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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

Die TYDSKRIF is die offisiële mondstuk en eiendom en word gepubliseer deur die Suid-Afrikaanse Veterinêre Vereniging. Dit verskyn kwartaalliks en word aan sake van algemene veeartsenykundige belang gewy. Bydraers tot hierdie Tydskrif maak hul stellings en lug hul menings slegs op eie verantwoordelikheid; sodanige stellings word nie noodwendig deur die Redaksiekomitee onderskryf nie en die menings gee nie noodwendig die Komitee se menings weer nie. Kopiereg word op al die letterkundige inhoud van die Tydskrif voorbehou.

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ALL CORRESPONDENCE: Manager, SAVA, Jl. S Afr. Vet. Ass., Box 26498, Arcadia 0007 (Tel. 26-6233)

ALLE BRIEFWISSELING: Bestuurder, SAVV, Tydskr. S Afr. Vet. Ver., Bus 26498, Arcadia 0007 (Tel. 26-6233)

REDAKTEUR/EDITOR: Prof. R.C. TUSTIN

ADMINISTRATIVE EDITOR/ADMINISTRATIEWE REDAKTRISE: Mrs. C. GROENEWALD

AGENTS IN GREAT BRITAIN: **AGENTE IN DIE VERENIGDE KONINKRYK:**

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PRESIDENT'S ADDRESS*

Mr. Vice President, honoured guests, ladies and gentlemen,

Once every year, and more specifically in those years that the South African Veterinary Association holds its biennial national congress we have the opportunity of taking stock of the veterinary activities during the preceding year. We may even look back further in the past and review our achievements with satisfaction. This has been ably and amply done by Dr D.W. Immelman, Director General of the Department of Agriculture and Fisheries in his opening address. I must concur that our profession has a proud tradition and I may state that I do not believe that there is another country in the world where the veterinary profession has had such a profound influence on a country's development as it has in South Africa.

Times have, however, changed and the era of spectacular successes, such as the eradication of epizootic diseases, is forever passed. For their achievements we honour the great names of the past and acknowledge the significant role of the Veterinary Research Institute and the Division of Veterinary Services in this respect. The time has now come when we must re-assess the role of the veterinary profession in a largely urbanised society and a technological era. We have to determine not only where we have been, but particularly where we are going and to take cognisance of those basic matters that fundamentally affect the veterinary profession. With this objective in mind, I wish briefly to consider the aspects that will form the cornerstones of our future, namely education, research, rural practice and urban practice and also to reflect on the impact that the new Veterinary Act will have on us.

Education

Because it is the mainstay of our profession, the SAVA has always placed the highest premium on sound basic and continued education. This is once again manifested by the present congress during which the recently appointed Dean of the Faculty of Veterinary Science at MEDUNSA, Prof. N.C. Owen will be one of the speakers in the first plenary session and is exemplified by the fact that the SAVA's Gold Medal for 1981 will be presented to the Dean of the Faculty of Veterinary Science, University of Pretoria for his achievements in the field of veterinary education. Our concern and involvement in education is further exemplified by the regular financial support that the Veterinary Foundation is giving to post graduate students who wish to further their studies locally and abroad.

The SAVA has always been, and I believe still is, a proponent of a second faculty for the training of veterinarians. We left no stone unturned in an attempt to persuade the authorities that in the light of the prevailing shortage of veterinarians the establishment of a faculty away from the existing one which is situated at Onderstepoort was desirable.** Despite all our efforts, it was, however, decided to enlarge the intake of students at Onderstepoort from 45 to 90 students. The first of these enlarged classes has already qualified and, on completion of their military training, will soon actively enter our ranks. At this tempo the number of veterinarians in South Africa will probably double during the next 10 years – a fact that will have a major impact on our profession. At the present moment there are numerous vacancies available in all fields, but as the numbers become larger, this might change and new avenues of employment will have to be found. By virtue of their wide training, I believe that this should present no problem, but it represents an exciting challenge for those veterinarians who will enter new fields that up to now have not been explored or exploited. This strengthening of our numbers will also offer the opportunity to re-establish our involvement in areas that have been neglected and that are presently largely occupied by animal scientists, zoologists, physiologists and similarly trained persons.

It is, however, disturbing that the present facilities for training 90 students at Onderstepoort are far from ideal and are certainly not conducive to inspired teaching. As a professional body, I believe that it is our responsibility to express our concern on this matter and to make a plea to the authorities to rectify the situation. The issue seems to be to decide whether the Faculty should be enlarged and modernized at its present site, or whether a completely new establishment should be built elsewhere in the vicinity of Pretoria. Both alternatives have merit and the SAVA has specifically refrained from expressing an official opinion on the matter. It is nevertheless imperative that a final decision should be taken soon. This is essential for the maintenance of the standards of training that the profession and the public have come to expect of veterinarians in South Africa.

The introduction of the various M.Med. Vet. courses has opened new dimensions for us. These courses create the opportunity for specialization and should also enable the Faculty to rationalize the undergraduate course and bring it back to realistic proportions. Despite the excellent work that is being done by the full-time Faculty staff, I believe that far greater use can be made of part-time lecturers particularly on the post graduate level. There are, for example, numerous individuals employed at the Veterinary Research Institute, Onderstepoort who possess a profound knowledge and expertise in specific disciplines and it is sad that, apart from periodically acting as external examiners, their expertise is not also exploited for formal training purposes. The same also applies to persons in private practice who

*Delivered at the Biennial National Congress of the South African Veterinary Association which was held in Cape Town during September 1981.

**Editor's note: At the Annual General Meeting of the SAVA which was held shortly after this address was delivered, a resolution was passed requesting the SAVA Council to re-investigate the need for a third veterinary faculty.

have established themselves in fields such as equine practice, poultry pathology, public health, veterinary jurisprudence and laboratory diagnostics. Although students are exposed to practices, it can only be to everyone's advantage if our profession's total teaching capacity is utilized to its full extent. In this respect I believe that we can be well guided by the principles employed by the medical and dental professions.

Research

Research is the very foundation of Veterinary Science and the work done at the Veterinary Research Institute has been the basis of the reputation that the veterinary profession has built up over the last 75 years. It is therefore disturbing to realise that, despite the contribution and achievements of a number of dedicated individuals, there has been no significant growth in this field over the last 25 years.

In 1958 there were 38 veterinarians employed at the Institute and this has only increased to 40 at the present moment. In fact, research in certain disciplines had virtually ceased, whereas in many others the investigations revolves around only one or two experienced individuals. Considering that the total number of veterinarians in South Africa now totals approximately 1 000, this means that only 4 % of our profession are actively involved in the pursuit of new knowledge. It must nevertheless be conceded that the Faculty has expanded appreciably since 1973 but, because of the teaching load, these colleagues cannot effectively commit themselves to long-term research programmes.

A small number of veterinarians have nevertheless established themselves in various fields of bio-medical research. This development is to be welcomed since it opens new fields for our profession, but their achievements do not directly benefit veterinary science as such.

The contributions of scientists such as chemists, entomologists and microbiologists to veterinary science must, however, not be ignored. As in the past their efforts still substantially expand our knowledge of basic biological processes, and as the need for more fundamental investigations become necessary, their expertise will become indispensable. Our appreciation of their role is in fact reflected by the several individuals in this category have been awarded honorary membership of the SAVA.

In the light of the fact that intensive farming practices are presenting problems that can only be solved by intensified and more extensive research, and considering that other fields such as equine medicine, aspects of reproduction, basic physiology, and pharmacology have been sorely neglected, the SAVA Council has decided to launch an investigation into the whole problem. We are deeply aware of the dedication of individuals and the efforts of the existing government and other establishments. Our intentions are not to criticize but we trust that, by an objective and comprehensive review, we may be able to come forth with proposals that will be of general benefit. We wish Dr N.P.J. Kriek and his committee success with their endeavour.

Stedelike privaat praktyk

Van die verskillende fasette van veeartsenykunde het die beoefening van private praktyke gedurende die afgelope dekades ongetwyfeld die grootste groei getoon, nie net in omvang nie maar veral ook in diepte. Daar is by my geen twyfel nie dat in hierdie verband die praktisyn in Suid-Afrika hoegenaamd nie by sy eweknie elders in die wêreld afsteek nie. Dit geld veral die standaarde wat in kleindier chirurgie en in gespesialiseerde perdepraktyke bereik is. Daar is, egter, twee aspekte wat nie uit die oog verloor moet word nie. Ten eerste, moet ons steeds besef dat met enkele uitsonderings enige dier 'n inherente ekonomiese of sentimentele waarde het wat 'n perk plaas op wat 'n eienaar vir die versorging van sy huisdier bereid sal wees om te betaal. Hierdie feit bring mee dat daar altyd realisme moet wees wanneer daar oor gelde besin word. Indien veeartsenykundige gelde peile sou bereik wat vir die algemene publiek onaanvaarbaar is, sal die noodwendige neiging wees om veeartsenykundige dienste op 'n kollektiewe basis aan te bied. Daar is trouens reeds tekens hiervan soos weerspieël deur die bedrywighede van bepaalde dierewelsynsorganisasies.

Ten tweede, sal die toevloei van 90 nuwe kollegas per jaar noodwendig ook 'n besondere invloed hê. Teen hierdie tempo sal die aantal veeartse in Suid-Afrika gedurende die volgende 10 jaar verdubbel, terwyl die stedelike dierebevolking na verwagting nie teen dieselfde koers sal toeneem nie. Tans is die werksgeleenthede onbeperk, maar dit sal nie noodwendig onbepaald so voortduur nie. Ten spyte van die verhoogde lewenstandaard van die Swart-, Kleurling- en Indiërbevolkings wat eise vir meer dienste sal meebring, moet ons aanvaar dat praktyke mettertyd wat getalle betref kan krimp. Dit is dus heeltemal gepas dat by 'n vorige geleentheid en weer by hierdie kongres tyd afgestaan word om te besin oor rasionalisering van praktykbestuur.

Landelike privaat praktyk

In teenstelling met die rooskleurige beeld wat vir stedelike praktyke geskets is, is die toestande wat landelike praktyke betref minder bevredigend. Trouens hierdie besef het daartoe gelei dat die SAVV op inisiatief van die Veterinêre Stigting en onder leiding van dr. Bill Sykes 'n indringende ondersoek na die aangeleentheid geloods het. Die resultaat van die landswye dinkskrums het uitgeloop op 'n besef dat indien die veearts hom steeds op die platteland wil handhaaf, hy 'n voorkomende kudde gesondheidsbenadering sal moet volg en weg moet beweeg van 'n suiwer kliniese benadering en die lewering van 'n nooddiens. Hierdie bevinding is stellig reg maar is nie eenvoudig om te implementeer nie. Die feit is dat die Suid-Afrikaanse boer reeds op 'n twee front indirek veeartsenykundig bedien word en ek wil graag enkele van hierdie aspekte noem:

1. Deur die betrokkenheid van die Afdeling Veeartsenydiens verseker die owerheid dat die land se veestapel van epizootiese en eksotiese siektes gevrywaar word. Voorts is daar skemas onderweg om tuberkulose en brucellose te beheer en boere met ernstige probleme het, deur hulle staatsveeartse, toegang tot diagnostiese laboratoria en voorligting.
2. Bykans alle navorsing op voedseldiere word deur die owerheid onderneem en hierbenewens word entstowwe teen 'n wye reeks infeksiesiektes teen baie billike pryse deur die Navorsingsinstituut vir Veeartsenykunde voorsien. Verder publiseer die Departement van Landbou en Visserye 'n uitgebreide verskeidenheid brosjures en inligtingstukke wat boere op die hoogte van die nuutste veeartsenykundige ontwikkelings hou.
3. Alle melk en vleis wat geproduseer word is aan veeartsenykundige ondersoeke onderworpe en indien

enige siektetoestand gevind word, word die produsent geadviseer hoe om dit reg te stel.

4. Deur die verspreiding van wurmmiddels, dipstowwe en ander geregistreerde geneesmiddels, speel die handel ook 'n groot rol om boere selfversorgend te maak.
5. Daar is ook 'n tendens vir instellings soos die Vleisraad, Wolraad, K.I.-Koöperasies en boerevereenigings om veeartse in diens te neem wat navorsing doen en dienste aan die lede van die instellings op 'n kollektiewe basis lewer.
6. Ten slotte moet gekonstateer word dat baie boere vandag formele opleiding bekom en gevolglik in staat is om verskeie siekte te identifiseer en te behandel, eerstehulp toe te pas en elementêre chirurgie te verrig.

Uit wat hierbo aangedui is, blyk dit dat daar vir die privaat praktisyn nie veel oorbly waarop hy hom kan toespits nie. Desnieteenstaande glo ek dat hy nogtans 'n belangrike rol het om te vervul mits hy bereid is om 'n persoonlike en gespesialiseerde diens te lewer. Dit gaan egter meebring dat hy hom veral op produksie-aspekte sal moet toelê. Hy sal hom in vakgebiede soos teling, voeding en bestuur moet inwerk en tog nie sy eerste funksie as arts nalaat nie.

Die kuddegesondheidsbenadering sal egter meebring dat die veearts al nader aan die werksgebied van die veekundige sal beweeg. Daar is al vrese dat dit tot botsings kan lei, maar dit is my oortuiging dat die beginsel van outonomiteit in eie kring ook hier geld. Indien die veearts en veekundige mekaar se opleiding respekteer kan die een beroep die ander aanvul en kan vrugbare samewerking tot wedersydse voordeel bewerkstellig word.

Dit is ongelukkig waar dat min boere besef wat 'n veearts vir hom kan doen. Dit is ook miskien daaraan te wyte dat ons te lank slegs 'n kliniese diens gelewer het wat min langtermyn ekonomiese nut gehad het. Indien ons met ons nuwe benadering wil slaag sal ons 'n besondere poging as SAVV maar veral as individue op plaaslike vlak moet aanwend om betyds die idee van 'n kuddebenadering wat langtermyn ekonomiese voordele inhou moet propageer. Die formulering van die idee was maklik. Om dit te implementeer gaan veel moeiliker wees.

Conclusion

The SAVA is an extremely diverse organization that is continuously involved in a large variety of activities. If it were not for the work done by numerous committees and particularly the chairmen of these committees this task could never be accomplished.

Since they have not made themselves available for re-election to council I wish to adress myself to two persons in particular.

The first is Prof. L.W. van den Heever. He has served the SAVA with dedication and distinction for many years, amongst others as President. His most recent major contribution was as chairman of the Veterinary Act Committee. The new Act will be the guiding document for the veterinary profession in South Africa for many years to come and will be a monument to him. For this and all else, we thank him.

It is also with sincere regret that we have to take leave of Dr R.D. Bigalke who, because of numerous commitments, is unable to continue as a member of Council. His balanced contributions at council meetings and particularly the role he played as chairman of the awards committee is gratefully recalled. He fulfilled this arduous task with the preciseness that we are accustomed to and his guidance will be missed. The SAVA has a true friend in the Director of the Veterinary Research Institute and we wish him every success in this most important task.

Ladies and gentlemen, thank you for your attention and may you all return home from this Congress enriched and inspired to serve our fine profession.

COLIN CAMERON

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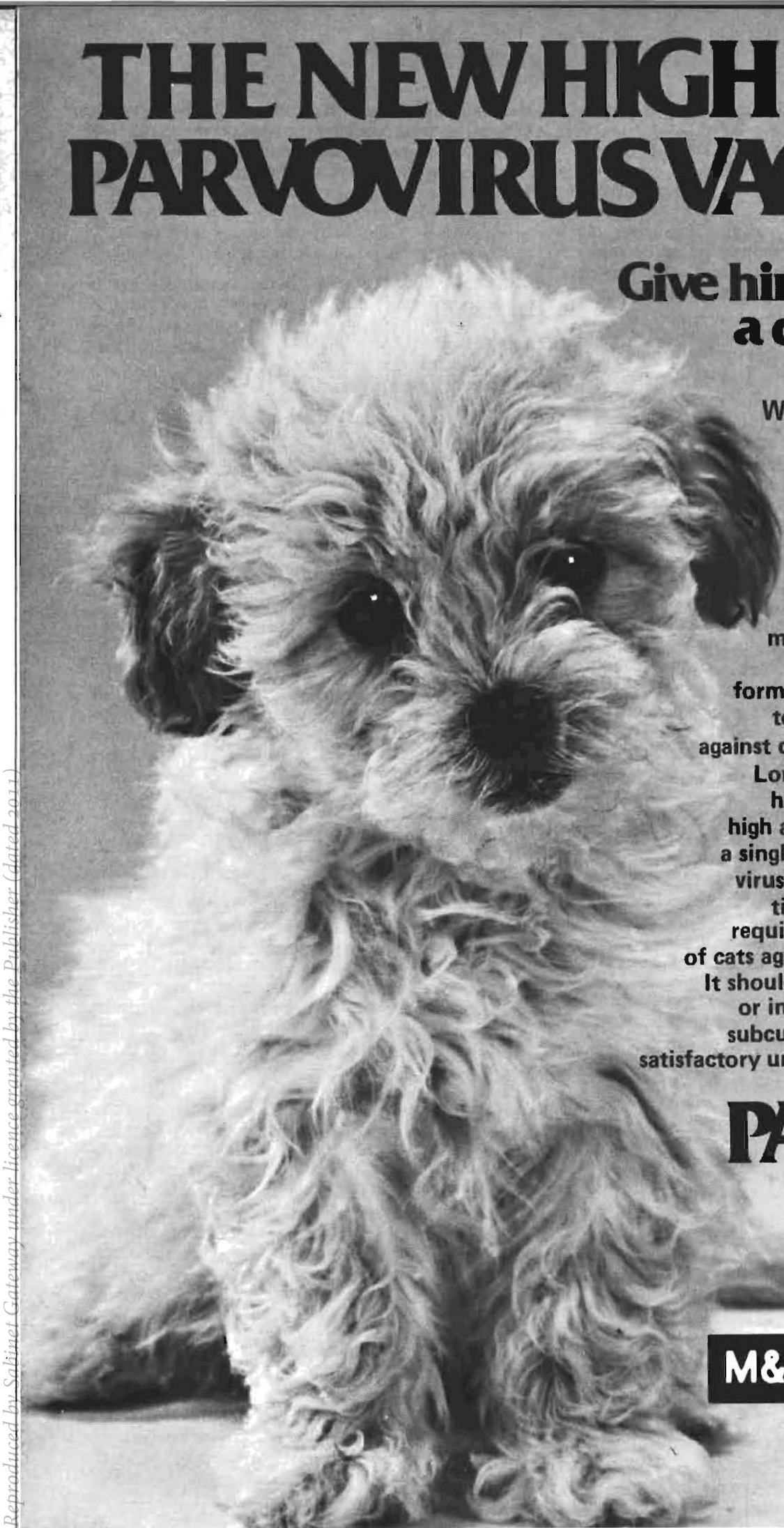

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VETERINARY RESEARCH

Because of Sir Arnold Theiler and his successors' association with Onderstepoort, the Veterinary Research Institute has become synonymous with veterinary research in the Republic of South Africa. It is, no doubt, because of this that the Institute must bear the brunt of the criticism whenever someone chooses to vent his or her anger or frustration because of some actual or imagined shortcoming in veterinary research. This type of criticism, from within and without the ranks of the profession, has become rather commonplace lately. Some of this criticism, however, does not bear close scrutiny and it is also rather embarrassing to find that, when pressed, the most outspoken critics tend to back down or do not respond at all. Contrarily, the most constructive and rational criticism emanates from the ranks of the researchers themselves and one is left with the impression that a large portion of the criticism is often directed against nobody in particular, without a grasp of the complications and difficulties of the day-to-day running of a research programme, and without a grasp of the fundamental directions towards which research should be directed.

However, the foregoing is not intended to create a false sense of well-being, because even the most ardent defender will have to admit that all is not well in the realms of veterinary research in RSA. One should also not be misled when referring to veterinary research, that reference is only being made to one specific institution. It should be understood that the entire spectrum of veterinary research, or the absence thereof, also encompasses that undertaken by private enterprise, other state concerns and in the veterinary faculties. It is only through a cooperative effort of all these associated institutions and concerns, that the problems of national importance can be tackled in any rational way.

If we had to ask ourselves what the impact of the current research is on our general veterinary problems, would we be able to claim without fear of contradiction that it is optimal, or even adequate? The SAVA's concern in this matter is reflected by the establishment of a committee to investigate and comment on the various aspects of veterinary research and the veterinary researcher in RSA. Since this inquiry is still underway it would be presumptuous at this stage to comment on the possible outcome of this investigation.

It may be necessary during this investigation to disturb some of the sacred cows of veterinary research in order to reach and implement the best possible solutions. Have we not as a profession and often as individuals not even associated with the Institute, too long basked in the glory of Sir Arnold Theiler and the name of Onderstepoort? Have we protected our legacy and developed it to the best of our ability, even when acknowledging the restrictions with which we have to contend? Have we not for too long been relying on the expertise, or in fact, demanded of a few one-man departments that they pursue the avenues of research on a multitude of problems that defy the efforts of teams of researchers elsewhere? Can we still consider ourselves to be the undisputed experts on the animal diseases of Africa, or to be adequately equipped to maintain ourselves in the fields that we traditionally consider to be ours, not only in the face of the ever-increasing international competition, but also in the local sphere of animal production and the shifting emphasis on the veterinary approach to these problems? Can we say without contradiction that we have kept up with the demands of modern technology and current problems, or do we still tinker with the problems of yesteryear unaware of the development or changing requirements in the world around us? Is it not time to re-assess our situation and our problems in the light of all these factors and not allow ourselves to be beaten by seemingly insurmountable obstacles of bureaucracy and professional jealousies?

The problems are not only those of the researcher, but of each individual member of the profession because the results of problem-orientated research bolster, or distract from, the image of the profession and the country as a whole. It may be that we are so awed by our so-called insurmountable obstacles that we often forget that "it is not the talents that we possess so much as the use that we make of them, that counts in the progress of the world[1]".

1. Robertson BT 1931. The spirit of research. Preece and Sons, Adelaide.

EDITORIAL — VAN DIE REDAKSIE

THE ROLE OF THE VETERINARY FOUNDATION IN THE ADVANCEMENT OF THE VETERINARIAN AND VETERINARY SCIENCE IN SOUTH AFRICA

The Republic of South Africa is one of those fortunate nations which can not only claim to being self sufficient in the production of food for its human population, but can also be numbered amongst that even more exclusive group of countries, the food-exporting nations of the world. Fortunate as we may be, this situation has not come about easily. Africa is plagued all too often by drought, an erratic climate and many infectious diseases of animals, several of which are enzootic to this continent. In spite of these adverse factors, thanks to the resilience and determination of the farming community and the contribution of its scientists, the RSA is in a strong position to fight the scourge of malnutrition and hunger which is the major threat to the health, productivity and stability of so many nations of the world. For this reason our scientists, including plant pathologists and geneticists, animal productionists and veterinarians, to name but a few, deserve the nation's continued support and recognition for the vital services they perform. As veterinarians we must always remain mindful of our great responsibility and obligations to the livestock industry, for we can justifiably be regarded as one of the key professions. We must not, however, despite the proud history of veterinary science in the RSA and the achievements of our predecessors, ever become complacent, self-satisfied or rest on our laurels; there are still too many problems on the scientific front or affecting our professional well-being which are yet to be resolved.

It has been encouraging in recent years to observe the greater awareness of veterinarians of the difficulties facing the profession. We have matured to the point where we realise and recognise many of our shortcomings and, more importantly, are searching for the solutions to overcome them. No one is in a better position to do this than veterinarians themselves. We cannot expect bodies outside the veterinary profession, such as the State or even individuals, to embrace out cause so fully that they will resolve all out own difficulties for us. This would be an abrogation of our duty. It is only when we have shown that we are capable of helping ourselves, that the assistance of sources outside the profession should be enlisted.

The enthusiasm and dedication required to deal with the problem areas concerning veterinarians are not lacking. However, as is so often the case, having indentified the area of concern, it is a lack of funds which stifles or retards the course of action which should follow. We are, as a profession, invariably deficient in financial resources. The Veterinary Foundation is a channel through which we as veterinarians can help ourselves, and through which we can appeal to and encourage those outside our profession to help us to help ourselves. It is a means by which we can attempt to provide the financial backing for our profession which we have lacked for so long, and which we have been in need of on so many occasions in the past.

The Veterinary Foundation was constituted by the South African Veterinary Association in 1966 to operate and control a trust fund under the patronage of the State President for the benefit of the Veterinary Profession. The profession at that time recognised the need for such a Foundation which could receive and administer grants and donations made by members of the veterinary profession, industry and the general public for the promotion and advancement of veterinary science in RSA.

The Veterinary Foundation is administered by a board of six trustees who may be proposed by the Board of the Foundation, but are nominated and approved by the SAVA. Each trustee serves the Foundation for a term of six years after which he or she retires, but may thereafter be eligible for re-election. The terms of office of the trustees are so arranged that each year one trustee retires in rotation. The Board of Trustees meets on a minimum of four occasions during the year, usually on the afternoon preceding a SAVA Council Meeting. At the first meeting of each year, the trustees propose a Chairman and Vice-chairman for the forthcoming year. These are finally nominated and approved by the Council of the SAVA. The Board of Trustees of the Foundation has the power to co-opt trustees at their discretion. At present, the Deans of the Faculties of Veterinary Science of the University of Pretoria and of Medunsa (Medical University of Southern Africa), the Chairman of the Finance Committee of the SAVA and an attorney to advise on legal matters are co-opted members of the Board.

Although the Foundation is in most aspects entirely independent of the SAVA, there exists a very close liaison between them. Both of these bodies have a basic common object which is the promotion and well-being of the veterinary profession in this country. The Chairman of the Foundation is co-opted onto the Council of the SAVA and reports on the developments within the Foundation at each of the Council's meetings. It must be emphasized that the funds of the SAVA to which its members contribute their annual subscriptions, are controlled by the Finance Committee of the Council of the SAVA. These funds finance the administration of the affairs of the Association. The funds administered by the Veterinary Foundation are entirely independent of those of the Association.

It is the aim of the Foundation to appeal to the veterinary profession for financial help and contributions, and having achieved the support of those it serves, to appeal on behalf of the profession to individuals and bodies outside of the veterinary profession. By increasing the awareness of others of the services provided by the veterinarian to the public, companion animals and livestock population, it is hoped that the Foundation's appeals for assistance will be sympathetically received by those for whom we provide a service.

The Foundation attempts to advance the cause of veterinary science in RSA in several directions:

1. Veterinary Education

Since its inception the advancement of veterinary education has been the chief priority of the Foundation. The provision of bursaries and scholarships at both undergraduate and postgraduate levels, the funding of experts to speak at short courses as part of a continuing education programme and the provision of a library containing audio-visual material and scientific publications are all projects deserving the attention and assistance of the Foundation. As the veterinarian's service becomes more and more sophisticated, and the tendency to specialization increases, the support of those concerned with the training of veterinarians will be more and more deserving of the Foundation's help.

2. Veterinary Research

In this country the majority of veterinary research is carried out at the Veterinary Research Institute at Onderstepoort, Regional Diagnostic Centres of the Division of Veterinary Services, and the Faculty Veterinary Science of the University of Pretoria and by veterinarians in industry. Discoveries made by the research worker today, pave the way for the developments of tomorrow. Thanks to those engaged in research and to the achievements attained in the past, the name of Onderstepoort is internationally known and respected. It is imperative that veterinary research remains an active ongoing process; it must never be allowed to lose its momentum for it is vital for the future of not only veterinary science but also the country as a whole.

On the diplomatic front, veterinary research and vaccine production together form one of the most positive and effective means whereby contact, cooperation and mutual respect can be fostered between our country and its neighbours on the African continent.

It is felt by some that veterinary research is not being expanded at a rate sufficient to meet the demands of the future. An investigation into the whole aspect of veterinary research is therefore at present being launched under the auspices of the SAVA and the sponsorship of the Foundation

3. Rural Veterinary Services

In recent years concern has frequently been expressed about the scarcity of rural veterinarians, and consequently the inadequacy of veterinary services in many country communities. Several symposia and meetings were held to investigate this problem. The conclusion was invariably reached that much of the blame could be laid at the door of economics and the fluctuating fortunes of the farmer. On the initiation and sponsorship of the Foundation, it was decided that the matter should be investigated in a more positive and aggressive manner, and a successful "think-tank" programme was set in motion in order to identify problem areas regarding rural veterinary services and to attempt to find solutions. Rural practitioners have commendably accepted the challenge and have actively promoted and pursued this most worthwhile project.

4. Small Animal Practice

Although the field of small animal practice has expanded at a much faster rate than that of other departments of the veterinary profession during the last twenty years, and outwardly, at least, does not seem to be plagued by the same problems encountered in rural areas and certain other fields of veterinary endeavour, those engaged in urban practice have also expressed the need to take an inward look at themselves and the services they provide. The Foundation has been approached to provide support for the initial stages of a Practice Management Survey which, it is hoped, will assist practitioners in assessing the efficiency of their practices, in costing and in the determination of salaries.

The veterinary profession has reached a vital stage in its development. As veterinarians we are being faced with new, at times exciting and at times disturbing, challenges. We may not ignore or circumvent them. The Veterinary Foundation, it is hoped, will assist us to meet these challenges. It is a channel through which those outside the veterinary profession can recognise and acknowledge the part played by veterinary science in the development of our nation, and it is a means by which the veterinary profession itself can show its support, dedication and commitment to its future.

BOEKRESENSIE

BOOK REVIEW

LAMENESS IN CATTLE

P.R. GREENOUGH, F.J. MacCALLUM and A.D. WEAVER

2nd Edn edited by A.D. Weaver

John Wright and Sons, Ltd, 42 Triangle West, Bristol BS8 IEX, England. 1981 pp 471. Price £25,00

As geheel gesien is dit 'n goeie en uitputtende werk oor mankhede by beeste. Die aanbieding is deurgaans van goeie gehalte en korrekte deurdagting. Alhoewel dit die enigste beskikbare boek oor hierdie onderwerp is, is dit outoritêr en kan moeilik verbeter word.

Een derde van die boek word gewy aan die anatomie van die bees met klem op die ledemate.

Hierdie boek word aan studente voorgeskryf en ek glo dat die praktisyn net voordeel kan trek deur die aankoop van so 'n boek.

S.S. van den Berg

THE AVERMECTINS: A NEW FAMILY OF ANTIPARASITIC AGENTS*

I.K. HOTSON**

ABSTRACT: Hotson, I.K. **The Avermectins: a new family of antiparasitic agents.** *Journal of the South African Veterinary Association* (1982) **53** No. 2, 87-90 (En) Merck Sharp and Dohme Research Laboratories, Ingleburn, NSW, Australia 2565.

The avermectins are macrocyclic lactones produced by fermentation of the soil micro-organism *Streptomyces avermitilis*. They show activity against a broad range of nematodes and arthropod parasites of domestic animals at dose rates of 300 μg/kg or less. Unlike the macrolide or polyene antibiotics, they lack significant antibacterial or antifungal activity.

By oral or parenteral administration, avermectins are active against gastrointestinal nematodes and lungworms, and important ectoparasites such as lice, mange mites, ticks and larval stages of flies. They show excellent activity against parasites resistant to existing anthelmintics or ectoparasiticides.

The avermectins appear to cause paralysis of nematodes and arthropods by opening *gamma*-aminobutyric acid-mediated chloride channels at the neuromuscular junction.

INTRODUCTION

The avermectins belong to a family of new compounds of microbial origin, which have shown remarkable activity against a broad range of internal and external parasites of animals. This paper reviews events leading to the discovery and characterization of these compounds, and reviews papers dealing with early efficacy studies.

DISCOVERY

For many years, the parasitology program at the Merck Institute has been devoted to finding new synthetic organic chemicals active against coccidia and helminth parasites of birds and animals. In more recent years, a search for radically different agents was instituted and soil micro-organisms have been isolated world-wide for laboratory screening. These organisms were cultured in the laboratory and the broths were then concentrated and fed to helminth-infected animals. Anthelmintic activity was assessed by the extent of worm removal and by observing changes in numbers of parasite eggs in faeces of mice carrying the nematode *Nematospiroides dubius*[12].

Evidence of activity was noted in a culture derived from a soil sample from Japan. Early tests showed that whole broth was active in mice over at least an eightfold dosage range without notable toxicity. When the unknown active portion of the culture was isolated by Burg et al.[11] it was shown to be active at levels down to 1 ppm in the diet, representing a very high level of potency.

ISOLATION

Isolation and purification of the active components from the broth was achieved by solvent extraction, solvent partition and absorption methods; novel partition chromatography systems were used to separate the components. The complex contains four major components, A_{1a}, A_{2a}, B_{2a} in varying proportions, and four minor components A_{1b}, A_{2b}, B_{1b} and B_{2b} each of which is a lower homolog of the corresponding major component[27].

Mixtures of homologs can be designated by omission of the subscript letter: for example B_1 will indicate a mixture of B_{1a} and B_{1b}, (Miller et al.[27]).

CHARACTERIZATION/STRUCTURE

The chemical structure of the avermectins was shown to be that of a macrocylic lactone[1]. Unlike the macrolide antibiotics, they do not possess significant antibacterial or antifungal activity[11].

Organic chemists subsequently modified the individual components of the avermectins and produced numerous analogues, on which studies were then done to determine safety and efficacy in relation to the parent compound.

One of the compounds was 22,23-dihydroavermectin B_1. This compound showed good characteristics of efficacy and safety in laboratory studies and was selected for further development[15 18].

IDENTIFICATION OF ORGANISM

The microbiologists had found that the new compounds were produced by a previously unknown species of *Streptomyces*, which they named *S. avermitilis*[11]. The specific name implies averminous, or "no worms".

It is of interest to note that of the many microbial fermentation products investigated over recent years, very few have been described as having anthelmintic activity. Hygromycin B is the only such product which has been commercialised.

MODE OF ACTION

From early efficacy studies in sheep, it was apparent that the avermectins were highly effective against many nematode parasites, including those showing resistance to existing anthelmintic "families" such as benzimidazoles and levamisole[17].

The avermectins paralyze worms, and do so in a unique way. In nematodes, the neurotransmitter which sends inhibitory signals from interneurons to motorneurons is *gamma*-aminobutyric acid. A series of studies[20 23 30] has shown that the avermectins potentiate the inhibitory effect of GABA. Signals from the central nervous system are therefore not perceived by the motorneurons and a state of paralysis ensues.

The avermectins have shown no activity against trematodes or cestodes, and these parasites are throught

*Presented at the Biennial Congress of the South African Veterinary Association, Cape Town, 7-11 September 1981.

**MSD Research Laboratories, P.O. Box 135, Ingleburn, NSW, Australia 2565.

not to use GABA as a neurotransmitter. This is therefore in keeping with the hypothesis regarding mode of action.

Arthropods use GABA as a neurotransmitter, not between two neurons as in nematodes, but between nerve and muscle cells – they inhibit neuromuscular transmisson of nerve impulses[20].

The avermectins have shown activity against several arthropod parasites of animals, and with this expanded spectrum of activity, the name chosen for the class of compounds was "anti-vermes" and "anti-ectoparasites", contracted to "a-verm-ect-ins".

SPECTRUM OF ACTIVITY

Endoparasites of ruminants

Early studies with the four major components of the avermectin family indicated that the B fractions were more active against nematodes than the A fractions and that fraction B_1 had a broad activity against nematode parasites of sheep at levels down to 100 μg/kg liveweight[17].

Activity against benzimidazole-resistant strains of *Haemonchus contortus* and *Trichostrongylus colubriformis* was confirmed in these studies (Table 1).

TABLE 1: ANTHELMINTIC ACTIVITY OF AVERMECTIN B_1a BY ORAL ADMINISTRATION AGAINST PATENT INFECTIONS IN EXPERIMENTALLY INFECTED SHEEP*

	Mean no. worms in controls	% Reduction in Worm Burden		
		25	50 μg/kg	100
H. contortus (BZA-R)[1]				
Inhibited L_4	70	96	96	96
Adults	442	96	99	98
O. circumcincta				
Inhibited L_4	1 421	38	94	95
Adults	1 137	74	99	98
T. axei				
Adults	2 852	49	99	99
T. colubriformis (BZA-R)[1]				
Adults	4 110	86	99	99
C. oncophora				
Inhibited L_4	193	11	98	97
Adults	1 761	40	93	94
Oes. columbianum				
Adults	61	97	100	100

[1]Benzimidazole-resistant strain.
*From: Egerton et al. 1979[17]

In cattle, as with sheep, there was very high activity against lungworm (*Dictyocaulus*) and nodular worm (*Oesohagostomum*) even at low dose rates, and Egerton et al.[19] showed excellent activity against several species of cattle nematodes using ivermectin at 200 μg/kg (Table 2).

A feature of many anthelmintics is poor or variable activity against a problem parasite of cattle – arrested early L_4 *Ostertagia*. In a recent review paper, Leaning[24] has summarized the work of MSDRL scientists and independent investigators which demonstrated consistently high activity against this parasite using ivermectin at 200 μg/kg by either the oral or parenteral route (Table 3).

TABLE 2: ANTHELMINTIC EFFICACY OF IVERMECTIN AS A SINGLE SUBCUTANEOUS DOSE AGAINST EXPERIMENTAL INFECTIONS IN CATTLE*

	Mean no. worms in controls	% Reduction in Worm Burden		
		50	100 μg/kg	200
Haemonchus placei adult	3 377	>99	>99	>99
Ostertagia ostertagi early L_4	926	97	99	>99
adult	6 301	>99	>99	>99
Trichostrongylus axei adult	3 489	97	>99	>99
Trichostrongylus colubriformis adult	1 629	0	27	90
Cooperia oncophora adult	5 482	15	86	98
Cooperia punctata adult	6 045	52	81	98
Oesophagostomum radiatum adult	192	100	100	100
Dictyocaulus viviparis adult	165	100	100	100

*From Egerton et al 1981[19]

Published studies by independent workers[2 7 31 34] have since confirmed the high efficacy of ivermectin at 200 μg/kg against both larval and adult stages of the common nematode parasites of cattle. Some variation in efficacy against adult *Cooperia oncophora*[31] and adult *Nematodirus helvetianus*[2] has been recorded.

Ectoparasite activity in ruminants

It was noted earlier that the avermectins exhibited activity against a number of economically-important arthropod parasites of domestic animals.

First suggestions of this activity were from early screening studies[29] with confused flour beetle (*Tribolium confusum*) and the larvae of the robust bot fly in mice (*Cuterebra* spp). Extension of testing to the sheep blowfly, *Lucilia cuprina*[22] gave further evidence of ectoparasite activity. Since then studies with a variety of host/parasite systems have demonstrated activity against lice[5 24], mange mites[3 5 26 33], ticks[14 18 21 28 32] and *Hypoderma* infestation[16 24]. Generally, a single subcutaneous treatment with ivermectin at 200 μg/kg gave excellent control against the mange mites, *Psoroptes* and *Sarcoptes*, *Hypoderma* spp., single host ticks and sucking lice of cattle. An example of the high efficacy against *Sarcoptes* infestation of cattle is shown in Table 4[5].

Activity in dogs, pigs and horses

Broad spectrum efficacy against nematodes and ecto-

TABLE 3: A SUMMARY OF TRIALS DEMONSTRATING EFFICACY OF IVERMECTIN IN CATTLE AT 200 μg/kg AGAINST *OSTERTAGIA OSTERTAGI**

	Number of Trials	Number of Animals		Range of Parasites in Controls	Mean Efficacy
		Control	Treated		
Adult	23	154	145	137-42 050	>99
Developing L_4	14	88	85	313-48 316	98
Inhibited L_4	8	53	51	8 008-225 354	>99

*From Leaning W.H.D. 1981[24]

TABLE 4: ARITHMETIC MEAN NUMBERS OF *SARCOPTES BOVIS* FROM CATTLE TREATED WITH IVERMECTIN* (TOTAL NUMBER OF LIVE MITES FROM 7 SITES)

Group	Number of Animals	Day of observation (Treated Day O)								
		−1	7	14	21	28	35	42	49	56
Control	3	504	551	551	493	564	487	502	506	483
100 μg/kg s.c.	3	457	105	13	13	31	78	28	3	17
200 μg/kg s.c.	3	467	0	0	0	0	0	0	0	0
400 μg/kg s.c.	2	469	0	0	0	0	0	0	0	0

*Barth D. and Sutherland I.H. 1980[5]

parasites is not limited to sheep and cattle. Extensive evidence is now available of activity against the major nematode parasites of dogs, pigs and horses, as well as evidence for ectoparasite control in these animals.

The avermectins are active against the common gastrointestinal parasites of dogs at 200 μg/kg. Blair and Campbell[8] demonstrated activity against adult *Ancylostoma* at doses as low as 3 μk/kg. However, doses of at least 200 μg/kg are needed for control of adult ascarids, *Strongyloides* and *Trichuris* (Egerton, unpublished information).

Avermectins are not active against adult *Dirofilaria*, the dog heartworm[13] but are highly active against microfilariae in the blood[9] and against early larval stages of the parasitic worm[10]. This means that control of heartworm infection can be achieved by a single dose of ivermectin given at approximately monthly intervals without any danger of killing adult parasites and therefore without the risk of also killing the dog. This will represent a tremendous advance in the prophylaxis of this disease.

In pigs, Barth et al.[6] reported high efficacy of ivermectin at 300 μg/kg against *Hyostrongylus, Oesophagostomum* spp, and *Metastrongylus* spp. *Strongyloides ransomi* was controlled either directly by treatment or by prevention of colostral transmission of the parasites by pre-farrowing treatment of the sow.

Activity against the pig louse, *Haematopinus suis* and the mange mite, *Sarcoptes scabei* var *suis* was excellent, following a single treatment at 300 μg/kg[4 6 25].

In horses, a single intramuscular injection of 200 μg/kg gives high efficacy against larval and adult strongyles and cyathostomes (including benzimidazole-resistant strains), and also *Parascaris, T. axei, Oxyuris* and *Habronema*. Treatment is also highly effective against larval stages of *Gastrophilus* spp[24].

CONCLUSION

The avermectins represent a new generation of antiparasitic agents for use in domestic animals. They show great promise not only for therapeutic use, but also for timely application in programs for prophylaxis of a number of important parasitic diseases.

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BOOK REVIEW — BOEKRESENSIE

ANIMAL DISEASE OCCURRENCE

Commonwealth Agricultural Bureaux, Commission of the European Communities, Farnham Royal, Slough SL2 3BN, UK. Published twice a year. Annual subscription £25-00. Volume 1, 1980. ISSN 0144-3879

Animal Disease Occurrence (ADO) is the printed form of the Animal Disease Information System which was recommended by the Round Table on Informaton and Documentation in Veterinary Medicine which met in Luxembourg between 1973 and 1978. The recommendation was implemented by the Directorate General "Information Market and Innovation" of the Commission of the European Communities. ADO is compiled and marketed by Commonwealth Agricultural Bureaux.

ADO is intended to provide worldwide coverage of information on disease occurrence and present it in a form which will be useful to its users. The information is collected from all that is recorded in the published and unpublished literature. ADO is not intended to provide immediate notification of animal disease status of a given country.

The journal is divided into two main sections. The first section consists of numbered abstracts (in English) with fully annotated references. This section has a similar format to *Veterinary Bulletin* (VB) but in ADO the abstracts are classified under 12 species headings. Some abstracts in ADO are identical to those appearing in VB, though they are often suitably prepared or edited to include information for ADO; but many abstracts are exclusive to ADO. All abstracts are listed in *Index Veterinarius*. The extra abstracts collected for ADO include abstracts of regular disease reports, relevant sections of annual reports (often abstracted as a whole for VB), individual papers in conference proceedings and non-conventional literature. The second section is a unique tabular section containing factual data from the original article. The tables may be searched easily for the occurrence of a particular disease, diseases occurring in a particular country and for diseases occurring in a particular species. Answers to questions on disease occurrence may be obtained without recourse to massive literature searches and laborious reading of source material. The entries in the tables are linked to the abstracts by the abstract number.

One of the purposes of this project is to create a computerized databank which will become accessible via the *EURONET* telecommunications network. This is being implemented by *Deutsches Institut fur Medizinische Dokumentation* (DiMDi) and should be available for online searching at the end of 1981.

THE EFFECT OF A SINGLE INJECTION OF NITROXYNIL AT 20 mg/kg LIVE MASS IN THE TREATMENT OF *PARAFILARIA BOVICOLA* INFESTATIONS IN CATTLE

A.C. WELLINGTON and L. VAN SCHALKWYK*

ABSTRACT: Wellington A.C., Van Schalkwyk L. **The effect of a single injection of nitroxynil at 20 mg/kg live mass in the treatment of *Parafilaria bovicola* infestations in cattle.** *Journal of the South African Veterinary Association* (1982) **53** No. 2, 91-94 (En) Animal Health Research Station, Maybaker (S.A.) (Pty) Ltd, P.O. Box 1130, 6000 Port Elizabeth, Republic of South Africa.

The efficacy of nitroxynil administered once by subcutaneous injection at a dosage regimen of 20 mg/kg live mass was evaluated against natural infestations of *Parafilaria bovicola* in cattle.

Trial animals were slaughtered 14 weeks after treatment. Treatment reduced the number of bleeding points by 97,8 %, eosinophil-positive carcass lesions by 85,7 % and eosinophil-positive lesion area by 92,8 %, compared with controls.

Key words: *Parafilaria bovicola*, nitroxynil, cattle.

INTRODUCTION

Wellington[1] treated cattle infested with *Parafilaria bovicola* with nitroxynil (Trodax, Maybaker) with 2 subcutaneous injections of 20 mg/kg live mass, given at an interval of 72 hours, and demonstrated a 95 % decrease in the mean affected lesion area and a 90,7 % decrease in the mean number of carcass lesions, when compared with those in untreated animals.

The present trial was carried out in order to examine the effect of nitroxynil when used as a single subcutaneous injection of 20 mg/kg live mass.

MATERIALS AND METHODS

(i) Selection of experimental animals and treatment

During September 1980, 22 animals (oxen and bulls) on a farm near Alldays (northern Transvaal) were selected from a herd of animals exhibiting lesions of *P. bovicola*, and were divided into 2 groups

1. A group of 11 control animals that were not treated.
2. A group of 11 animals that were injected subcutaneously with nitroxynil at 20 mg/kg live mass on 1 October 1980.

After treatment, the animals were transferred to the Maybaker Animal Health Research Station near Port Elizabeth on 3 October 1980, and stayed there for 3 months until they were slaughtered at the Port Elizabeth abattoir, where the carcasses were examined.

(ii) Examination of animals

The animals were examined every day from one month post-treatment to 3 days prior to slaughter. All lesions from which fresh blood exuded on each day, were recorded.

(iii) Carcass examinations

The control and treated animals were slaughtered 14 weeks after treatment during the period 6 to 8 January 1981.

During the examination of the carcasses, the number of lesions and the dimension of each lesion on the carcass were recorded. After skinning, the total skin area of each animal was estimated by measurement.

To differentiate between typical parafilarial lesions and bruised areas, impression smears were taken from all lesions, fixed with methanol and stained by the Giemsa method and examined microscopically to detect the presence or absence of eosinophils. In the final calculations, only eosinophil-positive areas were taken into account and lesions and lesion surface areas per animal in each group were calculated.

Grading of the carcasses was undertaken by the Control Inspector of the Department of Agriculture and Fisheries, Port Elizabeth.

In order to calculate the economic viability of the treatment, the following factors were taken into account: cost of treatment, cold dressed mass, mass of trimmings due to lesions removed from carcass, grading of carcass after trimming, financial loss due to trimming, calculated on the mass of trimming multiplied by carcass grading price prior to trimming, financial loss due to downgrading of carcass, and the price of each carcass at the auction sale.

RESULTS

The appearance of positive animals and the number of bleeding points recorded at weekly intervals from 33 (Day +33) to 95 (Day +95) days after treatment respectively are recorded in Table 1.

In Tables 2 and 3 the individual results obtained after examination of each carcass are indicated.

Table 4 is a summary of the effects of the treatment on the number of lesions and eventual lesion areas per carcass in each group.

A single treatment of nitroxynil at 20 mg/kg decreased the number of eosinophil-positive lesions by 85,7 % and decreased the eosinophil-positive lesion areas by 92,8 %. The results of the latter were shown by the Student's t test to be statistically significant ($P < 0,001$).

The financial implications with reference to the cost of treatment, its efficacy, and the financial savings with regard to the cost of treatment compared with the loss which would have been sustained if not treated, are recorded in Table 5.

DISCUSSION

Nitroxynil had the effect of decreasing the number of bleeding points for a specific period, when compared with the number noted in the control animals.

A single dose of nitroxynil, injected subcutaneously at 20 mg/kg, resulted in a decrease in the number of eosinophil-positive lesions and a decrease in the number of eosinophil-positive lesion areas. The single dose of 20 mg/kg thus has a similar effect to repeating nitroxynil at 20 mg/kg after 72 hours, reported earlier[1].

*Animal Health Research Station, Maybaker (S.A.) (Pty) Ltd, P.O. Box 1130, 6000 Port Elizabeth.

Nitroxynil decreased the number of positive animals showing bleeding points by 81,8 % and the total number of bleeding points by 97,8 % when compared with the control animals during the 9-week period from one month after treatment through to 3 days prior to slaughter.

Nitroxynil, when injected subcutaneously at 20 mg/kg into cattle infested with *P. bovicola*, resulted in a 92,8 % decrease in eosinophil-positive lesion area and an 85,7 % decrease in the number of eosinophil-positive lesions, when compared with unmedicated controls.

It must be emphasized that a period of 9 weeks after treatment and prior to slaughter should elapse to allow the lesions caused by *P. bovicola* to resolve.

TABLE 1: THE CUMULATIVE NUMBER OF POSITIVE ANIMALS AND OF BLEEDING POINTS FROM DAILY EXAMINATIONS RECORDED EVERY 7 DAYS OR 9 AND 6 DAYS ON 1 OCCASION, FROM 33 DAYS (DAY + 33) TO 95 DAYS (DAY + 95) AFTER TREATMENT RESPECTIVELY. THE PERCENTAGE DECREASE WHEN TREATED AND CONTROL GROUPS ARE COMPARED

Group	No. of Animals / Day	The cumulative number of animals after treatment									
		+ 33	+ 40	+ 47	+ 54	+ 61	+ 68	+ 75	+ 82	+ 89	+ 95
Controls	11	0	9	11	11	11	11	11	11	11	11
Nitroxynil	11	0	0	2	2	2	2	2	2	2	2
% Decrease			100	81,8	81,8	81,8	81,8	81,8	81,8	81,8	81,8
		The cumulative number of bleeding points after treatment									
Controls	11	0	77	139	210	261	324	388	420	469	505
Nitroxynil	11	0	0	2	3	4	7	9	10	11	11
% Decrease			100	98,6	98,6	98,5	97,8	97,7	97,6	97,7	97,8

TABLE 2: GROUP 1 INDIVIDUAL RESULTS OF THE UNTREATED CONTROL ANIMALS SHOWING THE NUMBER OF LESIONS THAT WERE POSITIVE FOR EOSINOPHILS, THE SUBCUTANEOUS AREA (CM^2) OF EACH ANIMAL, THE LESION AREA (CM^2) POSITIVE FOR EOSINOPHILS AND THE PERCENTAGE OF THE SUBCUTANEOUS AREA THAT WAS EOSINOPHIL-POSITIVE

Animal No.	Visible carcass lesions		Subcutaneous area cm^2	Lesion area cm^2		% Affected area	
	Total*	E + **		Total*	E + **	Total*	E + **
4	12	7	31 866	5 576	3 582	17,5	11,2
6	7	6	30 450	6 164	5 756	20,2	18,9
7	13	9	23 408	2 782	2 262	11,9	9,7
9	9	9	28 222	4 546	4 546	16,1	16,1
11	10	8	37 414	3 446	3 227	9,2	8,6
19	14	13	31 535	4 756	4 556	15,1	14,4
22	9	5	24 557	3 173	2 372	12,9	9,7
32	1	0	24 960	520	0	2,1	0
35	5	4	33 709	1 953	1 899	5,8	5,6
36	10	8	25 744	3 340	3 040	13,0	11,8
39	10	8	28 444	4 744	4 234	16,7	14,9
Mean	9,1	7	29 119	3 727,3	3 224,9	12,8	11,1

*Total = visible lesions or lesion areas
**E + = lesion or lesion areas positive for eosinophils

TABLE 3: GROUP 2 INDIVIDUAL RESULTS OF THE CATTLE TREATED WITH NITROXYNIL, SHOWING THE NUMBER OF LESIONS THAT WERE POSITIVE FOR EOSINOPHILS, THE SUBCUTANEOUS AREA (CM²) OF EACH ANIMAL, THE LESION AREA (CM²) POSITIVE FOR EOSINOPHILS AND THE PERCENTAGE OF THE SUBCUTANEOUS AREA THAT WAS EOSINOPHIL-POSITIVE

Animal No.	Visible carcass lesions		Subcutaneous area cm²	Lesion area cm²		% Affected area	
	Total*	E+**		Total*	E+**	Total*	E+**
5	5	1	32 480	837	180	2,6	0,6
12	5	0	33 915	998	0	2,9	0
13	5	0	28 461	1 181	0	4,1	0
14	5	3	26 637	1 261	860	4,7	3,2
20	9	5	33 214	3 236	994	9,7	3,0
21	3	0	25 760	508	0	2,0	0
30	7	0	23 561	1 114	0	4,7	0
34	3	1	37 363	1 136	756	3,0	2,0
41	3	0	29 909	312	0	1,0	0
42	4	0	20 703	1 027	0	5,0	0
43	1	1	29 070	100	100	0,3	0,3
Mean	4,6	1,0	29 188	1 064,5	262,7	3,6	0,8

*Total = visible lesions or lesion areas
**E+ = lesion or lesion areas positive for eosinophils

TABLE 4: EFFECT OF TREATMENT ON LESIONS PER CARCASS AND THE DECREASE OF THE AFFECTED CARCASS AREA, EXPRESSED AS A PERCENTAGE OF THE UNTREATED CONTROLS

Treatment	Animals slaughtered	Mean number of carcass lesions		Percentage Decrease	Mean subcutaneous area per animal cm²	Mean lesion areas cm²		Mean percentage affected areas		Percentage decrease in E+ lesion areas
		Total*	E+**			Total*	E+**	Total*	E+**	
Untreated controls	11	9,1	7,0	—	29 119	3 727,3	3 224,9	12,8	11,1	—
Nitroxynil 20 mg/kg	11	4,6	1,0	85,7	29 188	1 064,5	262,7	3,6	0,8	92,8

*Total = visible lesions or lesion areas
**E+ = lesion or lesion areas positive for eosinophils

TABLE 5: FINANCIAL IMPLICATIONS OF TREATING CATTLE WITH NITROXYNIL, SHOWING THE FINANCIAL SAVINGS (R c) POSSIBLE, COMPARED WITH THE FINANCIAL LOSS IN THE UNTREATED CONTROLS

Group	Mean dose of remedy per animal ml	Mean cost of treatment per animal R c	Mean live mass per animal at treatment kg	Mean cold dressed mass per carcass kg	Mean meat trimmed per carcass kg	Mean return ex sale of carcass R c	Mean loss due to downgrading R c	Mean loss due to trimming R c	Total financial loss per animal R c	Saving per animal less cost of treatment R c
Untreated controls	—	—	320,45	161,00	5,73	308,21	24,53	16,41	40,94	—
Nitroxynil 20 mg/kg	18,8	1,11	313,18	156,45	2,36	328,75	0	4,84	4,84	40,94 − 4,84 − 1,11 = 34,99

ACKNOWLEDGEMENTS

The authors are indebted to Dr G. Catton for identifying infested live animals and treating them, the Director of the Port Elizabeth abattoir and his staff for their cooperation in the slaughtering and examination of the carcasses, Mr L.A. Roeble for grading the animals, E.P. Livestock Agency for arranging the slaughter and sale of the animals, Drs P. van Schalkwyk and V. Rezin and Mr L. Geyser of Smith Kline Animal Health for helping with the examination of the carcasses, and Dr I. Carmichael for his help with the microscopic examination of the impression smears of the lesions.

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THE PATHOLOGY OF INFECTIOUS POLYARTHRITIS IN SLAUGHTER PIGS

G.V.S.TURNER*

ABSTRACT: Turner G.V.S. **The pathology of infectious polyarthritis in slaughter pigs.** *Journal of the South African Veterinary Association* (1982) **53** No. 2, 95-98 Department of Veterinary Public Health, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843, USA.

A study was conducted into the macroscopic and microcopic pathology of arthritic joints obtained from 37 freshly slaughtered pigs condemned for infectious polyarthritis. All the affected joints showed varying degrees of subacute to chronic serofibrinous to fibrino-purulent arthritis with or without pathology of the articular cartilage.

INTRODUCTION

Infectious polyarthritis is one of the most common causes of total and partial condemnation of pig carcasses. Because only animals which are normal and healthy are required to be sent for slaughter, the acute and subacute forms of porcine arthritis are seldom seen at the abattoir. Chronic arthitis is the most common form observed at the abattoir[9].

This paper deals with a study of the macroscopic and microscopic pathology of arthritic lesions in slaughter pigs with special reference to the degree of chronicity, severity and extent of the arthritis.

MATERIALS AND METHODS

Fore- and hindlegs were obtained from 37 freshly slaughtered pig carcasses which had been condemned for infectious polyarthritis. Arthritogenic agents were isolated from all the joints included in this study.

Affected joints were opened by severing the surrounding musculature and supporting ligaments. The gross pathology of the arthritic joint was then studied, taking the following into consideration: the amount and nature of synovial fluid; the degree of joint capsule involvement; the nature of the articular cartilage and the involvement of the peri-articular tissue. The pathological-anatomical diagnosis of each individual joint was recorded on a standard recording sheet.

Suitable samples for histopathological examination were then removed from the affected synovial membrane and placed in 10 % formalin for fixation and preservation. Histopathological specimens were processed in an automatic tissue processor and cut with a microtome at a thickness of 3 μm. All the tissue sections were routinely stained with haemotoxylin and counterstained with eosin. Microscopic examination of the tissue sections was made and the histopathological findings were recorded.

In order to observe and demonstrate macroscopically the actual bone involvement of grossly affected joints, the relevant bones were processed in a pressure cooker to remove the soft tissues and then air-dried. The bones were then immersed in trichlorethylene for 24 hours in order to defat and bleach then (Fig. 4).

RESULTS

Irrespective of the causative organism, the pathology of the arthritic joints was rather similar and in some cases the pathological changes in the joints of different pigs were almost identical. The gross pathology of the majority of the joints may be described as follows: an increase in the amount and viscosity of synovial fluid which was yellowish-brown in colour; hyperaemia and a marked fibrous thickening of the synovial membranes; the synovial villi showed varying degrees of proliferation, hypertrophy and hyperaemia (Fig. 1). All the pigs had polyarthritis, the stifle joint being most commonly affected.

The histopathology of the majority of these synovial membrane specimens was as follows: infiltration of lymphocytes, plasma cells, macrophages and, to a lesser degree, polymorphonuclear leucocytes; definite hyperplasia of the synovial lining cells; increase in the size of the villi; fibrinous exudate on the surface of the synovial membrane; increased vascularization; varying degrees of sub-synovial fibrosis (Figs. 5-10).

In spite of the similarity in the pathology of the affected joints, the severity of the arthritis varied. The degree of severity of the arthritis sometimes varied between the joints of the same pig. The variation in the gross pathology were as follows: the increase of synovial fluid varied from a negligible amount to approximately 15 ml in one joint; the colour of the synovial fluid varied from yellow-brown to reddish brown to red; the hyperaemia of the synovial membranes varied from slight to marked with varying degrees of thickening; the synovial villi showed slight to marked hypertrophy and on some occasions these enlarged villi had become detached from the joint capsule and were observed as free-floating "joint-mice" in the synovial fluid present within the joint cavity; the articular cartilages were sometimes eroded and in some cases the lessions were extensive and very severe; in some of the more chronic cases of arthritis, varying degrees of osteophyte formation were observed, especially around the edges of the joint (Figs. 2-4). The variations in the histopathology of the arthritic synovial membranes were mainly observed as a difference in the predominant cell types. Some affected joints showed plasma cells as the main type of infiltrating cell while in other sections either lymphocytes or polymorphonuclear cells were the main types involved. In some cases the lymphocytes were only perivascular or only appeared in the form of clones in the subsynovial tissues. Polymorphonuclear cells were always present, even if only in small numbers. These were usually situated near the synovial lining and, when in abundance, were also seen intermingled in the fibrinous exudate on the surface of the synovial membrane.

In the majority of the cases the degree of severity of the arthritis, as recorded from the macroscopic examination, were in good agreement with the histopathological changes. In a limited number of cases the histopathological examination showed a marked increase in polymorphonuclear cells indicating a purulent

*Department of Veterinary Public Health, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843, USA.

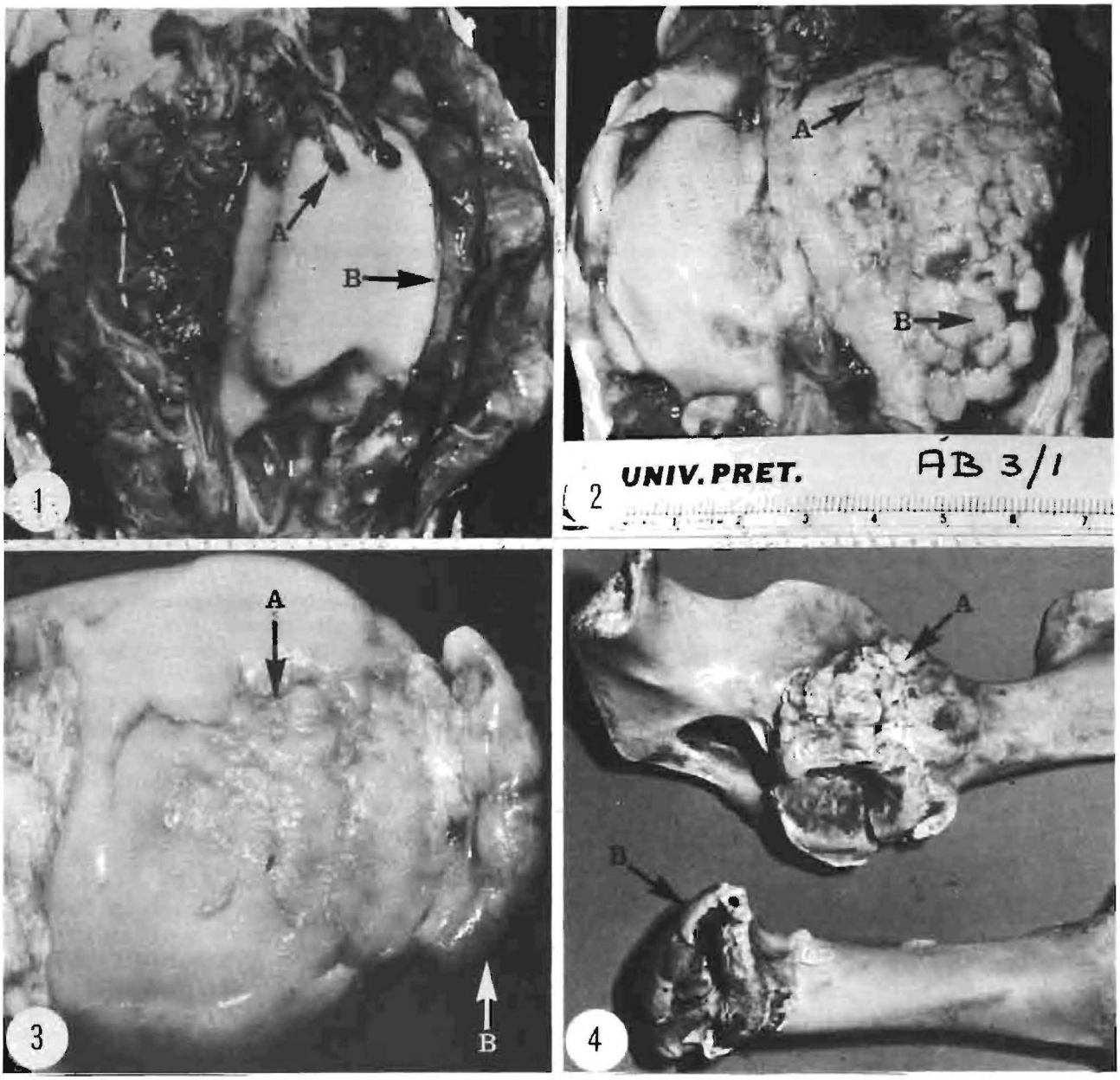

Fig. 1 Hypertrophy of the synovial villi (A) and thickening of the joint capsule (B).

Figs. 2 & 3 Severe chronic arthritis with erosions (A) and osteophytes of the articular cartilage (B).

Fig. 4 Bony structures of arthritic hip joint after removal of soft tissues. Osteophyte formation (A) and severe eroding of fermoral head (B).

arthritis whereas the exudate had appeared to be fibrinous.

DISCUSSION

In many countries *Erysipelothrix rhusiopathiae* is regarded as the major aetiological agent responsible for chronic polyarthritis in slaughter pigs.[9]. The pathogenesis of arthritis due to *E. rhusiopathiae* has been of interest because the joint lesions resemble closely those of rheumatoid arthritis of man[7 10]. There are differences in opinion as to the pathogenesis of chronic arthritis in swine erysipelas. The question arises as to whether the chronicity is due to the persistance of viable organisms or due to non-viable antigenic components of *E. rhusiopathiae.*

Some workers believe that the chronic lesion is caused by viable organisms persisting in the joints[10]. The possibility that immunological mechanisms might exacerbate and perpetuate the chronic lesions of *E. rhusiopathiae* arthritis should not be excluded[5]. Some workers consider that hypersensitivity, developed in the host by previous contact with the organism, is of primary significance in the persistance and further development of the arthritic syndrome[2 3 7-10]. Plasma cells in inflamed synovial membrans have been shown to contain antibody to *E. rhusiopathiae* antigen by means of immunofluorescence. It is thought that this could be a source of periodic or continuing allergic response[8 10]. An interesting feature noted in this study was the frequent presence of plasma cells, many of which contained Russel bodies in the cytoplasm (Fig. 10).

All the affected joints showed varying degrees of subacute to chronic serofibrinous to fibrino-purulent proliferative arthritis with or without pathology of the articular cartilage. The general pattern and variation in pathology of the arthritic joints examined in this study conforms with the findings of many other workers in this field[1 2 4-7]

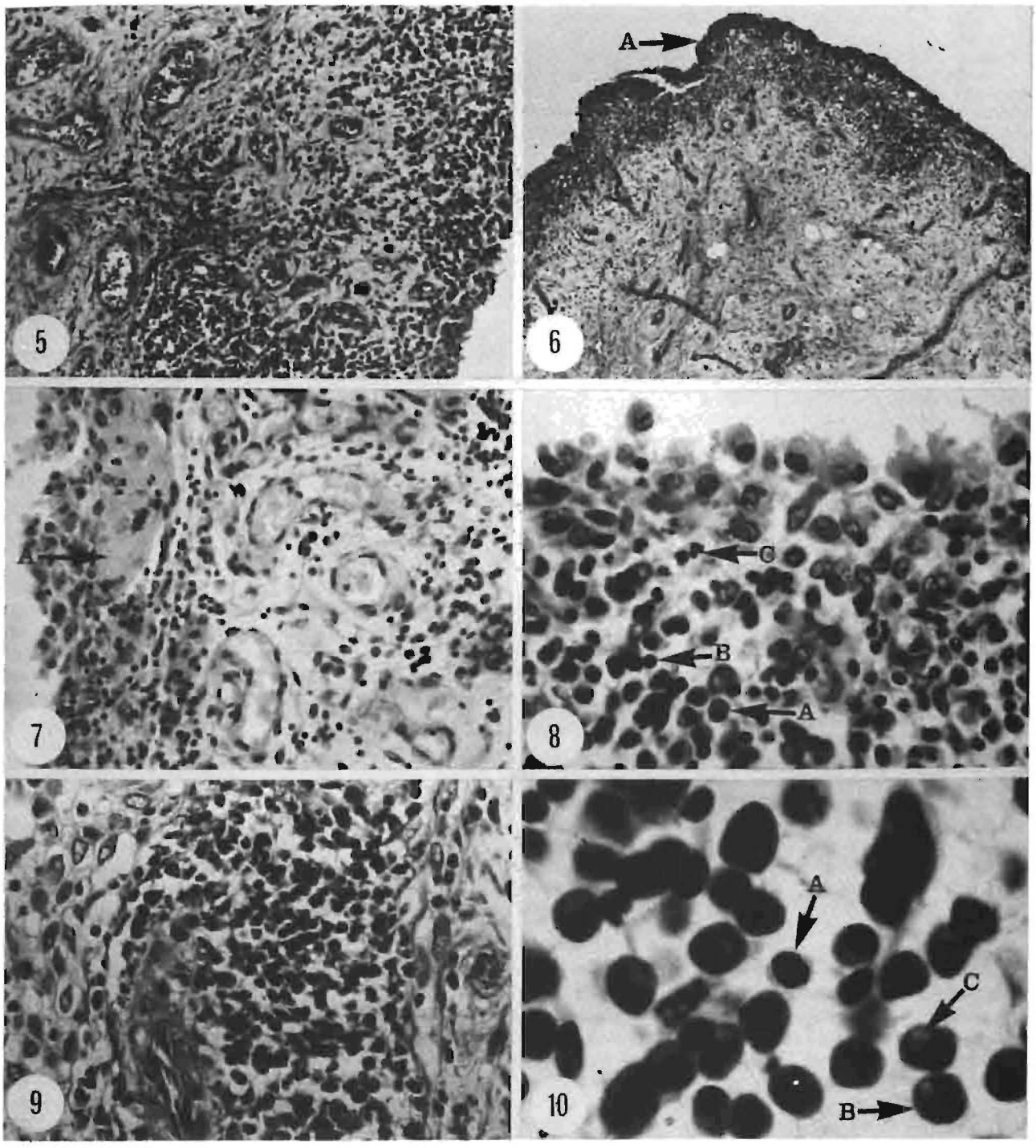

Fig. 5 Arthritic joint capsule showing cellular infiltration and vascularization. H.E. x 200.

Fig. 6 Tip of hypertropic villus with fibrin on surface (A), sub-synovial cellular infiltration and vascularization. H.E. x 75.

Fig. 7 Arthritic joint capsule with fibrin (A) in disrupted synovial epithelium, sub-synovial cellular infiltration and vascularization. H.E. x 300.

Fig. 8 Hypertrophy and hyperplasia of synovial epithelium with subsynovial infiltration of plasma cells (A), lymphocytes (B), and neutrophils (C). H.E. x 600.

Fig. 9 Focal lymphocytic and plasma cell infiltration in arthritic joint capsule. H.E. x 500.

Fig. 10 Lymphocytic (A) and plasma cell (B) infiltration. Some plasma cells have small Russel bodies in the cytoplasm (C). H.E. x 1000.

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ABSTRACT: Malan, F.S., Reinecke, R.K. & Scialdo, Rosina C., 1981; **The recovery of helminths postmortem from equines. II Helminths and larvae of *Gasterophilus* from the gastro-intestinal tract and oestrids from the sinuses.** *Onderstepoort Journal of Veterinary Research*, 48, 145-147 (1981).

The tongue, pharynx, oesophagus and gums are examined for larvae of *Gasterophilus* spp., and the nose and sinuses for oestrid larvae. The gastro-intestinal tract is divided into separate specimens – stomach, small intestine, caecum, ventral colon, dorsal colon, descending colon and rectum – and each is examined separately. Aliquots of ingesta of ¼ by mass of the stomach and 1/10 by mass of the small intestine, caecum, ventral colon and dorsal colon are collected for microscopic examination. Each part of the wall of the caecum, ventral and dorsal colon is washed and specimens are collected for subsequent examination. The gut wall of the caecum and colon is examined macroscopically for larval stages. Ingesta in the descending colon are examined macroscopically for *Gasterophilus* larvae.

ABSTRACT: Malan, F.S., Reinecke, R.K. & Scialdo, Rosina C., 1981. **Recovery of helminths postmortem from equines. I. Parasites in arteries, subperitoneum, liver and lungs.** *Onderstepoort Journal of Veterinary Research*, 48, 141-143 (1981).

The entire gastro-intestinal tract and viscera of the abdomen and thorax, including the heart, aorta and its branches to the viscera, are removed from the carcass. All the branches of the aorta, with the exception of the *A. gastrica sinistra*, are dissected from the intestinal tract, and subsequently each branch is isolated from the mesentery, fat, pancreas, kidneys, etc. Usually, the *A. ileocolica* is grossly enlarged due to chronic arteritis with thrombus formation caused by 4th stage larvae, 4th moult and 5th stage *Strongylus vulgaris*. Descriptions of methods to examine the subperitoneal tissues, liver and lungs are included.

ABSTRACT: Thomas, Shan E. & Mason, T.E., 1981. **Isolation and trasmission of an unidentified *Babesia* sp. infective for cattle.** *Onderstepoort Journal of Veterinary Research*, 48, 155-158 (1981).

Engorged adult female ticks submitted from farms in South Africa were routinely screened for protozoan parasites by examination of haemolymph smears. An unidentified *Babesia sp.* was found in *Hyalomma marginatum rufipes* and its transmission to susceptible cattle was achieved both biologically (tick feeding) and mechanically (injection of infected blood). Attempts to transmit this species to susceptible rabbits and a horse using similar methods did not produce evidence of infection.

This *Babesia sp.* was of low pathogenicity, even in splenectomized cattle. Morphologically, intra-erythrocytic piroplasms and merozoites in tick haemolymph resembled other bovine *Barbesia* spp. in many respects. Although it could be classified as a large *Babesia,* it was intermediate in size between the other species.

ABSTRACT: Prozesky, L., Joubert, J.P.J. & Ekron, M.D., 1981. **Paralysis in lambs caused by overdosing with parbendazole.** *Onderstepoort Journal of Veterinary Research*, 48, 159-167 (1981).

An experiment was undertaken to determine whether an overdose of the anthelmintic parbendazole could cause paralysis in lambs when given to ewes during the early stages of pregnancy. Out of a total of 68 lambs, born from ewes treated at various stages of gestation with parbendazole at 180 mg/kg, 5 showed the paralysis syndrome, 5 showed skeletal deformities, 2 were ataxic, while 1 foetus was aborted. The ewes giving birth to paralysed lambs had been treated with parbendazole at 30, 32, 37 and 53 days of gestation. Cerebral hypoplasia was observed in 2 of these lambs, while 2 others showed internal hydrocephaly. Histopathological lesions observed in the lambs with cerebral hypoplasia included gliosis and areas of encephalomalacia in the cerebral white matter. Lesions present in the spinal cords of the 2 ataxic lambs included hydromyelia, syringomyelia, duplication of the spinal canal and an abnormal position of the canal. This is the first report describing brain lesions in lambs as a result of an overdose of parbendazole.

ABSTRACT: De Vos, A.J. & Roos, J.A., 1981. **The isolation of *Theileria? taurotragi* in South Africa.** *Onderstepoort Journal of Veterinary Research*, 48, 149-153 (1981).

In 3 out of 4 attempts strains of a *Theileria* sp. of low virulence were isolated in the laboratory by feeding adult *Rhipicephalus appendiculatus* collected from the field on susceptible cattle. One of the strains, previously identified as *Theileria? taurotragi* (Tzaneen), was found to be serologically crossreactive with the other 2 strains. It was concluded that *T.? taurotragi* is prevalent in South Africa in those parts where the vector exists.

Infection was characterized by a transient fever and small numbers of macroschizonts and piroplasms. Subinoculation of the infection with small volumes of blood proved to be difficult.

ABSTRACT: Du Plessis, J.L., 1981. **The influence of dithiosemicarbazone on the immunity of sheep to heartwater.** *Onderstepoort Journal of Veterinary Research*, 48, 175-176 (1981).

Four out of 9 sheep, immune to heartwater and subsequently treated with gloxazone, showed a febrile reaction when they were challenged 6 months later, but the same number of untreated controls also developed this reaction. In a second group of treated immune animals challenged after 9 months, 8 out of 8 showed a febrile reaction, whereas only 3 out of 8 controls reacted. Furthermore, the blood of one of the 4 sheep that reacted to challenge at 6 months was infective to a susceptible sheep inoculated with it, whereas that of 4 out of the 8 challenged at 9 months was infective.

A MICROBIOLOGICAL STUDY OF POLYARTHRITIS IN SLAUGHTER PIGS

G.V.S.TURNER*

ABSTRACT: Turner, G.V.S. **A Microbiological study of polyarthritis in slaughter pigs.** *Journal of South African Veterinary Association* (1982) **53** No. 2, 99-101 (En) Department of Veterinary Public Health, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843, USA.

Arthritic joints obtained from 50 freshly slaughtered pig carcasses condemned for polyarthritis were studied microbiologically. A routine technique was developed for aseptically opening joints to obtain material for microbiological examination. A standard series of culture media for the primary isolation of arthritogenic agents were used in the examination of each affected joint. The microbiological study catered for isolation of the following microorganisms: *Erysipelothrix rhusiopathiae, Corynebacterium pyogenes, Staphylococcus aureus, Escherichia coli, Streptococcus* spp., *Haemophilus* spp., *Mycoplasma* spp., *Salmonella* spp., *Chlamydia,* and viruses.*E. rhusiopathiae* was found to be responsible for 48 % of the cases of arthritis and *Streptococcus* spp., *C. pyogenes* and *S. aureus* for 20 %, 4 %, and 2 % respectively; no microorganisms were cultured from 26 % of the arthritic joints.

INTRODUCTION

Polyarthritis in slaughter pigs is a universal problem. In other countries the occurrence, importance, and aetiology of porcine arthritis is well documented[17]. It is generally accepted that the following microorganisms are the main arthritogenic agents in swine: *Erysipelothrix rhusiopathiae, Corynebacterium pyogenes, Staphylococcus aureus, Haemophilus* spp., *Escherichia coli, Steptococcus* spp., Salmonella spp., and *Mycoplasma* spp.[16]. In many countries *E. rhusiopathiae* is regarded as the major aetiological agent responsible for polyarthritis in slaughter pigs and *Streptococcus* spp. are regarded as the second most common cause[17].

With swine erysipelas being a notifiable disease and a zoonosis and *E. rhusiopathiae* being the main arthritogenic agent encountered in slaughter pigs, it is difficult to understand why the high incidence of arthritis in pig carcasses and the economic implications thereof did not prompt an earlier investigation into the actual aetiology of the problem in South Africa. It therefore became apparent that it was necessary to ascertain the aetiology of arthritis in slaughter pigs encountered at the abattoirs. From the microbiological standpoint it was deemed necessary to develop an efficient and practical method for aseptically opening joints and obtaining microbiological samples for further testing.

MATERIALS AND METHODS

Fore- and hindlegs with unopened intact joints were obtained from freshly slaughtered pig carcasses which had been condemned for polyarthritis. In order to achieve satisfactory results, the fresh material was processed in the laboratory as soon after slaughter as possible. The microbiological examination procedure catered for isolation of the following microorganisms: *E. rhusiopathiae, Streptococcus* spp., *C. pyogenes, S. aureus E. coli, Haemophilus* app., *Mycoplasma* spp., *Salmonella* spp., *Chlamydia*, and viruses. No literature reference to *Chlamydia* or viruses as arthritogenic agents in swine was found. Nevertheless, because of the ubiquitous nature of the *Chlamydia* organisms and viruses, an isolation procedure for these types of microorganisms was included.

A standard series of culture media for primary isolation was used in the examination of each affected joint. The same procedure was carried out step-by-step on each aseptically opened joint. The following culture media were used: blood tryptose agar (BTA) for the isolation of *E. rhusiopathiae, C. pyogenes,* Streptococcus spp., *S. aureus, E. coli,* and *Salmonella* spp.; serum broth for the primary isolation of *E. rhusiopathiae, C. pyogenes, Streptococcus* spp., *S. aureus, E. coli,* and *Salmonella* spp.; chocolate agar for the isolation of strains of *Haemophilus* spp. requiring Factor X for growth; *Staphylococcus aureus* "feeder" strain, known to produce the V factor to ensure growth of *Haemophilus* spp. requiring the V factor; Hayflick's broth, a specific selective medium for the primary isolation of *Mycoplasma* organisms of porcine origin; Chalquest agar, a selective medium used for the primary isolation of *Mycoplasma* organisms, and for subcultures from Hayflick's broth; 7 to 8 day embryonated hen's eggs were used for the recovery of *Chlamydia* organisms from affected joint material[2 3 10]. Stice (PK15) porcine kidney cells were used to screen synovial membrane samples from affected joints for the presence of viruses.

Apparently normal joints were examined in order to adopt a standard routine method of opening the joints, to establish a practical aseptic technique, and to act as controls for the various isolation techniques performed. Ten joints, 2 from each of 5 control pigs, were opened and examined as described below.

In each case the porcine limb or part thereof was fixed in a carpenter's vice. The skin over the affected joint was heat sterilized by thoroughly scorching the area with an iron spatula which had been heated over a Bunsen burner. Using sterile rat-tooth forceps and scalpel, a section of the scorched skin overlying the joint to be opened was removed. The underlying tissues were removed aseptically, and the distended joint capsule exposed. A small incision was made through the distended joint capsule and an aseptic sample of the synovial fluid was taken with a sterile bacteriological swab. Three swabs of synovial fluid were routinely taken per affected joint. The 3 bacteriological swabs were routinely processed as follows: (i) one swab was placed in serum broth. (ii) The second swab was placed in Hayflick's broth. (iii) The third swab was streaked over specific media in the following order: two BTA plates, chocolate agar, and finally, a Chalquest agar plate. The one BTA plate was inoculated by means of a few closely placed parallel strokes of the swab. Using a sterile platinum loop the *Staphylococcus aureus* "feeder" was then streaked at right angles to this[6].

*Department of Veterinary Public Health, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843 USA.

The joint capsule was further incised and a small piece of capsule was aseptically transferred to serum broth and another placed in Hayflick's broth. A slightly large piece was then removed and firmly streaked sequentially across BTA, chocolate agar, and Chalquest agar. The plating on the Chalquest agar was always performed last because the medium contains pencillin, and it was undesirable for the antibiotic to contaminate antibiotic-free BTA and chocolate agar. For *Chlamydia* and virus detection a piece of joint capsule was ground finely with a sterile pestle and mortar. The ground material was inoculated into the yolksac of 7-8 day embryonated eggs for the isolation of *Chlamydia*, and roller tube cultures of Stice (PK 15) porcine kidney cells were infected as described by Turner[17].

The inoculated solid media were placed in a sealed candle jar in order to create an atmosphere of ± 10 % carbon dioxide. All the media were incubated at 37 °C. Indentification of any growth on BTA was based on the morphological and haemolytic characteristics of the colonies, the production of catalase, the microscopic appearance of a Gram stained smear, and specific biochemical tests based on those recommended in Bergey's Manual of Determinative Bacteriology[1]. The serum broth was examined for signs of growth 24 hours after inoculation. If there was no growth, the serum broth was incubated for an additional 24 hours before subculturing onto BTA. The BTA plate was then examined as described above.

The Chalquest agar was examined under a dissecting microscope for typical *Mycoplasma* colonies. On the second day after inoculating the Hayflick's broth, a subculture was made from the broth onto Chalquest agar. The agar was then examined 7 days later as described above. The Hayflick's broth was discarded at this stage. Where no growth was noted on the Chalquest agar after 7 days the medium was incubated for an additional 7 days.

RESULTS

None of the ten apparently normal joints used as controls yielded any microbiological growth. The results of the microbiological investigation are summarized in Table 1. *E. rhusiopathiae* was found to be responsible for 48 % of the cases of arthritis and *Streptoccus* spp., *C. pyogenes,* and *S. aureus* for 20 %, 4 % and 2 % respectively; no microorganisms could be cultured from 26 % of the arthritic joints. The microorganisms were isolated from both the synovial fluid and synovial membrane samples which had been inoculated onto BTA and into serum broth.

No isolation of *Haemophilus* spp., *Mycoplasma* spp., and *Chlamydia* organisms was made. No evidence of cytopathic changes was noted in the Stice porcine kidney cell cultures and the material was therefore considered free of virus.

DISCUSSION

Certain *Mycoplasma* spp. are known arthritogenic agents in swine and can bring about joint lesions which are difficult to differentiate from those caused by other arthritogenic agents [9 14]. For this reason it was considered necessary to screen the affected joints in this survey for the possible presence of *Mycoplasma* organisms as the causative agent. The complex growth requirements of *Mycoplasma* organisms necessitated the use both liquid and solid media. Hayflick's broth is a suitable enrichment medium for most *Mycoplama* organisms. Chalquest agar is also a suitable medium, especially for the subculture from PPLO broth and facilitates the microscopic detection of the characteristic small colonies[10].

The fact that no *Mycoplasma* were isolated from these media makes it unlikely that *Mycoplasma* were present in the arthritic lesions. *Haemophilus* spp. are known arthritogenic agents which can cause lesions very similar to that caused by other arthritogenic agents[9]. For this reason 2 selective media were utilized for the isolation of *Haemophilus* spp. The fact that no *Haemophilus* spp. could be isolated from the joint material is therefore considered of some signifcance. *Chlamydia* organisms are known to cause polyarthritis in lambs and calves and have been isolated from affected joints in these animals[9 14]. To establish whether *Chlamydia* were causing the arthritis in this study, cultural methods capable of supporting growth of these organisms were included. The negative findings are of significance.

To facilitate the isolation of organisms it is generally recommended that both synovial fluid and a portion of the affected synovial membrane be used as inocula[18]. Because of the scarcity of organisms in some of the more chronic forms of arthritis, it is also recommended that an enrichment medium such as serum be used for primary isolation[15].

It is interesting to note that in this study there was no significant difference between the results obtained from the use of either serum broth or BTA for the primary isolation of *E. rhusiopathiae, Steptococcus* spp., *C. pyogenes,* and *S. aureus.* Similary, no significant difference resulted from the use of either synovial fluid or synovial membrane as inoculum.

Various techniques have been employed for the isolation of *E. rhusiopathiae.* Some workers advocate the use of enrichment media to enhance the possiblity of isolating *E rhusiopathiae* from affected material[15 18]. The use of selective media containing substances such as sodium azide and crystal violet are widely advocated, especially in order to eliminate much of the difficulty in culturing *E. rhusiopathiae* from contaminanted material[11 18]. The fluorescent antibody technique is considered less accurate that cultural methods for the detection of *E. rhusiopathiae* and is not regarded as a satisfactory test for routine use in the diagnostic laboratory[8]. In spite of the fact that in some cases the number of colonies on primary plates may be rather low, the use of blood agar is regarded as a useful medium for the primary isolation of *E. rhusiopathiae* organisms[11]. Because of the reliability of the aseptic technique adopted in this study, it was thought unnecessary to use an additional selective medium merely for the specific isolation of *E. rhusiopathiae.* The use of BTA and serum broth was adequate for the isolation of *E. rhusiopathiae,* as well as catering for organisms such as *C. pyogenes, Streptococcus* spp., *S. aureus,* and *E. coli.*

In this study *E. rhusiopathiae* was found to be the most common isolate. Some workers have, however, also observed that a substantial number of joints showing arthritis failed to yield organisms on culture[5 12 13]. Connell et al. found that a significant percentage of pathological joints did not yield *E. rhusiopathiae* even though the arthristis was initiated by this organism[4].

There are various theories on why the causative organisms cannot be isolated from a numbe of cases of chronic porcine arthritis. It has been postulated that although primarily due to *E. rhusiopathiae,* the arthristis is perpetuated by immunological phenomena[7]. The failure to isolate organisms has also been attributed to the lack of sensitivity of the bacteriological techniques employed[15 18]. With chronicity, bacteria are commonly more difficult to recover from arthritic joints and there may be an element of chance attached to the presence or absence of viable organisms at the site where the bacterial specimen is taken[15]. Based on the above criteria it may be assumed that *E. rhusiopathiae* was responsible for at least some of the arthritic processes in the 26 % culturally negative joints ecountered in this survey.

Based on the fact that the joints of all 10 control pigs were found to be sterile and the absence of obvious contaminants during the entire course of the study, the routine technique used for aseptically opening joints must be regarded as being efficient and practical. For primary isolation of the arhritogenic agents listed, the standard series of culture media employed proved to be the most practical for this study.

Table 1: RESULTS OF MICROBIOLOGICAL EXAMINATION OF ARTHRITIC PORCINE JOINTS

Isolations	Number Carcasses Affected	%
Erysipelothrix rhusiopathiae	24	48
Streptococcus spp.	10	20
Corynebacterium pyogenes	2	4
Staphylococcus aureus	1	2
Negative	13	26
Total Number of Carcases Examined	50	100

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ABSTRACT: Boomker, J., Horak, I.G. & de Vos, V., 1981. **Paracooperiodes peleae gen. et sp. n. (Nematoda: Trichostrongylidae) from the vaal ribbok, *Pelea capreolus*** (Foster, 1790). *Onderstepoort Journal of Veterinary Research*, 48, 169-174 (1981).

A new genus and species of trichostrongylid nematode, *Paracooperioides peleae,* was collected from the small intestines of vaal ribbok, *Pelea capreolus* (Forster, 1790), from the Bontebok National Park, Swellendam, Cape Province.

These nematodes are small and slender with a small cephalic inflation. The cuticle bears numerous transverse striations which are more pronouced anteriorly. The dorsal ray is long and is similar to that of *Gazellostrongylus* Yeh, 1956, and *Cooperioides hepaticae* Ortlepp, 1938, but differs in that it bifurcates in its distal quarter. Each branch divides again, giving rise to a thinner, outer branch and a thicker inner branch. The latter recurves upon itself to form a small, elongated knob. The spicules of *Paracooperioides peleae* resemble those of *C. hepaticae* but can be differentiated from them in that they bear small lateral barbs on their tips. Ten longitudinal ridges, supported by sclerotized rods, are present at the middle of the body. In transverse section, *Paracooperioides peleae* is intermediate between *Cooperioides* Daubney, 1933 and *Paracooperia* Travassos, 1935.

ABSTRACT: De Vos, A.J., Bessenger, R. & Banting, L.F., 1981. ***Theileria? taurotragi:* a probable agent of bovine cerebral theileriosis.** *Onderstepoort Journal of Veterinary Research*, 48, 177-178 (1981).

A case of bovine cerebral theileriosis was confirmed at autopsy on a farm where 4 animals out of 70 died. All were less than 2 years old and all showed nervous signs.

Serologically, no evidence was found of *Theileria mutans* or the *Theileria parva* group in young animals born on the farm. Six out of 13 calves 6-9 months of age were, however, serologically positive for *Theileria? taurotragi* and it was concluded this species was the probable cause of death of the 4 animals.

THE USE OF DETERGENTS AND SANITIZERS IN DAIRY FARM SANITATION – AN UPDATED PERSPECTIVE

P.H. GILBERT

ABSTRACT: Gilbert P.H. **The use of detergents and sanitizers in dairy farm sanitation – an updated perspective.** *Journal of the South African Veterinary Association* (1982) **53** No. 2, 103-106 (En) Technical Services Department, Klensan (Pty) Ltd., P.O. Box 55, 1624 Chloorkop, Republic of South Africa.

Raw milk quality in South Africa is poor and standard plate counts in the millions per ml are common. This is largely due to inefficient cleaning and sanitizing of dairy equipment. The basic constituents in milk are described and various soils are classified as soluble in water, alkali, acid, solvent or surfactant or as insoluble. The importance of water quality is highlighted and the influence of mineral salts on soil deposition described. Dairy detergents are broadly classified as alkaline or acid, the former being most effective against fatty and proteinaceous soils and the latter effective against mineral salts. Typical detergent ingredients and their properties are described. Chlorine is incorporated into alkaline detergents not as a sanitizing agent, but as a peptizing agent to aid in protein soil removal. At high pH values the antimicrobial activity of chlorine is greatly diminished. The use of a daily acidified rinse (pH 3,0-5,0) is preferred to the periodic acid wash, since the acid rinse prevents mineral deposition rather than removing accumulated milkstone.

All cleaning programmes follow the same fundamental steps – Pre-rinse (40-50 °C), wash (60-70 °C), rinse (pH 3,0-5,0) and sanitize (25 ppm iodine and 100 ppm chlorine). Farms following such a programme are able to achieve Standard Plate Counts of < 10 000/ml and coliform counts of < 10/ml for raw milk.

INTRODUCTION

It is accepted without question by food technologists, veterinarians, and public health officials, that thorough cleaning and sanitizing of milking machines and ancillary equipment, is an essential pre-requisite for the production of top grade raw milk. The microbiological quality of raw milk in South Africa is extremely poor and tests in the author's laboratories have revealed that standard plate counts in the millions per ml are common. This is largely due to the inefficient cleaning and sanitizing of dairy equipment. The situation is further aggravated by the fact that many of the personnel supplying technical advice to the dairy farmer (veterinarians, public health officials, extension officers) are themselves poorly informed about the newer developments and concepts in dairy farm sanitation. This paper reviews current concepts, practices and procedures.

CLEANING

Cleaning is defined as the removal of soil. The chemical agent or detergent makes such removal easier, thereby reducing labour. Time, temperature, detergent concentration, water quality and mechanical action are among the factors which affect the cleaning efficiency of detergents.

TYPE OF SOILS

When the removal of soils is the fundamental purpose of cleaning, an understanding of the types of soils that may be encountered is essential in developing a successful cleaning programme. Soil is best identified by characteristics that provide information on how it may be dissolved or suspended because this is the object of cleaning. Soils may be broadly classified as follows:

A. SOLUBLE:

(1) in water (e.g. salt, sugar)
(2) in alkali (e.g. fats, protein)
(3) in acid (e.g. mineral scales)
(4) in surfactant solution (e.g. oil, grease)
(5) in organic solvents (eg. oil, grease)

B. INSOLUBLE (e.g. carbonized soils)

CONSTITUENTS OF MILK

The chemical components of milk may be classified in the following groups:

(1) *Water* – in which all other components are in solution, emulsion or suspension;
(2) *Lipids* – the constituents of milk which are extractable with ether, these consist principally of a suspension of the so-called "milk-fat": globules;
(3) *Proteins* – of which there are 2 major groups, the caseins and the whey proteins, which exist in the colloidal state;
(4) *Lactose* – which is the true solution;
(5) *Salts* – which consist mainly of the inorganic components which remain when the milk is "ashed";
(6) *Miscellaneous* – which includes the vitamins, pigments, enzymes, gases and other components present in small quantities.

TABLE 1: GROSS COMPOSITION OF MILK*

Breed	Composition				
	Water	Fat	Protein	Lactose	Ash
	(%)	(%)	(%)	(%)	(%)
Holstein	88,12	3,44	3,11	4,61	0,71
Ayrshire	87,39	3,93	3,47	4,48	0,73
Brown Swiss	87,31	3,97	3,37	4,63	0,72
Guernsey	86,36	4,5	3,6	4,79	0,75
Jersey	85,66	5,15	3,7	4,75	0,74
Range (All breeds)	84,5-98,6	3,5-5,9	2,9-3,8	4,4-5,0	0,67-0,77
Average	87,44	3,85	3,3	4,7	0,72

*Based on values for herd milk (Armstrong, T.V., Journal of Dairy Science, 42:1. 1959)

WATER QUALITY

Water, the primary solvent in detergency, is never "pure" since it contains dissolved and suspended matter. The falling rain dissolves carbon dioxide from the air, making a dilute solution of carbonic acid. As this dilute acid percolates through soil, it dissolves various alkaline metals, primarily calcium and magnesium, to lend "hardness" to the water. The U.S. Geological Survey classifies water hardness as follows:

TABLE 2: CLASSIFICATION OF WATER HARDNESS

Hardness	Parts per Million (ppm)
Soft	0- 60
Moderately Hard	60-120
Hard	120-180
Very Hard	Over 180

Many of the minerals present in water may be precipitated by factors such as heat, alkalinity and even poorly formulated or incorrectly used detergent compounds. The prevention of mineral deposition on equipment is essential for effective cleaning. When minerals precipitate on a surface, they entrap organic milk constituents within the crystalline film and as this process is repeated over a period of time a milkstone deposit eventually becomes apparent. Such soils typically harbour substantial bacterial populations and contribute high levels of contamination to the milk. Water quality varies from area to area and it is imperative that an appropriate water analysis (pH, hardness) be conducted to accurately assess the specific needs and problems of the farmer in question.

TYPES OF DETERGENTS

Most detergents utilized on dairy farms can be classified as either (1) alkaline detergents or (2) acid detergents.

Alkaline Detergents

These are so named because they are principally composed of alkaline compounds. These are the oldest and most familiar of the dairy cleaners. Their primary function is to provide the chemical action required for the removal of organic soils (fats, proteins) from equipment surfaces. Alkaline cleaners are not designed or formulated for removing mineral films.

The principal ingredient materials used in formulating alkaline detergents are basic alkalies, phosphates, wetting agents and chelating agents. A well balanced cleaner will combine these available materials in proper proportion according to the nature and quantity of organic soil to be removed.

Each of the ingredients performs one or more specific functions in the cleaning operation. The basic alkalies (sodium hydroxide, sodium carbonate, trisodium phosphate and the alkaline silicates) are used for their ability to provide alkalinity and serve to solubilize fats and pro-

TABLE 3: RELATIVE CLEANING VALUES OF VARIOUS DETERGENT INGREDIENTS*

KEY TO CHART
A HIGH VALUE
B MEDIUM VALUE
C LOW VALUE
D NEGATIVE VALUE
* VIA PRECIPITATION
• VIA SEQUESTRATION
✡ ALSO STABLE TO HEAT

	INGREDIENTS	COMPARATIVE ABILITY											
		EMULSIFICATION	SAPONIFICATION	WETTING	DISPERSION	SUSPENSION	PEPTIZING	WATER SOFTENING	MINERAL DEPOSIT CONTROL	RINSABILITY	SUDS FORMATION	NON-CORROSIVE	NON-IRRITATING
BASIC ALKALIS	CAUSTIC SODA	C	A	C	C	C	C	C	D	D	C	D	D
	SODIUM METASILICATE	B	B	C	B	C	C	C	C	B	C	B	D
	SODA ASH	C	B	C	C	C	C	C	D	C	C	C	D
	TRI-SODIUM PHOSPHATE	B	B	C	B	B	B	A*	D	B	C	C+	C−
COMPLEX PHOSPHATES	SODIUM TETRA-PHOSPHATE	A	C	C	A	A	A	B•	B	A	C	AA	A
	SODIUM TETRA-PHOSPHATE	A	C	C	A	A	A	A•	B	A	C	AA	B
	SODIUM HEXAVETAPHOSPHATE	A	C	C	A	A	A	B•	B	A	C	AA	A
	TETRASODIUM PYROPHOSPHATE	B	B	C	B	B	B	A•	B	A	C	AA	B
ORGANIC COMPOUNDS	CHELATING AGENTS	C	C	C	C	C	A	AA✡•	A	A	C	AA	A
	WETTING AGENTS	AA	C	AA	A	B	B	C	C	AA	AAA	A	A
	ORGANIC ACIDS	C	C	C	C	C	B	A✡	AA	B	C	A	A
	MINERAL ACIDS	C	C	C	C	C	C	A✡	AA	C	C	D	D

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teins. In addition the alkaline silicates are useful as corrosion inhibitors to protect soft or easily corrodible metals. The phosphates provide a number of useful properties to alkaline cleaners. While there are variations among the different types, in general they are all good water softeners, free rinsers, peptizing agents, emulsifiers, dispersants, deflocculants and soil suspending agents. The relative cleaning values of various detergent ingredients are outlined in Table 3.

The wetting agents (surfactants) are surface active agents and are used in detergents to provide wetting action and penetration of cleaning solutions. Also these materials are good emulsifiers of fats, act as dispersants and suspending agents and impart good rinsing properties to detergent solutions.

Acid Detergents

These are products that are composed of organic and inorganic acids individually or in various combinations. Common ingredients are nitric, phosphoric and sulphamic acid. Their principal function in a balanced cleaning programme is to prevent the accumulation of mineral deposits and, in some cases, to remove established deposits.

In terms of chemical action, the acids react with insoluble salts to convert these to water soluble salts for easy removal. At normal use concentration the acids have little effect on organic soils.

Chlorinated Cleaners

Chlorine is often included in alkaline detergents to assist in the removal of proteins from equipment surfaces. In this role, chlorine functions as a peptizing agent and reduces the molecular size of proteins to render them more soluble. It is a misnomer to refer to chlorinated cleaners as "detergent-sanitizers" or "detergent-disinfectants" since the chlorine in the detergent is not an effective sanitizer. Chlorine's primary purpose in the detergent is to break down the protein, and this requires alkaline conditions. On the alkaline side (above pH 7) chlorine is an effective detergent but forms little or no hypochlorous acid (HOCl) and consequently has limited bactericidal activity. Many chlorinated alkaline detergents have pH values in the range 10-12 at use-dilution. Under neutral or slightly acidic conditions (pH of 7 or below) chlorine is a highly efficient bactericide but displays little or no detergent ability.

It is also worth noting that chlorine is highly reactive material and combines rapidly with organic materials such as protein. It therefore makes little sense to "clean and disinfect in one operation" as is so frequently recommended by detergent suppliers. The chlorine is rapidly dissipated, the detergent solution has an elevated pH and the chances of efficient destruction of bacterial populations are consequently remote. The pursuit of the mythical "detergent-sterilizer" is undoubtedly one of the principal causes of South Africa's poor quality raw milk.

TABLE 4: SOIL DEPOSITS AND THEIR REMOVAL

Film/Deposit	Description of Identification	Cause	Removal	Prevention
Protein	Blue rainbow hue varnish-like "apple sauce"	1. Non-chlorinated cleaner 2. Inadequate pre-rinse 3. Improper (sporadic) cleaning	Initial clean-up equal parts chlorinated alkaline detergent, hot water	1. Chlorinated alkaline detergent 2. Proper cleaning with recommended use dilution after each use
Milk or Waterstone	White to yellow	1. Mineral from milk 2. Mineral from water	Initial cleaning Acid wash	Regular and proper cleaning procedures coupled with acidified rinse
Fat, grease	Water droplets hanging greasy appearance	1. Same as protein 2. Low temperature 3. Low detergent conc. 4. Regular use of acids in washing	Initial clean-up	Regular and proper cleaning procedure coupled with acidified rinse
Mineral (Calcium, magnesium)	White (waterstone) chalky to grey	1. Improper rinsing 2. Precipitation of minerals from water 3. No acidified rinse 4. Non-compatible alkaline detergent	Acid wash	1. Acid rinse 2. Product used must have good water conditioning properties 3. Water softener or treatment
Iron	Brown to red	1. Water supply	Acid wash	1. Regular effective acid rinse 2. Water treatment
Silica	White to grey	1. Poor rinsing 2. Water supply	Special acid wash	1. Complete post-rinse 2. Regular effective acid rinse. 3. Water treatment
1. Inking (Blacking)	1. Back rubber parts	1. Reaction between chlorinated compound and rubber	Acid wash	Acid rinse
2. Black	2. Black residue deposit	2. Rubber migration		
3. Wetting agent	Blue	Poor, inadequate rinsing	Initial clean-up	1. Recommended compound
4. Factory soil	Grease, factory dirt, black deposit, rusting	Lack of initial clean-up	Initial clean-up	2. Proper rinsing Thorough cleaning before initial equipment use

ACID WASHES AND RINSES

Standard instructions by many detergent suppliers are that dairy equipment should be given an acid wash (typically using 1% acid detergent) every 7 or 14 days to remove milkstone or scale build-up! This implies that the deposition of scale or milkstone is an unavoidable situation and also, presumably, that the attendant progressive deterioration in the micro-biological quality of the milk is unavoidable. This makes very little sense and the alternative daily acidified rinse, which is standard practice in the USA, should be employed.

This acidified rinse is an important part of each and every cleaning cycle. After the alkaline cleaning cycle has been completed an acid detergent is added to the rinse water to produce a pH of 3,0-5,0. The acid solution is allowed to contact the previously cleaned equipment surfaces to accomplish the following:

(1) To react with and neutralize any residual alkaline wash solution in order to remove it from equipment.
(2) To remove scale or mineral deposits to aid in preventing focal points for possible pitting corrosion.
(3) To prevent the precipitation of water hardness ions from the rinse water to eliminate water spotting and scale build-up
(4) To react with iron to aid in the prevention of Fe filming.
(5) To ensure that any residual moisture in the equipment has a low pH and will not favour microbial growth.

CLEANING PROCEDURES

Regardless of the type of cleaning procedure employed, i.e. manual or recirculation, the same fundamental steps are followed.

(1) **Pre-rinse.** Immediately after use, all equipment should be rinsed with water, preferably at 45-50 °C. These temperatures are above the melting point of butterfat and will improve the efficiency of rinsing. Avoid excessively high temperatures which may coagulate milk proteins. Extremely cold water may cause fat crystallization resulting in the formation of greasy films on surfaces. Discharge the rinse to drain.
(2) **Washing.** Prepare an appropriate cleaning solution. Follow directions and use the correct detergent at the correct concentration. Maintain the wash solution temperature within the recommended range. Perhaps the most common cause of failure in recirculation cleaning is allowing the temperature to drop below 40 °C. Fats redeposit and the entire cleaning programme breaks down.

For manual washing operations, use appropriate brushes for the type of equipment to be cleaned. Do not use metal sponges, wire wool or abrasive scouring pads as these will damage equipment surfaces. For circulation cleaning, maintain solution flow rates at the recommended solution temperature for the recommended time. (N.B. Do not clean for too long as temperatures will drop below 40 °C.)

(3) **Rinse.** After washing, rinse all equipment to remove suspended soils and traces of cleaning compounds. Acidify the water to pH 3,0-5,0 (typically 0,15% acid detergent). This is particularly important in hard water.
(4) **Storage.** Following rinsing, all utensils and equipment should be stored in a manner that permits water to drain and the equipment to become air dry with protection from possible gross re-contamination.
(5) **Sanitizing.** Equipment should be sanitized just before it's next use to reduce residual bacterial contamination of surfaces. Cleaning by itself does not eliminate all types of bacteria although good cleaning considerably reduces the initial bacterial population. Furthermore, equipment which has not been thoroughly cleaned cannot be adequately sanitized since residual soils protect bacteria from sanitizer action.

Chemical sanitizers are normally used and iodophors (25 ppm iodine) and chlorine (100 ppm chlorine) are the most widely used. Allow a minimum contact time of two minutes for best results.

CONCLUSION

The use of a well balanced cleaning and sanitizing programme will aid in the production of raw milk of exceptionally high microbiological quality. Farms with well disciplined and carefully organized sanitation programmes which extend from cow preparation to bulk tank cleaning, are easily achieving Standard Plate Counts of < 10 000/ml and coliform counts of < 10/ml for raw milk – standards usually not reached for fluid pasteurized milk by most processing plants in South Africa.

TREATMENT OF THE LARVAL STAGE OF *TAENIA MULTICEPS* WITH PRAZIQUANTEL

ANNA VERSTER* AND R.C. TUSTIN**

ABSTRACT: Verster, A.; Tustin, R.C. **Treatment of the larval stage of *Taenia multiceps* with praziquantel.** *Journal of the South African Veterinary Association* (1982) **53** No 2, 107-108 (En) Section of Helminthology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Five sheep were infested orally with 5 500 eggs of *Taenia multiceps* and were treated with praziquantel (Droncit, Bayer) when they showed typical nervous signs of coenuriasis. Two sheep were treated with praziquantel at 100 mg/kg live mass per day for 5 days, 2 sheep at the same dosage for 2 days and one animal at 50 mg/kg per day for 5 days. No viable coenuri were recovered from any of these animals. This is the only anthelmintic that is known to be effective against this parasite, but its cost is such (R200 at the lowest dose rate) that it can be economically used only for the treatment of valuable stud animals.

INTRODUCTION

Verster, Tustin & Reinecke[2] treated sheep infested with the larval stage of *Taenia multiceps* (*Coenurus cerebralis*) with mebendazole (Multispec, Ethnor) at 2 dosages and by 2 routes: intraperitoneally at 40 mg/kg live mass in a single dose or orally at 100 mg/kg live mass daily for 14 successive days. Neither of these treatments was effective. Gallie & Sewell[1] studied the effect of praziquantel (Droncit, Bayer) on the cysticerci of *Taenia saginata* and reported that oral treatment at 50 mg/kg live mass for 4 successive days killed all the cysticerci that were 12 weeks old, but was inefective against those that were 4 weeks old.

The use of praziquantel in sheep experimentally infested with the larval stage of *T. multiceps* is described in this paper.

MATERIALS AND METHODS

Five thousand five hundred eggs of *T. multiceps* were dosed orally to each of 5 sheep. When the animals showed typical nervous signs of coenuriasis from 6-11 months after infestation, they were treated with praziquantel as is summarized in Table 1. The drug was obtained in 2 formulations: tablets which each contained 50 mg of the active principle and an aqueous suspension which contained 5 % of the anthelmintic. To treat Sheep 1-3 the required number of tablets was pulverized, suspended in water and dosed by means of a stomach tube. Sheep 4 and 5 were treated with the 5 % suspension administered via a stomach tube.

TABLE 1: TREATMENT OF THE SHEEP INFESTED WITH THE LARVAL STAGE OF *TAENIA MULTICEPS*

Sheep No.	Age of Infestation at treatment (months)	Treatment with Praziquantel		
		mg/kg live mass	Duration of treatment (days)	Total dose (mg/kg live mass)
1	6	100	5	500
2*	7	100	2	200
3	7	100	5	500
4	10	50	5	250
5	11	100	2	200

*This animal caught its horn in the gate and died of shock 2 days after treatment commenced.

*Helminthology Section, Veterinary Research Institute, 0110 Onderstepoort.
**Faculty of Veterinary Science, University of Pretoria.

With the exception of Sheep 2 which died 2 days after treatment commenced, all the animals were killed by exsanguination and autopsied 30 days after treatment. The coenuri recovered from Sheep 1 and 2 were fed to 2 dogs to determine the viability of the scolices; the animals were euthanased 28 days later and the small intestine examined for tape worms.

RESULTS

Sheep 1

A single large coenurus about 40 mm in diameter, was recovered from the brain of this animal and it was noticed that its scolices appeared to be disintegrating. The whole of this coenurus was fed to a dog but no tapeworms were recovered at necropsy 28 days later.

Sheep 2

This animal caught its horn in a gate and died after 2 doses of praziquantel had been administered. One apparently normal coenurus approximately 30-40 mm in diameter, was present in the brain. This coenurus was fed to a dog but, once again, no cestodes were present when it was examined 28 days later.

Sheep 3

When the animal was examined 30 days after the last treatment, a small degenerate coenurus was present in the cerebral cortex.

Sheep 4

There were 4 partially calcified necrotic lesions in the cerebrum and one each in the cerebellum and hypothalamus of this animal. One of the lesions in the cerebrum was approximately 10 mm in diameter while the other lesions varied from 2-4 mm in diameter.

Sheep 5

Three small degenerate coenuri were present immediately beneath the meninges of the cerebrum of this sheep.

DISCUSSION

Despite the relatively small numbers of sheep in this experiment, there can be no doubt about the efficacy of praziquantel in the treatment of *T. multiceps* larvae. In our experience degenerate coenuri of this parasite are extremely rare in the brain of sheep and it may be assumed that the praziquantel was responsible for the degeneration of the coenuri in the brains of the sheep.

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It is of special interest to note that the drug killed the larvae at an oral dose of 100 mg/kg for 2 days within a period of 2 days of commencement of treatment (Sheep 2). The same dosage rate given to another sheep (No. 5) on each of 2 days again proved effective.

Praziquantel is the only drug that has shown any promise for the treatment of coenuriasis, but it is much too expensive to be used on a flock basis and can economically only be used in treating valuable stud animals. The lowest dose rate at which we achieved success (200 mg/kg live mass) cost R200. Further investigations may prove that a lower dose rate may be effective.

ACKNOWLEDGEMENTS

We are most grateful to Bayer S.A. for providing some of the praziquantel used in this trial. Mr. B.P. Maartens is thanked for dosing the animals and Prof. R.K. Reinecke for helpful criticism of the manuscript.

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2. Verster Anna, Tustin R C, Reinecke, R K 1978 An attempt to treat the larval stage of *Taenia multiceps* and a resume of its neural and extraneural distribution in sheep. Onderstepoort Journal of Veterinary Research *45: 257-259*

ABSTRACT: Potgieter, F.T. & Bester, J.B., 1981. **Freeze-drying of *Anaplasma marginale*.** *Onderstepoort Journal of Veterinary Research*, 48, 179-180 (1981).

Heparinized whole blood, heavily parasitized with *Anaplasma marginale*, was collected from 3 splenectomized oxen. Buffered lactose peptone (BLP) was added in equal volumes as a stabilizer and the mixture lyophilized in 2 mℓ aliquots after rapid freezing. The dried material was reconstituted with 2 mℓ sterile water and inoculated without delay. The product remained infective for at least 6 months when stored in an ordinary household deep-freeze unit.

ABSTRACT: Hoogstraal, H. & Wassef, Hilda Y., 1981. **Notes on African *Haemaphysalis* ticks. XIII. Identity of *H. (Rhipistoma) cooleyi*, a parasite of the rock hyrax in South Africa (Acarina: Ixodidae).** *Onderstepoort Journal of Veterinary Research*, 48, 135-140 (1981).

The lectotype male, allotype female, and paratype nymph of *Haemaphysalis (Rhipistoma) cooleyi* Bedford, *1929*, a parasite of the rock hyrax, *Procavia capensis*, in the Transvaal, are redescribed and illustrated to provide reliable criteria for differentiating between haemaphysaline parasites of hyraxes. Keys are included for identifying adults of these *Haemaphysalis* spp. (*orientalis* Nuttall & Warburton, 1915; *bequaerti* Hoogstraal, 1956; *cooleyi* Bedford, 1929; *hyracophila* Hoogstraal, Walker & Neitz, *1971*).

ABSTRACT: Herr, S. & Marshall, C., 1981. **Brucellosis in free-living African buffalo (*Syncerus caffer*): a serological survey.** *Onderstepoort Journal of Veterinary Research*, 48, 133-134 (1981).

The rose bengal and complement fixation tests were successfully applied to buffalo (*Syncerus caffer*) sera. An overall occurrence of 23 % positive reactors was obtained, but *Brucella* infection does not appear to act as an effective culling agent in buffalo. Any eradication programme must take free-living buffalo into account as a possible source of re-infection for cattle.

BOOK REVIEW — BOEKRESENSIE

ANIMAL ANATOMY AND PHYSIOLOGY

JESSE F. BONE

1st Edn. Reston Publishing Company, Inc., Reston, Virginia 22090. 1979 pp 560, numerous illustrations and tables, Price R26,60 (ISBN 0-8359-0220-X)

As explicitly stated in the preface by the author this book was written to serve as an introduction to anatomy and physiology of domestic animals. It is intended for the non-professionally orientated student or layman, and viewed in this light the book can be recommended.

The author has partly succeeded in integrating structure and function of the animal body. The anatomy of the domestic animal is covered in a fair degree of detail, some of which extends beyond the bounds of the interested layman or superficial student. The author does not, however, use the modern formal Nomina Anatomica Veterinaria terminology. Instead he uses translations thereof and lay-terms. Certain terms are antiquated or derived from human anatomy and are therefore anatomically incorrect for animals. One diagram (Fig. 3-1) is not labeled. A very useful section is given on the ageing of domestic animals.

The physiology of domestic animals is covered fairly well in this book, with the exception of endocrinology, which is dealt with very superficially. Certain aspects of physiology appear at first sight not to be covered because they do not follow on sections where one would expect to find them. In many cases this disrupts the continuity and understanding of the section concerned. The physiological terminology used is often outdated, not to mention SI units and symbols which are seldom used.

The section on intermediary metabolism is fairly comprehensive with the exception of the control of metabolism. There are a number of errors, most of them are, however, of a minor nature. The control of metabolism and the all important role played by DNA and RNA, is hardly mentioned.

The final chapter deals with the anatomy and physiology of the domestic fowl which once again is extremely useful.

H.J. Bertschinger and G.J. Louw

THE LIFE CYCLE OF THE LUNGWORM, *PNEUMOSTRONGYLUS CALCARATUS*

IRMGARD G. HEINICHEN ANDERSON*

ABSTRACT: Heinichen Anderson I.G. **The life cycle of the lungworm, *Pneumostrongylus calcaratus*.** *Journal of the South African Veterinary Association* (1982) **53** No. 2, 109-114 (En) Department of Zoology, University of Zululand, 3886 Kwa-Dlangezwa, Republic of South Africa.

Further data on the life cycle of the lungworm, *Pneumostrongylus calcaratus*, including the morphology of a larva in third moult found in the lung of a guinea pig, are presented. Transmission experiments in sheep, a goat and guinea pigs are recorded and the whole life cycle of *P.calcaratus* is decribed and illustrated.

INTRODUCTION

The life cycle of *Pneumostrongylus calcaratus* was decribed by Heinichen[9] and Heinichen Anderson[10]. It was reported that the yellow slug, *Elisolimax flavescens* (Keferstein, 1866), which is common in the coastal regions of Natal, is the intermediate host of this lungworm. The morphology of the first, second and third stage larvae (L_1, L_2 and L_3) was described[9] [10].

In the present paper further data are presented: all the measurements of the L_1, L_2 and L_3 are listed and compared with those of *Pneumostrongylus tenuis* (Dougherty, 1945) and the L_1 of *Pneumostrongylus cornigerus*, (Ortlepp, 1962); the morphology of the fourth stage larva (L_4) is described, and the results of experimental transmission to mammalian hosts, other than the impala, are recorded.

MATERIALS AND METHODS

For the experimental infestation of sheep, a goat and guinea pigs, the foot of naturally and experimentally infested *E. flavescens* was cut into fine pieces and fed to the animals.

A 5-month-old lamb and a 4-month-old goat were infested with L_3 of *P. calcaratus* harvested from experimentally infested slugs. The lamb and goat were killed 10 and 17 days after infestation respectively. All their internal organs and also the spinal cords were examined for larvae of *P. calcaratus*. Subsequently another lamb was similarly infested, but killed after 51 days.

Seven young guinea pigs were treated *per os* with 25 mg mepyramine maleate (Anthisan, Maybaker S.A., Ltd.) 2 days before experimental infestation with L_3 collected from infested slugs. Two of the guinea pigs were killed after 4 days and the remainder 7, 10, 11, 16 and 42 days after infestation.

Two groups of 4 and one group of 5 guinea pigs received subcutaneous injections of 2,0, 1,25 and 1 mg methylprednisolone acetate (DM = Depo-Medrol: Tuco Products) respectively, 3 and 1 day prior to infestation with L_3 of *P. calcaratus*. The latter group was also treated every second day with 50 mg oxytetracycline (Boehringer) per kg live mass. A minimal dose, approximately 0,05 mg of DM, was given to another 2 guinea pigs 2 days before infestation and these were also treated every second day with 50 mg oxytetracycline per kg live mass.

All internal organs as well as the spinal cords were examined in the guinea pigs which survived the treatment with DM and the subsequent infestation with L_3 of *P. calcaratus*.

*Department of Zoology, University of Zululand, 3886 Kwa-Dlangezwa, Republic of South Africa.

Measurements of L_4 were taken with an eyepiece micrometer and the drawing was done with the aid of a camera lucida.

RESULTS

(a) Observations

Although some of the measurements of L_1, L_2 and L_3 were partly described and compared with those of *P. tenuis* in the previous publications[9] [10], more detailed data on these measurements are given in Table 1 where L_1 is compared with *P. cornigerus*[14].

Transmission tests on the young impala, the goat and sheep gave negative results. Only one of the lambs, killed 51 days after infestation, had developed lungworm lesions. G.D. Imes (1976 unpublished work, Veterinary Research Institute, Onderstepoort) kindly examined these lungs and found that the lesions were similar to those caused by *Muellerius* spp. in sheep.

Ten of the 15 guinea pigs examined for *P. calcaratus* larvae gave positive results. Of these, the 2 treated with mepyramine maleate and killed 4 days after infestation were found to be infested with only 1 L_3 each. Seven treated with DM and killed between 4 and 6 days after infestation, had several L_3 in their lymph nodes. Only one guinea pig also treated with DM survived for 8 days after infestation and a single male larva in its third moult was found in the lung. Seven guinea pigs treated with DM did not survive longer than 2 to 4 days.

The larva of *P. calcaratus* in its third moult found in the lung of a guinea pig was 729 μm long and its width varied from 16 μm anteriorly to 42 μm across the oesophagus and 26 μm at the level of the anus. The oesophagus was 194 μm in length and 23 μm wide. The distance of the excretory pore and nerve ring from the anterior end measured 130 μm and 113 μm respectively. The distance from anus to tail was 49 μm and the sheath extended 4 μm beyond the larval tail. The genital primordium had divided into 5 cells and its tail had a small, pointed appendage (Fig. 1).

(b) Life cycle

The life cycle of *P. calcaratus* is illustrated in Figure 2. The L_2 was found 6 to 7 days, and L_3 13-14 days respectively, after L_1 entered the foot of the slug.

Slugs occur on various trees in the Nyala Game Ranch mainly during the summer months but are most abundant on *Ziziphus mucronata* (Willd, 1809), a tree regularly browsed by impala and other antelope.

The parasitic L_3 of *P. calcaratus* was found in the mucosa of the small intestine of some adult impala. These larvae were probably migrating to the lymph nodes.

Experimental infestations were carried out to com-

TABLE 1: DIMENSIONS (μm) OF THE L_1, L_2 AND L_3 OF *PNEUMOSTRONGYLUS CALCARATUS* AND *PNEUMOSTRONGYLUS TENUIS* AND THE L_1 OF *PNEUMOSTRONGYLUS CORNIGERUS*

SPECIES	*P.calcaratus*		*P.cornigerus*	*P.tenuis*		*P.calcaratus*		*P.tenuis*	*P.calcaratus*		*P.tenuis*	
HOST	Impala		Bontbok	White-tailed Deer		Impala		White-tailed Deer	Impala		White-tailed Deer	
AUTHOR	These results		Ortlepp (1962)	Anderson (1963)		These results		Anderson (1963)	These results		Anderson (1963)	
STAGES OF DEVELOPMENT	L_1		L_1	L_1		L_2		L_2	L_3		L_3	
NUMBER MEASURED	10		20	---		10		1	10		10	
DIMENSIONS	RANGE	MEAN ± s.d.	RANGE	RANGE	MEAN	RANGE	MEAN ± s.d.	RANGE	RANGE	MEAN ± s.d.	RANGE	MEAN
TOTAL LENGTH *	314-341	325,60 ± 8,90	285	310-380	348	520-807	707,17 ± 87,10	715	604-780	699,30 ± 52,00	900-1080	971
Width: At anterior end	5-10	7,40 ± 1,60	---	---	---	5-18	12,90 ± 3,70	---	8-27	16,50 ± 6,70	---	---
Across Oesophagus	11-14	12,50 ± 1,80	---	---	---	20-34	25,30 ± 5,00	---	20-32	22,90 ± 3,20	---	---
Maximum	15-19	17,20 ± 1,30	15	16-19	18	39-57	47,50 ± 6,00	43	30-92	50,80 ± 23,20	36-45	42
At anus	9-12	11,00 ± 3,20	---	---	---	23-37	29,80 ± 4,50	---	20-54	33,70 ± 4,30	---	---
Length of oesophagus	141-163	152,30 ± 6,30	80	132-181	165	176-289	224,80 ± 43,10	242	169-260	212,80 ± 27,10	300-400	352
Distance from excretory pore to anterior end	72-95	87,60 ± 7,50	---	80-112	94	98-176	121,50 ± 44,70	113	100-157	126,50 ± 16,30	122-149	133
Distance from nerve ring to anterior end	74-108	94,50 ± 9,00	---	80-112	94	118-182	147,50 ± 23,90	101	125-189	162,00 ± 25,70	100-125	114
Genital primordium: Length	---	---	---	---	---	11-16	14,20 ± 1,80	---	9-49	22,50***	---	---
Width	---	---	---	---	---	7-11	8,40 ± 1,30	---	6-20	11,40***	---	---
Distance from genital primordium to tail	---	---	---	100-134	124	221-322	279,20 ± 28,50	277	163-329	235,00***	331-373	342
Distance from anus to tail (with sheath if present)	19-39	30,10 ± 6,90	8	29-41	34	46-72	55,80 ± 8,20	32	39-113	62,70 ± 25,30	31-45	35
Distance from tail to sheath	No sheath					4-27	10,20 ± 9,00	---	1-52	20,20 ± 24,30	---	---
Distance of sheath from anterior end	No sheath					11-49	17,80**	---	7-22	51,00**	---	---

* The total length includes the sheath if present
** Only six worms measured
*** Only seven worms measured

plete the life cycle. Parasitic L_3 were found in the lymph nodes of some guinea pigs, but no further development took place there. In one guinea pig, 1 larva found in the lung, was in its third moult, showing L_4 in the sheath of L_3 (Fig. 1). The small nodules found in the visceral pleura of impala lungs contained 5th stage worms.

A very heavy infestation of lungworm is illustrated in Fig. 3. Many grey lesions can be seen in the distal, thin edges of the lung, indicated by an arrow (Fig. 3). When these lesions are opened they are filled with thin, thread-like adult *P. calcaratus* and numerous L_1.

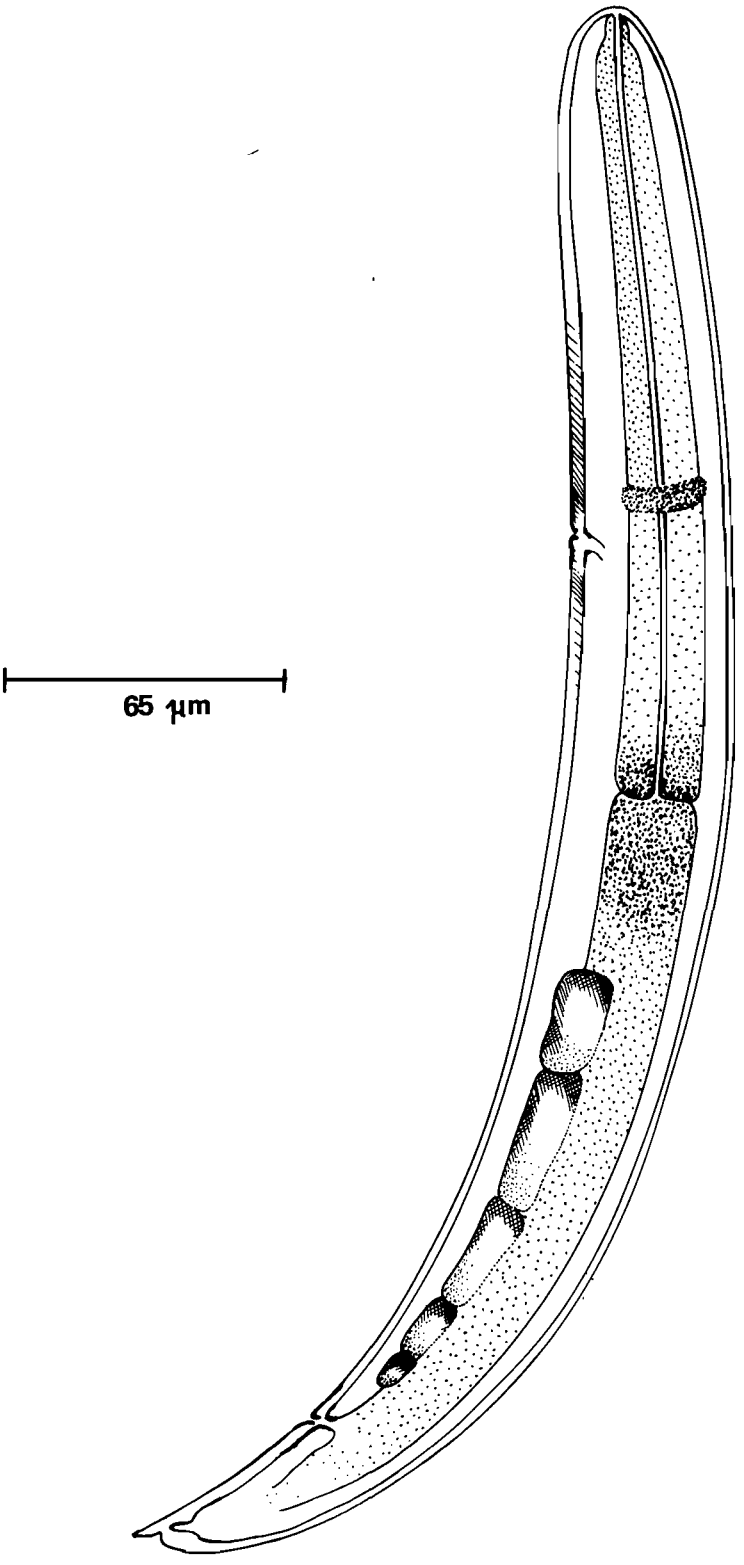

Fig. 1: *Pneumostrongylus calcaratus* in third moult

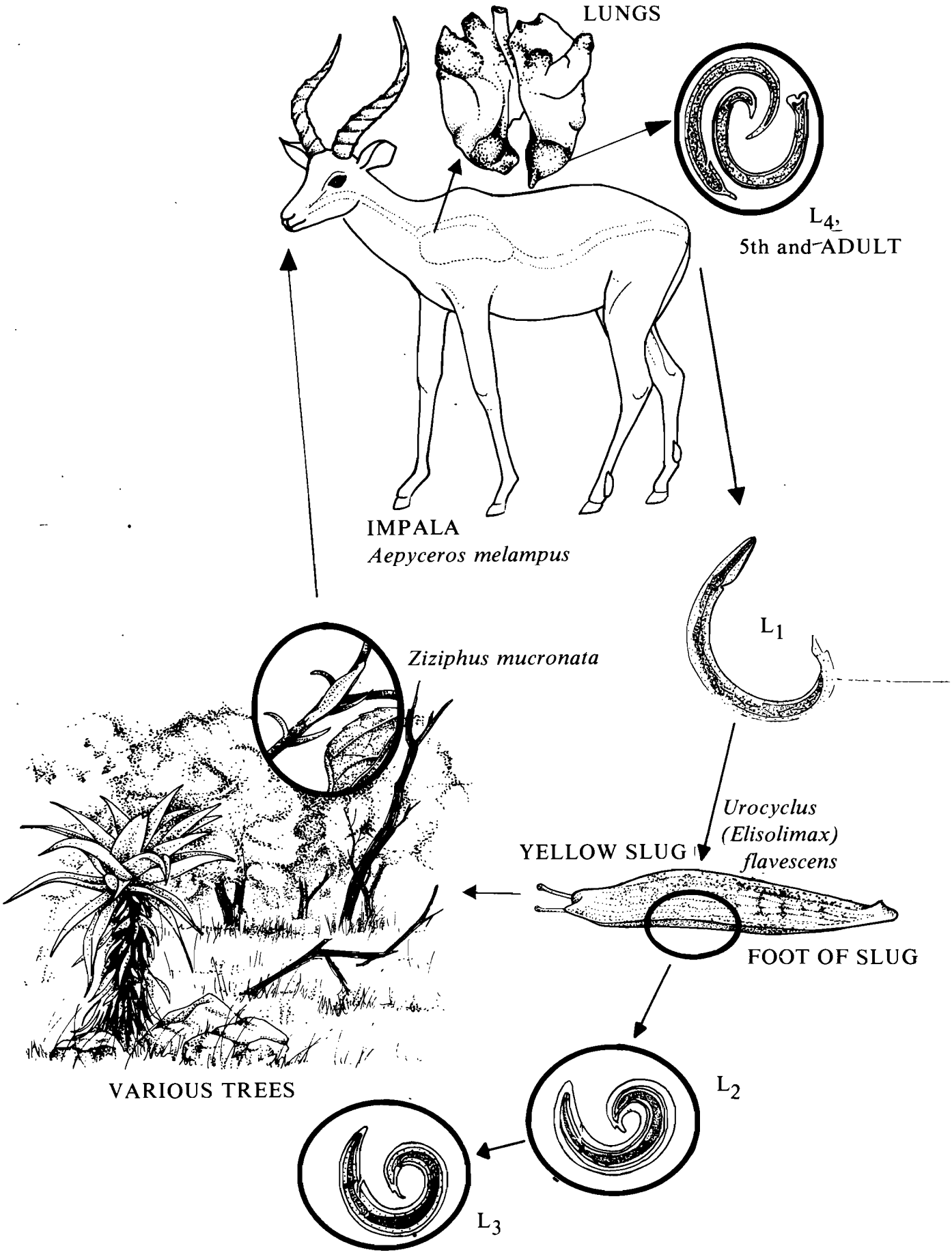

Fig. 2: Life cycle of *Pneumostrongylus calcaratus*

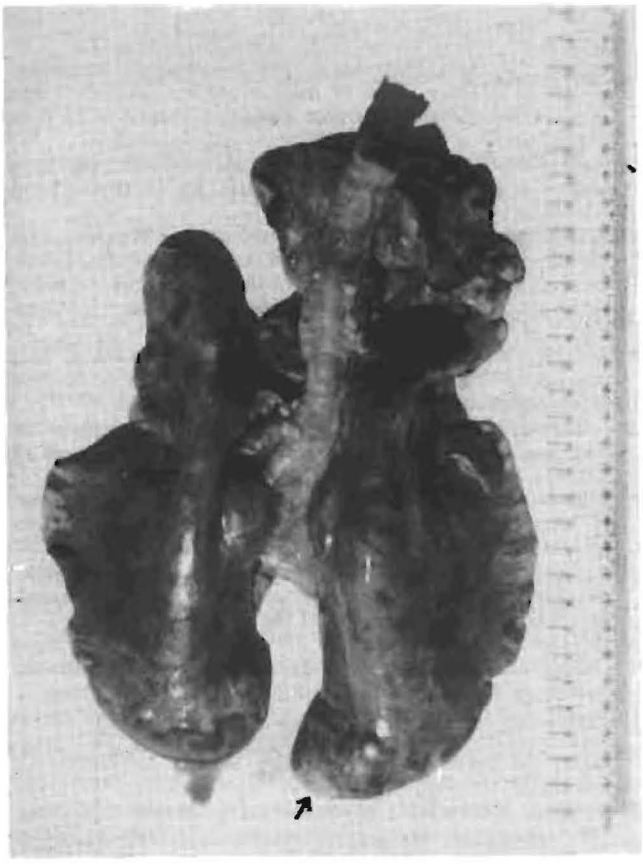

Fig. 3: Impala lung showing the grey lesions on the distal edges of the lung *Pneumostrongylus calcaratus* infestation

DISCUSSION

Alicata[1] isolated infective larvae from the foot of molluscs by breaking up the tissues and wrapping the small pieces of tissue in gauze placed in a waterbath (37 °C) for 1 to 2 hours. This method was tried repeatedly without success with the tissues of *E. flavescens*. The foot of the slug was therefore cut into fine pieces and fed to the mammalian hosts.

Various abnormal hosts have been used to study the life cycle of lungworms. In studying the migration of parasitic larval stages of *Dictyocaulus filaria* (Hudophi 1809) Kauzal[12] used guinea pigs, while Soliman[15] used rabbits, mice and guinea pigs. More recently, Anderson & Strelive[5] successfully used guinea pigs for their studies on the life cycle of *P. tenuis*.

One sheep examined 51 days after infestation with *P. calcaratus* had lungworm lesions and Imes (1973, personal communication) suggested that *P. calcaratus* reached the lung, but that the tissue was probably not suitable for any further development.

Since no *P. calcaratus* larvae were found in the artificially infested impala[10], nor in one lamb and a young goat[2], the studies were repeated on guinea pigs, but successful transmissions occurred only when the animals were treated with an immuno-suppressant. The mortality rate, however, was approximately 47 % during the first 8 days after infestation as treated animals were susceptible to various infectious diseases. Guinea pigs treated with immuno-suppressants should be kept under pathogen-free conditions, but this was not possible during this investigation. When they were treated with mepyramine maleate they neither acquired infectious diseases, nor were they susceptible to infestation with the lungworm.

The parasitic L_3 found in the mesenteric lymph nodes of the guinea pigs 4 to 6 days after infestation had not increased in size. Brenner[8] found in *Haemonchus placei* (Place, 1893) and Sommerville[16] in *Cooperia curticei* (Giles, 1892) that no growth took place in the parasitic L_3 during the first 2 days of infestation. Archer & Hopkins[7] also found a lag in growth for the initial period of infestation in the cestode *Diphyllobothrium* (Cobbold, 1858) and they suggested that this was due to a period of physiological adaptation. The fact that, in the present study, growth of the L_3 of *P. calcaratus* only commenced 6 to 8 days after infestation, is possibly also due to a period of physiological adaptation in the guinea pig, which is not its normal host.

The single L_4 found in the lung of a guinea pig, 8 days after infestation, shows that little if any growth took place during the development from L_3 to the L_4. The total length of the L_3 varied from 604-780 μm (699,30 ± 52,00 μm), whereas the L_4 was 729,00 μm long.

Mönnig[13] gave the measurements of the "longest portion of a male recovered" as 29,7 mm. The one complete male recovered from a small nodule on the outside of a lung and measuring 35 mm[10] was probably a 5th stage (immature adult).

A study of the morphology of *P. calcaratus* has shown that it is closely related to the lungworm *P. tenuis* of the white-tailed deer[3]. The life cycle of *P. calcaratus*, however, is also similar to that of other lungworms of sheep viz. *Muellerius capillaris* (Müller, 1889) and *Protostrongylus rufescens* (Leuckart, 1865).

Anderson[3] found L_4 and 5th stage of *P. tenuis* in the spinal cord of the white-tailed deer and in his transmission tests of *P. tenuis* to guinea pigs he also recovered larvae from the spinal cord. The spinal cords of a few impala from the Nyala Game Ranch, as well as from the guinea pigs used for the transmission tests, were examined for *P. calcaratus*, but no parasites were found. These observations showed that the third moult of *P. calcaratus* took place in the lung and although it passed through the mesenteric lymph nodes, there was no development of the L_3 there.

Soliman[15] and Anderson & Verster[6] experimentally infested guinea pigs and sheep respectively with the lungworm *D. filaria* and they found that there was a marked decrease in the number of parasites in the lymph nodes 7 to 8 days after infestation, while the number of parasites in the lung tissue increased. The single male L_4 of *P. calcaratus* that was found, was recovered from the lung of the guinea pig 8 days after infestation.

Anderson & Strelive[5] recorded that some of the guinea pigs infested with *P. tenuis* showed neurological signs 27-28 days after infestation. In the present study no such signs were observed in any of the guinea pigs not even the animal which survived for 5 weeks after infestation.

Young & Wagener[18] reported that in the Kruger National Park almost all the impala studied were infested with *P. calcaratus*. Young & Wagener's article[18] is illustrated with a photograph of a thick, white nematode in the bronchi of a lung, which they identify as *P. calcaratus* and describe this parasite as being a "very thin, thread-like, black nematode (Fig. 1)". During this study it was found that when a lung lesion is opened to expose the adults, eggs and L_1 of *P. calcaratus* the thin, thread-like adults are only visible as a dark greyish mass.

Anderson[4] observed that natural infestations of *P. tenuis* did not occur in aquatic snails, but he could infest *Lymnaea* spp. in the laboratory. *Lymnaea collumella* (Say, 1817), which occurs on the Nyala Game Ranch, was also examined, but no *P. calcaratus* larvae were found in it.

To date no work has been done on the infestation of other molluscs with *P. calcaratus*. According to the distribution of *E. flavescens*[17], this slug occurs in the Kruger National Park where all the impala studied were infested with *P. calcaratus*[18]. In his studies on the helminths of impala in the Nylsvlei Nature Reserve, Horak[11] did not find any impala infested with *P. calcaratus*. Since no investigations were carried out as to whether *E. flavescens* occurs in this area, the absence of the lungworm amongst impala from the Nylsvlei Nature Reserve may be due to the absence of the intermediate host. Great care, however, should be taken not to introduce *P. calcaratus* into the Nylsvlei Nature Reserve until the occurrence of *E. flavescens* has been investigated, nor until it has been determined what other molluscs can act as intermediate hosts.

CONCLUSIONS

These and other recent studies[9, 10], have culminated in the elucidation of the entire life cycle of *P. calcaratus*. It is easy to detect the presence of L_1 of *P. calcaratus* in impala faeces as well as L_2 and L_3 in *E. flavescens*. Care therefore should be taken not to introduce this parasite into another area through media such as transportation of impala. It is difficult to control the intermediate host, but through biological control measures, such as large birds feeding on the slug, their numbers may be reduced.

ACKNOWLEDGEMENTS

Grateful appreciation is expressed to Messrs I. and R. Scott-Barnes for helping me to obtain the material. My sincerest thanks go to Dr A. Verster and Prof. J. Heyns for their assistance and advice, to Lt Col G.D. Imes for the autopsies carried out on the sheep infested with *Pneumostrongylus calcaratus* and to Prof. H.P.A. de Boom for his contribution towards the preparation of this paper. Grateful thanks also go to the University of Zululand for the facilities provided and to the Council for Scientific and Industrial Research for their financial support.

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CASE REPORT **GEVALVERSLAG**

ACUTE RENAL FAILURE IN A DOG FOLLOWING EXERTIONAL RHABDOMYOLYSIS

ELIZABETH W. HOWERTH* and CHERYL M.E. McCRINDLE**

ABSTRACT: Howerth E.W.; McCrindle C.M.E. **Acute renal failure in a dog following exertional rhabdomyolysis.** *Journal of the South African Veterinary Association* (1982) **52** No. 2, 115-117 (En) Section of Pathology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Rhabdomyolysis with myoglobinuria occurred in a dog following extreme physical exertion. Acute renal failure subsequently developed. This case serves to emphasize the importance of screening for renal dysfunction following exertional rhabdomyolysis.

Key words: Muscular disease, kidney disease, myoglobinuria, oxalosis.

INTRODUCTION

Rhabdomyolysis resulting from severe crushing injuries and associated with myoglobinuria and acute renal failure was first reported in man during World War II[4]. Since that time, numerous and diverse causes of rhabdomyolysis with myoglobinuria have been recognized in man. In addition to crushing injuries, the following causes have been reported in the literature: extreme muscular exertion, hereditary muscle enzyme deficiencies, convulsions, electric shock, ischaemia as the result of vascular occlusion, exogenous toxins and drugs, malignant hyperthermia, hypothyroidism, hypokalaemia, hypophosphataemia, diabetic acidosis, and systemic infection and fever[7 11-13 18].

Reports of rhabdomyolysis and/or myoglobinuria in dogs are rare. In this report we describe the clinical and pathological findings in a dog with exertional rhabdomyolysis, myoglobinuria, and subsequent acute renal failure.

CASE HISTORY

An adult male Bull Terrier was presented in a state of collapse. The dog had been chasing sheep and was found prostrate in the vicinity of a dead sheep. On admittance he had a temperature of 38,5 °C and there was fresh blood in the faeces. Muscular rigidity was marked and the dog was unable to walk. Immediate supportive therapy, including intravenous fluids, antibiotics and vitamins, was administered.

During the first day of hospitalization, brown pigmented urine was voided. Three days later, the conjunctivae appeared brownish and the muscles of the head began to swell; intravenous fluids and antibiotics were again administered. On the fifth day of hospitalization he was able to walk and was discharged, although the muscles of the head were still swollen.

Seven days later the dog was readmitted in a state of collapse. While at home he had refused to eat or drink and had become progressively weaker. Therapy on readmittance consisted of the administration of intravenous fluids to correct the dehydration which had developed. Fluid therapy was repeated the following day and corticosteroids were administered. The blood urea nitrogen was > 32,6 m mol/ℓ (Azostix, Ames Co.) on the third day following readmittance and, due to the poor prognosis, the owner requested euthanasia.

PATHOLOGY

A complete necropsy was performed and selected tissues were fixed in 10% neutral buffered formalin. Representative samples were embedded in paraffin, sectioned at 5-7 μm and stained with haematoxylin and eosin (HE). In addition, sections of muscle were stained with von Kossa's technique and sections of kidney were stained with von Kossa's, periodic acid – Schiff (PAS), Perls', Hall's, Alizarin Red S and Benzidine methods.

Macroscopic Findings

Dried dark tarry faeces covered the perineal area. Multiple ulcers and erosions, ranging from 5 to 10mm, were present in the mucosa of the stomach. Digested blood and mucus coated the surface of the stomach and the small and large intestines. The kidneys were outwardly pale and swollen. The cut surfaces of the renal cortices were pale and bulged slightly. Multiple, small, greyish-white, chalky-appearing streaks were present in the left ventricular myocardium at the apex. The skeletal muscles had numerous large and confluent pale areas, which were mottled by greyish-white, chalky-appearing streaks. The distribution of the muscle lesions was bilaterally symmetrical. Muscles most severely involved included the epaxial, diaphragmatic, intercostal, abdominal, caudal brachial, cranial thigh, rump, and the temporal groups. Other organs and tissues were grossly unremarkable.

Microscopic Findings

All skeletal muscles that had exhibited gross lesions showed extensive degeneration and necrosis (Fig. 1). The necrotic fibres were swollen, hyaline or granular in appearance, and often fragmented. Many of the necrotic fibres were mineralized. A mild to moderate infiltration of macrophages often accompanied the muscle fibre necrosis.

Small foci of myocardial necrosis consisted of swollen, usually mineralized, myocardial fibres and a small number of macrophages.

Severe diffuse atrophy of the renal cortical tubules was characterized by the presence of numerous dilated tubules lined by flattened epithelial cells. Many tubules contained light to dark red granular or hyaline casts

*Department of Pathology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.
**Private Practitioner, Pretoria.

(Fig. 2). Some of these casts were intensely PAS positive with a few being benzidine-positive. A small number of yellow-brown birefringent crystals, made up of needles in a spoke-like arrangement, were scattered throughout the cortical tubules. The crystals manifested a positive reaction for calcium.

Evidence of ongoing necrosis was scant and tubular regeneration, although minimal, was observed. Reactive inflammatory changes were slight.

Gastric ulceration did not extend beyond the submucosa. Infiltrating neutrophils and thrombosed veins were present at the base of these lesions.

DISCUSSION

Exertional rhabdomyolysis (ER) is a condition which results from exteme physical exercise, occurs primarily in untrained individuals, and is characterized by muscle necrosis and myoglobinuria[18]. The clinical signs in this case are comparable to those observed in man where the syndrome is characterized by muscular pain, swelling, induration, and limitation of activity[7]. The exact pathophysiology of ER is not known but it is presumably related to energy metabolism. Glycogen is virtually depleted from muscle of normal individuals after exhaustive exercise suggesting that ER may occur secondary to indequate or abnormal production of energy during stress[18].

Equine paralytic myoglobinuria (azoturia) is the most widely recognized form of ER in animals[16]. Other syndromes similar to ER have been described in wild animals[2], cattle[1], sheep[17], in a racing greyhound[6], and in a dog with seizures[20].

In retrospect, the pigment observed in this dog's urine on the first day of hospitalization was most likely myoglobin. The severe widespread skeletal muscle necrosis could easily account for the release of large quantities of myoglobin, resulting in myoglobinuria. Histochemical evidence of haem pigment in some of the renal tubular casts also indicated that myoglobin (and/or haemoglobin) had recently been excreted through the kidney. Myoglobinuria, haemoglobinuria, haematuria, porphyria, and ingestion of certain dyes or drugs that may discolour the urine should be considered in the differential diagnosis of pigmenturia[8] [18]. A presumptive diagnosis of myoglobinuria can be based on detection of haem pigments in the urine and absence of erythrocytes in the urine sediment. In addition, the plasma should be clear and normal coloured because myoglobin is a small molecule which is poorly bound to serum proteins and is rapidly excreted by the kidneys before reaching levels high enough to discolour the plasma[8]. Specific identification of myoglobin in urine is best achieved by immunologic or electrophoretic techniques[11].

Renal changes similar to those decribed in this case are commonly associated with ER and myoglobinuria in various species[2] [18] [20]. Although the pathogenesis of the nephropathy associated with the excretion of haem pigments (myoglobin/haemoglobin) is not fully understood, the following mechanisms have been suggested: (1) haem proteins either cause or are associated with a decrease in or redistribution of renal blood flow or glomerular filtration rate, or both, with resultant areas of renal ischaemia; (2) haem proteins form casts that plug renal tubules; and (3) haem proteins and/or their

Fig. 1: Skeletal muscle fibre necrosis with mineralization (arrows). HE X 200

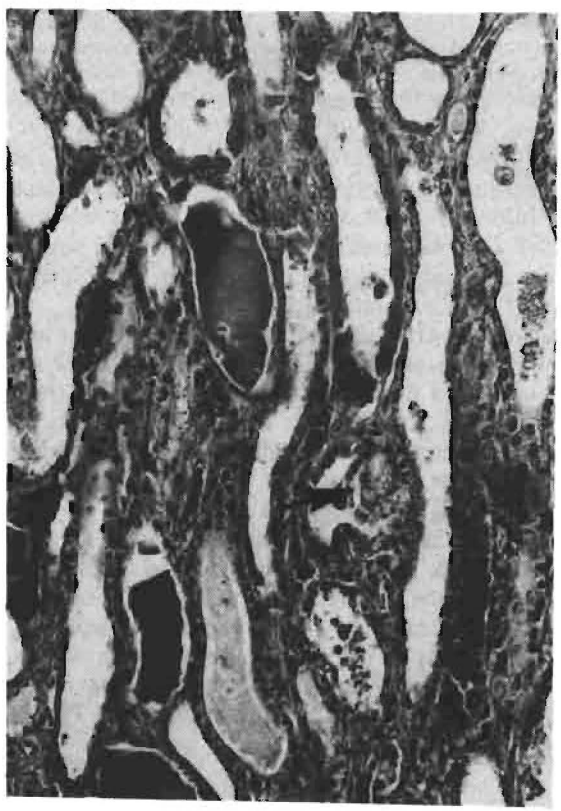

Fig. 2: Renal cortex showing dilated renal tubules, hyaline and granular casts, and oxalate crystals (arrow). HE X 200

breakdown products are directly toxic to the renal tubular epithelium. The pathophysiology of renal failure associated with ER may involve any of these mechanisms, singly or in combination[3 14].

Based on their morphological appearance, histochemical properties, and birefringent nature, the crystals in the kidney tubules were considered to be calcium oxalate[10]. Renal oxalosis secondary to renal insufficiency is not an unusual occurrence in man[19]. The origin of the oxalate crystals is unknown but two processes may be involved: (1) retention of calcium oxalate resulting from anuria and/or (2) an overproduction of calcium oxalate as a result of an abnormality in metabolism[10]. We suspect that the formation of oxalate crystals in this case was associated with renal failure and that the oxalate deposits, although minimal, may have contributed to renal parenchymal damage.

Gastric ulceration is frequently encountered in uraemic dogs and has been attributed to anoxia as a result of vascular injury[5].

The possibility of the myocardial changes being related to those in the skeletal muscle cannot entirely be excluded but they can probably best be attributed to the presence of renal disease. Similar myocardial lesions have been described in uraemic dogs, humans and rats and although electrolyte disturbances, metabolic disorders and vascular changes have been suggested as possible factors involved in the genesis of uraemic myocardial lesions, the basic mechanism is unknown[9 15].

Exhaustive and unfamiliar exercise associated with chasing sheep is considered to have been the precipitating cause of rhabdomyolysis in this case. Although the myoglobinuria is presumptive, the history of recent physical exertion and the gross and histopathological findings strongly suggest that the pigmenturia observed was caused by myoglobin. Concomitant occurrence of a positive urine occult blood test, pigmented granular casts in the urine sediment, and elevated serum creatine phosphokinase levels is considered strongly suggestive of rhabdomyolysis with myoglobinuria in man[11]. This triad of abnormalities might also prove useful in the presumptive diagnosis in dogs. Subsequent screening for renal dysfunction is important in ER since myoglobinuric associated nephropathy, as seen in this dog, is a frequent complication which can lead to renal failure.

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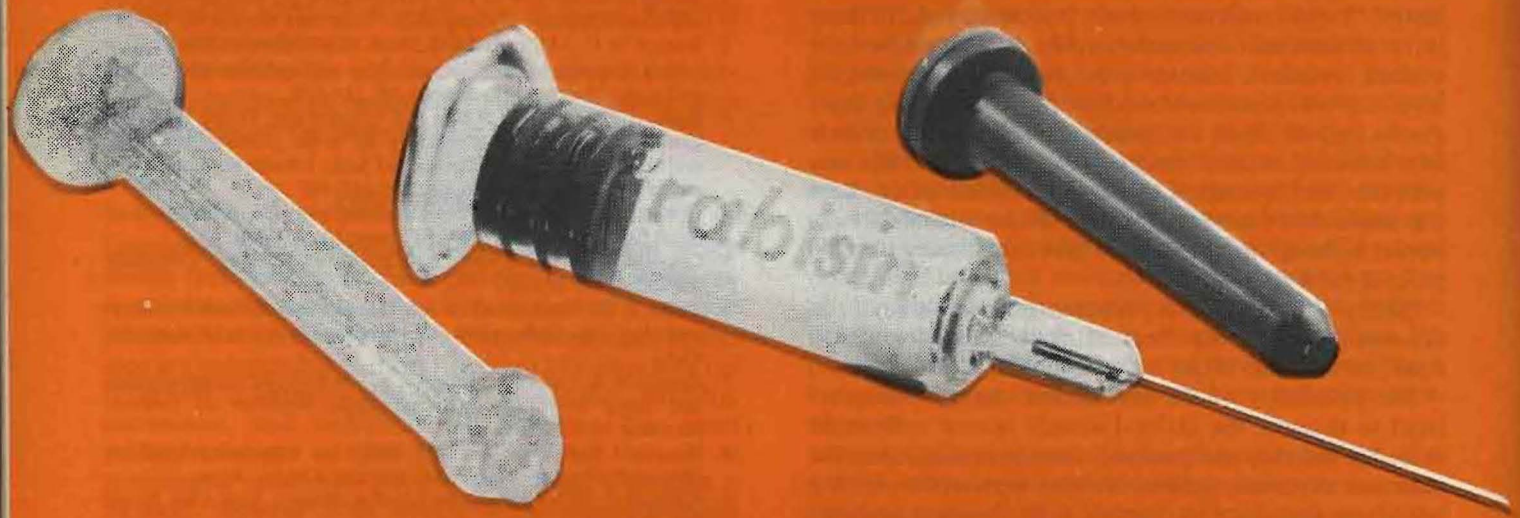

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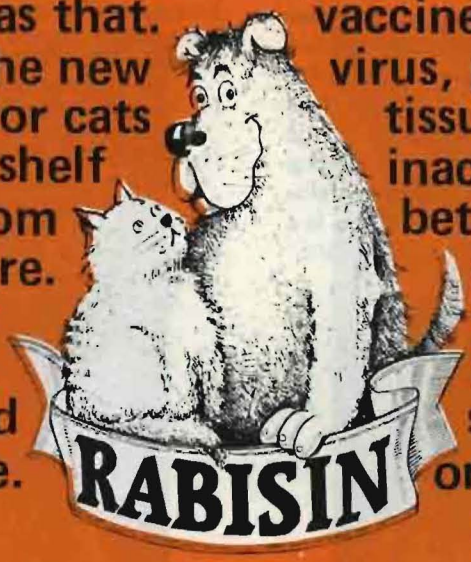

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CASE REPORT **GEVALVERSLAG**

THE SURGICAL REPAIR OF ATRESIA ANI IN A DOBERMANN BITCH

G.J. LOUW* and SELMA J.E.M. VAN SCHOUWENBURG**

ABSTRACT: Louw G.J.; Van Schouwenburg S.J.E.M. **The surgical repair of atresia ani in a Dobermann bitch.** *Journal of the South African Veterinary Association* (1982) No. 2, 119-120 (En) Department of Anatomy, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The surgical repair of atresia ani accompanied by a large rectovaginal fistula was successfully performed in a 4-month old Dobermann bitch. As a young pup, the dog had been able to pass liquid stool through the vagina via a rectovaginal fistula, but the gradual change to a adult diet had resulted in considerable obstipation and tenesmus. The operation was performed experimentally, since the dog would have had to have been euthanased at this stage, and its success has allowed the dog to continue a satisfactory existence.

Key words: Atresia ani, rectovaginal fistula, surgery, dog.

INTRODUCTION

This report deals with the successful surgical correction of atresia ani accompanied by rectovaginal fistula in a young Dobermann bitch. The pup was born without an externally visible tail (Fig. 1). The breeders of the dog also realised that she was smaller and a slower grower than the rest of the pups from her litter. When she was a few weeks old, they observed her passing stool through her vulva. Since the animal could not have lead a normal existence it was decided to reconstruct an anus and close the rectovaginal fistula surgically.

HISTORY AND CLINICAL FINDINGS

The pup was able to pass liquid stool through the rectovaginal fistula prior to weaning. At 6 weeks of age, when her diet changed from liquid to a more solid one, the owners noticed that she would not pass faeces for three or four days, exhibit excessive tenesmus, and then pass large volumes of stool. The owners were advised to maintain her on a low fibre, high water-content, high quality diet until she would be old enough for an anaesthetic and surgery.

*Department of Anatomy, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort.
**Private Practitioner, Pretoria.

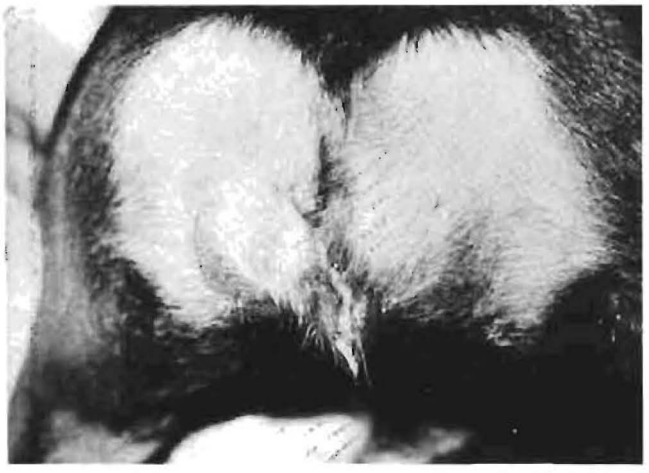

Fig. 1: The perineal area of the bitch pre-operatively, showing the absence of a tail.

When the dog was 4 months old, barium (Nov-Umbrose, Glaxo-Allenbury) was introduced into the rectum via the rectovaginal fistula for radiographic examination (Fig. 2 & 3). The radiographs indicated a large mass of stool accumulating in a blind-ending descending colon. Some barium had leaked through the rectovaginal fistula into the bladder. The coccygeal vertebrae were flexed tightly ventrally, within the pelvis.

The dog was starved for a day and dosed continuously with laxatives in preparation for general anaesthesia and the surgery.

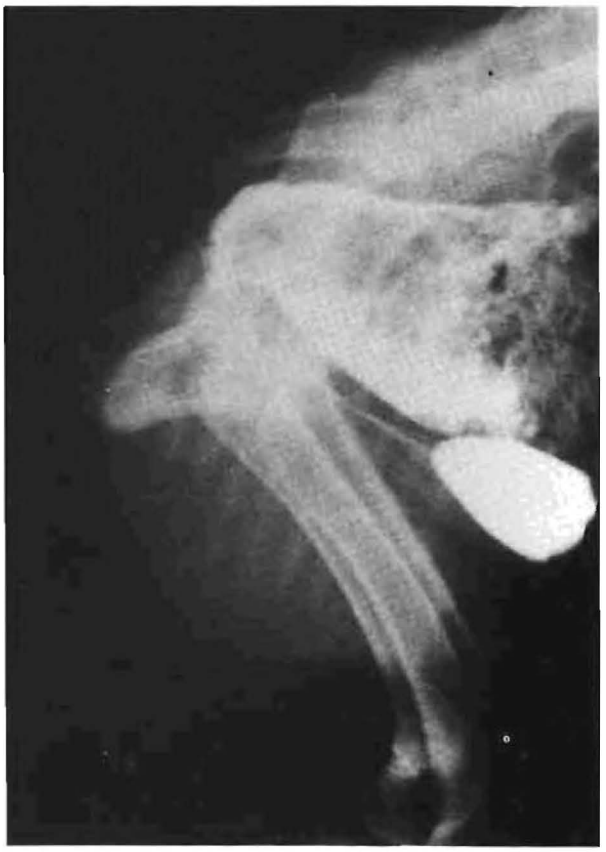

Fig. 2: Radiograph of the pelvic area after barium contrast medium had been instilled into the colon.

SURGICAL PROCEDURE

A midline incision was made along the perineum at the level of the absent anus. Blunt dissection through the subcutaneous tissues revealed the blind-ending tip of the colon descendens lying deep intrapelvically. The tip was grasped with tissue forceps and drawn caudally, whilst being freed from its pelvic attachments by blunt dissection. When once it lay freely within the pelvis, it was sutured to the skin incision by a few stay stitches. The tip was then incised and the exposed wall of the colon was firmly sutured to the skin incision. The internal anal sphincter and perineal muscles were present and well developed, but there was no evidence of an external anal sphincter (Fig. 4). The rectovaginal fistula was closed by numerous pursestring sutures along its length.

The bitch recovered well, the only complication being scar tissue formation around the newly created anus. To prevent contraction of this tissue and stenosis, the anus was stretched daily with a gloved, lubricated forefinger. This was done for several weeks until it was evident that this complication had been overcome.

DISCUSSION

The dog was saved from euthanasia by this surgical procedure, and she now leads a normal life. Her diet comprises softened commercially available dog cubes and no bones. She passes stools normally, 2 or 3 times daily, when once there is a large volume of faeces in the colon. Due to the absence of a functional external anal sphincter, she is unable to terminate the passage of stool easily. The problem is kept at bay by the fact that the faeces remain loose.

At the age of one year, the bitch remained far smaller in stature than the average Dobermann of her age, but came into oestrus normally. Due to the fact that a small amount of vaginal epithelium was included in the floor of the reconstructed anus, this area became reddened and oedematous, as well as the vulva. The dog then underwent an ovariohysterectomy and and the swollen area of the anus returned to its former appearance after a couple of weeks.

Johnson, et. al.[3] described surgical repair of a similar condition in a goat and commented that this condition is thought to have a genetic basis. Three foetal developmental factors that are listed as causes of this are failure of the anal membrane to become perforated, failure of the colon to become canalized, and an interruption of the foetal blood supply to the perineum.

ACNOWLEDGEMENTS

We wish to thank Mrs S.E. van der Hoven of the Faculty of Veterinary Science, University of Pretoria, for preparing the photographs.

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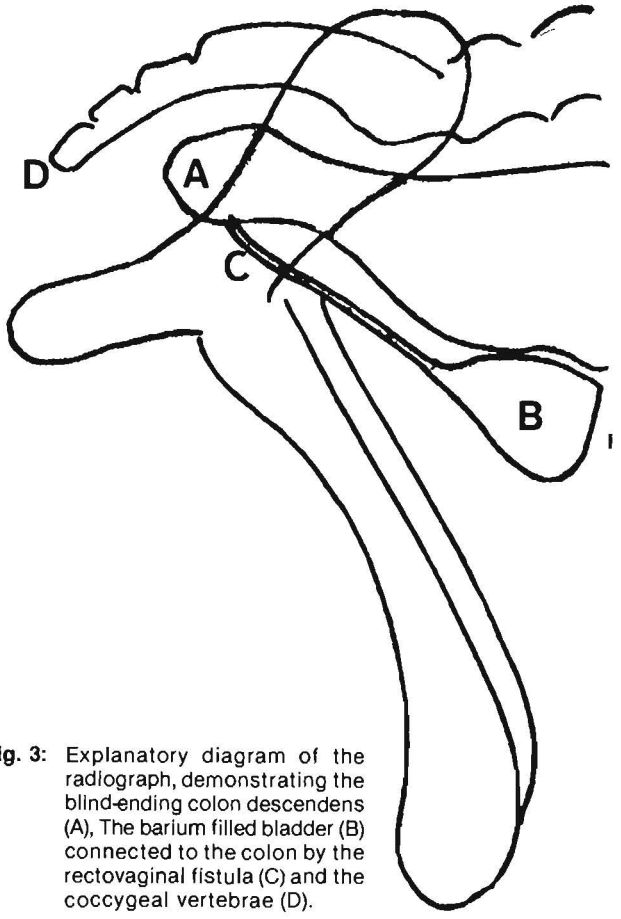
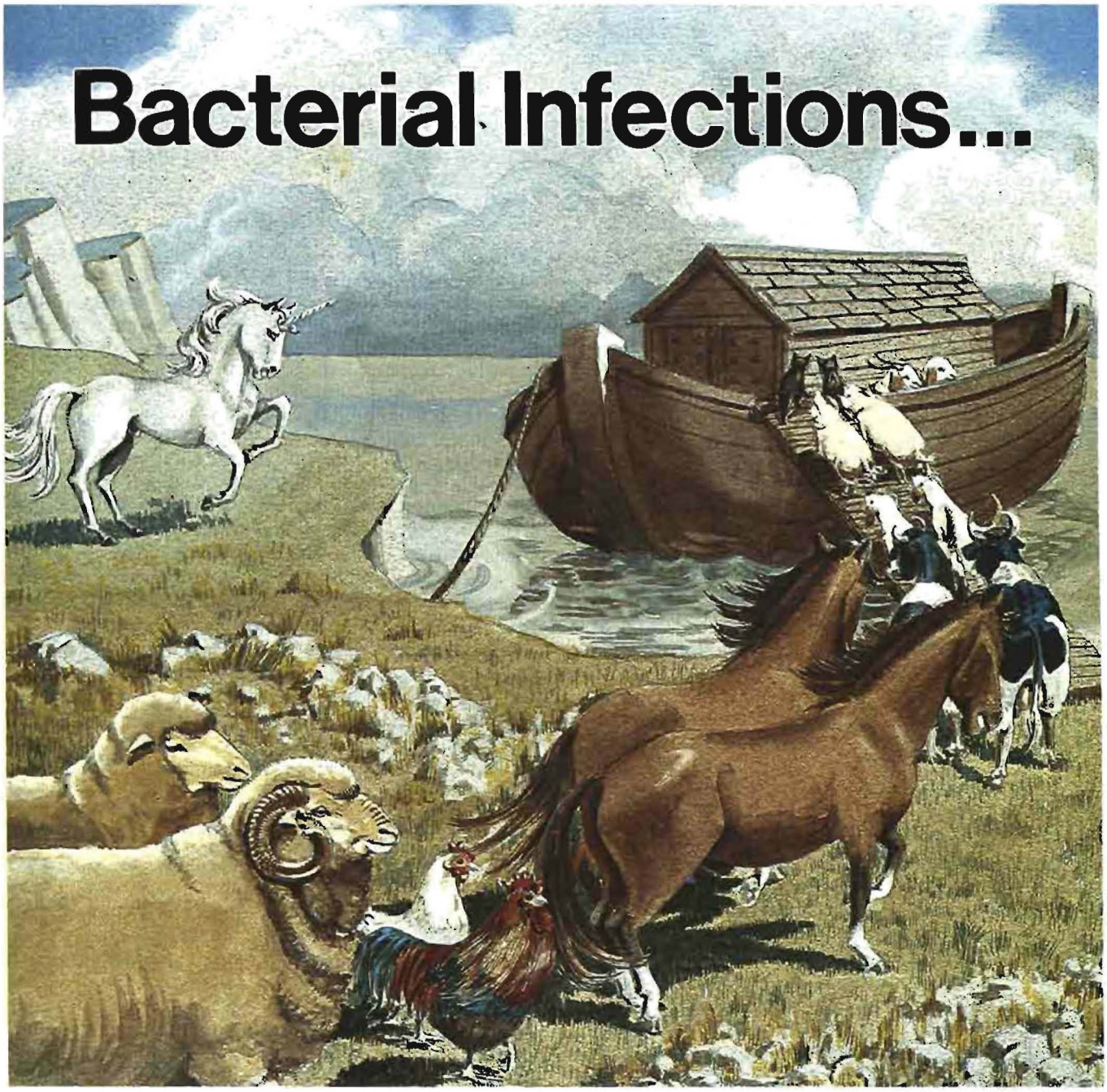


Fig. 3: Explanatory diagram of the radiograph, demonstrating the blind-ending colon descendens (A), The barium filled bladder (B) connected to the colon by the rectovaginal fistula (C) and the coccygeal vertebrae (D).

Fig. 4: The perineal area of the bitch post-operatively, showing the vulva (arrowed V) and the anus (arrowed A).



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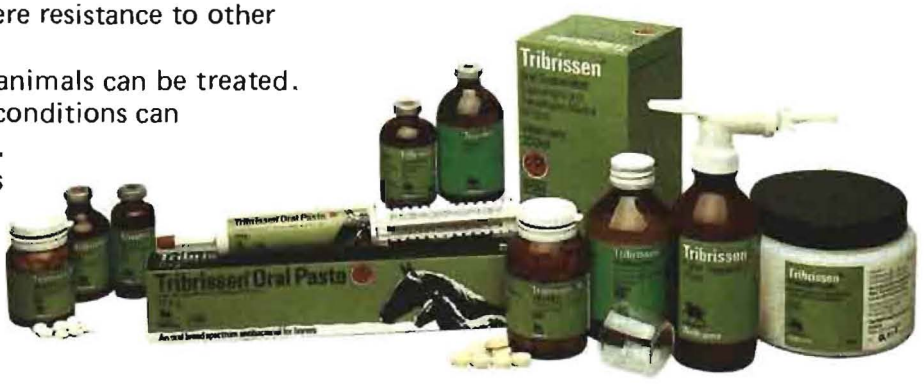

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CASE REPORT **GEVALVERSLAG**

MALIGNANT OEDEMA CAUSED BY *CLOSTRIDIUM PERFRINGENS* TYPE A IN A HORSE

R.F. HORNER*

ABSTRACT: Horner R.F. **Malignant oedema caused by** *Clostridium perfringens* type A **in a horse.** *Journal of the South African Veterinary Association (1982)* **53** No. 2, 122-123 (En) 52 Durban Road, 3201 Pietermaritzburg.

The clinical progression of a case of acute hind leg lameness in a horse resulting from infection with *Clostridium perfringens* type A is reported. The lesion produced involved a large localized area of muscular tissue with gas formation, purulent exudation and tissue necrosis. The successful response to medical and surgical treatment is outlined.

Key words: Malignant oedema, *Clostridium perfringens* type A, horse.

INTRODUCTION

Malignant oedema is defined by Blood & Henderson[2] as an acute wound infection and subsequent toxaemia caused by organisms of the genus *Clostridia*. According to Smith et al.[7] it is found to occur most frequently in cattle, sheep and horses. Organisms which have been isolated from lesions typical of malignant oedema of animals include *Clostridium septicum, Cl. chauvoei, Cl. perfringens, Cl. sordelli* and *Cl. novyi.*

Niilo[5] states that in Canada only 2 clearly defined acute diseases appear to be caused by *Clostridium perfringens* namely haemorrhagic enteritis of the newborn calf caused by type C and enterotoxaemia of sheep caused by type D. Dickie et al.[3] have recorded enterotoxaemia in foals caused by type C.

Clostridium perfringens type A may play a role as a secondary pathological agent in various disease conditions[5]. It is associated with gas gangrene the equivalent of malignant oedema in human medicine, infections of traumatic injuries of animals and also with some food poisoning outbreaks in man.

The alpha-toxin produced by *Cl. perfringens* type A results in either haemolysis or necrosis of cells depending on the tissues accessible to the toxin[5]. Niilo & Avery[6] and Sterne & Batty[8] have reported type A as causing enterotoxaemia in sheep and calves and in Sweden Wierup[10] has associated type A with an acute enteric disease of horses.

Cl. perfringens type A is an ubiquitous organism and frequent resident of the alimentary canal being isolated by Ackermann & Kleine[1] from rectal faecal samples in 20% of horses examined in 3 stables in Germany.

CASE HISTORY

A 3-year old Thoroughbred stallion in training was found to be lame in the right hind leg immediately after rolling in the stable yard. The animal had behaved normally at early morning training. A hot, focal area of swelling 50 mm in diameter was observed by the trainer on the proximal caudo-lateral aspect of the biceps femoris-semitendinosus muscle groups of the right hind leg. The area was painful on palpation and a spirit based liniment was applied.

*52 Durban Road, Pietermaritzburg.

Clinical examination was undertaken later that day (Day 1). The horse had an anxious demeanour with severe lameness and reluctance to bear weight on the right hind leg. The local swelling had subsided but extreme tenderness was apparent on attempted palpation of the right hindquarter. Contraction of the musculature in this area occurred in anticipation of palpation. Slight oedema of the leg proximal to the hock was evident. No sign of any wound or bite was observed and no sweating or muscle tremor noted. Attempts to take the animals rectal temperature were resisted.

The diagnosis was of acute myositis and treatment was isopyrin and phebuzine (Tomanol, Byk Gulden) intravenously and prednisolone (Deltacortril, Pfizer) intramuscularly.

During the next two days the horse was disinclined to move and was stabled. Appetite, fluid intake and habitus were good. Treatment during this time was isopyrin, phenylbutazone and dexamethazone (Dexa Tomanol, Byk Gulden) intravenously, vitamin E and selenium (Injacom E Selenium, Roche) intramuscularly and phenylbutazone (Equipalazone, Arnolds) per os.

By Day 7 the biceps femoris and gluteal muscle areas were noticeably swollen. The hair coat over the affected area of 200 mm in diameter appeared slightly darker than the surrounding coat. On sterile needle puncture of this swollen area there was a release of gas plus a very slight exudation of a sero-sanguineous fluid. By this time oedema was pronounced over the entire length of the leg and the animals appetite was declining. Treatment instituted was acepromazine (Acetylpromazine, Milvet) and Xylazine (Rompun, Bayer) given intravenously as tranquilization plus procaine penicillin and dehydrostreptomycin (Streptopenicillin Vet, Novo) intramuscularly to be repeated daily.

On Day 9, 3 focal areas of skin necrosis each 20 mm in diameter were observed in the centre of the darker hair coat area. Under tranquilization and restraint the three necrotic areas were surgically incised. A drainage hole was opened surgically 200 mm distally in the caudal aspect of the skin and sub cutaneous tissue between the semimembranosus and semitendinosus muscles. Swabs were taken of the exudate for bacterial culture.

A large quantity of purulent material was drained from the lesion over the ensuing few days. The darkened area of hair enlarged to 30 mm in diameter and the skin became necrotic. The affected area was depressed inwardly with its perimeter forming a ridge. Three pieces

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of grey fibrous and necrotic tissue 40 mm long x 10 mm thick were removed from between adjacent muscle groups. Leg oedema remained pronounced.

Topical treatment consisted of thorough irrigation of the lesion with centrimide and chlorhexidine gluconate (Savlon, ICI), a desloughing agent consisting of trypsin, balsum peru and castor oil (Velzyme, Centaur) and tail bandaging.

On Day 10 the ventral abdominal and scrotal area were swollen and oedematous. Necrotic skin over the wound was debrided back to the perimeter ridge creating an open wound 300 mm in diameter. The horse was now bearing some weight on the leg. Daily treatment was ampicillin sodium (Penbritin, Beecham) intravenously with topical treatment of the wound.

The scrotal and ventral abdominal swelling subsided within 36 hours. Healthy granulation tissue subsequently became evident over the wound.

By Day 16 the horse was much improved with a good appetite and was willing to use the leg normally. All oedema of the leg had subsided and there was no resentment to cleansing or palpation of the affected area. After 6 weeks the horse was back in training.

BACTERIOLOGY

Microscopic examination of stained smears of tissue exudate revealed gram positive bacilli. Bacterial culture produced a light pure growth of *Cl. perfringens*. Bacterial typing identified the organism as *Cl. perfringens* type A. The organism was sensitive in vitro to penicillin, erythromycin, tetracycline and clindamycin.

DISCUSSION

Although clostridial infections are generally found to result from contamination of wounds, in this case no external entry point for the organism could be detected. Over-vigorous rolling immediately before the onset of lameness could have resulted in muscle bruising and haematoma formation if contact was made with a stone or similar projecting hard object. This could have allowed a suitable environment for the lodging and multiplication of the organism. Infarction resulting from thrombosis of a blood vessel supplying an area of muscle may also have provided a suitable site for infection.

The sudden severe lameness and muscle pain may have lead one to suspect large scale muscle damage as might occur with azoturia. However, the horse worked normally during the early morning training session and remained so until rolling.

The initial local swelling may have been a superficial haematoma overlying the muscle or a possible tissue reaction to the presence of the organism. The time interval from the initial onset of infection until gas formation and exudation were observed was 6 to 8 days. A period of 5 days was recorded from infection until exudation in a similar equine case involving *Cl. septicum* and *Cl. chauvoei*.[9]

Initial treatment was empirically directed at the reduction of inflammation and pain and although later antibiotic treatment may have limited the spread of infection, the surgical opening and drainage of the affected area appeared to make the most significant contribution to recovery of the animal.

The most common of all the *Cl. perfringens* types and the most variable in toxigenic properties is type A. However in the horse it appears only to have been associated with an acute enteric disease in Sweden[10].

To my knowledge there is no reported cases of *Cl. perfringens* type A causing malignant oedema in the horse. Fatal malignant oedema in a horse caused by *Cl. chauvoei* in which there was no evidence of external wounds or trauma has been reported by Murphy[4]. Westman et al.[9] recorded another case in a horse caused by a mixed infection of *Cl. septicum* and *Cl. chauvoei* which responded to therapy.

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CASE REPORT GEVALVERSLAG

DIE GEBRUIK VAN AMIKASIEN IN DIE BEHANDELING VAN *PSEUDOMONAS AERUGINOSA* VEROORSAAKTE ENDOMETRITIS IN DIE MERRIE

ENETTE VAN DYK, A. IMMELMAN en J.S. VAN HEERDEN*

ABSTRACT: Van Dyk E.; Immelman A.; and Van Heerden J.S. **The use of amikacin in the treatment of endometritis caused by *Pseudomonas aeruginosa* in the mare.** *Journal of the South African Veterinary Association* (1982) **53** No. 2 124-126 (Afrik) Department of Genesiology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

After isolation of *Pseudomonas aeruginosa* from endometrial biopsies of 6 mares they were treated with amikacin sulphate. Three were treated by intra-uterine application of the drug, in one the drug was given by intramuscular injection, in another the intravenous route was used while in the last mare simultaneous local and intravenous treatment was applied. An intra-uterine Tris-EDTA instillation preceeded the uterine amikacin instillations to aid in the breakdown of the capsule around the bacterium. Serum concentrations of amikacin were determined after intravenous and intramuscular administration. The highest concentration of 15 µg/ml was reached 1 hour after intravenous injection. After 12 hours the blood concentration was below the therapeutic level. To maintain effective levels injection must be repeated 6-8 hourly. All mares treated by the intra-uterine or intravenous routes recovered. The importance of obtaining cultures from uterine biopsies is stressed as *Pseudomonas* was cultured only once from the conventional cervical swab while the other 5 mares had negative cervical swabs.

INLEIDING

Intra-uteriene besmetting met *Pseudomonas aeruginosa* kom voor in merries met 'n verlaagde endometriale weerstand óf tydens dekking óf as gevolg van aerovagina of beide. Indien dit kronies word is die besmetting moeilik om te genees en gee dit aanleiding tot onvrugbaarheid.

Vorige antibiotiese behandelings en ouderdom speel 'n rol in die vatbaarheid van die merries vir die organisme[6]. Die algemene gebruik van antibiotika in die uterus laat die relatiewe nie-patogene *P. aeruginosa* toe om te vermeerder en die rol van 'n patogeen te speel sodra sy opponente verdwyn of in aantal verminder[9].

Amikasiensulfaat (Amikin-Bristol Lab.) is 'n semisintetiese aminoglikosied antibiotikum wat aktief is teen 'n breë spektrum van Gram negatiewe organismes, insluitende *Pseudomonas*, en sommige Gram positiewe organismes.

Op grond hiervan is die gebruik en effektiwiteit van hierdie middel in die behandeling van *Pseudomonas* veroorsaakte endometritis in merries ondersoek.

PROSEDURE EN RESULTATE

P. aeruginosa besmetting is in 6 merries gediagnoseer met behulp van uterus biopsies en deppers. 'n Deeglike ondersoek van die geslagstelsel word gedoen deur vaginale ondersoek, sorgvuldige palpering van die buitelyne en deursnit van die uterus. 'n Weefsel monster word geneem met 'n Hauptner instrument wat 'n monster van 5×8×3 mm kan neem.

Die merrie word voorberei vir die biopsie deur die met gaas verbinde stert uit die pad te hou en die perineum deeglik met seep te was en daarna af te spoel met skoon warm kraanwater. Die punt van die steriele instrument word in die hand geneem wat met 'n steriele handskoen bedek is en met die indeks vinger deur die cervix begelei tot in die uterus. Sodra die instrument in die uterus is word die hand onttrek en in die rektum geplaas waar dit dan op die verlangde plek fikseer word en die biopsie dan geneem word. Primêre kulture is gemaak op bloed triptose agar (Difco, Michigan, V.S.A.) met 5 % beesbloed. Deppers en biopsie materiaal was direk op die agar gesmeer, daarna is kulture aerobies by 37 °C geïnkubeer. Na 24 h was daar 'n oormatige groei wat geïdentifiseer is as *P. aeruginosa* in ooreenstemming met standaard tegnieke[3]. Antibiogramme is hierna van die primêre kulture gemaak.

Merries no. 1, 2 en 3 is slegs intra-uterien behandel. Tris-EDTA is opgemaak deur 1,2 g EDTA, en 6,05 g Tris in 1 ℓ gedistilleerde water op te los. Die pH is op 8 gebring deur die byvoeging van ys asynsuur. Die oplossing is daarna gesteriliseer deur dit te autoklaaf. Ongeveer 250 ml Tris-EDTA word in die uterus van die merrie geplaas en 4 h later opgevolg met 500 mg amikasiensulfaat opgelos in 100 ml steriele water. Die behandeling is 3 maal toegepas met 48 h tussenposes. Stilboestrol dipropionaat (Maybaker) is tydens elke behandeling teen 'n dosis van 20 mg intramuskulêr toegedien.

Merrie no. 4 is behandel deur amikasien sulfaat teen 'n dosis van 6,5 mg/kg twaalf-uurliks vir 4 dae intraveneus toe te dien. In die geval van Merrie no. 5 is dieselfde dosis met dieselfde tussenpose vir 4 dae intramuskulêr toegedien.

Van hierdie 2 merries is 10 ml veneuse bloed in heparien versamel vir die bepaling van plasma konsentrasies van amikasien. In Merrie no. 4 is bloedmonsters gekollekteer 1, 6 en 12 h na toediening en in Merrie no. 5 is bloedmonsters 2, 6 en 12 h na elke toediening van amikasien gekollekteer. Plasma konsentrasies van amikasien sulfaat is bepaal deur middel van 'n mikrobiologiese toets met *Bacillus subtilis* (AT CC 6633) as toets organisme.

In Merrie no. 6 is die intrauteriene behandeling soos hierbo geskryf 3 maal toegepas met 48 h tussenposes en terselfdertyd is intraveneuse behandeling 12 uurliks teen 'n dosis van 6,5 mg/kg toegedien. Geen bloedmonsters

*Fakulteit Veeartsenykunde, Universiteit van Pretoria, Posbus 12580, 0110 Pretoria.

is in hierdie geval gekollekteer vir die bepaling van die amikasien konsentrasies nie. Die geskiedenis, kliniese bevindings en reaksie op behandeling word saamgevat in Tabel 1.

BESPREKING

Dit is reeds aangetoon dat 'n biopsie slegs 0,2 % van die hele endometrium verteenwoordig en tog verteenwoordigend van die hele oppervlakte kan wees[7]. Die belang van uterus biopsies as diagnostiese prosedure in teenstelling met servikale deppers spreek duidelik uit die verskillende gevalle. Slegs in die geval van Merrie no. 1 was die deppers ook positief vir *P. aeruginosa*. Die ander deppers was almal negatief, terwyl die uterus biopsies positief was.

Amikasien word geklassifiseer onder die aminoglikosied antibiotikas. Die aminoglikosiedes word hoofsaaklik onveranderd deur die niere uitgeskei, gewoonlik deur glomerulêre filtrasie. Hierdie groep is tot 50 maal meer bakteriododend onder alkaliese toestande[8].

Die skadelike reaksies van die aminoglikosied groep kan in drie groepe opgesom word, naamlik ototoksisiteit, nefrotoksisiteit en neuromuskulêre blokkade. Dit is belangrik om daarop te let dat in perde die gebruik van Furosemide (Lasix-Hoechst) die ototoksisiteit van die groep middels wel verhoog[1 2]. In die geval is die toestand stabiel of progressief nadat behandeling gestaak is. Nefrotoksisiteit wat deur die aminoglikosied antibiotika veroorsaak word kom voor as 'n akute nekrose van die proksimale tubulêre selle van die nier. Dit is

TABEL 1: GESKIEDENIS EN KLINIESE BEVINDINGS VAN MERRIES MET INTRA-UTERIENE *P. AERUGINOSA* BESMETTING NA BEHANDELING MET AMIKASIEN.

Merrie no Ras Ouderdom	Vuldatum	Vorige besmetting	Vorige behandeling	Depper/ Biopsie t.o.v. Pseudomonas	Antibiogram	Histopatologie	Behandeling	Biopsie na 4 dae/ 2 weke	Verdere Teelgeskiedenis
No 1 Nooitgedacht 10 jr	Nooit	Vorige teelseisoen herhaaldelik gedek		+ +	4+ Amikasien	Effense hiperemie; kliere onaktief. Kompakte kerne.	Lokaal (intrauterien) Tris + Amikasien.	– –	Volgende oestrus siklus gedek. Dragtig. Vul In Okt. '80
No 2 Volbloed 10 jr	4 vullens 1977	Staph aureus	Ampicillien (Penbritin V-Beecham) Caslick's.	– +	4+ Amikasien	Geen akute of kroniese inflammasie. Geen fibrose. Effense edeem.	Lokaal (intrauterien). Tris + Amikasien.	– –	Nie beset. Geen bakteriese groei word gevind.
No 3 Volbloed 12 jr	1977 (distokie)	Serviks groot hoeveelheid fibrose. Herhaaldelike dekking.		– +	4+ Amikasien	Dieper gedeeltes van sommige kliere toon nekrobiotiese veranderinge; kliere is met debris gevul. Effense subepitheliale limfosiet infiltrasie. Hiperemie. Klier epiteel in aktiewe stadium.	Lokaal (intrauterien) Tris + Amikasien.	– –	Merrie is dragtig. Vul in Okt. '81.
No 4 Volbloed 9 jr	1 vul. 1977	Klepsiella β-haemolitiese Strep	Tetrasikliene (Polyotic V-SA Cyanimid) Ampicillien Eritromisien (Erythrocin-Abbott).	– +	4+ Amikasien	Kliere onaktief met geko-aguleerde afskeiding in sommige. Heelwat fibrose in dieper dele van endometrium. Oppervlakkige dele wys edeem en hiperemie.	6,5 mg/kg intraveneus 12-uurliks vir 4 dae (Plasma konsentrasie – sien Fig. 1)	– –	Onbekend
No 5 Volbloed 10 jr	4 vullens 1977	Streptococcus Staphylococcus	Chlooramfenikol (Chloramphenicol Panvet). Ampicillien gentamisien (Garamycin-Scherag).	– +	4+ Amikasien	Lae graad edeem. Geen fibrose. Geen inflammasie.	6,5 mg/kg intramuskulêr 12-uurliks vir 4 dae (Plasma konsentrasie – sien Fig. 1).	+	Is later suksesvol lokaal (intrauterien) behandel. Onbekend.
No 6 Volbloed 6 jr	Van baan af; enkele dobie klein vul gehad.	Pseudomonas.	Polimiksien (polymixin B Novo). Kanamisien (Kantrex-Bristol) metronidazole (Flagyl-Maybaker).	+ +	4+ Amikasien 4+ Sulfa.	Weefsel is oedemateus, kliere lyk aktief, party bevat neutrofiele. Kliere is verleng en effens vergroot. Effense hiperemie.	Trimethoprim Sulphdoxine (Trivetrin-Wellcome) 15 mg/kg i/v daagliks vir 5 dae, 250 ml Tris/EDTA i/u, 4 uur later 5 g Trimethoprim + sulfadiazine (Tribrissin-Wellcome) i/u in 100 ml H_2O i/u. Hierna lokale Tris + Amikasien behandeling en Amikasien i/u.		Merrie steeds nie dragtig maar geen verdere bakteriese groei is verkry nie.

omkeerbaar as die middel gestaak word met die eerste tekens van 'n renale wanfunksie[1,2].

Alle aminoglikosied antibiotikas het die vermoë om 'n neuromuskulêre blokkade te veroorsaak maar gewoonlik net onder spesiale omstandighede. In post-chirurgiese gevalle waar neuromuskulêre blokkerende middels wel gebruik is, óf waar aminoglikosiedes direk in die peritoneale holte toegedien is, het dit gelei tot 'n apnea. Dit kan moontlik toegeskryf word aan die ongewoon hoë konsentrasies van die middels, wat direk toegang tot die neuromuskulêre aansluitings in die diafragma het en 'n respiratoriese versaking veroorsaak. Die vermoë om 'n neuromuskulêre blokkade te veroorsaak van die verskillende aminoglikosiedes is varieërend en kan volledig of gedeeltelik herstel word deur die intraveneuse toediening van kalsium soute, maar die effek van cholinomimetiese middels soos neostigmine en edrophonium varieer[8]. Die spesifieke farmakologiese werking van amikasien in die mens is wel bekend maar huidig is dit nog nie in die perd opgeklaar nie.

Amikasien het aktiwiteit teen kliniese isolate van Gram negatiewe bakterieë insluitende *P. aeruginosa* en 'n wye reeks van Enterobacteriaceae. *Pseudomonas* infeksies in die mens word teen 'n dosis van 7,5 mg/kg wat elke 8 h herhaal word behandel. Dit is bekend dat amikasien vinnig geabsorbeer word na intramuskulêre inspuiting. Piek serum peile van ongeveer 11 μg/ml word bereik nadat 250 mg intra-muskulêr toegedien is en na intra-muskulêre dosis van 500 mg is die piek serum peil 20 μg/ml.

In die perd was die middel irriterend na intramuskulêre toediening en het dit 'n lokale reaksie, 'n swelsel, veroorsaak. Die plasma konsentrasies wat bereik is was nooit hoër as 6,5 μg/ml nie. Volgens Tobin[8] versprei aminoglikosiedes nie goed deur die liggaam na intramuskulêre toediening nie, alhoewel dit vinnig opgeneem word.

Met intraveneuse behandeling is 15 μg/ml na 1 h gemeet maar na 12 h was dit onder terapeutiese vlak, dus om 'n betekenisvolle plasma konsentrasie te behou moet die inspuiting 6-8-uurliks herhaal word in teenstelling met 12-uurliks soos dit wel toegedien is, alhoewel die enkele hoë piek direk na toediening blykbaar tog effektief was om die organisme uit te skakel.

In die geval van lokale behandeling is gepoog om die pH van die uterus te alkaliniseer deur die gebruik van die Tris-EDTA buffer, ten einde die groter doeltreffendheid van die antibiotikum by 'n hoër pH te benut.

Gram negatiewe bakterieë is beskerm teen die aksie van antibiotika as gevolg van die ondeurdringbaarheid van die lipopolisakkariedlaag van die selwand. Divalente katione soos Mg en Fe is nodig om die integriteit van die laag te behou. Antibiotiese weerstand kan egter afgebreek word deur die blootstelling aan EDTA wat skynbaar werk deur die ione te bind en sodoende te lei tot die disintegrasie van die polysakkaried laag. Vir dié rede moet daar dus ook 'n tydsverloop wees tussen die instillasie van Tris-EDTA en die daaropvolgende antibiotiese instillasie[9]. Die gebruik van stilboestrol dipropionaat was ten doel om die ontwikkeling van endometriale kliere en bloedvate in die endometrium te bevorder en daardeur groter blootstelling van bakterieë in die kripte te kry.

Lokale behandeling is aan te beveel omrede die sukses wat behaal is. Dit sal ook die mees ekonomiese behandeling wees. Indien parenterale behandeling nodig blyk te wees moet huidig aanbeveel word dat 6,5 mg/kg elke 3-6 h intraveneus toegedien word.

Bakteriese groei het na behandeling slegs in no. 5 voorgekom en dit moet daarop gelet word dat sy met daaropvolgende lokale behandeling negatiewe bakteriese groei getoon het. Slegs 2 merries, naamlik no. 1 en 3, het na die behandeling weer beset geraak. Hierdie 2 merries is nie van te vore behandel nie; die amikasien en Tris-EDTA was die enigste terapie na diagnose. Inteenstelling het die ander merries, wat herhaaldelike kursusse van verskillende antibiotikas ontvang het, nie weer beset geraak nie. Volgens die histologiese snitte is daar geen voor die handliggende rede waarom enige van die merries nie weer beset sou kon raak nie.

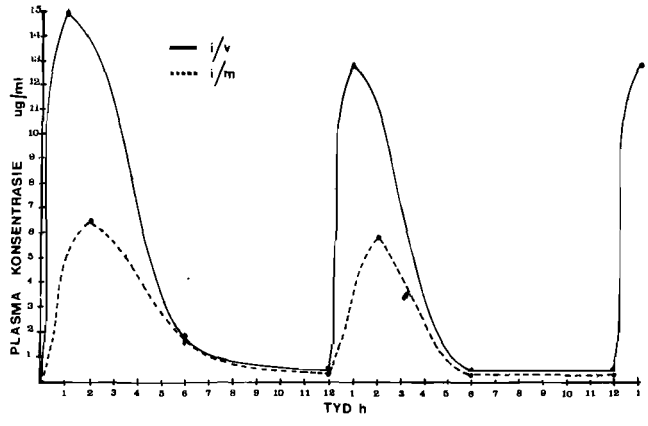

Fig. 1: Plasma konsentrasies van Amikasien na intramuskulêre en intraveneuse toediening.

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REVIEW OORSIG

IVERMECTIN AS AN ANTIPARASITIC AGENT IN HORSES*

J. SCHRÖDER AND G.E. SWAN**

ABSTRACT: Schröder J.; Swan G.E. **Ivermectin as an antiparasitic agent in horses.** *Journal of the South African Veterinary Association (1981)* **53** No. 2, 127-128 (En) MSD Research Centre, Private Bag 3, 1685 Halfway House, Republic of South Africa.

Ivermectin, described as 22,23-dihydroavermectin B_1, was the compound chosen from the avermectin group of compounds for development as an antiparasitic agent in horses. A review of the literature indicates that parenteral administration in horses at 200 μg/kg body mass is highly effective against the strongyles *Strongylus vulgaris, Strongylus edentatus* and *triodontophorus* spp., and adult and immature cyathostomes, including strains resistant to benzimidazole anthelmintics. Other nematodes controlled in horses include *Oxyuris equi, Parascaris equorum, Trichostrongylus axei,* and *Habronema* spp.

Ivermectin is also highly effective against stomach bots (*Gastrophilus* spp.).

INTRODUCTION

Anthelmintics are the most widely and frequently used drugs in horses[2]. The benzimidazole group constitutes the largest segment of currently used equine anthelmintics. Cambendazole (Equiben: MSD) and mebendazole (Telmin: Janssen) are registered for horses under Act 36/1947[13].

The emergence of strains of cyathostomes ("small strongyles") tolerant to benzimidazoles (including febantel[1]: Rintal, Bayer) may pose a threat to their efficacy[2 6]. When this occurs, alternation with anthelmintics of different chemical classes becomes necessary. Possible alternatives would include pyrantel pamoate (Nemex H: Pfizer), dichlorvos (Equigard: Shell) and trichlorphon (Neguvon P: Bayer).

Ivermectin has been called the most impressive anthelmintic of a new class to emerge in the past 19 years[2]. Its antiparasitic activity in horses and ponies has been described by various authors[2 3 5 7-12].

ANTIPARASITIC EFFICACY

Ivermectin was administered subcutaneously or intramuscularly to horses in several trials in the US[2 3 5 7-10] and shown to be effective at dosages of 100 to 200 μg/kg against bots and nematodes which parasitise the gastrointestinal tract. These parasites included *Gastrophilus intestinalis, Gastrophilus nasalis* (second and third instars), *Habronema muscae, Habronema majus, Draschia megastoma, Trichostrongylus axei, Oxyuris equi* (fourth larval stage and adults) and *Parascaris equorum*. Strongylinae, including *Strongylus spp., Triodontophorus spp.* and *Oesophagodontus robustus*, and cyathostominae were also removed at these dosages. In one trial[11], the efficacy of ivermectin was demonstrated against population-B benzimidazole tolerant cyathostomes (*Cyathostomum spp., Cylicostephanus spp* and *Cylicocyclus nassatus*) following oral administration.

In addition to the above parasites, *Onchocerca cervicalis* and *Setaria equina* are also removed by ivermectin[2 3 5 7 9 10]. Although Klei et al[10] and Klei & Torbert[8] describe activity against *Anoplocephala perfoliata*, this is refuted by Lyons et al[11] and by the knowledge that ivermectin is inactive against cestodes at the above mentioned dosages[4].

Slocombe and McCraw[12] in Canada injected ivermectin subcutaneously in ponies 7 days after infestation with *Strongylus vulgaris*. Dosages as low as 100 μg/kg resulted in disappearance of the clinical signs of infestation (increased body temperature, lethargy, depression, recumbency and colic) within 3 to 4 d. The treatment also produced a "radical cure", in that no lesions (arteritis and thrombosis) could be found in the cranial mesenteric artery and its major branches at slaughter 3 weeks later.

MODE OF ACTION

Ivermectin is a new and unique chemical compound. Its mode of action differs from those of organophosphates (blocking of cholinergic nerve transmission), pyrantel (depolarization or hyperpolarization of muscle cell membranes), levamisole (immobilization through nerve ganglion stimulation) and benzimidazoles (inhibition of the formation of microtubules).

Ivermectin immobilizes parasites by blocking the signal transmission from ventral cord interneurons to the excitatory motor neurons in those parasites which have gamma-amino-butyric acid (GABA) in their nervous systems. The discoordinated, immobilized parasite is expelled from its site of infestation[4].

SAFETY

Ivermectin has a wide safety margin in horses. Signs of toxicity (mydriasis only) can be expected to appear at dosages of 3 mg/kg, i.e. 15 times the recommended dosage. Fatalities appeared only at much higher dosages (12 mg/kg, 60 times recommended level).

At the recommended dosage, reactions at the injection site occurred in a small proportion of treated horses. These were soft tissue swellings and of a transitory nature.

*Presented at the Biennial Congress of the South African Veterinary Association, Cape Town, 7-11 September 1981.

**MSD Research Centre, Private Bag 3, 1685 Halfway House, Republic of South Africa.

Breeding stallions were injected intramuscularly at 600 μg/kg. No deleterious effect on semen characteristics, spermatozoal morphology, testicular measurements, concentrations of testosterone, or libido was seen. Until the results of current studies became available, pregnant mares must not be treated.

Ivermectin has been formulated for deep intramuscular injection and should not be given intravenously. Intravenous injections of polysorbate 80, a surfactant contained in the formulation, can cause allergic or anaphylactic reactions, which can be fatal, in hypersensitive horses (unpublished results, Merck Sharp and Dohme Research Laboratories, Rahway, New Jersey, USA).

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BOOK REVIEW **BOEKRESENSIE**

VETERINARY TOXICOLOGY

MYRA L. CLARKE, D.G. HARVEY and D.J. HUMPHREYS

2nd edition. Bailliére Tindall, London 1981 pp 328. Publ. Price R29,85

The first book with the title Veterinary Toxicology was written by G.D. Landers in 1912. This was followed by a book with the same title and written by R.F. Garner. The third edition was revised by E.G.C. Clarke and Myra Clarke. In 1975 the first edition of Veterinary Toxicology written by Clarke and Clarke appeared and in 1981 the second edition was published. Over all these years this publication has been a standard reference book in veterinary toxicology.

The information is grouped into 10 groups as it was done in the previous edition. The first part deals with the basic concepts of toxicology and the classification of poisons. The other groups are inorganic and mineral substances, toxic gases and vapours, organic compounds, poisonous plants, mycotoxins, venomous bites and stings and radio-active materials.

The information given in these different parts corresponds to a large extend with that of the previous edition. The format has been changed and the pages now have double columns.

Toxic plants of South Africa are mentioned, and the basic syndrome and lesions caused by them are discussed. As we may expect from a book dealing with this subject on a worldwide basis, fine details cannot be given. The information on organic and inorganic substances is applicable to us here in South Africa and is very useful information.

Mycotoxins are becoming more and more important as a cause of toxicity in our animals. The descriptions of the mycotoxicoses are very brief but serve as an introduction into a vast and rapidly developing field of study. Should one require more detail then books dealing specifically with them are available.

Five appendices have been added to this new edition. The first appendix lists the standard works which are referred to in this book. These references are very useful should one wish to read further in a specific field.

Appendix 2 is not even a full page in length and comprises a list of pesticides and herbicides that may affect honey bees. The third appendix is a list of similar compounds and their effect on fish.

The second last appendix is a list of plants associated with teratogenic effects, infertility and abortions while the last appendix is a short note on carcinogenesis.

Veterinary Toxicology will remain a standard reference book for the veterinarian, veterinary student and toxicologist. As the changes in the new edition are slight, the previous edition on the bookshelf need not be replaced.

A. Immelman

TO THE EDITOR **AAN DIE REDAKSIE**

ACTINOBACILLUS SEMINIS INFECTION IN A WALRICH RAM

Actinobacillus seminis is a well known cause of clinical and subclinical forms of epididymitis in rams. As this organism reveals a high susceptibility to tetracyclines in vitro, it was argued that this antibiotic could be the logical treatment for rams suffering from subclinical epididymitis due to *A. seminis*.

During an examination for fertility in a flock of rams in the Tweeling area, a Walrich ram of about 18 months of age was found to be excreting large numbers of neutrophils in its semen. The 2 significant features at this stage were that the animal was clinially sound and that there was an absence of pathogenic bacteria in its semen. A subsequent semen sample was examined after the animal had received a course of 5 intramuscular injections of long-acting oxytetracycline (Terramycin LA Pfizer) at 3 day intervals and at a dosage rate of 1 ml/10 kg body mass. This semen sample also contained large numbers of neutrophils, but in addition, the presence of *A. seminis* could be demonstrated bacteriologically.

A second series of Terramycin LA injections at the same dosage levels and time intervals as previously was administered but the subsequent semen picture remained as before. At this stage the ram was euthanased for post mortem examination purposes. Samples for histological examination of the vesiculae seminales and the tails of the epididymi were taken in 10 % formalin.

The vesiculae seminales revealed no pathological changes. In some sections from one of the epididymi, some tubuli were packed with neutrophils while others contained necrotic cellular masses. Spermatozoa were absent in all the tubules examined. Chronic fibrotic changes with lymphocytic and plasma cell infiltrations in the interstitial tissues were also present. These changes indicate an acute flare up of chronic lesions. It is evident that the acute inflammatory process had persisted and *A. seminis* organism continued to be excreted in the semen despite the antibiotic treatment. It appears therefore that the dosage rate of Terramycin LA for the treatment of this type of lesion should be increased to levels more than 1 ml/10 kg body mass and that the poor response to treatment could well be the result of a poor transfer of tetracyclines from the circulation to the testes and epididymi. Before an effective dosage rate of tetracyclines for the treatment of subclinical epididymitis caused by *A. seminis* can be suggested, the threshold value for the transfer of tetracyclines to the relevant organs should be investigated.

J.A. Erasmus
Veterinary Laboratory
P.O. Box 625
Kroonstad
9500

J.A.L. de Wet
Veterinary Laboratory
Private Bag X502
Bloemfontein
9300

L. Prozesky
Veterinary Research
Institute
Onderstepoort
0110

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BOOK REVIEW **BOEKRESENSIE**

POISONOUS PLANTS OF SOUTHERN AFRICA

J. VAHRMEIJER

1st Edn. Tafelberg Publishers Limited, 28 Wale Street, Cape Town, 8000. 1981 pp 168, 120 colour plates and distribution maps. Price R20,00 excluding GST. (ISBN 0-624-01459-2)

Available in both English and Afrikaans (under the title GIFPLANTE VAN SUIDER-AFRIKA) this unique publication subserves a long-felt need. The first 4 chapters of the text comprise a brief discussion of the scope, predisposing factors, prevention and types of plant poisoning in Southern Africa. In a book with an essentially botanical bias, these serve as an adequate introduction to the problem. The major portion of the text consists of a description of 67 of the most important poisonous plants. Each description is complemented by distribution maps and colour plates which can only be described as superb. An error (mismatching of the plant descriptions and colour plates on pp 76, 77 and 80, 81) has marred an otherwise laudable exercise. The concluding chapters contain information on identification of gifblaar and gousiektebossie and despatching of plant material for identification.

Overall this book is a valuable addition to the veterinary literature and is recommended especially for the veterinary student and large animal practitioner.

J.W. Nesbit

TO THE EDITOR — AAN DIE REDAKSIE

WHEN IS A MURMUR NOT A MURMUR?

We wish to draw the attention of colleagues to the difficult, occasionally embarrassing, situations which may arise from the inappropriate use of the word "murmur" when giving opinions on the heart sounds of horses for the benefit of owners, trainers or other connections.

By definition the word "murmur" means a normal or abnormal heart sound produced by turbulence[2 6 9]. Murmurs are classified by human and animal cardiologists as innocent, physiologic or pathologic[2 9]. The intensity of the murmur is graded I to V according to the ease with which it is heard on auscultation with a stethoscope[5]. In horses, such usage might be acceptable were it not for the fact that the word "murmur" tends to raise doubts about a horse's soundness, even when it is qualified by "innocent" or "physiological" or "ejection".

In man, "It is of great importance to recognize and remember that most systolic murmurs do not indicate the presence of any structural or organic heart disease[9]". The same is true of horses. Many veterinarians have reported that low-grade early systolic murmurs can be auscultated in up to 66 % of clinically normal horses[3-8]. This is a blowing sound of Grade I to Grade III intensity, occupying up to three-quarters of the systolic interval[5]. With heart surface or intracardiac phonocardiography it has been demonstrated in all horses examined to date[1 8].

We are thus faced with the situation that a blowing aortic or pulmonary systolic murmur of Grade I to Grade III intensity *is a normal heart sound in the horse.* In our opinion, this sound should not be designated a murmur *unless there is irrefutable evidence of heart disease.*

What then should we call this sound which is present in the majority of horses if we listen hard enough? For some years we have used the term "systolic flow sound" to describe it, qualified by an assessment of Grade I to Grade III intensity. We consider that the use of the above term to denote blowing early systolic "murmurs" of Grade I to Grade III intensity in horses prevents doubts about soundness and/or depreciation of value arising from the use of the word "murmur".

It follows from our argument that the phrase "normal heart sounds" in a certificate for a horse with no signs of heart disease includes the presence of a blowing early systolic sound of low to medium intensity.

A. Littlejohn and C. Button
Department of Physiology, Pharmacology and Toxicology
Faculty of Veterinary Science
University of Pretoria

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MULTIPLE MYELOMA

The author of a recent paper[1] in your journal indicated that "multiple myeloma" originated "from the myeloblasts or myelocytes of the bone marrow." This is incorrect. In fact, multiple myeloma is a neoplasm of plasma cells[2] which are themselves derived from lymphocytes rather than from myeloid cells (myeloblasts, myelocytes and their progeny).

A.D.J. Watson
Department of Veterinary Clinical Studies
The University of Sydney, NSW
2006, Australia

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TO THE EDITOR — AAN DIE REDAKSIE

A TRIBUTE TO THE SA VETERINARY FOUNDATION'S CONTRIBUTION TO THE HERD/FLOCK APPROACH BY RURAL PRACTITIONERS

The Rural Practitioners' Group is deeply indebted to the South African Veterinary Foundation for the financial assistance, encouragement and stimulation that has been rendered to the Group over the past 18 months. Without this assistance the rural practitioner's approach and his involvement in the livestock production-industry would probably have remained static.

With this assistance, however, the rural veterinarian has now adopted an outgoing and production-orientated approach and rural practice has undergone some extremely positive changes.

One of the ongoing results of the various meetings that have been held within the profession is the fact that we have realized that we will continually have to assess the development of large animal practice over the medium term and to prepare the veterinary profession for changes in the agricultural industry for our mutual benefit.

On the 10 May 1979 an initial meeting was held in Pretoria under the chairmanship of Dr R. Coubrough. There was grave concern about the declining involvement of the rural veterinarian within the livestock industry. At this stage the Veterinary Foundation realized that the rural practitioner's future was not very bright and took the lead by instigating numerous meetings to launch an in-depth examination of rural practice in the Republic. The meetings brought many problems to the fore and helped to define the direction of future meetings.

On 21 February 1980 a further "brain storming" session was held under the leadership of Messrs M. Scott and P. Miller which highlighted many of the problems as seen by veterinarians, but fortunately also emphasised many avenues of increasing the veterinarian's involvement in the livestock industry. As a result, regional meetings of rural practitioners took place during which many positive avenues of involvement within the livestock industry emerged together with the awakening of a positive identity.

The overall impression was that veterinarians have a very positive role to play in the livestock industry and that there is an under-utilization of veterinary manpower within this industry in the Republic.

Conclusions and recommendations formulated were:

1. It is generally accepted that the future lies in giving an economically viable production-orientated service to the livestock industry by adopting a herd/flock approach which has measurable results. We believe that it is within the scope of the practising veterinarian to adapt his knowledge and to make a success of this approach.

1.1 A continuing system of refresher or retraining courses is of paramount importance and should be made available to practitioners.

1.2 Of necessity, attention should be given to under- and post-graduate training, both of content and of elective choice.

2. Inter-disciplinary contact within the livestock production industry on national, regional and local levels. A herd/flock health approach depends on many factors such as management, nutrition, health and disease prevention, breeding, genetics, etc., of which we are a very important and indispensible link, and communication with other allied links is of great importance.

3. To counteract the under-utilization of veterinary manpower within the farming community, it is essential that methods be devised whereby our production-orientated herd/flock approach can be propagated. Lines of communication must be opened with the following instances:

3.1 Organised Agriculture
   (a) South African Agricultural Union, together with its various provincial branches and farmers' associations
   (b) The various agricultural boards – meat, dairy and wool

3.2 State Agricultural Extension Services

3.3 Agricultural Cooperatives

3.4 Individual farmers

3.5 State veterinary department

3.6 Agricultural press, etc.

It is envisaged that personal contact will be made at committee level with the above national and regional organisations by nominated veterinarians to promote the herd/flock approach. Regional veterinary committees are also being formed to foster this approach. It is also necessary for individual veterinarians to adopt this approach within their practice area.

A start was made when the Veterinary Foundation donated money towards a meeting which was held with the Meat Board in Pretoria on 4 June 1981. It was attended by Dr C. Cameron (President SAVA), Dr R.D. Sykes and myself as the chairman of the Rural Practitioners' Group. Many matters were discussed, including the problems of communicating our herd/flock approach to the farming community. Two positive thoughts emerged:

(a) We were asked to address both the Transvaal Meat Committee and the Free State Red Meat Committee. Dr D. Barry and Dr R.D. Sykes were nominated to address the meetings and they received a very enthusiastic response. This was also funded by the Veterinary Foundation.

(b) The Meat Board agreed to circulate a brochure outlining the herd/flock approach to their members.

Amongst the many objectives we wish to achieve during the coming year, the following take priority:

1. A meeting with the committee of the Society of Animal Productionists.
2. Further meetings with organized agriculture and the agricultural boards, etc.

3. Printing of pamphlets outlining the veterinary herd/flock health approach which will be circulated to organized agriculture, etc. and through them to individual farmers. (It might be expedient to work through an advertising agency.)
4. It is also essential that memoranda be drawn up after discussion with the above bodies.

One thus realizes the tremendous contribution and stimulus the Veterinary Foundation's support has given to the rural veterinarian. However, I believe that this stimulus has filtered through to the entire profession and that every section has been critically looking at their role within the profession. At last we are becoming an outgoing profession.

When one looks at the 4 objectives we have set out to achieve this year one realizes a common problem that arises out of the outging approach: It is essential to meet with the committees of the various organizations and this means that we will have to fit into their dates and venues for meetings – and that costs money.

Having outlined the achievements which have resulted from the support given to the rural practitioner, it is sincerely hoped that the Veterinary Foundation will be able to keep the momentum going by their continued support.

R.A. Wilson
P.O. Box 30
Caledon
7230

BOOK REVIEW **BOEKRESENSIE**

PROSTAGLANDINS IN ANIMAL REPRODUCTION

L-E. EDQVIST and H. KINDAHL

H. Lundbeck & Co Ottiliavej 7-9 2500 Valby Kobenhavn. Acta Veterinaria Scandinavica Supplementum 77 1981 pp 390. Price not quoted. ISBN 87-88085-00-7

This book combines the papers of a symposium held on this subject at the Swedish University of Agricultural Sciences, Uppsala, Sweden. The book is divided into 6 sections, viz. a general section, a section on the non-pregnant animal, a section on practical uses in the non-pregnant animal, a section on the pregnant animal, a section dealing with practical uses in the pregnant animal and a section considering therapeutic applications of prostaglandins. A general discussion is also included after the last main section.

The general section consists of 7 contributions ranging from prostaglandin chemistry, metabolism and clearance, pharmacology and assay methods to residues in beef and dairy cattle and the effects of prostaglandins on the genital tract and on hormone secretion. It provides a basic insight into these fields which will be of benefit to the student, practitioner and research worker.

The second section dealing with the non-pregnant animal also consists of 7 contributions. These cover the subject of luteolysis including the identification of prostaglandins as luteolytic agents, the importance of corpus luteum blood flow and the utero-ovarian relationships, the role of luteal prostaglandin receptors and the control of luteolysis in the mare. The student, practitioner and research worker will benefit tremendously from this section.

The practical uses of prostaglandins in the non-pregnant animal is discussed in the third section. These uses are applied to dairy cattle, sheep and goats, pigs and mares and also contains an article on the use of prostaglandins in cattle breeding in general. This section will be of value mainly to the student and practitioner.

Five contributions are included in the fourth section on the pregnant animal. These deal mainly with the role of prostaglandins during late pregnancy and its role in parturition. The student, practitioner and research worker will benefit from this section.

The practical uses in the pregnant animal are limited to induction of parturition in cows and sows. These will be of benefit to the student and practitioner. Unfortunately, no contribution on the induction of parturition in mares has been included.

The last section deals with the use of prostaglandins in the treatment of cystic ovaries in cows, unobserved oestrus in cows and puerperal infections such as pyometra, endometritis and metritis. This section is indicated for the student and practitioner.

The general discussion at the back of the book provides interesting reading and contains questions and answers from the various participants at the symposium. It contains a lot of information for the post-graduate student and research worker.

In conclusion, this book will be a meaningful source of reference for the post-graduate student, lecturer, practitioner and research worker. It will be of value to the undergraduate student interested in supplementary reading.

H.M. Terblanche

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