THE VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to yon by:
the Department of Primary Industry, Tasmania,
Mt. Pleasant Laboratories,
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DEADLINE FOR NEXT VET. PATH. REPORT IS NOVEMBER 1

PRESIDENT'S PAGE

The ASVP executive is frequently requested to become involved in matters concerning veterinary diagnostic laboratories e.g. accreditation of laboratories and quality control programs.

Some members of the ASVP consider ASVP has a clear role to play in the development and role of veterinary diagnostic laboratories in Australia. Many veterinary pathologists play key roles in veterinary diagnostic laboratories. Veterinary laboratory diagnoses is much broader than pathology encompassing all disciplines involved in the delivery of laboratory diagnosis e.g. technical disciplines like bacteriology, virology, clinical pathology, toxicology, parasitology and pathology, but also a host of non-technical issues like staff training and development, billing, marketing, quality control, and courier services. It has been suggested that only specialist veterinary pathologists are equipped to manage veterinary diagnostic laboratories. This view fails to recognise the skills and expertise for instance of veterinary microbiologists. One of the most successful managers of a veterinary diagnostic laboratory was John Moxham at Ruakura in New Zealand. John had experience in veterinary pathology but used many of the skills acquired as being a veterinary representative for Tasman Vaccine Laboratory to run a very successful laboratory.

The ASVP' a core role must be to promote and encourage an interest in veterinary pathology e.g. through newsletters, slide of the month and annual scientific meetings. Resources available to the ASVP executive are appropriate to enable it to perform this role. From time to time, ASVP will take the opportunity to comment on matters concerning veterinary pathology e.g. its submission on registration of specialist veterinary pathologists.

Issues concerning broader veterinary laboratory matters e.g. accreditation and quality control systems are outside the scope of ASVP's core role. These matters were handled for State laboratories by the Sub Committee of Principal Laboratory Officers (now disbanded). A proposal to establish a Sub-Committee on Animal Health Laboratory Standards to fulfill a similar role for State veterinary diagnostic laboratories is being submitted to Standing Committee on Agriculture.

If ASVP members consider the ASVP should play a more active role in matters of diagnostic laboratories, there would need to be a substantial increase in resources for the ASVP executive. Alternatively, consideration could be given to the formation of an Australian Association of Veterinary Laboratory Diagnosticians.

Rod Oliver
President

SECRETARY'S REPORT

*** 1991 ASVP SCIENTIFIC MEETING***

Elizabeth Macarther Agricultural Institute 11 & 12 May 1991

Programme:

Oncology:	Terry Rothwell	Small Animals
	Tony Ross	Large Animals
Liver Pathology:	Roger Kelly et al	
Case presentations:	Please include cases associated with rare or endangered species.	

Note that the ASVP meeting immediately precedes the Australian & New Zealand Veterinary Association's Pan Pacific Conference 13-17 May 1991 at the Darling Harbour Conference Centre.

I understand that small animal oncology and the roles of zoos in regenerating endangered species are topics in the programme of the Pan Pacific Conference.

Because the period during which the ASVP meeting has been organised is within university term time accommodation will not be available at Nepean Hall (University Farms). Motel/hotel accommodation is available at Crown Hotel/Motel, 191 Argyle St. Camden, NSW, Telephone (046)552200. Tariffs quoted as of 24th July are -

Hotel section	Single $45.00
	Double $55.00
	Twin $60.00
	Extra person $15.00
Motel section	Standard Single $55.00
	Double $65.00
	Twin $70.00
Deluxe	Single $70.00
	Double $85.00
	Extra person $15.00

Possible 10%-17% increase in Tariffs by May 1991.

ASVP LOGO

Don't forget this challenge from Peter Phillips. Put your talent to work. Entries will be displayed at the May 1991 meeting and a choice considered for adoption as the **ASVP** logo. While you are in a creative mood you could also put your mind to a motto.

ASVP MEMBER TELEPHONE NUMBERS

The majority of location/contact details on the **ASVP** member data base do not include member contact telephone numbers. When sending in your **ASVP** subscriptions, for example, would you please also include your place of work telephone number, including the telephone area code.

CHANGE OF ADDRESS

If you are moving please advise the secretary of your new address so that the member data base can be up-dated. It is from this data base that the address stickers for the Pathology Report etc. are printed.

Roy Mason

EDITOR'S REPORT

This issue contains some further items on Veterinary Specialist Registrations (pages 9 to l8) and several responses from readers on accreditation of veterinary pathologists and veterinary pathology laboratories (pages 6 to 8). The Letters to the Editor section also contains Sue Friend's letter on this subject, this time in full (sorry Sue!).

I understand some members have failed to receive recent copies of the VPR. 1f you are one of them please contact us and we'll check our mailing lists and send on any back issues required (Nos. 25, 26 or 27).

Virtually every Australian state is faced with a financial dilemma - how to provide the range of public services with a dwindling allocation from the federal government. I expect this has implications for managers of government veterinary laboratories as well.

Perhaps the now is the time to review not only the way government vet. pathologists see themselves but also how clients see them. Being a "clever pathologist" in terms of diagnostic acumen is one thing. To be resourceful and adaptable in servicing the changing needs of our Australian community is another form of cleverness.

For farming Australia, veterinary pathology is seen as a useful science which has implications for the way animal owners manage their livestock for realistic production. Diagnostic veterinary pathology could be considered as an indicator science of not only animal health and welfare but also of overall farm management. The rural community is being asked to accept some new concepts: whole farm planning, minimal input farming, integrated pest control, and sustainable animal production. Clearly the government veterinary pathologist can make valuable contributions in the development of these strategies.

In this context, veterinary pathologists might be thought of as "environmental scientists" with skills to be applied to any one of a number of situations facing agricultural Australia.

Perhaps the challenge facing government vets. Pathologists are to apply their talents to a changing Australia. In essence being clever.

NATIONAL PATHOLGY REGISTRY

*** A PLEA TO A.S.V.P. MEMBERS *** (W.J. Hartley)

Over the last two or three years repeated approaches have been made to individuals and institutions for materials to be sent to either or both of the National Pathology Collections. With a few exceptions, the response has been poor.

You will all know that the Domestic Pathology Collection is an A.S.V.P. project and that it has been financed by the States and Commonwealth for two years. It's present and future survival depends completely on the support it receives from all A.S.V.P. members. To be effective as a reference and teaching facility the collection must have many more examples of common and less common pathologic entities. So to help achieve its survival it is imperative that each month or so each institution select up to, say, five current cases with good pathology for inclusion in the collection. To data there are only 2,000 cases filed. By the end of 1990 it is hoped that the number will be close to 3,000 if A.S.V.P. members co-operate.

The non-domestic animal collection funded by the Zoological Parks Board of N.S.W. has gradually developed into something reasonably comprehensive with 4,000 cases on file. However, still more materials will be welcome. As some experience has been gained in this area, Bill Hartley will be very pleased to give opinions on disease processes in any native, species including caged and aviary birds.

Send any interesting domestic cases to Dr. W.J. Hartley, C/- Elizabeth Macarthur Agricultural Institute, Woodbridge Road, Menangle, N.S.W. 2568.

Send wildlife cases to Dr. W.J. Hartley, C/- Taronga Zoo, P.O. Box 20, Mossman, N.S.W. 2088.

REGISTRY NEWS

Pathologists wishing to "brush up" in a particular area of expertise are invited to visit the Registry for a free short course of instruction. If that is not possible, you can write to the Registrar requesting study sets in the area of your interest.

Remember, each state's government laboratories are entitled to one week of course work in the state during 1990 and again in 1991. Dr. Bill Hartley's salary and expenses have been prepaid. All your state needs to do is to choose areas of interest, consider a course structure and make arrangements with Bill Hartley.

Bill will be in the USA working at Poisonous Plants Research Institute in Utah and visiting the Armed Forces Institute of Pathology in Washington. He will be away from 20th August to 20th October, 1990.

LETTERS TO THE EDITOR

*** LETTER TO THE EDITOR**

(From Ruth Reuter, Regional Veterinary Laboratory, Albany W.A.)

I am happy to see that the ASVP is becoming more active in areas other than routine submission of case reports to the VPR, as illustrated by the recent discussion on accreditation and specialization. The membership also appears to be growing. Perhaps it is time once again to raise the question of the venue for the AGM, particularly since Sydney has been nominated as the site for the 1991 meeting.

At the risk of being judged parochial, I would like to make some comments on this subject. From the March 1990 VPR it appears that of 144 Australian members, 63 reside in states other than New South Wales and Victoria. If my calculator is operating correctly this comes to approximately 44% of the membership, or roughly the figure which saw our Prime Minister back in Canberra! Having spent approximately 7 years in beautiful Melbourne, I am aware of the arguments used to keep the AGM rotating between the two aforementioned states. Of these, the most pressing appears to be financial.

The fact that it is just as far and just as expensive to fly from Perth to Sydney as it is to fly from Sydney to Perth seems to be conveniently overlooked when the venue for meetings is decided. With the debut (and rapid demise) of "W.A. Inc.", it has become decidedly more difficult for a struggling West Australian public servant to make the journey. Access to continuing education courses and seminars of various types is much easier for our cousins in the cities of Sydney and Melbourne, and contact with peers far more convenient and less expensive.

With so few veterinarians in general and pathologists in particular in such a big country, segmentation of the profession is of concern since it interferes not only with rapid transmission of information but social contact, with people with similar interests. With such great distances to cover at significant expense it becomes very difficult to attend more than one conference a year. The situation is not helped when a person is a member of more than one organization.

I feel strongly that it is not unreasonable for the AGM of this group to be held in a venue other than Sydney or Melbourne at least one year in three, possibly in the same location as the A.V.A., or closely related to that meeting. This would be of great assistance to those members who belong to both organizations and who would then be able to justify attendance at both meetings. If such a consideration is not possible, those members who are relatively isolated might have to look at the formation of regional organizations. Such a move would obviously weaken the profession, but relieve some of the frustration experienced by those of us who feel left out!

I would be most interested in comments from the membership.

*** LETTER TO THE EDITOR**

(From Richard Whittington, EMAI Menangle NSW 2568)

In response to the information provided in the VPR No. 27, and the letter from Miller **et al**, I would - like to offer the following comments.

The discipline of veterinary pathology, as practiced world-wide, is extremely diverse. We have on the one hand practiced anatomical and histopathologists dealing with all species and on the other pathologists working in research often in extremely narrow fields. We must not lose sight of the spectrum in considering the registration of veterinary specialists.

The issues raised by Miller **et al** relate, in my opinion, to an argument of exclusivity. There is a small group of veterinarians in Australia with Fellowship of the Australian College and\or American Board Certification. They are no doubt, extremely well qualified and competent. There is a much larger group, also well qualified by virtue of postgraduate qualifications, and also competent. Members of both groups are scattered in the various disciplines referred to above. In my opinion, the argument of exclusivity does little more than protect commercial interests. It does not assist the profession of veterinary pathology, or the aims of the ASVP. The medical profession successfully applies a similar argument of exclusivity to limit the number of medical specialists at an artificially low level.

Insistence on rigid criteria for registrations as a specialist, for example Fellowship of the Australian College, is unworkable. Doyens of veterinary pathology in this country have no intention of submitting themselves to examination, nor should they be expected to. In addition, what would be the reaction of those practicing commercial veterinary pathology, to registration as a specialist veterinary pathologist, a fellow examined in veterinary microbiology or immunology? Do we later face arguments that certain registered specialists should not be permitted to practice commercially?

The only standards worth achieving are those we set ourselves as individuals. The service and professionalism we offer to the public does not depend solely on the letters that follow our name.

The current proposal of the ASVP includes minimum qualifications and experience. It recognises the expertise of those currently practicing veterinary pathology, be they in diagnostics, teaching or research. It proposes representation of the ASVP on the committee reviewing applications.

The proposal may need some fine tuning. For example, would ASVP members consider time spent in the meat inspection service, or even general practice, as worthy of consideration as part-relevant experience?

I support the position taken by the ASVP, but would encourage further debate. It would, however, be a pity if the unity achieved within the ASVP over the years is tarnished by this issue.

* LETTER TO THE EDITOR

(From Jeremy Langdon, Animal Health Laboratory, Perth W. A.)

Registration or Obfuscation?

Herewith a few whimsical thoughts after reading the stentorious promulgations of my friends and colleagues Drs Mitchell and Rahaley, and their fellow pathologists in private practice, supporting a stringent system of examination for specialist registrants (Vet Path Report, March 1990). Firstly, I was struck by the coincidence that the high and admirable standards of examination they commend fall just a notch or two below their own qualifications, with which they could equally pursue lofty academic positions. Dismissing nagging doubts about exam passing skills and commercial advantage, I decided this was perhaps, as they write, also in the interest of the recipients of pathology services.

Now this got me wondering just what services are offered by private veterinary pathology laboratories, and from my remote position of ignorance - which all and sundry are welcome to improve - it appeared to me that the bulk of their work would be analysis, nay autoanalysis, of blood, faeces and other tit-bits of clinical pathology. I daresay there is call for histopathology but rarely a necropsy, and from time to time a taxing slide would require the highest skills in morphologic histopathology, such as our above colleagues possess. From time to time that is, for one sees only the common diseases commonly.

A component of clinical pathology is included in Australian and North American specialties in pathology, but how much more appropriate might a clinical pathology specialty be for those who run private veterinary pathology services? My plea is that such questions remain alive while we search our souls for a workable, equitable and consistent registration system, applicable in both public and private sectors, which does not exclude the bulk of practising and competent pathologists.

In the meantime, lest we price or specialize ourselves out of the market, we should wrest our highly focused eyes from the microscope and search the wider industrial scene for guidance. As the airline boss said to the pilot, we do not need astronauts to fly aeroplanes.

* LETTER TO THE EDITOR

(From R N Thornton MAF New Zealand, Batchelar Animal Health Laboratory, Batchelar Agricultural Centre, State Highway 57, PO Box 1654, Palmerston North, NZ)

I have read with interest, mingled with shades of benign cynicism, some of the views about specialist pathologist status and what one needs in order to qualify. Membership by examination of the pathobiology chapter appears to be regarded as hardly a qualification at all and is supposedly little better than an undergraduate qualification leading to better things and in my view is wrong. The only qualification worth studying for is one that is useful and the only one that is useful in this context is one that confers specialist status. A membership examination pitched almost at undergraduate levels serves only one purpose to my mind and that is to swell the ranks and therefore the coffers, of the College. I believe a single, worthwhile, qualification conferring specialist status should have been the aim from the start. A good example is the examination for the American Boards which is hard but which if you get it is worth having. Why should we have to study for two lots of exams?

*** LETTER TO THE EDITOR**

(From Sue Friend, Central Veterinary Diagnostic Laboratory, 166 Union Rd., Surrey Hills, Victoria 3127.)

Re: Accreditation of Veterinary Pathologists and/or Veterinary Pathology Laboratories.

I would like to express my concern at some aspects of the statement promulgated by the executive of the Australian Society for Veterinary Pathology (ASVP) on accreditation of Veterinary Pathologists in the Veterinary Pathology Report #26, of October 1989.

I do not agree that the Veterinary Surgeons' Board of each state is best placed to decide if a person is qualified as a Veterinary Pathologist. Surely the Australian Society for Veterinary Pathology and the Australian College of Veterinary Scientists (ACVSc) Pathobiology Chapter would be the appropriate bodies to make such a decision. The board may be involved in the final decisions, but only after seeking advice from the ASVP and ACVSc as to whether a person is suitably qualified.

Certainly the. essential qualifications should include a degree in Veterinary Science Veterinary Medicine, recognised by the Veterinary Surgeons Board and post graduate qualifications such as fellowship of the ACVSc in pathology or equivalent i.e. diplomat of the American College of Veterinary Pathologists. In my opinion, membership of the ACVSc in pathobiology is not an adequate basis on which to be called a Veterinary Pathologist.

Section (ii) which states that a person should have demonstrated ability and at least 3 years practising Veterinary Pathology is not specific enough. There must be adequate supervision by appropriately trained individuals in suitably equipped training centres. There should be critical appraisal of the progress of the trainee and of the credentials of the training programme, personnel and centre. Who is going to do the training and who is going to critically evaluate the trainee and the programme? This is where a National Training Programme, under the auspices of the ACVSc, would be of great value.

Also, I believe that we should not put accreditation of veterinary pathology laboratories in the "too hard basket". As mentioned, there are established guidelines for lab. accreditation and laboratories should be encouraged to investigate and follow the guidelines which are most appropriate from them. Quality, assurance programmes for biochemistry, haematology, microbiology and histopathology used by laboratories across Australia would be a start. Just as registration for specialists in Veterinary Pathology has taken time to evolve, so will accreditation of veterinary laboratories, and the ASVP should be promoting this.

In my opinion, there is no role for medical pathologists in the field of veterinary diagnostic pathology other than in the comparative research areas. Medical pathologists should not be involved in the diagnosis of animal diseases, unless they have a degree in Veterinary Science/Veterinary Medicine and appropriate post graduate qualifications or are supervised by a veterinary pathologist. Indeed, the ASVP should actively discourage medical pathologists from practicing "Veterinary diagnostic work" or providing a veterinary biopsy service.

I believe that the ASVP and the ACVSc are crucial for the advancement of Veterinary Pathology in Australia and we should strive for excellence, not mediocrity.

* SPECIALIST REGISTRATION FOR VETERINARY PATHOLOGISTS *

At the AGM of the ASVP in Launceston in February a sub-committee of Tony Ross and Keith Walker was formed to explore the issue of Specialist Registration.

After discussions with the NSW Board of Veterinary Surgeons a submission by the ASVP (printed in the Vet Pathology Report of April 1990) was made to the Australian Veterinary Board's Conference which met in Townsville in May 1990.

The thrust of the ASVP submission was to broaden the criteria for registration as a Specialist Veterinary Pathologist. It also wished to obtain input from the ASVP into the decision making by the Advisory Committee on Registration of Veterinary Specialists which advises each of the State Boards of Veterinary Surgeons on specialist registration. The Committee had no pathologist on it until Mary Barton joined the committee in May 1989.

Reprinted below for information of members are:

1. a summary of the objectives of registration of Veterinary Specialists;

2. a report by Jakob Malmo, Chairman of the Advisory Committee on the recent discussion at the Australian Veterinary Boards Conference;

3. the amended criteria for Specialist Registration.

It is important to recognise that the Advisory Committee and the Australian Veterinary Board's Conference have no legal status. Each State Board of Veterinary Surgeons has the regulatory power and autonomy to make its own decisions on Specialist Registration.

There is good reason to believe that the Advisory Committee will consider broadly based PhD's and coursework Masters Degrees as suitable academic qualifications for Registration, provided that the candidate can fulfill the other requirements. Obtaining a Fellowship of the Australian College of Veterinary Scientists or membership of the American Veterinary Boards in Pathology will not be necessary when broad based training in veterinary pathology has taken place during post-graduate studies.

Those ASVP members who wish to apply for Specialist Registration should contact the Registrar of their relevant Board of Veterinary Surgeons for an application form.

South Australian pathologists are reminded that new legislation in that State requires that Veterinary Specialists only be appointed on the recommendation of the Advisory Committee on Registration of Veterinary Specialists.

Evolution and standardisation of Specialist Registration standards will hopefully occur over the next 12 months. ASVP members are encouraged to air their views in the Vet Path Report, to the ASVP Executive and to their State Board of Veterinary Surgeons.

Tony Ross

* JOBLINE *

VETERINARY PATHOLOGY SERVICES

2 DALY STREET
KURRALTA PARK SA 5037

PO BOX 355
PLYMPTON SA 5038

PATHOLOGIST REQUIRED

A unique opportunity exists for a pathologist to join a private veterinary group. Veterinary Pathology Services P/L is seeking an additional pathologist primarily based at our Adelaide laboratory. Some interstate travel will also be required.

The successful applicant will be a person with ambition, true entrepreneurial skills and a good communicator who is prepared to work hard for appropriate reward. Eligibility for registration as a Veterinary Specialist (pathology or clinical pathology in South Australia is ESSENTIAL. Salary package negotiable front $55K with shareholder status possible after a trial period. Prospective applicants should contact Drs Richard Miller, Geoff Mitchell or Chris Belford (008 777 372) or Dr Rob Rahaley (008 882 515).

ADVISORY COMMITTEE ON REGISTRATION OF VETERINARY SPECIALISTS

The Objectives of Registration of Veterinary Specialists

To provide the public and the veterinary profession at large with access, for purposes of consultation and referral, to specially experienced and qualified veterinarians who have demonstrated superior knowledge and skill in specified veterinary techniques and disciplines.

The ways in which their superior knowledge and skills are demonstrated, and the specified techniques and disciplines in which they are superior, are set down in the Australian Veterinary Boards Conference approved document entitled "THE GENERAL CRITERIA WHICH CAN BE APPLIED IN THE CONSIDERATION OF ANY QUALIFICATION WHICH IS PRESENTED BY AN APPLICANT TO THE BOARD AS A SUITABLE SPECIALIST QUALIFICATION".

The objectives of specialist registration can be summarised as follows:

1. Specialists are to be registered for the purpose of practice. Practice is defined as the exercising of a profession and in this context shall refer to veterinarians in private practice, government practice or university practice.

2. Practicing specialists may provide service either to the public or to other members of the profession.

3. The standard required for specialist registration is comparable to the standard of specialist registration in other countries (notably, the United Kingdom and the United States of America), but allows for local needs for referral specialists.

4. To be registered as a specialist the veterinarian shall have an agreed standard of training and shall have submitted to a suitable broad based examination.

A REPORT ON THE DISCUSSION AT THE AUSTRALIAN VETERINARY BOARD'S CONFERENCE REGARDING SPECIALIST REGISTRATION

Townsville
18 May 1990

1. The objectives of Specialist Registration

Our document "The objectives of registration of Veterinary Specialists was considered and accepted with some minor modifications. This document includes a brief history detailing the development of the Advisory Committee on Registration of Veterinary Specialists.

From this document, a summary paper outlining the objectives of registration of Veterinary Specialists was prepared

2. Uniformity of Standards

The need for uniformity of standards regarding specialist registration was re-affirmed by all Boards.

There was considerable discussion as to whether or not all applications for specialist registration should be forwarded to the Advisory Committee on Registration of Veterinary Specialists. Two differing views were put:

(i) That all applications for specialist registration should be forwarded to the Advisory Committee on Registration of Veterinary Specialists for assessment. The supporters of this view believed that such a mechanism would ensure uniformity of standards and would ensure that a single body maintained expertise in this area by virtue of the number of applications which it would consider.

(ii) That it was not necessary that all applications should be forwarded to the Advisory Committee on Registration of Veterinary Specialists, but that applications which obviously met the criteria could be registered (with a copy of the application forwarded to the Advisory Committee on Registration of Veterinary Specialists) and only problem applications forwarded to the A.C.R.V.S.

A compromise proposal was accepted whereby:

* For the next 12 months all applications for specialist registration shall be forwarded to the Advisory Committee on Registration of Veterinary Specialists for assessment.

* The Advisory Committee on Registration of Veterinary Specialists should present to the next Veterinary Surgeon's Board Conference (May 1991) a list of qualifications in which it recommends that Boards can accept as suitable qualifications for Specialist Registration without referring directly to the Advisory Committee on Registration of Veterinary Specialists for assessment.

  Whether or not this step is desirable can be reviewed at the May 1991 Australian Veterinary Surgeon's Board Conference.

3. Broadening of Criteria

The Conference accepted our suggestion that the THE GENERAL CRITERIA WHICH CAN BE APPLIED IN THE CONSIDERATION OF ANY QUALIFICATION WHICH IS PRESENTED BY AN APPLICANT TO THE BOARD AS A SUITABLE SPECIALIST QUALIFICATION: should be broadened to include as acceptable qualifications certain broad based PhDs and Masters.

It was suggested that the following clause should be added to the General Criteria document:

4. Broadening the Range of Specialties and Sub-specialties

The Australian Veterinary Board's Conference was of a view that it could handle the widening range of specialist areas by retaining the agreed 7 major specialty areas, but allowing newer areas to be accepted under these areas.

As examples –

Veterinary Medicine - Goats
Veterinary Medicine - Aquaculture

I suggested that it would assist the Advisory Committee on Registration of Veterinary Specialists if the Boards would nominate the area in which they wished an applicant for specialist registration to be considered.

My personal view is that this was a very worthwhile meeting and that the decisions made should assist the consideration of applications for specialist registration.

Jakob Malmo
Convenor
Advisory Committee on Registration of Veterinary Specialists

THE GENERAL CRITERIA WHICH CAN BE APPLIED IN THE CONSIDERATION OF ANY QUALIFICATION WHICH IS PRESENTED BY AN APPLICANT TO THE BOARD AS A SUITABLE SPECIALIST QUALIFICATION.

1.1 **Minimum time from graduation to submitting for specialist registration**

An applicant must have practiced as a veterinary surgeon for at least 5 years before being eligible to be registered as a specialist.

1.2 **Minimum time working in specialty before registration**

An applicant must have worked for at least 3 years full-time equivalent in his/her specialist area before being eligible to be registered as a specialist.

1.3 **A period of supervised training is an essential part of preparation for the final examination.**

This may involve either:

(a) a formal course such as a University residency course

or

(b) an alternative to the formal residency course - the alternate approach allows candidates to work in an approved centre to provide for a period of supervised training.

While undergoing training in an approved centre, 50% or more of the candidate's work will be in the field of the specialty for which registration is sought.

1.4 **The period of supervised training required**

The formal residency program includes a normal expectation that the candidate applying for registration as a specialist will have carried out at least a 2 year full-time equivalent defined program under the direction of an approved supervisor, preferably prior to the examination.

or

The alternate program includes a normal expectation that a candidate applying for registration as a specialist will have carried out at least a 2 year equivalent training program under the direction of an approved supervisor, preferably prior to the examination.

This means, for example, that a veterinarian in practice spending two thirds of his/her time working in the specialty under supervision will have a 3 year period during which time he/she shall obtain a 2 year full-time equivalent. If he/she is working, 50% of his/her time in the specialty under supervision, this will require a 4 year period to obtain a 2 year full-time equivalent. Less than 50% of the time spent working in a specialty shall not be acceptable.

1.5 **The selection and role of supervisors**

Under both the formal and the alternate system, approved supervisors are necessary.

Selection: In University programs the supervisor will be appointed by the Dean. Supervisors in alternative training centres will be appointed by the Australian College of Veterinary Scientists.

(NOTE: In the case of the Royal College of Veterinary Surgeons Diplomas and American Board Diplomas, there can be no control as to the supervisors who are appointed.)

Supervisors shall be recognised specialists in the area of the candidatures and must be working in the specialty.

Recognised specialists shall be either those registered by the Boards as such or those with qualifications considered equivalent to Board requirements.

A supervisor in an approved training centre shall, before accepting appointment, assess a candidate's written proposal for a training program. An assessment will take into account the centre's workload, facilities and resources and their adequacy for the proposed program.

Supervisors shall be responsible for both instruction and continuing assessment of candidates. Progress reports will be given to the candidate every 6 months. A final written report on the candidate and the program will be submitted to examiners of the candidate.

1.6 **Prerequisites of an Approved Centre**

Approved centres for the purpose of training under the alternate program require:

* An adequate workload in the specialty
* Adequate facilities for the performance of the specialty
* Adequate access to other professional expertise
* The written permission of the principal of the centre providing the facility

1.7 **Case reports, dissertations and publications**

Case reports, dissertations and preparation of publications shall be included as part of the period of supervised training and these case reports etc. shall be assessed by the final examiners.

At least 2 of the 3 (that is 2 out of the triad of case reports, dissertation and preparation of publications) shall be acceptable. The number and type of these reports may vary between specialties.

1.8 **Examinations**

The examinations will be of a comprehensive and searching nature and will include:

(a) Written examinations - these shall consist of two 3 hour papers. The first paper will cover the basic science of the specialty and the second paper will cover clinical applications of the specialty. The 2 areas must be discrete and clearly identifiable within the examination process.

(b) Clinical and practical examinations. The difficulty of conducting 'hands on' practical examinations is recognised, but by making use of aids such as suitable slides and specimens the examiners will develop the basis for a discussion and evaluation of practical skills.

(c) Oral examinations.

1.9 **PhD's and Course Masters**

PhDs and Course Masters will be considered as suitable qualifications to support a claim for Specialist Registration, provided that during the Masters of PhD program the applicant also received in depth supervised training for an equivalent of 2 years full time across the breadth of the discipline in which the applicant claims specialist status, followed by written examinations, clinical and practical examinations and oral examinations as defined in part 1.8 of this document.

STATE REPORTS

WESTERN AUSTRALIA - Ruth Reuter

REGIONAL VETERINARY LABORATORY ALBANY (Ruth Reuter)

Bovine Mucosal Disease

A newborn heifer calf was submitted to the laboratory from a mob of 70 Murray Grey cows. The calf was moribund, with eyes rotated downward and head extended dorsally. It was unable to stand or suck. Nine other calves from the mob were stillborn. On post mortem examination, gross lesions were restricted to the musculoskeletal and nervous systems. The humeri were shortened, there was excess fluid in the cranial cavity and only a very small vestige remained of the cerebellum. On microscopic examination the small amount of cerebellar tissue showed severe hypoplasia, as did the cortical region of the thymus and there was a complete lack of germinal centre development in the spleen. A sample of blood was positive for mucosal disease/virus diarrhoea antibody by immunodiffusion. Since the animal had not received colostrum, this was considered to indicate mucosal disease virus infection.

The property had not experienced a similar problem previously. Discussion with the owner revealed that all stillbirths had occurred in a group of 40 animals which had been raised on the property. Six months prior to the stillbirths, 11 pregnant cows and 1 bull had been bought in and mixed with this herd. All of the newly purchased cows calved normally. It is probable that one or more of these animals were the source of the infection.

Lupinosis in Weaner Sheep

A hogget from a mob of 750 Merinos was submitted live to the Regional laboratory in early April. Approximately 15 animals had died during the previous week, and a large percentage were showing signs of illness. The sheep had been grazing "Danja" lupin pasture earlier in the year for a period of 3 weeks. They were removed from this pasture at the end of January. Late in March they were yarded for sorting for sale. When a sudden storm came up, they were put in the lupin paddock for 24 hours because this was the best shelter on the property, then placed back in their original paddock. Losses commenced 4 days later.

The sheep submitted was in lateral recumbency with opisthotonus, salivation and dyspnoea. There was yellowish discolouration of mucous membranes. On post mortem there was extensive jaundice of the carcase with gross distension of the gallbladder and a bright ye1low, firm 1iver. The brain appeared swollen. There was a strong chemical odour to the carcase. Histology on the liver revealed hepatic necrosis with pigment in sinusoidal macrophages, pleomorphic nuclei in hepatocytes, lymphoid foci in periportal regions. In the brain there was astrocyte swelling, spongiform encephalopathy in the white matter particularly of the brainstem and cerebellum. A diagnosis of toxic hepatopathy and hepatic encephalopathy due to phomopsin, the causative agent of lupinosis, was made. The lesions were much older than 4 days and were probably the result of the earlier exposure to the toxin with deaths precipitated by the stress of yarding and exposure.

Hepatopathy in a Princess Parrot

One dead bird was submitted from an aviary which had contained 6 Princess parrots. No clinical signs had been observed at 8.00a.m. when the birds were fed. Two hours later one was found dead on the floor of the aviary. Shortly after noon the owner found the second bird dead and brought it in for examination. The bird was in good body condition. The blood was watery, the liver yellow in colour with dark areas, the muscle pale. The small intestinal content was blood-stained. On histology the only changes observed were in the liver, where hepatocyte nuclei were enlarged with prominent nucleoli and the cytoplasm of cells in

some areas contained large amounts of golden-brown pigment. A toxic hepatopathy was suspected. On questioning, the owner recalled that a large amount of damp seed from the feeder had been discarded on the floor of the aviary several days before. The aviary was cleaned out and no further losses have occurred.

State Footrot Reference Laboratory

With the ideal climatic conditions this year, the laboratory has had a record number of submissions of samples from cases of suspected footrot. At present there is a focus of infection in the Southwest of the state and a large scale effort is in place to eradicate this. The farmers in the area are greatly concerned and have been most cooperative.

ANIMAL HEALTH LABORATORIES. SOUTH PERTH

Bovine hyperthermia. illthrift and deaths associated with ergotism (Ron Peet).

15 of 1,700 yearling steers died and 40 appeared sick in a feedlot at Esperance. The syndrome was characterised by hyperthermia with animals wanting to stand and actually wallow in water like pigs, profuse salivation and clear nasal discharge, inappetance with laboured breathing. The morbidity approached 85% and growth rate was markedly retarded. The condition appeared 3 weeks after feeding commenced and serological tests were negative for IBR but positive for PI3 and mucosal disease. However, there were1 no rising titres in paired samples and post mortem examination of 2 animals revealed no gross or histological evidence of disease. The only lesions observed were focal haemorrhages in the brainstems of both animals.

Examination of the barley feed mix revealed rye grass seed contamination which was infected with probable Claviceps spp at a concentration of 0.06% w/w ergots or sclerotia in the feedstuff. Screenings of the barley grain yielded 0.5% w/w ergots. A diagnosis of bovine hyperthermia probably due to ergotism was made on clinical symptoms, lack of evidence of other differential diagnoses and the presence of sclerotia (ergots) in the feed. Cattle in N.S.W. showed symptoms of the disease when feed contained 0.02%-0.8% w/w ergots.

References

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Contagious ecthyma tongue lesions resembling foot and mouth disease. (Trevor Ellis and Ron Peet)

3 of 12 tongues from sheep slaughtered at a small abattoir north of Perth were condemned by the local health inspector for tongue lesions. The lesions were circular erosions approximately 0.5 to 1.0cm in diameter on the surface and edges of the tongues (2 to 3 on each tongue). They were considered to be suspicious of foot and mouth disease although contagious ecthyma was suspected. Fresh samples were forwarded to AAHL that same evening and the property and butcher' shop placed in quarantine with traceback procedures instituted. These were completed the following day and AAHL reported negative FMD ELISA tests the following evening.

Meanwhile, frozen and rapidly processed paraffin sections had been examined at AHL, Perth. The lesions were characterised by marked erosion of squamous epithelium leaving granulation tissue markedly infiltrated with eosinophils, plasma/lymphoid cells and macrophages. The intact epithelium on the edges of the erosions showed some ballooning degeneration with microvesiculation, microabscessation and occasional eosinophilic intracytoplasmic inclusion bodies consistent with contagious ecthyma. This diagnosis was confirmed by electron microscopic examination of tongue scrapings which showed numerous virions consistent with orf.

Field examination of the remaining killer flock of sheep revealed approximately 60% had tongue lesions. The animals had been fed hay heavily infested with the spiny seed-heads of the "double-gee" plant (Emex Australis or Emex spinosa) which may have predisposed to the "scabby mouth" infection of the tongue.

These tongue lesions must be distinguished from foot and mouth lesions (Yager and Scott, in Jubb, Kennedy and Palmer). Similar tongue lesions of contagious ecthyma have previously been compared to bluetongue infection (Gardiner et al 1967).

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Myoliquefaction in fish post mortem (Jeremy Langdon)

Myoliquefaction or "milky flesh" is caused by myofibre infections with myxosporean protozoans mostly in the genera Kudoa, Chloromyxum, Henneguya and Unicapsula. Yellowtail kingfish and barracouta are perhaps the most commonly affected species. The liquefactive process is caused by histiolytic enzymes released by the parasites, and may occur in fish held at room or refrigerator temperatures, after death. With some species, liquefaction and crumbling of the flesh begins upon warming such as occurs in slow cooking, triggering serious nervous disorders in the cook.

Wild and cultured mahi or dolphin fish have developed the condition in some locations in Western Australia. The flesh becomes softened, opaque and sticky within 24 hours of storage at 4 degrees C. Histopathology reveals numerous intramyofibre spore masses of Kudoa thyrsitis but quite remarkable preservation of myofibre nuclei, sarcolemma and myofibrils. Van Gieson staining reveals hypochromicity and rupture of the collagen fibres in the intramuscular connective tissues, so collagenase activity may produce some of the gross changes.

The enlarging spore masses incite an inflammatory response only when the sarcolemma is breached, with encapsulation by fibroblasts and epithelioid macrophages. Many spores degenerate within a few months of encapsulation.

Kudoa thyrsitis has long been known to cause "milky flesh" in southern Australian barracouta, and occurs in at least a dozen fishes world-wide, but its life cycle and mode of transmission are unknown. The use of filtered sea-water for fish farms prevents infection.

Urea poisoning in goats (David Forshaw)

23 of 40 Angora goats were found dead or close to death around their water trough. The goats had been in the paddock for a year and at the time were being hand fed meadow hay with no access to green feed. They had been vaccinated with PK/tet 4 months previously and drenched for worms 4 weeks previously.

Post mortem examination of two goats, one of which was killed, revealed only moderate bloating and dark, liquid, small intestinal contents.

As no specific PM findings were obvious, the owner was questioned about possible toxins. He admitted to using water previously used to wash out a tank containing urea to prime a pump with which he had then pumped dam water into the water trough. Urea levels in the drinking water were 326 emol/1 – approximately 20g/l. (The toxic dose for sheep is 0.3-0.5 g/kg). However, the tank water urea was 190 mmol/1 - approximately 11g/1. No dam water was submitted. How the urea had become more concentrated in the drinking water was not clear.

Histological examinations revealed no significant lesions. Despite careful searching, no lesions were observed in the thalamus as described in experimental acute ammonia intoxication of sheep.

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SCHOOL OF VETERINARY STUDIES, MURDOCH UNIVERSITY.

Plasma Cell Myeloma (M. Burrows)

A 7 year old male Doberman dog presented with a history of lethargy, anorexia, abdominal guarding, hind limb ataxia and urinary incontinence. There were cutaneous ulcerations on the prepuce, around the anus and on the feet.

Haematology, biochemistry and urinalysis investigations revealed an anaemia, hyperglobulinaemia, hypercalcaemia and proteinuria. Serum electrophoresis confirmed a monoclonal gammopathy and the owners requested euthanasia. No skeletal radiographs performed.

On necropsy, the dog was in fair condition. There were no gross abnormalities evident.

Histological examination revealed multifocal aggregates of plasmacytoid cells with pale, finely vacuolatad cytoplasm, an eccentrically located nucleus with central nucleoli and peripheral chromatin granules in the liver, spleen, pancreas, bone narrow, lymph nodes, small intestine and skin. The kidneys had periglomerular and interstitial fibrosis containing multifocal aggregates of plasma cells within the interstitial spaces. There was dilation of renal tubules with protein casts.

Multiple myeloma is a rare condition. This is an interesting case as cutaneous lesions associated with plasma cell myeloma have not been previously documented in the dog.

Feline Enteritis-like Syndrome (B.Chadwick)

In a household of 8 cats, 3 developed an acute enteric illness within a 7-day period, with clinical signs of pyrexia, lethargy, anorexia and haemorrhagic diarrhoea. Two of the cats were littermates (5 Months old); the third was the grand dam of the former (7 years old). The cats were treated with IV Hartmans solution for dehydration, and with parenteral antibiotics. The Older cat and one of the kittens died and were subsequently sent to Murdoch University for post mortem. No clinical pathology data was available.

Post mortem examination of the 7 year old cat found severe haemorrhagic enteritis and colitis with frank blood in the lumen. Histologically, the colitis was associated with severe crypt necrosis. Bone marrow was mildly hypocellular. The kitten showed moderate enteritis and colitis with crypt necrosis, and mild hypoplasia of bone marrow including both myeloid and erythroid elements. These changes generally and the severe crypt epithelial necrosis in particular were considered characteristic of feline infectious enteritis. However, both kittens had received a recent full vaccination course; the 7 year old cat had been vaccinated every year.

The third cat recovered from the acute illness, but remained ill-thrifty. Follow-up clinical pathology was performed 6 weeks after recovery. Serum was positive for feline leukaemia virus antigen but negative for FIV antibody. Complete blood count showed a mild non-regenerative anaemia, mild lymphopaenia and mild thrombocytosis. All other cats in the house were tested for FeLV: all were sero-negative.

The kitten was euthanased on the basis of the positive FeLV result. Post mortem found atrophic villi in the small intestine and evidence of fusion of villi-crypts with fibrosis of the lamina propria. This was considered evidence of recovery from the acute disease. Bone marrow was mildly hyperplastic, and lymphoid tissue was very reactive in all lymph nodes examined.

A diagnosis of FeLV associate acute enteritis was made despite incomplete clinical pathology on the two cats that died acutely. A feline enteritis/panleukopaenia-like syndrome has been reported sporadically, in vaccinated cats, and is usually associated with FeLV. It does not have the age predilection of FIE for young animals necessarily. It is considered likely that the syndrome involves an acute infection in cats chronically infected with FeLV, although the identity of the acute agent remains unknown. It is normally a sporadic disease of older animals and indeed the outbreak form of this disease is rather unusual.

NORTHERN TERRITORY - Lorna Melville

BERRIMAH AGRICULTURAL RESEARCH CENTRE

Type C botulism in a horse (L. Melville)
Several cases of suspect botulism in adult horses were investigated following a suggestion there might be a link between a lead product produced in SA and suspect botulism cases in that state. The feed was also sold in the NT.

Culture of the feed was negative for botulism, however, the large intestine contents of one horse yielded type C botulism toxin following culture. The method used is fairly long and involves initial isolation in a fortified egg meat media, followed by screening for toxin in mice and follow-up protection tests with anti C and anti D antitoxin.

Suspect Cobalt deficiency in weaner goats (L Melville)
A group of kids weaned onto lush pangola pasture showed severe weight loss over a number of months, with terminal nervous symptoms.

The kids were anaemic and emaciated and failed to respond to treatment with a vitamin/mineral mix. They had no evidence of parasites or ooccidiosis on post mortem examination. Selenium copper levels were normal in these animals. Histologically the kids showed spongy vacuolation in the white matter of the brain with variable lesions of chronic liver damage.

The liver lesions varied from widespread centrilobular necrosis to individual hepatocytic necrosis, centrilobular fatty change and centrilobular pigmented macrophages. The changes seen were considered highly suggestive of cobalt deficiency and the nervous signs due to hepatic encephalopathy.

Melioidosis (Morton Bell)
Meloidosis is seen as a seasonal problem in the top end of the Northern Territory. The first case this season was in a sheep in January. On post mortem abscesses were observed in the lungs which yielded a pure culture of Pseudomonas pseudomallei. The second case was a 2 year old female goat with mid shifting joint swellings. This goat was suspected of having melioidosis and had been receiving antibiotic therapy when it died suddenly. The post mortem revealed a severe haemothorax, several abscesses, one on the anterio-ventral sternum and two associated with the aorta. One was dorsal to the heart and the other was anterior to the diaphragm. In both these locations the aorta showed signs of inflammation, haemorrhage and necrosis. Further investigation showed the lesion dorsal to the heart was an aneurism which had ruptured. Pseudomonas pseudomallei was isolated in pure culture of this lesion.

Histology of the aorta from affected areas showed severe diffuse suppurative arteritis and thrombosis formation on the exposed tunica where the endothelium had been lost.

Urolithiasis in Replacement Pullets (M Bell)
A 20% mortality in replacement pullets at 17 weeks of age, using to 30% by 28 weeks was found to be associated with Kidney lesions and urate crystal deposition on liver and heart.

The gross kidney lesions were atrophy of the left kidney with dilated ureters packed with mucous and white concretions. Histologically there was crystal deposition within the kidney with associated necrosis and inflammation, fibrosis, dilated tubules and cellular casts.

Despite extensive investigations no aetiology could be established for these lesions. Areas looked at included IB vaccination responses, vitamin A levels, feed and water analysis and strain of chicken.

This case is discussed in more detail in the March Slide of the Month.

QUEENSLAND - Fraser Trueman

ANIMAL RESEARCH INSTITUTE

BOTULISM (Fraser Trueman, Peter Ketterer)

For many years botulism has been recognised as a serious constraint to cattle production in areas of NW Queensland. Cattle grazed under extensive conditions in these phosphorus deficient areas display osteophagia leading to botulism. In recent years several cases have been diagnosed in Southern Queensland in cattle receiving supplements or in feed lots (See Vet Path Report No 24). Some recent cases are summarised.

Two outbreaks were investigated in dairy herds where several animals displayed recumbency and death over a course of 2-3 days. No gross or microscopic pathology was seen, and botulinum toxin was not detected on mouse inoculation. Botulism was diagnosed on clinical symptoms, and in both cases access to pasture that had been fertilised with poultry litter. Numerous decomposing poultry carcases were present in the litter.

Seven Brahman animals died in a feed lot, and botulism was confirmed on mouse inoculation test. The source of toxin was not determined.

Forty-five of 70 dairy cattle died with symptoms of botulism. High levels of botulinum type D toxin were demonstrated in the decomposing carcase of a snake caught up in the bail feeding mechanism (from Rockhampton Veterinary Laboratory).

Massive mortalities occurred simultaneously on two feed lots with 5,472 deaths recorded over a 3 week period. From 215 animal samples, of which 139 were sera, botulinum type D toxin was finally demonstrated in one serum sample collected from an affected animal early in the outbreak. The source of toxin was found to be a common source of chicken litter included in the feed. There was some doubt that the litter had been processed and heat treated before being supplied. Type D toxin was readily found in poultry carcases in the litter (from Toowoomba and Yeerongpilly Laboratories).

Poultry litter has now been prohibited as an animal feed in Queensland.

Xanthorrhoea johnsonii Poisoning of Calves (R. McKenzie)

Three Brahman-cross calves (4-5 months old, mixed sexes) in a total herd of 30 cattle were affected with posterior ataxia, decreased skin sensitivity over the hindquarters and urinary incontinence. The calves were affected for about 4 weeks. The herd had access to a stony ridge on which grew a small number of Xanthorrhoea johnsonii. Their flower spikes had been broken off and eaten, some of their foliage had been eaten as well. Necropsy of one calf revealed Wallerian degeneration of scattered nerve fibres of both sciatic nerves and the spinal cord with some nerve fibres in tracts in the medulla oblongata and cerebellar peduncles being similarly affected. The spinal cord lesion was more intense dorsolaterally, that is in the proprioceptive spinocerebellar tracts. This case is noteworthy for the occurrence of degenerative lesions in the nervous system where no lesions of this type were described in the original work on poisoning by Xanthorrhoea spp. in Queensland. Such lesions have been seen in cases in Tasmania (Munday et al. 1976 Aust vet J **52**:92) and in a suspected case at Pomona some years ago.

Purulent Arthritis in Sheep (G. Storie)

Several outbreaks of purulent arthritis were reported from the Cunnamulla district during the quarter. On one property, 50 of 500 lambs were affected of which 30 died, and 200 mortalities from a group of 2000 were reported on another property.

A 4-month-old lamb from each property was submitted for necropsy. The right shoulder joint of the first lamb was swollen to 3 times normal size. There was a severe purulent arthritis with cellulites and abscessation of periarticular soft tissues. Destruction of articular cartilage, pannus formation and developing osseous ankylosis, together with severe periarticular fibrosis, had resulted in complete joint immobility. The second lamb had a similarly affected right elbow and in addition, a 4cm diameter abscess in the caudal lobe of the right lung. Histological examination of synovial membrane from the affected joints revealed a severe chronic diffuse purulent and ulcerative synovitis with marked proliferation of fibrovascular tissue and chondroid and osseous metaplasia. The animals were hypoalbuminaemic (22 and 20g/l), hyperglobulinaemic (58 and 66 g/l) and leukopaenic (1.4 and 25 10^9/l) . Both joints yielded Fusobacterium necrophorus in pure culture. Culture for aerobes, Erysipelothrix and Mycoplasma was negative. A FAT for Chlamydia psittaci was also negative.

Outbreaks of purulent arthritis have occurred regularly in Paroo Shire over the last 2 years, mostly associated with mulesing. Affected animals are first noticed 10-14 days after mulesing. Lameness, fever and joint swelling usually lead to death. The occasional survivors are poor doers and crippled. Anecdotal information puts losses on some properties at 1000-1500 animals last year. Poor surgical hygiene is presumed to be the cause of the problem.

Vitamin A deficiency in hand-fed cattle (R. McKenzie)

Vitamin A deficiency was diagnosed in a group of 12-15 month-old Friesian steers being fed a ration of 83% rolled barley, 10% Rhodes grass chaff, 5% molasses, 1% urea and 1% ground limestone. Clinical signs noted included excessive salivation, apparent blindness and inco-ordination. Two died and a third was submitted for necropsy. Subsequently a further steer had convulsions while being handled. Significant findings at necropsy and histologically were papilledema of the optic discs, stenosis of the optic foramina of the skull with constriction and degeneration of the optic nerves, ulceration of the reticular groove and

omasal groove and some leaves, abomastitis and ulceration of the upper duodenum. The liver contained 0.3mg vitamin A/kg (normal > 2.0 mg/kg). The mode of association of the alimentary tract lesions with the vitamin deficiency was unclear.

NEW SOUTH WALES - Tony Ross

REGIONAL VETERINARY LABORATORY. MENANGLE

Is there still a vital place for pathology in the modern world? Pathologists know there is, but what about others! Here are two cases worth quoting. (K Walker)

GN90/1546
Suspect generalised TB in an aged Friesian cow from a major stud herd with nationwide sales and current attendance at major shows. Lung, thoracic and retro-pharyngeal/prescapular lymph nodes involved. Avian intradermal positive. Meat Inspector 99% certain it is bovine TB!

Pathology

Disseminated adenocarcinoma with no evidence of granulomata. Slides and smears also negative for acid-fast organisms. Culture to come.

Outcome

Huge relief in 56 hours for field staff; minimal follow-up ramifications for owner and the TB Eradication Program. Phew!

P.S. Anzac Day in between.

GN90/2071

29/5/90 am. Exotic disease alert. Diagnostic team directed to a coastal dairy herd. One cow salivating with lip, tongue and nasolabial skin lesions of a type not familiar to the clinicians involved but familiar to an experienced pathologist.

Pathology

Multiple (30+) peripherally erythematous plaques, no vesiculation, lesion biopsy result in 48 hours showed a ballooning degeneration of the stratum spinosum with eosinophilic large inclusions. Bovine papular stomatitis confirmed in conjunction with demonstration of parapox virus particles on E.M. (EMAI and AAHL Geelong).

Outcome

Emergency called off on day of visit and endemic disease confirmed within 48 hours.

P.S. Four other cows and a week old suckling calf were also found affected on the farm on the day of inspection. They had isolated oral lesions only.

REGIONAL VETERINARY LABORATORY. ORANGE

TB IN CATTLE (J. Seaman. D. Seward and M. Carrigan)

Tuberculosis was diagnosed in a large beef herd at Mudgee during February. The initial case was detected at Blayney abattoir in a yearling heifer with tuberculous lesions in the liver, lung, mediastinal, pharyngeal and bronchial lymph nodes. Traceback information indicated the herd of origin had a previous history of tuberculosis in 1982. The herd provided the opportunity to evaluate the gamma-interferon assay being

developed by CSIRO as a serological test for tuberculosis. Four hundred and sixteen cattle were TB tested using both the tuberculin skin test and the gamma-interferon assay. A total of 37 reactors were detected of which 24 had lesions. The tuberculin skin test detected 21 of these lesioned animals while the gamma-interferon assay detected 19. One lesioned animal detected at slaughter was negative to both tests, thus 23 ex 24 lesioned cattle were detected by the 2 tests. Culture of lymph nodes from reactor animals is underway and to date 18 sets of lymph nodes are culture positive for M. bovis. The sensitivity of the 2 tests will not be fully evaluated until all cultures are completed. The herd has been totally depopulated.

CHLAMYDIOSIS (J. Seaman)

Chlamydial infections - the new **IMAGEN** fluorescent stain for detecting Chlamydia organisms has had quite a workout in recent weeks with positive results in cattle (Sporadic Bovine Encephalomyelitis}, parrots (Psittacosis) and even in a guinea pig. The cattle cases were supported by good pathology and serological reactions and it appears **SBE** was active in some areas during early autumn. Chlamydial polyarthritis was also confirmed serologically in lambs during autumn.

CHRONIC CHOLECALCIFERON TOXICITY IN A DOG (D. Seward)

An 8 month old German Shepherd pup was euthanased following the development of intermittent vomiting and pneumothorax requiring repeated aspiration.

The pup had been treated 9 weeks earlier for acute cholecalciferol toxicity following the ingestion of between half and one cup (60-120 gm) of Quintox$^{(R)}$ pellets.

Formalin fixed sections of stomach, intestine, kidney, heart and lung were submitted.

Histologically diffuse, severe, chronic interstitial pneumonitis with septal mineralisation and subepithelial mineralisation of medium and small bronchioles was present.

Kidney sections revealed severe chronic nephropathy with intestinal fibrosis, tubular dilation, presence of proteinaceous casts with mineral deposits within the glomeruli and some mineralisation of cortical and medullary tubules.

Scattered small foci of mineralisation were identified within the myocardium and intimal mineralisation of most medium and larger vessels was evident.

Severe confluent multifocal necrosis with mineralisation and peripheral replacement macrophage and fibrous tissue response was present in all gut sections examined being most pronounced in sections of stomach and duodenum.

The LD50 for cholecalciferol in the dog has been stated as 88 mg/kg. Quintox pellets contain 0.75 g/kg cholecalciferol. One cup (approx. 120 gm) contains 88 mg which is equivalent to one-fifth of the LD50 for a 5 kg dog. Although owner assessment of the amount consumed may be inaccurate it would appear that the pup consumed well below the recognised LD50.

Experimental poisonings of dogs at one-quarter and one-eighth of the LD50 have been reported (Gunther *et al* 1988).

Similar histopathology has been described in a cat dying following the consumption of only a few pellets (Wellcome Fact Sheet - New Rodenticide - Toxicology).

It would appear that the danger of this relatively recent rodenticide to non-target species may have been understated.

References

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ROCK FERN POISON1NG IN CATTLE (M. Carrigan)

During May and June there were many reports of rock fern (Cheilanthes sp.) poisoning in cattle from the Condobolin, Nyngan and Narromine areas of western NSW. The deaths followed the heavy rains of April which resulted in rapid growth of rock fern. Animals involved had often been severely stressed, by periods of trucking or droving, in the 1-2 weeks prior to the occurrence of clinical signs. Although there were many reported cases, only a few were referred to the Regional Veterinary Laboratory.

Specimens were received from 4 properties on which deaths occurred in cattle grazing pastures containing abundant rock fern. The mortalities were 5 ex 150, 33 ex 230, 37 ex 500 and 45 (possibly more) ex 160. Clinical signs of weakness, depression, laboured breathing and bloody faeces were present in sick animals. Necropsy findings were similar in all cases and included petechial and ecchymotic haemorrhages throughout the carcase, but particularly subserosally in the thorax and abdomen. There was haemorrhage into the lumen of the intestines and in some animals blood clots in the colon. A number of cattle had infarcts involving the full thickness of the intestinal wall. Formalin fixed tissues were the usual samples submitted to the laboratory, however, in one case fresh tissue were forwarded for bacteriology and Pasteurella haemolytica was recovered from the lung, liver and kidney in heavy growth. Histologically the main findings were congestion and multiple foci of haemorrhage in heart, lungs, kidneys and intestines.

In all cases rock fern poisoning could only be suspected because of the limited range of specimens submitted to the laboratory. As the toxic principle of rock fern is considered to be similar to that of bracken fern, blood samples from sick and moribund animals would have helped in establishing a diagnosis. However, the histories of grazing rock fern, the necropsy findings and the lack of alternative diagnosis strongly suggested rock fern poisoning in the cases examined.

REGIONAL VETERINARY LABORATORY. WOLLONGBAR (P.A. Gill)

INTRAHEPATIC PORTOCAVAL SHUNT IN A BRAHMAN

A 4-month-old Brahman heifer had a 2.5 month long history of lethargy and paresis. There were no significant gross lesions at necropsy. Histological changes included severe, diffuse microcystic cavitation of the white matter from cortex to cerebellum, symmetrical in distribution. In the portal triads, there was proliferation and hypertrophy of branches of the hepatic artery, the portal veins were thin walled and sometimes absent. Mesangio-capillary glomerulitis was evident in the kidney. The brain lesions were attributed to status spongiosus and the liver lesions were consistent with an intrahepatic shunt. We gratefully acknowledge the assistance of Roger Kelly and Bill Hartley with this case.

BACULVIRUS INFECTION IN PRAWNS

Investigations into mortalities in Penacus monodon postlarvae found eosinophilic intranuclear inclusion bodies in the hepatopancreas. These lesions are consistent with plebejus baculovirus infection. The epidemic was controlled by de-population, the raceways were dried out and spelled prior to re-population.

OYSTER MORTALITIES ASSOCIATED WTTH FRESHWATER INFLUXES

Several cases of increased mortalities in adult oysters were associated with influxes of freshwater after rains. Low grade to moderate filtration of haemocytes throughout the connective tissue and degenerative changes in digestive gland epithelium were a common histological lesion in affected oysters.

REGIONAL VETERINARY LABORATORY. WAGGA WAGGA (John Glastonbury)

In early January 1990 we were sorry to say farewell to Jeff Marshall upon his departure for the University of Nebraska, Lincoln to study for a PhD. After the customary delay, we were pleased to welcome Jocelyn Godwin, a 1988 graduate of Murdoch University as his replacement in June.

Our veterinary staff has achieved some milestones recently. John Searson and Ian Links have obtained registration as specialists in the area of Pathobiology. Adrian Philbey was appointed to the NSW Divisional Committee of the Australian Veterinary Association.

HUMANS

SUSPECTED ANTHRAX

On 28 March 1990 a local veterinary practitioner developed a circumscribed inflammatory lesion on the skin of his right forearm. This progressed over the following week to form a necrotic black central area surrounded by a zone of erythema and fluid-filled vesicular eruptions. The lesion was not painful but the entire arm became swollen, oedematous and uncomfortable. The veterinarian was hospitalised and received fluids intravenously. Antibiotic therapy was instituted on the second day and, after the acute phase, the inflammation gradually subsided leaving a raised eschar which persisted for several weeks at the initial site of inflammation. Anthrax was strongly suspected on clinical grounds but cultures, taken from the superficial skin and after antibiotic therapy had been initiated, yielded only Staphylococcus spp.

The practitioner had necropsied a cow from a local farm 2 days prior to the occurrence of the initial lesion and had sent specimens, including fresh and fixed tissues, swabs and smears of intestine, to the RVL Wagga Wagga. Enterotoxaemia was suspected in the field because the gastrointestinal tract was reddened while the spleen was not swollen. The laboratory findings were not consistent with anthrax. Despite vaccination against enterotoxaemia, several cattle died on the farm over the next 2 weeks. Following the suspected human case of anthrax, the herd was vaccinated against anthrax. Deaths ceased 8 days following vaccination.

CATTLE

NEUROLOGICAL DISORDER OF ANGUS CALVES (Adrian W. Philbey)

In a herd of purebred Angus cattle about 1% of 500 calves died in successive years at 3 to 6 weeks of age. Two 4-week-old Angus calves were laterally recumbent, had muscular tremors, opisthotonus, bruxism, strabismus and nystagmus and went into tetany and convulsions when stimulated. No gross lesions were detected at necropsy. On histological examination in one calf there was focal symmetrical polioencephalomalacia of the olivary nuclei in the medulla oblongata and early oedema and vacuolation of the substantia nigra. In the other calf the dorsolateral medulla oblongata was affected by bilaterally symmetrical malacic changes and there was segmental poliomyelomalacia.

SHEEP

SUSPECTED FACIAL ECZEMA (John Glastonbury}

In a flock of 800 mixed age Corriedale breeding ewes, 100 deaths (12.5%) occurred over a period of 5 months from December 1989 to April 1990. The farm had a history of chronic copper poisoning but there was no response to 2 oral drenches of molybdenum. About 100 ewes were in poor condition, despite supplementation with hay and oats, at the time of investigation. Deaths occurred suddenly or were preceded by a period during which affected ewes were listless and lost condition. Photosensitisation was not recorded. One ewe necropsied was emaciated and had lambed recently. The subcutaneous tissues and viscera were icteric and the liver was enlarged, pale and had rounded borders. Histologically chronic degenerative hepatopathy, characterised by severe bile stasis, biliary fibrosis and granulomatous cholangiolitis, was accompanied by renal bile pigment staining. A change in weather conditions mitigated against a search for Pithomyces chartarum.

GELATIN GEL TEST - DIAGNOSIS OF VIRULENT FOOTROT (Ian Links)

The gelatin gel test measures the heat stability of proteases produced by Bacteroides nodosus in broth culture. The percentage reduction in protease activity is measured after heating 3-day broth cultures at 68°C for 16 minutes. Virulent and intermediate strains of B. nodosus produce heat stable proteases, the stability being correlated with degree of virulence. The gelatin gel test produces results more quickly than the elastase test.

The gelatin gel test was compared with the elastase test for 522 isolates of B nodosus from 71 outbreaks of footrot in 1989 and 1990. Protease activity was eliminated by heating in 141 isolates which tested negative at 21 days in the elastase test and were considered benign. Thirty five isolates which were negative at 21 days in the elastase test yielded reductions of 22 to 84% in the gelatin gel test. The remaining 346 isolates were intermediate or virulent in the elastase test and retained protease activity in the gelatin gel test but it was not possible to clearly differentiate intermediate from virulent isolates. Work on the gelatin gel test is continuing.

TASMANIA - Judith Handlinger

ANIMAL HEALTH LABORATORY, MT. PLEASANT LABS. LAUNCESTON.

NEOSPORA CANINUM INFECTION DETECTED IN A BOVINE ABORTED FOETUS - David Obendorf & Roy Mason

Abortions caused by sporozoan parasites which don't cause clinical disease in cows have been reported in several parts of Australia and New Zealand. This form of abortion is characterised by numerous inflammatory granulomas associated with tissue necrosis; occasionally sporozoan cyst stages are associated with these lesions. The aetiology of the condition has never been completely resolved.

Paraffin-embedded foetal tissue sections containing these lesions were sent to J.P, Dubey of the USDA Beltsville Maryland. Sections were reacted with anti N. caninum serum in the peroxidase-antiperoxidase test. Based on this test, N. caninum organisms have been identified from foetal tissues in USA, New Zealand and now, Tasmania.

Currently, a serological test for Neospora infection is being developed by US researchers; we're helping by sending them freeze-dried sera from dams and foetuses.

Neospora caninum is closely allied to Toxoplasma gondii. It is the cause of limb paralysis, neuritis and generalized infections in dogs, abortion and paralysis in cattle, and perinatal infections in kittens. The complete life cycle is still unknown, however, immuno-compromised mice may be infected

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Shivaprasad HL, Ely R and Dubey JP (1989) A Neospora-like protozoan found in an aborted bovine placenta. Vet Parasitol **34**:145-148.

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BOVINE COCCIDIOSIS caused by Eimeria zuerni (David Obendorf)

Recently we investigated an outbreak of scouring and illthrift in beef calves at foot on King Island. Each autumn and winter, a significant number of calves developed a mucoid, progressing to bloody, scour. Badly affected calves became depressed, developed tenesmus (sometimes prolapsing their rectums) and failed the suckle. Twenty calves from a mob of 300 breeding calves died.

At post mortem , gross pathology was restricted to the terminal ileum and large intestine. In young recently affected calves, the mucosa of the ileum, caecum and colon was thickened and corrugated. Areas of ulceration and erosion with haemorrhage were present in the caecum and crown colon. Numerous schizont and gametocyte stages of Eimeria zuerni were present in the large bowel sections. Chronically affected

calves showed severe mucosal ulceration with diphtheritic membrane formation. Mild blood loss, anaemia and hypoproteinaemia were detected in these long-standing infections.

Faecal consistency changed through the course of the disease, commencing as a white scour, progressing to mucoid, and finally haemorrhagic. Ante mortem diagnosis is assisted if blood stained faeces are submitted for examination.

King Island is flat and lies on the western boundary of Bass Strait. Exposure to wet windy conditions combined with low temperatures is thought to have contributed to this outbreak. In our experience, Eimeria bovis and E. zuerni are most commonly associated with blood scouring and colitis.

* JOB LINE *

NSW AGRICULTURE & FISHERIES

VETERINARY RESEARCH OFFICER

REGIONAL VETERINARY LABORATORY
NORTH COAST AGRICULTURAL INSTITUTE
WOLLONGBAR

POSITION NO. AG 90/

Equal Employment Opportunity Is NSW Agriculture & Fisheries policy.

SALARY:	$28,484 p.a. rang· $40,372 p.a. with progression to $46,232 p.a. subject to certain conditions.
DUTIES:	Undertake diagnostic pathology investigations into diseases of farm livestock.
QUALIFICATIONS:	Essential
	Degree In veterinary science registerable in NSW. Willingness and capacity to implement EEO policies.
	Desirable
	Post-graduate experience in diagnostic pathology.
NOTE:	Temporary appointment under Section 33/34 or 38 under the Public Sector Management Act up to 20th May, 1991.
ENQUIRIES:	Dr. Roger Cook (066) 240261
APPLICATIONS:	Applications should be, submitted on a Form 59 available from all State Government offices to the Recruitment Officer, NSW Agriculture & Fisheries, North Coast Agricultural Institute, Bruxner Highway, Wollongbar by 6th July, 1990.

GA2 186949

* JOBLINE *

POSITION ANNOUNCEMENT

Department of Veterinary Science
The Pennsylvania State University
University Park, PA 16802

Position: Veterinary Pathologist

Available: 1st October 1990

Salary and Rank: The appointment is a tenure-track position at the Assistant Professor/Associate Professor level depending on qualifications on a 48-week salary plan. Salary and precise rank will be commensurate with the qualifications of the applicant.

Duties and Responsibilities: The Veterinary Pathologist is responsible for the development and co-ordination of an independent and collaborative research program; for provision of pathology consultation to Departmental diagnostic programs and pathology support for the Laboratory Animal Resources Program; and for teaching at the graduate level.

Qualifications: All candidates must possess a D.V.M. or V.M.D. degree from an AVMA accredited College of Veterinary Medicine, board certification by the American College of Veterinary Pathologists and a Ph.D. degree in a biomedical science. Experience In morphologic pathology is highly desirable.

Application Procedure: All applications will be accepted until 1st September 1990 or until the position is filled. Interested persons should send a letter of application with detailed curriculum vitae and the names of three references to:

Dr. F. G. Ferguson, Chairman, Search Committee
101 Centralized Biological Laboratory
The Pennsylvania State University
University Park, PA 16802

Further information and a more complete position description are available from Dr. F. G. Ferguson.

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