of condition due to inability to graze was observed in a dairy herd. A high grain concentrate with Christmas Island phosphate as a mineral supplement was fed. Biochemical analysis of urines showed fluoride levels of 56,120 and 82 mg/1 in 3 animals which confirmed the diagnosis of fluorosis. Normal cattle have 2-6 mg/1 fluoride and levels of 15-20 mg/1 are associated with moderate clinical signs.

Ichthyobodosis in fish (P. Ketterer)

5% of a batch of 800 silver perch (Bidyanus bidyanus) flngerlings was affected by a skin condition consisting of large focal white areas of dorsal body and dorsal fin. More advanced lesions had strands of mucus adhering.

Organisms were not detected microscopically on skin and gill wet preparations but histology of skin sections demonstrated protozoan parasites with the morphology of *Ichthyobodo necator* (Costia). The parasites were present (in large numbers) in only one of several skin sections examined.

Significant mortality in a batch of fingerling Australian short finned eels (*Anguila australis*) was caused by a combined infection with *Ichthyobodo necator* and *Ichthyophthirius sp*. Heavy infestation of the gill lamellae and the branchial cavity was identified on histological examination.

Botulism in Wildfowl (L. Dowling)

Deaths and illness were reported among wildfowl on three properties in the south coast hinterland in late February and early March 1990. Affected birds included black duck (*Anas supeciliosa*), plumed whistle-duck (*Dendrocygna eytoni*), magpie geese (Anseranas semipalmata) and black swan (*Cygnus atratus*). Twelve died on one property. Clinical signs observed included flaccid paralysis and botulism was strongly suspected. Type C botulinum toxin was detected in the intestinal contents of an affected black swan and the serum of a black duck from separate properties. The source of the toxin was unclear but recent rain may have flooded and killed some vegetation, leading to the right anaerobic conditions in the rotting plants to allow proliferation of the organism. Type C botulinum toxin was detected in rotting vegetation from one property. Once outbreaks of botulism start among waterfowl, the resulting carcases themselves and the maggots feeding on them become sources of more toxin. Type C botulinum toxin was detected in a rotting duck carcase from one property.

Suspect Enterotoxaemia in Cattle (P. Ketterer)

Five deaths occurred over a period of 1 month in a group of 74 heifers grazing kikuyu on a dairy farm. A field post mortem carried out within 4 hours of death, showed blood tinged peritoneal fluid, haemorrhagic decomposing small intestinal content and pulpy decomposed kidneys. On histological examination, all tissues showed advanced post mortem decomposition and post mortem invasion by large Gram positive bacilli. Gram positive rods with subterminal spores were present in smears of small intestine content and toxins consistent with *Clostridium perfringens* type C were identified by mouse inoculation. Enterotoxaemia is rarely diagnosed in cattle but *Cl. perfringens* type C is reported as a cause of disease in feedlot cattle. The kikuyu paddock was used as a dump for old fertiliser and lush growth was thought to be a predisposing factor.

Reduced palatabilitv of pearl millet (Pennisetum americanum) (R. McKenzie)

In late January 1990, a herd of beef cattle were let into a forage crop of pearl millet (*Pennisetum americanum*) [Nutrifeed, Pacific Seeds] which had some areas of yellowed plants in it. The cattle refused to eat the plants over a large part of the paddock. Alkaloids in pearl millet have been reported to be associated with unpalatability in the USA (Rouquett *et al* 1980. Characteristics of the occurrence and some factors associated with reduced palatability of pearl millet. *Agronomy J* **72**:173-174), but there were no published reports in Australia. The unpalatable plants contained 12% nitrate and about half the alkaloid content of the palatable millet which had a nitrate content of 1.6% (there is a possibility that the samples were mislabelled). Neither sample gave a positive test for cyanide. No illness was noted in the cattle.

Jute Seed Poisoning (R. McKenzie)

Eleven cattle aged 6 months to 8 years died within 24 hours, two of them during mustering. One animal was examined clinically and had fever of 40°C, blood in the faeces and urine and jaundice. Post mortem of 3 animals showed jaundice; swollen gall bladders and numerous black seeds in the rumen. Kidney, heart, spleen, liver and lung samples from 3 animals were examined histologically but the only significant changes were congestion and slight oedema in one lung and degeneration of small blood vessels in one myocardial sample. *Corchus olitorius* (jute) seeds were identified as the most numerous seeds in the rumen. Clinical and histological findings were consistent with cardiac glycoside intoxication from jute seeds. Sudden death, particularly during exercise and bloody faeces are typical findings.

Avocado (*Persea Americana*) Poisoning (R. McKenzie)

Thirteen horses were affected (9 severely) in a herd of 16, 2 days after they were given access to avocado trees (*Persea americana* - probably variety Hass of the Guatemalan horticultural race), in a disused orchard. The trees had new leaf shoots on them following recent rain and the horses had eaten these and some remaining fruit. The main clinical signs were oedematous swelling of the head and face, dyspnoea, fluid accumulation in the thorax and hypersensitivity to touch about the head. In severe cases, there was marked subconjunctival oedema with weeping of fluid from the conjunctival sac. Inappetance, reluctance to move, some mild signs of colic and tenderness over the lumbar region were noted in some horses. The worst affected were treated with anti-inflammatory drugs and diuretics by the attending veterinarian and recovered clinically over the next few days. Weight loss over several days was noted in one horse. Blood samples were taken from 3 affected horses on days 3, 5 and 12 after signs of poisoning were first noted. Increased activities of CPK and AST indicated muscle damage while increased bilirubin concentrations suggested liver damage. By day 12, only the most severely affected horse had significant abnormalities of clinical chemistry. Increased leucocyte counts were seen in one horse on day 3 and in another on day 5. This degree of avocado poisoning had been seen in horses in the USA (Carlson GP unpublished data, University of California, Davis, 1987) but had not been reported in Australia to date.

Bovine pestivirus infection (P. Ketterer)

Mucosal disease was diagnosed in a 6-month-old calf from a herd where 3 other animals had died with similar signs over a 6-month period. A crusted exudate was adherent to the borders of the muzzle and at the medial cantus of the eyes. Maceration of interdigital skin and mild coronitis were seen on the feet. Scattered small erosions were present on the anterior tongue and on the hard palate. The rumen mucosa had a large area with numerous circular ulcers which were almost confluent. The abomasal mucosa was oedematous and had scattered focal erosions and one deep ulcer in the pyloric region. No gross changes were seen in the small intestine, which was unusual, but the colonic mucosa bad 2-4mm focal nodular swellings. Histology of tongue, muzzle and rumen mucosa showed necrosis and erosion or ulceration of epithelium with infiltration of lymphocytes in the propria. The colon showed focal atrophy of the epithelium with loss of glands and infiltration of the propria with lymphocytes.

Bovine pestivirus was isolated from blood, colon mucosa, rumen mucosa, spleen, nasal and ocular swabs. The serum was negative for bovine pestivirus antibodies.

Late respiratory disease in meat chickens (L. Dowling)

High mortalities from birds in good condition with a tracheitis caused heavy losses. The birds were meat chickens ranging in age from 28 to 40 days of age. The condition had a rapid onset usually from 35 days of age and the birds would die quickly. Mortality would average 1% per day.

One submission of 32 day old birds had damaged Bursa of Fabricius, with depletion of lymphocytes in the medulla of follicles, heterophil infiltration in the cortex and interlobular septa. This is suggestive of Infectious Bursal Disease which may be immuno-compromising the flocks. Other results showed *E. coli* to

be the predominant bacteria cultured from hearts, lungs, tracheas and livers. *Salmonella Group B4* and mycoplasmas were also cultured on occasions. No significant viruses were cultured. The histopathology of the tracheitis commonly showed focal necrosis of mid-epithelial cells, intra-epithelial oedema, lymphocytic infiltration of lamina propria and hyperplasia of epithelial cells. Purulent pneumonias were occasionally present. A possible pathogenesis of this respiratory disease/high mortality syndrome may be immunosuppression of the birds (Infectious Bursal Disease, possible Chicken Anaemia Agent), followed by mycoplasma infection and tracheal damage with subsequent invasion by *E. coli* causing septicaemia and death.

Systemic Rickettsia-like infestation in farmed fresh water crayfish (P. Ketterer)

High mortality occurred in pond reared redclaw crayfish (*Cherax quadricarinatus*) on a large farm. Histological examination demonstrated a heavy gill cuticle infestation by temnocephalid eggs (30-40% of the gill area was occupied by the eggs). Interlaminar spaces of the gills were filled with a fine granular detritis . In addition prominent hyperplasia and hypertrophy of cells lining haemolymph spaces was seen histologically in gills and other body organs (heart, hepatopancreas, skeletal muscle). The affected cells contained large granular basophilic cytoplasmic inclusion bodies and electron microscopic examination of ultra thin sections demonstrated dense aggregates of rickettsia-like organisms. The sludge deposits in the gills and the temnocephalid egg infestation of the gills must have severely compromised respiration and predisposed to the rickettsia-like infection.

University of Queensland (Roger Kelly)

Sea Turtle Research in Moreton Bay

Anita Gordon has enrolled in a Masters program to investigate causes of disease in sea turtles (mostly greens and loggerheads) in Moreton Bay. The work is being done in conjunction with population studies being run by Colin Limpus of the Queensland National Parks and Wildlife Service. He has noticed that the prevalence of skin lesions and ill-thrift appears to be higher in these species in Moreton Bay (the sump for the Brisbane River) than in areas further north. We have to team all sorts of basic anatomy and techniques (can you bleed a turtle?)

Typhlocolitis in Immature Laboratory Rabbits

A recent outbreak of acute, usually fatal typhlocolitis occurred recently in a specific-pathogen-free rabbit breeding colony. Affected animals were either found dead, or died after a short illness characterised only by diarrhoea. At necropsy, there was severe erosive and ulcerative typhlocolitis with not much significant bacterial involvement, even as secondary invaders. Remaining crypt cells sometimes showed nuclear changes suggestive of virus infection. The appendix was affected to a lesser degree, while the small intestine seemed to have escaped any damage.

Mass Mortality in Fruit Bats in Queensland

There have recently been high mortalities in fruit bats (mostly *Pteropus poliocephalus*) in coastal districts. Affected animals showed signs of depraved appetite (chewing on stems of pawpaw, etc) before convulsing, acting as if blind, etc. and dying. Necropsy findings have been disappointing, and pesticide and heavy metal levels have not been significantly elevated. Nor has there been any evidence of rabies. The animals have been in reasonable body condition which doesn't support the contention that acute starvation is responsible.

Nocardiosis in Homo Sapiens (Dick Sutton)

Perhaps not a "true" veterinary case, but a personal experience of a *Nocardia brasiliensis* skin infection causing enforced absence from work may be of interest. The first signs were a couple of pimples on the left leg which were "squeezed". Within 46 hours there was a sizeable suppurative reaction and swelling and pain in the inguinal nodes. A smear from a swab revealed masses of gram positive filamentous organisms and culture subsequently confirmed the initial *Nocardia* diagnosis. Altogether there were 5 lesions on the leg which showed abscessation with an extensive zone of swelling and hyperaemia. The inguinal node area became very reddened and swollen. Loss of weight-bearing function was present for about 4-5 days. There was no systemic reaction such as temperature elevation or loss of appetite. Treatment was with heavy dosage of trimethoprim and sulphamethoxazole (Bactrim). Initially the dose was 6 double strength tablets per day which is 0.96g and 4.8g respectively. This has subsequently been reduced to 4 tablets per day. Treatment will continue for another two months because of the persistence of the organism. Relapses are frequently septicaemic. Sensitivity tests showed the organism to be resistant to most antibacterials; Bactrim or its equivalents are the drugs of choice. Fluid intake has to be maintained at a high level because of possible nephrotoxic effects (there has to be some bonus arising out of this).

Nocardia brasiliensis is a soil organism and infection in people has been associated with gardening. While the victim has been known to pull a few weeds, the association was at best tenuous. It is an unlikely organism to have come from PM Room; animal cases involving skin or thoracic cavity are usually due to *Nocardia asteroides*. Nor is immunological compromise believed to be a factor in this case.

TASMANIA - Judith Handlinger

ANIMAL HEALTH LABORATORY MT PLEASANT LABORATORIES, LAUNCESTON.

Winter Mortalities in Juvenile Platypus (David Obendorf)

This year, three dead platypus have been examined; all were males with 'type 1' spurs making them less than 6 months old. Significant post mortem findings were low body weight for age, absence of subcutaneous tail fat and little or no food in the gut. Two animals had cytomegalovirus infections of the kidney tubules; one had acute suppurative pneumonia and another had alopecia and ulceration of the ventral tail. A locally invasive Mucor sp. was isolated from the tail lesions.

In 1989, we examined another platypus with Mucor sp. dermatitis and subcutaneous granuloma formation. Several large wounds were found at the edges of the tall and the unfurred areas of the feet and bill. This condition was first reported by Barry Munday and Beavan Peel (Reference: J. Wildlife Disease **19**:365, 1985). From reports from freshwater fishermen in Tasmania, we suspect this condition may be widespread.

Interesting conditions seen in Tasmanian Wildlife (David Obendorf)

* Trichinellia paeudospiralis muscle infections in two Masked Owls and a swamp harrier. The owl species is a forest dwelling bird, reputed to predate young native cats. The harrier is a recognised carrion feeder capable of eating virtually any dead mammal.

* Acute Toxoplasmosis is a common case of winter mortality. This year has been no exception with macropods, wombats and bandicoots all dying as a result of this infection. As the natural food availability drops off and cold conditions develop, wildlife parks with high stocking densities begin to lose animals. A growing concern is the detection of acute Toxoplasmosis in free-ranging barred bandicoots (Perameles gunnii). This species is now an highly endangered species on mainland Australia due to a combination of habitat loss, introduced carnivore predation. It appears that the remnant population at Hamilton, Victoria may also be further dwindled by the effect of Toxoplasma infection.

Currently we have been encouraged by the use of a direct and modified agglutination test to detect IgG and IgM-specific for Toxoplasma gondii infection in a range of wildlife species. Highly elevated IgM titres are correlated with "acute onset" infection. More cases may help us differentiate between newly acquired and recrudescent infections.

* Coccidiosis in common wombats (Eimeria arundeli)has generally been considered a "low pathogenicity parasite". Recently we received two cases involving juvenile hand reared wombats showing weight loss and depression. The faeces were quite abnormal being pasty and smelly, diarrhoea was not a feature. The oocyst count in one animal was 50,000\gram. Grossly, the small intestinal enteritis was characterised by thickened grey, almost textured mucosa with hypertrophied villi. Microscopically the lamina propria of villi was distended with various micro and macrogametocytes and oocysts. The opinion of a Canadian pathologist with some pretensions to wombat protozoology will be sort.

* Cryptococcal pneumonia in an echidna. A road killed echidna had a mucopurulent unilateral fungal pneumonia. Cryptococcus neoformans was isolated. We have also seen cryptococcal infections in wild ring-tailed possums associated with granulomatous meningoencephalitis.

UNABLE TO ADD Peter Phillip's ASVP LOGO

JOBLINE

North Carolina State University
College of Veterinary Medicine

Visiting Instructor - Anatomical Pathology. Department of Microbiology, Pathology and Parasitology, College of Veterinary Medicine, North Carolina State University has an opening for a visiting instructor in Anatomical Pathology. This is a non-tenure-track position with an annual appointment. Candidates should possess a D.V.M. or equivalent degree and have completed formal training (residency or graduate program) in anatomical pathology. Candidates should have partially or completely met the eligibility requirements for certification by the American College of Veterinary Pathologists. This position is intended for an individual wishing time to prepare for board certification without having the full responsibility of an academic position. About 50% of time will be devoted to supervising anatomical pathology residents and participating in the necropsy and surgical pathology service program of the College of Veterinary Medicine. Remaining time may be devoted to preparation for board certification, pursuing research interest, training with industrial and government toxicologic pathologists in the Research Triangle area, etc. Salary range - $27,000 to $30,000 per year. Starting date, July 1, 1991. Applicants should send letter of application, curriculum vitae and names of 3 references to Dr. Talmage T. Brown, Jr., Professor of Pathology, Department of Microbiology, Pathology and Parasitology, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough Street, Raleigh, North Carolina 27606, (919) 329-4258.

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Toxicologic Pathologist. The College of Veterinary Medicine at North Carolina State University is seeking a pathologist who has interest, training and experience in toxicology. A DVM and preferably a Ph.D. degree with postdoctoral training in toxicologic pathology are required. Board certification in pathology will be given preference. The applicant must present evidence of research productivity in toxicology by way of research publications and be capable of developing a high quality, fundable research program in toxicologic pathology. Individuals with specific interests in either respiratory, gastrointestinal or hepatic toxicology are encouraged to apply. The successful candidate will be expected to participate in the pathology and toxicology teaching and research programs in the College of Veterinary Medicine and in the University. The rank of the. position is a tenured-track assistant or associate professor, depending on qualifications. Please send application or nominations to Dr. Talmage T. Brown, Jr., Professor of Pathology, College of Veterinary Medicine, North Carolina State University, Raleigh, North Carolina 27606. Applications should include a curriculum vitae and the names of 3 references. Closing date for applications is December 1, 1990, or until a suitable candidate is chosen.

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